Farzeen Firoozi *Editor*

Female Pelvic Surgery

Springer

Female Pelvic Surgery

Farzeen Firoozi
Editor

Female Pelvic Surgery

Springer

Editor
Farzeen Firoozi
Hofstra North Shore—LIJ School of Medicine
The Smith Institute for Urology
Lake Success, NY, USA

ISBN 978-1-4939-1503-3 ISBN 978-1-4939-1504-0 (eBook)
DOI 10.1007/978-1-4939-1504-0
Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2014949890

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

To my wife, Kelly, who, in addition to contributing a chapter for the book, also gave generously in allowing me those late nights and weekends to complete my first book. Her support is the driving force behind everything I have been able to accomplish in life.

To my boys, Sam and Alex, who have asked me earnestly every night to read them my new book when it's completed. I am thankful for their genuine excitement and support for their father's book.

Finally, I also dedicate this book to Tahmoures Firoozi, MD, FACS: my best friend, my mentor, and my father. Thank you for teaching me, above all, everything that cannot be learned from a book.

Foreword

Medicine is an ever-changing world, and the specialty of Female Pelvic Medicine and Reconstructive Surgery (FPMRS) is a product of that world. Practice as we know it now is developing rapidly in ways we never imagined. Value-based care delivery and optimization of a patient's throughput are clearly the center pieces of the new health system, and all the while quality of care and patient-centered outcomes are more important considerations than ever. Thus, the timeliness of this book is so significant.

Dr. Firoozi has put together an outstanding list of authors and contributors to this book who truly believe in this new-age philosophy of collaborative innovation and practice state-of-the-art Female Pelvic Medicine and Reconstructive Surgery. Our continued evolving understanding of female pelvic health disorders and of available new management techniques is highlighted in this book. Furthermore, there is tremendous balance in the recommendations made herein, as there are always pros and cons in surgical and interventional therapies. One of the more striking attributes of this book is that it details some areas that are not as commonly discussed and offers tremendous insight into subjects such as neovaginas and complex reconstructive surgery of the female lower urinary tract.

This book should serve as a valuable reference for the practicing pelvic surgeon and aid in dealing with the increasingly complex patient whom one sees in practice. It should serve as a model for the continued cross-collaboration of the disciplines of urogynecology and female urology as the team concept continues to evolve on behalf of all of our patients and their improved quality of lives.

Cleveland, OH — Sandip P. Vasavada, M.D.

Preface

The subspecialty of female pelvic medicine and reconstructive surgery, while still in its infancy, has grown in leaps and bounds over the last decade. There have been significant innovations in the fields of research and treatment of female pelvic floor disorders. This growth has been due in large part to the collaboration of urologists and gynecologists.

Technology and regulatory requirements are constantly evolving; thus, this book was envisaged to provide a compendium of information for the practicing urologist, gynecologist, female urology specialist, and urogynecologist. It draws from all these areas of medicine, with experts from varied training backgrounds, and truly represents a collaborative effort. The goals of this book are twofold: to report an updated account of the current literature and to provide the basic and advanced surgical techniques for the management of common and uncommon diseases within the domain of female pelvic medicine and reconstructive surgery.

It is my hope that the internationally recognized contributors to this book have created a text that is both accessible and integral to the readers. I genuinely believe that this book is a significant and valuable addition to the library of those who practice in or aspire to enter the field of female pelvic medicine and reconstructive surgery.

Lake Success, NY — Farzeen Firoozi, M.D., F.A.C.S.

Contents

Contributors

Nitya E. Abraham, M.D. Female Pelvic Medicine and Reconstructive Surgery, Glickman Urology and Kidney Institute, Cleveland Clinic Foundation, Cleveland, OH, USA
abrahan@ccf.org

Chad Baxter, M.D. Department of Urology, David Geffen School of Medicine at UCLA, Santa Monica, CA, USA
cbaxter@mednet.ucla.edu

Jerry G. Blaivas, M.D. Department of Urology, Weill Cornell Medical College, New York Presbyterian Hospital, New York, NY, USA

SUNY Downstate College of Medicine, New York, NY, USA
jgblvs@gmail.com

Kimberly L. Burgess, M.D. Division of Urology, Department of Surgery, Greenville Health System, Greenville, SC, USA
Kburgess2@ghs.org

Kai-Wen Chuang, M.D. Department of Urology, North Shore—Long Island Jewish Health System, New Hyde Park, NY, USA
Kaiwen.chuang@gmail.com

Teresa L. Danforth, M.D. Department of Urology, State University of New York at Buffalo, Buffalo, NY, USA
danforth@buffalo.edu

Farzeen Firoozi, M.D., F.A.C.S. Female Pelvic Medicine and Reconstructive Surgery, Hofstra North Shore—LIJ School of Medicine, The Smith Institute for Urology, Lake Sucess, NY, USA
ffiroozi@nshs.edu

Kelly A. Garrett, M.D., F.A.C.S., F.A.S.C.R.S. Department of General Surgery, Division of Colon and Rectal Surgery, Weill Cornell Medical College, New York Presbyterian Hospital, New York, NY, USA
Keg9034@med.cornell.edu

David A. Ginsberg, M.D. Department of Urology, University of Southern California, Los Angeles, CA, USA
ginsberg@med.usc.edu

Howard B. Goldman, M.D. Female Pelvic Medicine and Reconstructive Surgery, Glickman Urology and Kidney Institute, Cleveland Clinic Foundation, Cleveland, OH, USA
goldmah@ccf.org

Sender Herschorn, M.D.C.M., F.R.C.S.C. Division of Urology, Department of Surgery, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, ON, Canada
s.herschorn@utoronto.ca

Michael Ingber, M.D. Saint Clare's Health System, Denville, NJ, USA

Weill Cornell Medical College, New York, NY, USA
inberMD@aol.com

Forrest Jellison, M.D. Female Pelvic Medicine and Reconstructive Surgery, Department of Urology, San Antonio Military Medical Center (SAMMC), Uniformed Services University of the Health Sciences, Fort Sam, Houston, TX, USA
fjellison@gmail.com

Kelly C. Johnson, M.D. Chesapeake Urology, Owings Mills, MD, USA
kjohnson@cua.md

Elizabeth Kavaler, M.D. Department of Urology, Lenox Hill Hospital, New York, NY, USA

Assistant Professor of Urology, Weil Cornell Medical College, New York, NY, USA
drkavaler@nyurological.com

Ryan M. Krlin, M.D. Department of Urology, Louisiana State University Health Sciences Center, New Orleans, LA, USA
Rkrli1@lsuhsc.edu

Yaniv Larish, M.D. Department of Urology, Lenox Hill Hospital, New York, NY, USA
ylarish@nshs.edu

Melissa A. Laudano, M.D. Department of Urology, Weill Medical College of Cornell University, New York Presbyterian Hospital, New York, NY, USA
Melissa.laudano@gmail.com

Richard Lee, M.D., M.B.A. Department of Urology, Weill Medical College of Cornell University, New York, NY, USA
Ril9010@med.cornell.edu

Courtenay K. Moore, M.D. Lerner College of Medicine, Cleveland Clinic, Cleveland, OH, USA

Cleveland Clinic, Glickman Urological and Kidney Institute, Cleveland, OH, USA
Moorec6@ccf.org

Geneviève Nadeau, M.D., M.Sc., F.R.C.S.C. Division of Urology, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, ON, Canada
Genevieve.nadeau.6@ulaval.ca

Marie Fidela R. Paraiso, M.D. Department of Obstetrics and Gynecology, Women's Health Institute, Center for Urogynecology and Pelvic Reconstructive Surgery, Cleveland Clinic, Cleveland, OH, USA
paraism@ccf.org

Nirmala Pillalamarri, M.D. Division of Female Pelvic Medicine and Reconstructive Surgery, North Shore—Long Island Jewish Health System, Great Neck, NY, USA
npillalama@nshs.edu

Rajveer S. Purohit, M.D., M.P.H. Department of Urology, Weill Cornell Medical College, New York Presbyterian Hospital, New York, NY, USA
rajpu@yahoo.com

Shlomo Raz, M.D. Department of Urology, Division of Pelvic Medicine and Reconstructive Surgery, UCLA School of Medicine, Los Angeles, CA, USA
sraz@mednet.ucla.edu

Eric S. Rovner, M.D. Department of Urology, Medical University of South Carolina, Charleston, SC, USA
rovnere@musc.edu

Stephan A. Seklehner, M.D. Department of Urology, Weill Medical College of Cornell University, New York, NY, USA
St.seklehner@gmx.net

Steven W. Siegel, M.D. Metro Urology, Center for Female Urology and Continence Care, Woodbury, MN, USA
pnedoc@comcast.net
siegel.steven@gmail.com

Karen Soules, M.D. Department of Urology, Louisiana State University Health Sciences Center, New Orleans, LA, USA
karensoules@gmail.com

Christopher F. Tenggardjaja, M.D. Female Pelvic Medicine and Reconstructive Surgery, Glickman Urologic and Kidney Institute, Cleveland Clinic, Cleveland, OH, USA
tenggac@ccf.org

Cecile A. Unger, M.D., M.P.H. Department of Obstetrics and Gynecology, Women's Health Institute, Center for Urogynecology and Pelvic Reconstructive Surgery, Cleveland Clinic, Cleveland, OH, USA
ungerc@ccf.org

Sandip P. Vasavada, M.D. Female Pelvic Medicine and Reconstructive Surgery, Cleveland Clinic, Glickman Urologic and Kidney Institute, Cleveland, OH, USA
vasavas@ccf.org

James M. Weinberger Institute for Bladder and Prostate Research, New York, NY, USA
jmweinberger@mednet.ucla.edu

Harvey Winkler, M.D., F.A.C.O.G. Division of Female Pelvic Medicine and Reconstructive Surgery, North Shore—Long Island Jewish Health System, Great Neck, NY, USA
hwinkler@nshs.edu

Jack Winters, M.D. Department of Urology, Louisiana State University Health Sciences Center, New Orleans, LA, USA
cwinte@lsuhsc.edu

Anatomy of the Female Genitourinary Tract

1

Yaniv Larish and Elizabeth Kavaler

Introduction

The following chapter will give the reader a fundamental understanding of female pelvic and anterior abdominal wall anatomy, including the relationships between the genital, urinary, colorectal, and musculoskeletal systems. In addition, we will describe the vascular and nervous supply to each organ and explore the interrelationships and function between the pelvic organs and their support mechanisms.

Abdominal Wall Musculature

Embrology (Fig. 1.1)

By the third week of gestation, the epiblastic layer of the bilaminar embryonic disc transforms into three layers. The middle layer is composed of mesoblastic cells that give rise to the embryonic mesoderm. The embryonic mesoderm divides into the paraxial mesoderm, the intermediate mesoderm, and the lateral plate mesoderm. The lateral plate mesoderm forms into the somatic mesoderm and the splanchnic mesoderm. The somatic mesoderm becomes the lateral and ventral body wall.

Anatomy

The first layer encountered just deep to the skin and subcutaneous tissues of the anterior abdominal wall is a fascial plane consisting of two layers: a fatty superficial layer which is called Camper's Fascia and a deeper membranous layer, Scarpa's Fascia. Scarpa's Fascia runs inferiorly to attach to the thigh laterally and fuses medially with the perineal membrane as the inferior border of the superficial perineal pouch through which the urethra passes.

Deep to the superficial fascia lies the anterolateral muscles, which run in three separate directions, creating a strong latticework of muscle fibers. The most superficial of these muscle groups is the external oblique muscle, which runs caudal and medially from the lower eight ribs to the pelvis. The inferior border of the external oblique forms the inguinal ligament. A defect in the inguinal ligament, the superficial inguinal ring, affords passage of the round ligament from the abdomen to the pelvis. Deep to the external oblique, the internal oblique muscle fibers run

Y. Larish, M.D.
Department of Urology, Lenox Hill Hospital, New York, NY, USA
e-mail: ylarish@nshs.edu

E. Kavaler, M.D. (✉)
Department of Urology, Lenox Hill Hospital, New York, NY, USA

Assistant Professor of Urology, Weil Cornell Medical College, New York, NY, USA
e-mail: drkavaler@nyurological.com

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_1,

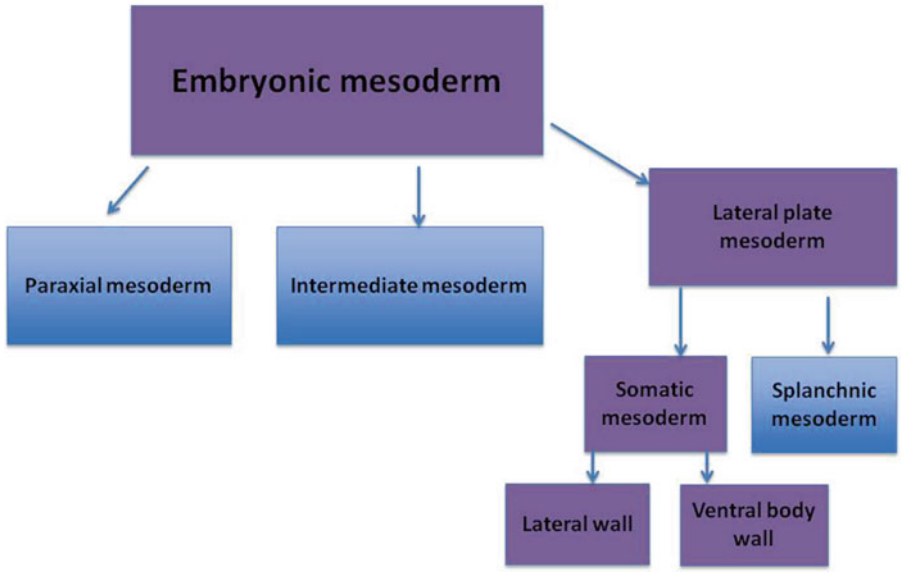


Fig. 1.1 Embryology of abdominal wall musculature

cranially and laterally from the lower six costal cartilages to the pelvis, where they fuse with the muscle layer deep to it, the transversus abdominus, forming the conjoint tendon.

Medially, the rectus abdominus muscle originates at the pubic bone and runs superiorly to insert in the fifth, sixth, and seventh costal cartilages and the xiphoid process. It is encircled by the rectus sheath, which varies in composition depending on its location. Superiorly, the internal oblique splits to cover the rectus muscle both anteriorly and posteriorly. These layers are reinforced by the external oblique anteriorly and the internal oblique posteriorly. Caudally, all three anterolateral muscle groups pass anteriorly to the rectus. Above the coastal margin, the external oblique covers the rectus along its costal cartilage insertion. Together, these muscle fibers provide support for the abdominal organs. The ability to contract these muscles results in a rise in intraabdominal pressure, which aids processes of micturition and defecation.

Blood Supply

Running along the posterior aspect of the rectus sheath, the superior epigastric artery comes off of the internal mammary artery and anastomoses with the last five intercostal arteries above, and the inferior epigastric artery below. It supplies the upper abdominal wall. The inferior epigastric artery, a branch of the external iliac artery, comes through the abdominal inguinal ring, where it courses medially toward the superficial inguinal ring between the transversalis fascia and the peritoneum. This vessel passes through the transversalis fascia into the rectus muscle and meets the superior epigastric artery. The inferior epigastric artery supplies the lower abdominal wall.

Branches of the external iliac artery, mostly by way of the deep circumflex iliac artery, supply the lower abdominal wall. Traveling along the anterior superior iliac spine between the transversus and the internal oblique muscles, it anastomoses

with the iliolumbar artery. The deep circumflex iliac artery also anastomoses with the inferior epigastric artery.

Nerves

The lower six thoracic nerves run between the internal oblique and the transversus muscles, penetrare the rectus sheath and muscle, and end as cutaneous branches. The 12th thoracic and first lumbar nerves join to form the iliohypogastric nerve, supplying the psoas and the quadratus lumborum before veering anteriorly to supply the transversus and the internal oblique muscles. The ilioinguinal nerve also comes off of the last thoracic and first lumbar nerve and communicates with the iliohypogastric nerve. The iliohypogastric nerve divides into the iliac branch, which supplies the lower internal and external oblique muscles above the iliac crest, and the hypogastric branch, which supplies the inferior portions of the transversus and the internal oblique muscles, the external oblique aponeurosis , the skin, and the symphysis pubis. This nerve then meets the ilioinguinal nerve near the anterior superior iliac spine.

Bony Pelvis

Embryology

The embryologic development of the bony and muscular pelvis is similar to that of the abdominal wall musculature. During the third week of gestation, the bilaminar embryonic disc transforms into the trilaminar embryonic disc, composed of ectoderm, mesoderm, and endoderm. A thickening of cells forms at the caudal region (primitive streak) and migrates cephalad, creating a depression on the ventral aspect of the trilaminar disc. This area of cells is called the embryonic mesoderm. Three clusters of mesodermal cells form by the end of the third week of gestation. They are the paraxial mesoderm, the lateral plate mesoderm, and the intermediate mesoderm. The paraxial mesoderm gives rise to the axial skeleton. The lateral plate mesoderm becomes the lateral and ventral body wall as well as the gut. The intermediate mesoderm will evolve into the urogenital ridge, and ultimately the reproductive and urinary tracts.

Anatomy

The pelvis can be separated into two compartments separated by the iliopectineal line: the greater (false) pelvis and the lesser (true) pelvis (Fig. 1.2). The iliac fossa and the sacrum form the boundaries of the greater pelvis, which houses the sigmoid colon and segments of the small intestine. The lesser pelvis lies below the pelvic brim and contains the pelvic organs, including the bladder, reproductive organs, the rectum, and the anus. It is bordered by the sacrum and coccyx posteriorly, the pubic symphysis anteriorly, and the obturator internus laterally. The pelvic brim demarcates the roof of the lesser pelvis while the pelvic musculature constitutes its floor.

The bony pelvis is composed of three bones: the ilium, the ischium, and the pubis (Fig. 1.3). The amalgam of these three bones is called the innominate bone. The ilium sits posteriorly, and articulates with the acetabulum, the ischium, and the pubis. The superior border of the ilium is called the crest, extending inferiorly, ending at the iliac spines. The components of the ischium include the body, the superior and inferior rami, and the tuberosity. The posterior border joins with the ilium to form the greater sciatic notch. The inferior ramus meets the pubis to form the ischiopubic arch. The pubis is made up of a body and two rami. The obturator sulcus is located inferior to the superior ramus. The symphysis is a synarthrodial joint within the pubic bone.

The muscular attachments (Fig. 1.4a, b) to the ilum, ischium, and pubis create the bowl of the pelvic floor. The external oblique, the internal oblique, the transversus abdominus, latissimus dorsi, quadratus lumborum, tensor fascia latae, and Sartorius muscles all attach to the ilium. The rectus femoris muscle connects to the iliac spine. The iliacus muscle sits in the iliac fossa, and the

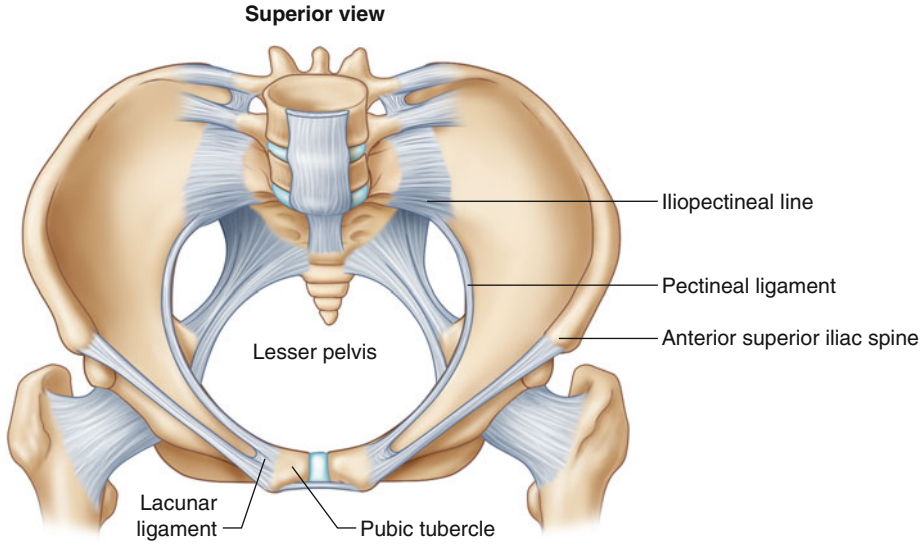


Fig. 1.2 The bony pelvis, divided into two compartments divided by the iliopectineal line: the greater (false) pelvis and the lesser (true) pelvis

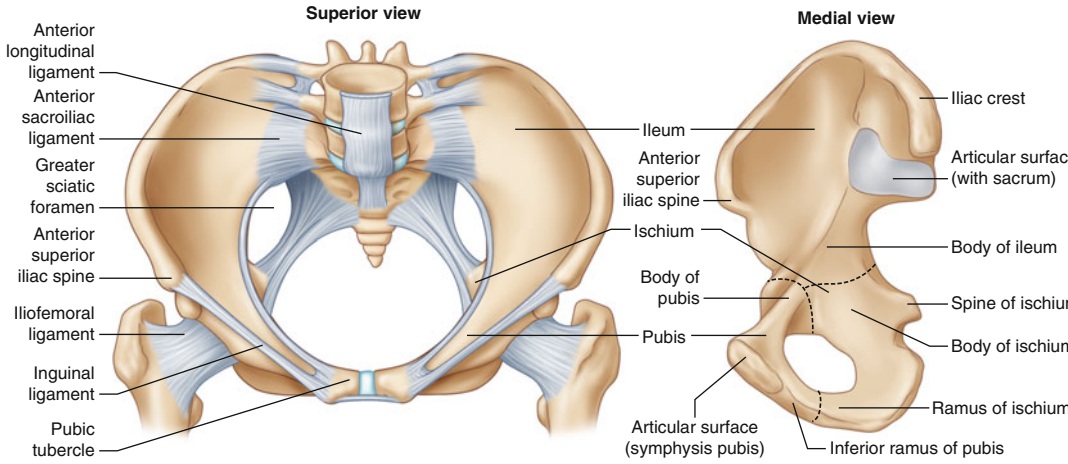


Fig. 1.3 The bony pelvis is composed of three bones: the ilium, the ischium, and the pubis

tendon of the psoas inserts between the iliac spine and the iliopectineal eminence. Both the gluteus maximus and minimus muscles attach to the ilium.

The ischium gives rise to the obturator internus muscle. The coccygeus and levator ani muscles attach to the ischial spines deep in the pelvis. The transversus perinei muscle originates from the lower border of the ischium. Laterally, the adductor magnus muscle connects to the ischial tuberosity. The obturator externus muscles and adductor longus muscles come off of the superior ramus of the pubis, while the obturator internus muscles and levator attach on the anterior surface. The rectus and pyrimidalis muscles attach on the superior border. The anterior surface of the inferior ramus attaches to the abductor brevis, magnus, and the obturator internus. See Fig. 1.5a, b.

The sacrum is formed by the fusion of 5–6 sacral vertebrae. The body of the sacrum

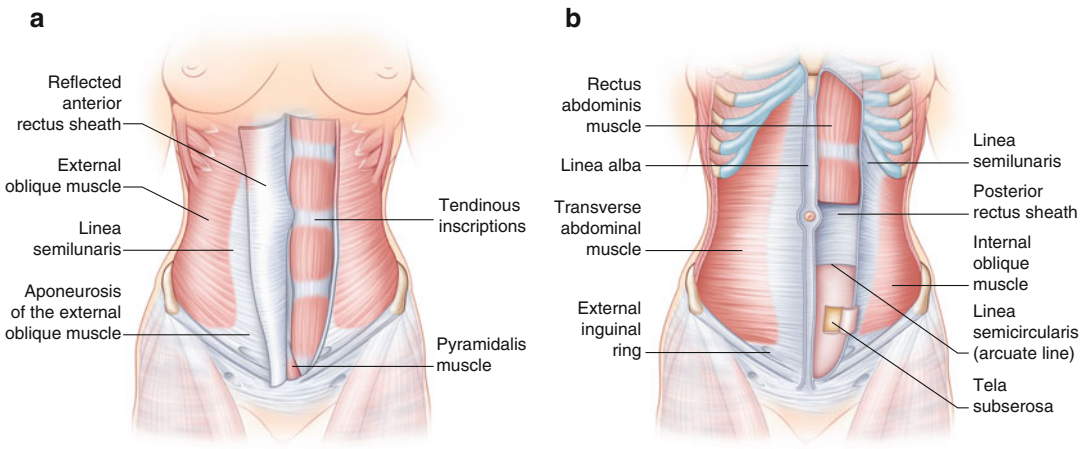


Fig. 1.4 The muscular attachments to the bony pelvis

articulates with the fifth lumbar vertebrae through a fibrocartilage disk, where it juts out anteriorly and is labeled the sacral promontory. The lamina and spines of the third, fourth, and fifth sacral vertebrae are absent, leaving a wide space called the sacral hiatus. The coccyx is formed by 3–5 caudal vertebrae that fuse and join the sacrum at the sacrococcygeal joint. Muscular attachments to the sacrum include the piriform muscle, sacrospinalis muscles, and the gluteus maximus muscle. Muscles that connect to the coccyx include the coccygeus and the iliococcygeus musles.

The sacrospinalis ligament connects the lateral border of the sacrum and the coccyx to the ischial spine. The sacrotuberous ligament extends from the sacral vertebrae to the tuberosities of the ilium. This is a deep posterior ligament with fibers that connect to the hamstrings. The iliolumbar ligament joins the iliac crest with L4-5. The iliofemoral ligament attaches the crest of the ilium to the acetabulum. See Fig. 1.6.

Blood Supply

The blood supply to the innominate bones comes from branches off of the iliolumbar, deep circumflex iliac, and obturator arteries, which all come off of the hypogastric (internal iliac) artery. The lower part of the bone receives nutrients from branches of the gluteal arteries, which also comes off of the hypogastric artery.

The sacrum receives its blood supply from the middle sacral artery, a branch of the aorta, and the lateral sacral arteries, which comes off of the hypogastric artery (internal iliac). These vessels anastomose with the lumbar arteries and the superior gluteal artery.

Nerve Supply

The innominate bones derive their nerve supply from the superior hypogastric plexus, which sits over the sacral promontory. It receives nerve fibers from inferior mesenteric and lumbar sympathetic ganglia. The superior hypogastric plexus gives rise to the middle hypogastric plexus and then divides into two lateral hypogastric nerves. They travel in the uterosacral ligament into the rectum and the vagina. At this juncture, they are labeled the pelvic plexuses. The fibers that make up the pelvic plexuses come from the S2-4 parasympathetic and sympathetic trunks. As the nerves descend into the pelvis, they innervate the bones and ligaments through which they pass.

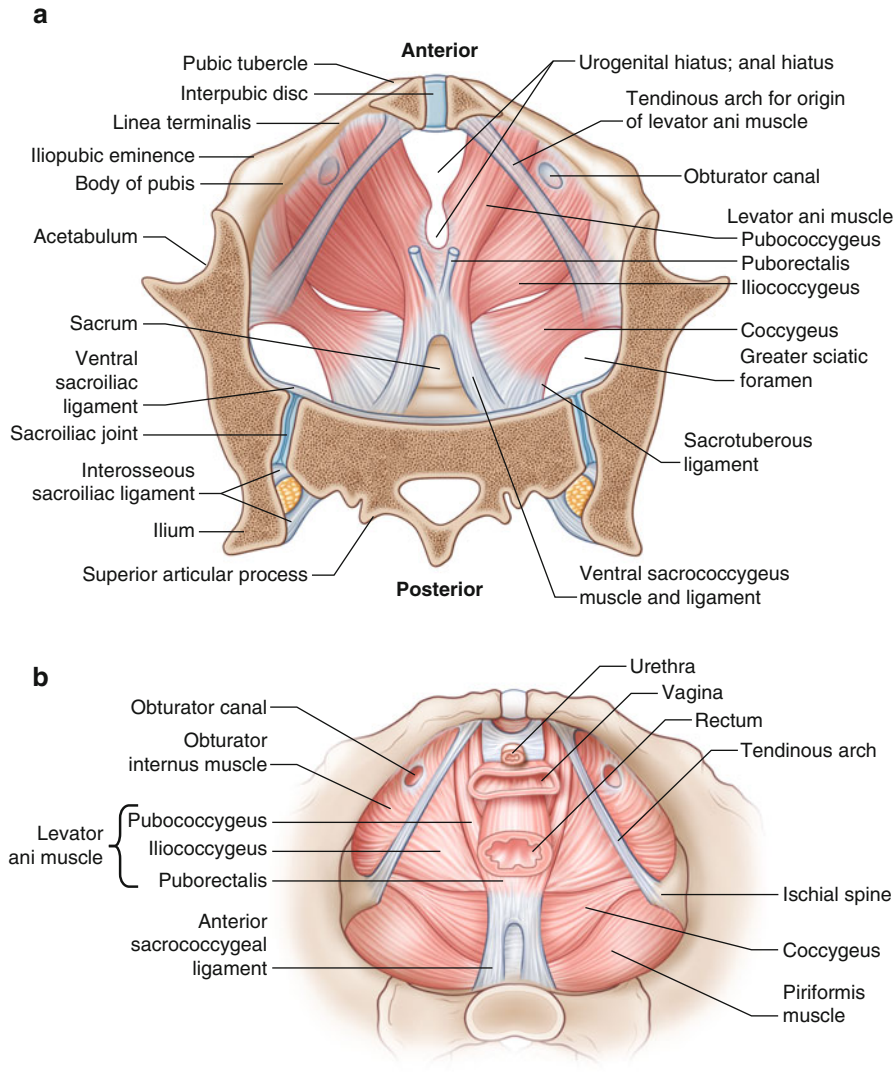


Fig. 1.5 The musculature of the bony pelvis, with an abdominal view

Pelvic Muscles

Embryology (Fig. 1.7a, b)

The pelvic diaphragm arises out of all three layers of the embryonic tissue: the endoderm, the mesoderm, and the ectoderm. The endoderm develops into the primitive gut, which gives rise to the cloaca. At week 6 of gestation, the cloaca divides into the urogenital sinus and the anorectal canal. The mesoderm gives rise to the urogenital ridge, which becomes the urinary tract, the uterus and upper vagina, and the lateral plate mesoderm. The lateral plate mesoderm divides into the somatic and splanchnic mesoderm. The somatic mesoderm gives rise to the lateral and ventral walls, and the splanchnic mesoderm evolves into the visceral peritoneum. The ectoderm gives rise to the skin of the external genitalia and the nervous system.

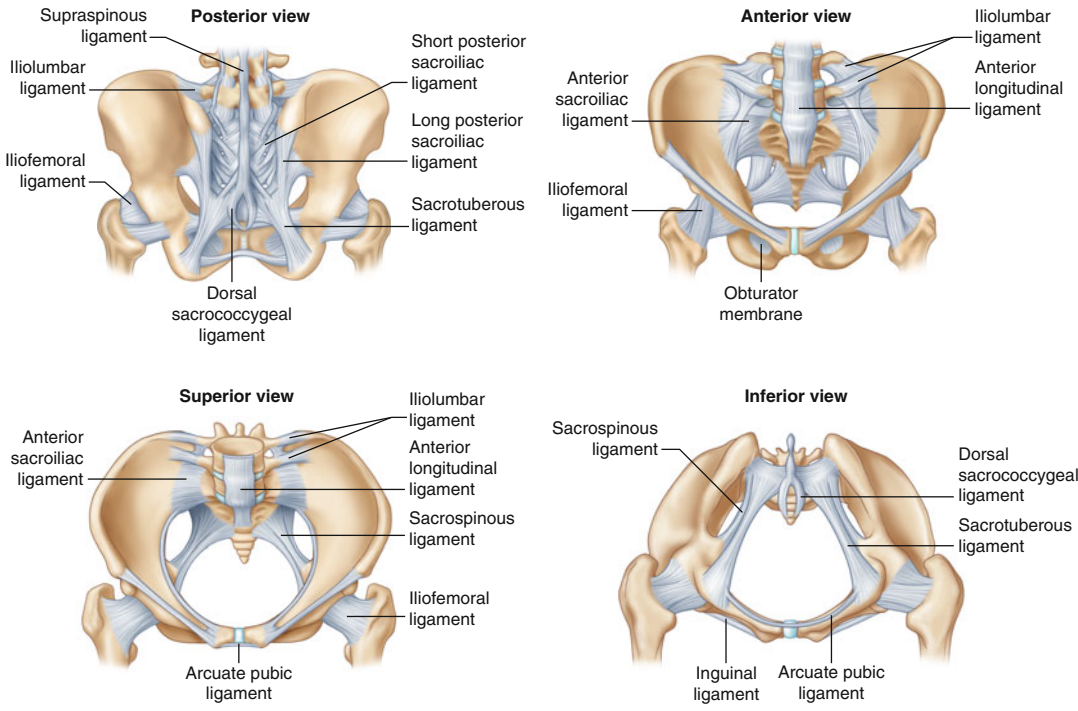


Fig. 1.6 The ligaments of the bony pelvis

Anatomy

Pelvic Floor Musculature

In addition to providing support to the pelvic and abdominal organs and ensuring that they stay in their proper anatomical locations, the pelvic floor apparatus aids in the evacuation of both urine and feces. Additionally, the female pelvic floor has a central role in the acts of conception and parturition.

The pelvic floor is comprised of two main components; a visceral fascial layer, the endopelvic fascia (comprised of connective tissue), and a muscular layer, collectively referred to as the levator ani and coccygeus muscles. The endopelvic fascia provides a passive support mechanism when the genital hiatus is open, and provides an apical tethering point for the pelvic viscera. Conversely, the musculature provides an active support mechanism by contracting and closing the genital hiatus.

The pelvic diaphragm refers to the bowl of muscle that provides a supportive plate on which the bladder, uterus, and rectum sit. Perforated by the urethra, the vagina, and the rectum, these muscles serve as the support structures of the pelvic floor. The bony supports include the pubic bone anteriorly, the sacrum posteriorly, and the ischium bilaterally. The three main muscle groups that comprise the pelvic support include the levator ani, anteriorly and inferiorly, the obturator muscles laterally, and the coccygeus muscle, posteriorly.

Divided into three parts, the levator ani muscle is comprised of the Iliococcygeus muscle, the pubococcygeus muscle, and the puborectalis muscle. The iliococcygeus muscle arises laterally from the arcus tendineus it extends from the symphysis to the ischial spine, where it meets the obturator internus muscle. Medially it meets the pubococcygeus, and posteriorly, it inserts into the coccyx. The pubococcygeus is a strap of muscle that runs anterior to posterior from the pubis to the coccyx where it meets the sacrococcygeal ligament, forming a strong raphe over the sacrum. The puborectalis muscle creates a band of tissue around the rectum, which supports the posterior pelvis.

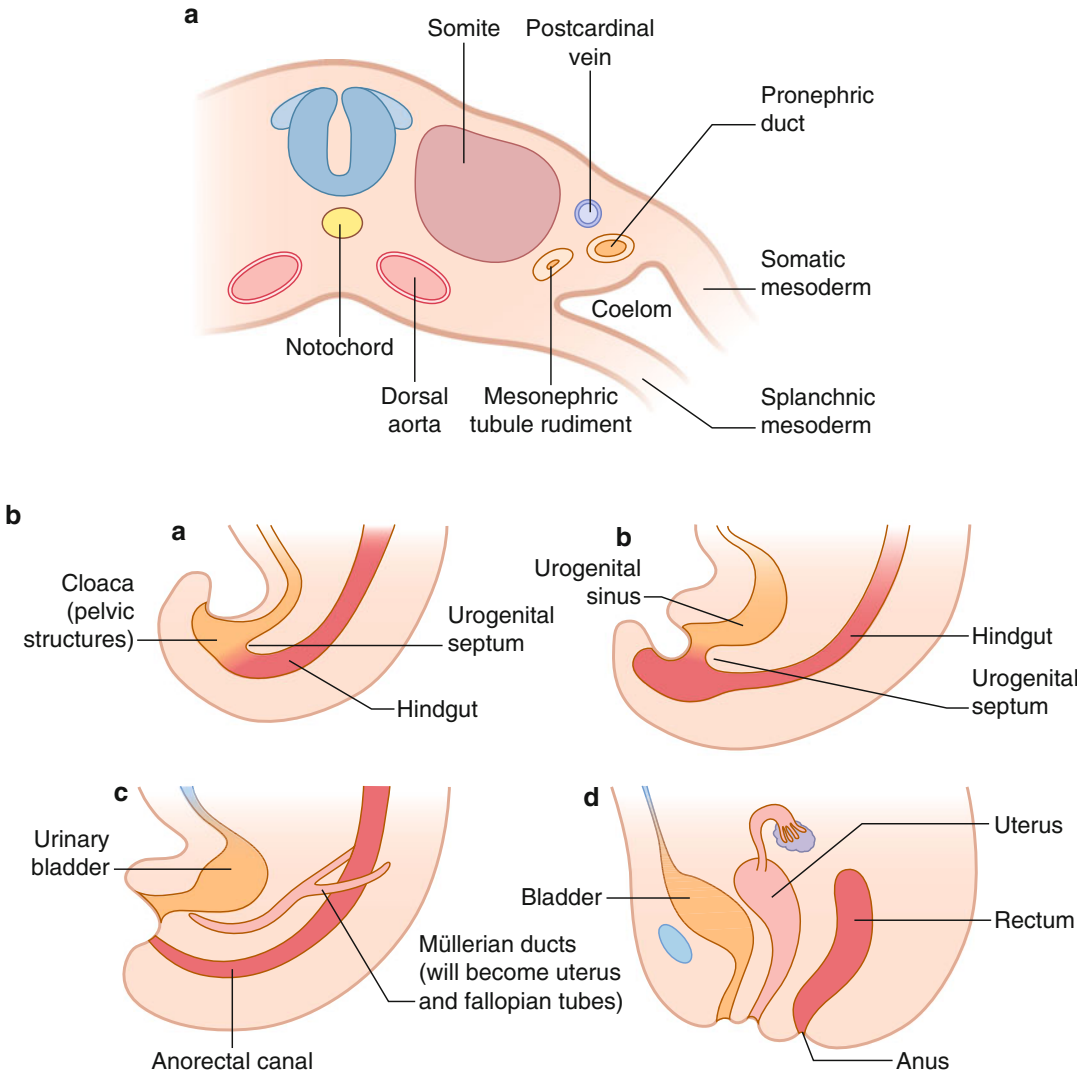


Fig. 1.7 (**a**) Embryology of the pelvis muscles. (**b**) Embryology of the pelvic floor musculature

The levator ani muscles function to both contract around the urethra, vagina, and rectum, and to counteract intraabdominal and intrathoracic pressures on the pelvic organs. See Fig. 1.8.

Arising from the pubic rami and the ischium, the obturator internus muscle connects the pelvis to the hip, where its fibers insert into the trochanter. The obturator vessels and nerves traverse the notch on the lateral aspect of the obturator foramen. Intrinsic to the functioning of the inner thigh, the obturator muscle is involved in lateral rotation, abduction, and extension of the upper leg.

Not comprising many muscle fibers, the coccygeus appears more as an aponeurosis than as a muscle. It arises from the ischial spine and extends into the fourth and fifth sacral vertebrae, as well as the coccyx. Besides supporting the pelvic organs, it helps with flexing and abducting of the coccyx.

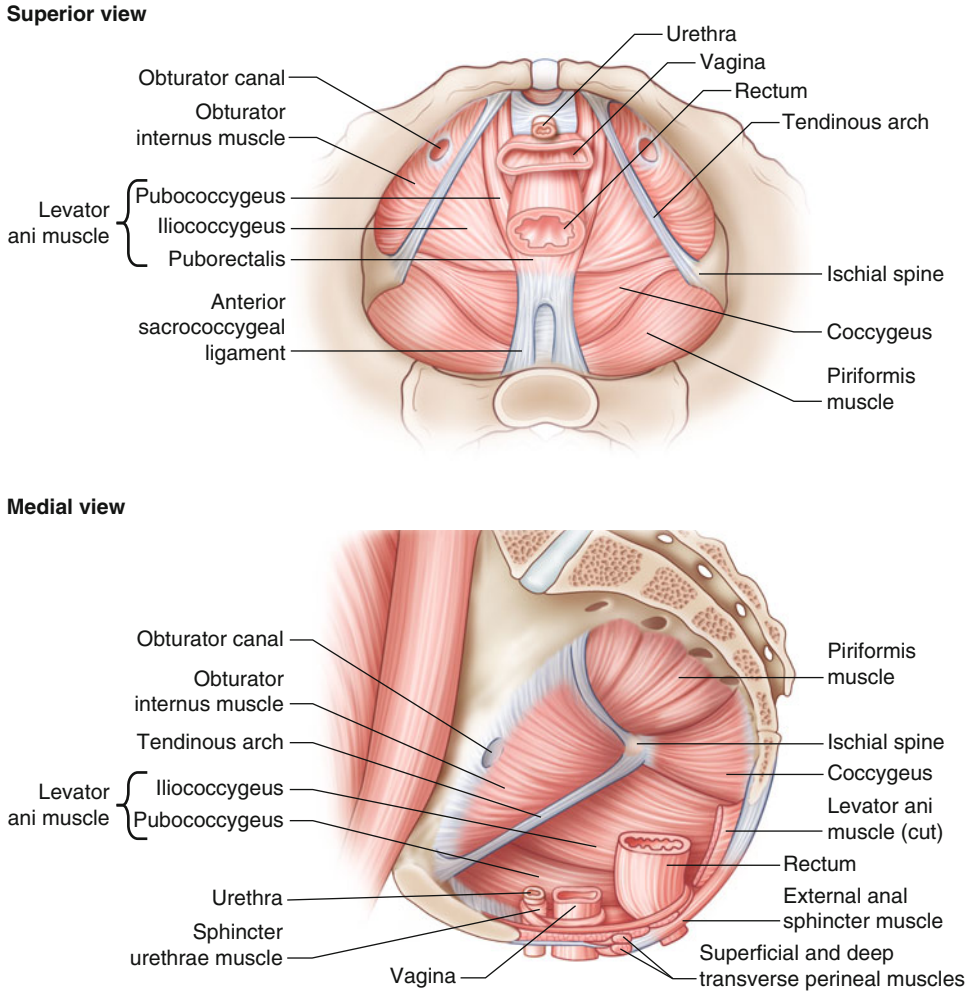


Fig. 1.8 Pelvic muscles groups and their attachments

Perineum and Structures from Below

From the perineal perspective, the fascial supports overlying the organs play a crucial role in both the pathophysiology of pelvic organ prolapse and the methods of repairing those defects. An extension of the transversus abdominus muscle, the endopelvic fascia lays over the abdominal portion of the pelvic musculature. The levator ani muscles are covered from the perineal side by the same facial material but it is divided into three sections depending on what structures are being covered.

From the vaginal view, looking at the *anterior* vaginal wall, the urethropelvic (also called the pubourethral) ligament creates a bridge extending from the inferior aspect of the pubic ramis from side to side, thus supporting the urethra. If there were any weakness or damage to this fascial layer, stress incontinence would be the end result. The pubocervical fascia (sometimes called the vesicocervical fascia) is the component of the sheet of support under the bladder. It is not discrete and cannot be visually separated from the urethropelvic ligament, except by determining the organ under which it sits. This fascial layer can be torn, causing a central defect, which results in a cystocele. Correcting central defects in the pubocervical fascia requires replacing that fascia support from below. See Fig. 1.9.

The urethropelvic fascia and the pubocervical fascia insert into the pelvic sidewall where the

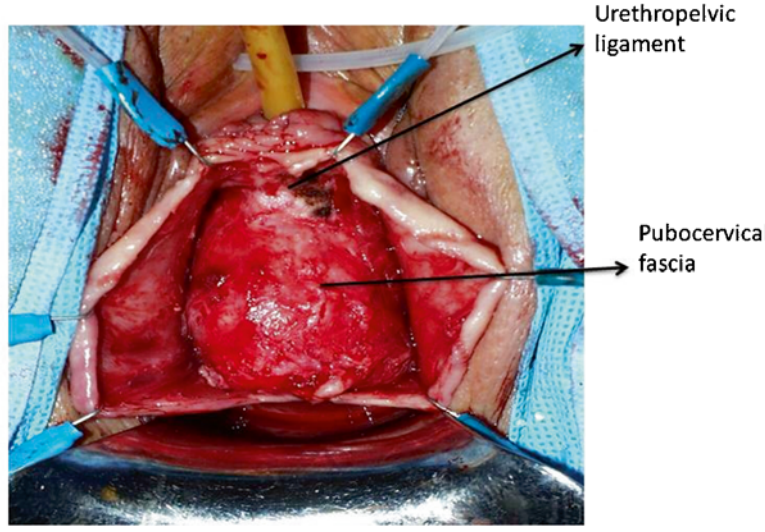


Fig. 1.9 Fascial layers of the pelvic organs: vaginal view

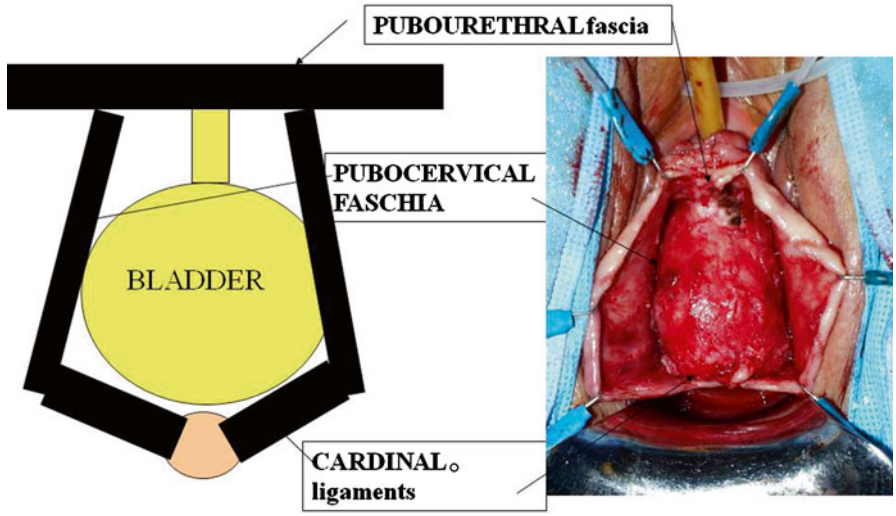


Fig. 1.10 Ligaments of vaginal support

levator ani muscles meet the obturator internus. From the view looking into the pelvis from above, the endopelvic fascia can be seen inserting into the same place. This condensation of both fascial layers into the same insertion point is called the arcus tendineus. An important support for the pelvic organs, the arcus tendineus can be damaged in surgery, pregnancy, or childbirth, resulting in the pelvic organs sliding down the sidewall and causing a prolapse. See Fig. 1.10.

The posterior section of the support under the anterior vaginal wall is the cardinal ligament, which supports the uterus and the apex of the vagina. The cardinal ligament extends from the sciatic foramen, encircles the cervix, and fuses with the sacrouterine ligament posteriorly. The sacrouterine ligament is a non-discrete condensation of fibers that originates from the second through fourth sacral vertebrae and merges with the pubocervical fascia at the vaginal apex. If the

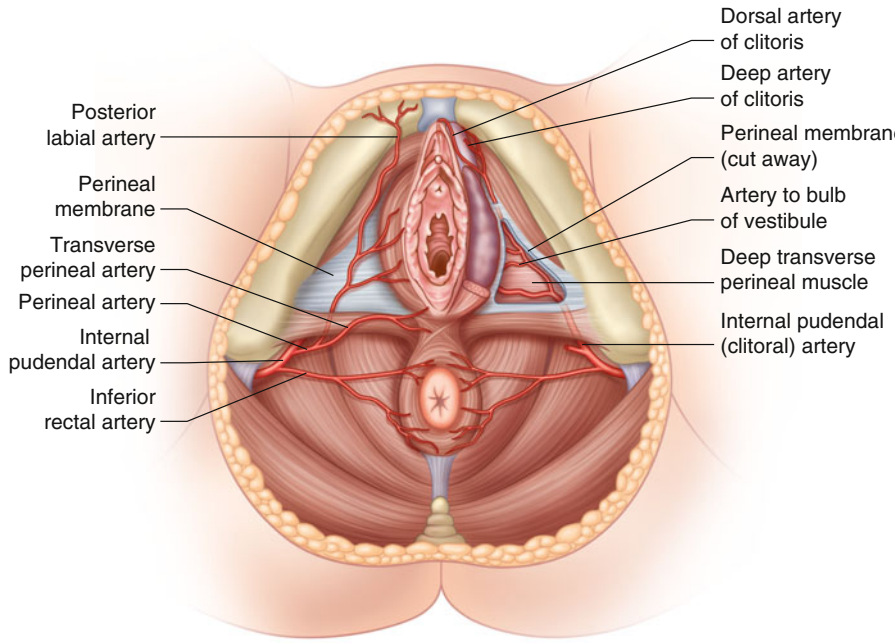


Fig. 1.11 Posterior vaginal support structures

fibers of the sacrouterine ligament are intact, they can be used to support a prolapse repair. Deep to the sacrouterine ligament is the sacrospinous ligament, which runs from the sacrum to the ischial spine. It is a strong, fibrous *true* ligament, in that it joins two bony structures.

The posterior vaginal wall support structures include the rectovaginal septum, the pararectal fascia, and the perineal body. At the apex of the vaginal, the rectovaginal septum fuses with the cardinal ligament/sacrouterine ligament complex. Disruption of the cardinal ligaments through surgery, pregnancy, or childbirth can lead to weakness of the septum and posterior apical prolapse. The perineal body is a central point where the deep and superficial transverse perineal muscles, the anal sphincter, the bulbocavernosus muscle, and the levator ani muscles all converge. Flexibility of the perineal body allows for absorption of pressure during valsalva, and contraction increases both vaginal compression and stability. See Fig. 1.11.

Blood Supply (Fig. 1.12)

The common iliac artery branches into the internal (hypogastric) and external iliac arteries at the lumbosacral articulation. The internal iliac artery divides into three parts once it crosses through the psoas and piriformis muscles: the anterior branch, the posterior branch, and the visceral branch. Arising first, the posterior branch gives rise to the iliolumbar, the lateral sacral, and the superior gluteal arteries. The anterior branch of the internal iliac artery (hypogastric artery) divides into three branches: the obturator, inferior gluteal, and the internal pudendal arteries. The visceral branch becomes the superior vesical, middle hemorrhoidal, the uterine, and the vaginal arteries.

Sometimes coming off of the external iliac artery, the obturator artery, which usually arises from the anterior branch of the internal iliac artery, terminates in iliac, vesical, pubic, anterior, and posterior branches. These vessels

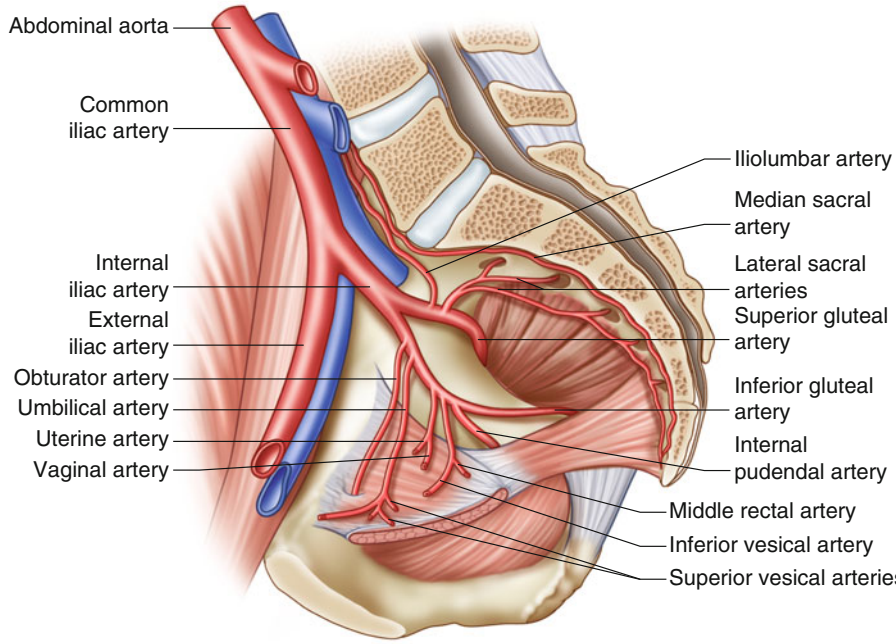


Fig. 1.12 Branches of the internal iliac artery (hypogastric artery)

supply the muscles and bones of the deep pelvis. These branches communicate with the inferior epigastric artery and other divisions of the external iliac artery. The umbilical artery comes off of the inferior gluteal artery from the anterior branch of the internal iliac artery. It gives off branches to the bladder, including the middle vesical artery.

The visceral portion of the internal iliac artery includes the middle hemorrhoidal artery which anastomosis with the superior mesenteric artery through the superior hemorrhoidal artery. The uterine artery branches off early in the pelvis close to the common iliac artery. The vaginal artery has variable origins. It can arise from the hypogastric artery directly, the uterine artery, or the superior vesical artery.

Nerve Supply (Fig. 1.13)

Innervation of the pelvis arises from the second, third, and fourth sacral nerves that enter the pelvis through the greater sciatic foramen, between the piriformis and coccygeus muscles beneath the ischial spines. It enters the perineum through Alcock's canal, where it branches into three parts, the inferior hemorrhoidal nerve, the perineal nerve, and the dorsal nerve of the clitoris. The inferior hemorrhoidal nerve supplies the anal sphincter and peri-anal skin. The perineal nerve supplies the levator ani muscles before it enters through the urogenital diaphragm to innervate the ischiocavernosus and bulbocavernosus muscles, and the urinary sphincter. A superficial branch of the perineal nerve supplies the labia major. The dorsal nerve of the clitoris enters through the urogenital diaphragm before heading toward the glans of the clitoris.

Embryology of the Urinary Tract (Fig. 1.14)

In utero, the kidneys mature through a successive and predictable series of phases prior to reaching maturity. The first two stages, the pronephros and mesonephros, regress. The final stage, the

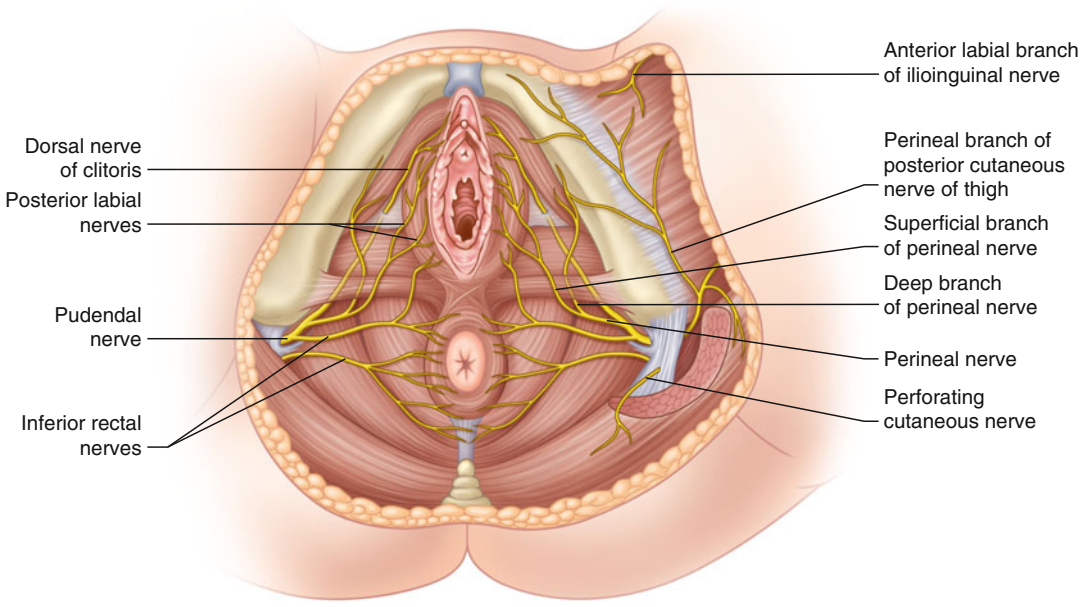


Fig. 1.13 Nerve supply to the pelvis

metanephros, persists. The pronephros is nonfunctional, appears late in the third week, and completely degenerates by the start of the fifth week. The mesonephros serves as primitive excretory organ while the metanephros develops. The mesonephros regresses by the fourth month. Meanwhile, the metanephros forms near the sacrum as the ureteric buds and metanphros mesenchyme.

The ureteric buds and mesenchyme exert reciprocal biochemical effects on each other. The metanephric mesenchyme develops into the glomerulus, proximal tubule, loop of Henle, and distal tubule, while the ureteric bud forms the collecting ducts, calyces, pelvis, and ureter. Despite renal maturation continuing postnatally, the functioning elements of the kidney has fully developed by 32–34 weeks of gestation.

The terminal portion of the hindgut, the cloaca, is divided into the dorsal primitive rectum and ventral primitive urogenital sinus by the urorectal septum. The urinary bladder and urethra are derived from the primitive urogenital sinus and the surrounding splanchnic mesenchyme. The urogenital sinus gives rise to the urinary bladder, which is continuous cranially with the allantois, and caudally with the urethra. The allantois is obliterated during fetal development and forms a fibrous cord, the urachus, which following birth becomes the median umbilical ligament. The trigone of the bladder is formed in a complex process of Wollfian duct involution, while the bladder wall is of endodermal origin. The smooth musculature of the bladder develops during the 12th week of gestation and is ultimately coated on the inside by epithelial cells of endodermal origin.

The Kidneys

Anatomy

The kidneys are paired retroperitoneal organs approximately 10 cm long and weighing 135–150 g. They lie opposite T12-L3 and the right kidney lies slightly lower than the left. The upper pole of the kidney is nearer to the midline than the lower pole, and the medial border of the kidney faces slightly anterior. The right kidney is

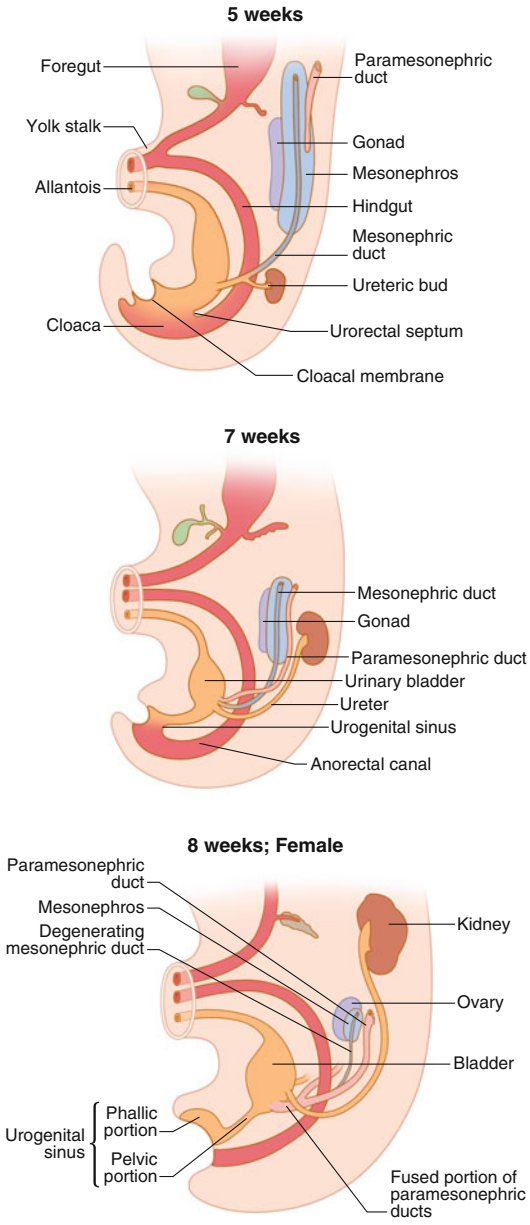


Fig. 1.14 Embryology of the urinary tract

bordered superiorly by the adrenal gland, medially by the second portion of the duodenum, laterally by the posterior abdominal wall, and inferiorly by the mesocolon and small intestine. The left kidney is bordered by the spleen and stomach and adrenal superiorly, the descending colon laterally, the aorta medially, and the mesocolon and small intestine inferiorly. The medial portion of the kidney is concave and houses the renal hilum. The renal hilum transmits the renal vein, renal artery, and ureter into the kidney (anteriorly to posteriorly). The kidneys and adrenals are surrounded (except inferiorly) by a perinephric fat capsule, called Gerota's fascia. The capsule invades the kidney medially to surround the structures of the renal hilum, and runs inferiorly over the course of the ureter.

Blood Supply (Fig. 1.15)

The renal arteries branch from the aorta below the superior mesenteric artery. The right renal artery is longer than the left, and usually courses beneath the inferior vena cava. At the renal hilum, the renal artery branches into several segmental branches, acting as the end-arterial supply to the various segments of the kidney. Small segmental veins coalesce to form the renal vein. The vein of the left is longer and, in addition to draining the kidney, drains the left adrenal and gonadal vein. The renal vein on the right is short, and drains only the kidney, while the adrenal and gonadal veins drain directly into the inferior vena cava.

Nerve Supply

The innervation of the kidneys includes sympathetic, parasympathetic, and visceral afferent nerve fibers. The sympathetic (originating in the sympathetic chain) and parasympathetics (originating from the vagus) nerves join to form an autonomic plexus which runs with the renal artery. The visceral afferents, conveying sensation of pain, travel along the path of the autonomic plexus to the spinal ganglia and spinal cord.

Ureters

Anatomy

The ureters start at the level of the ureteral pelvic junction where the renal pelvis tapers into a narrow tube within the renal hilum posterior to

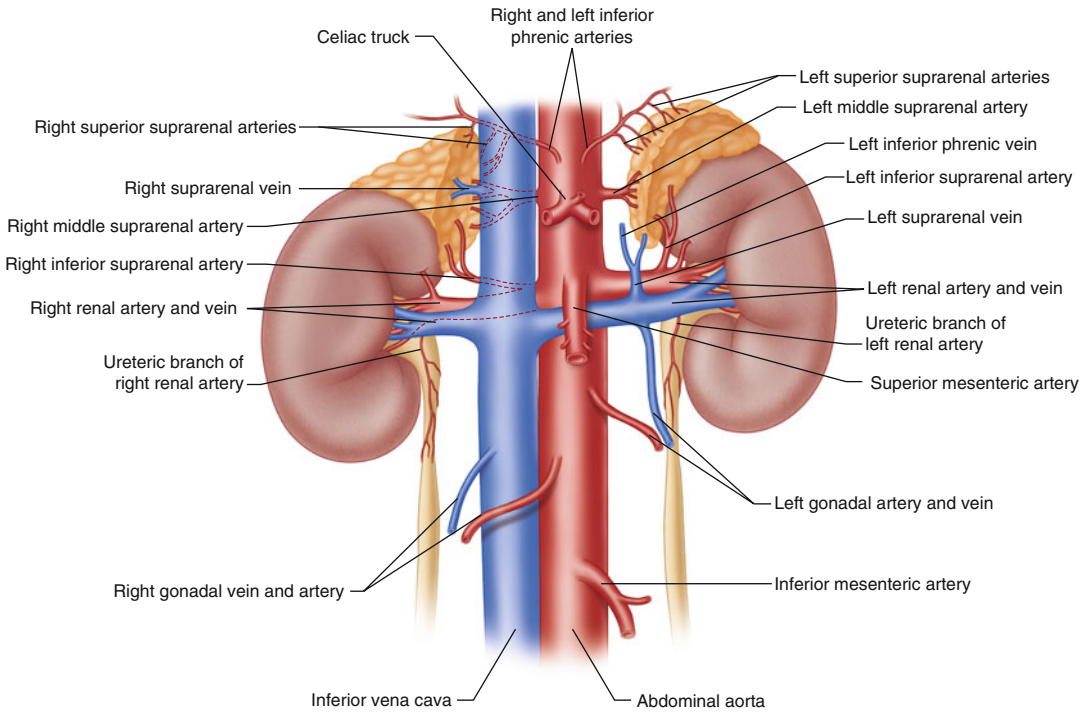


Fig. 1.15 Blood supply to the kidneys

the renal artery and vein. The lumen of the ureter is lined by transitional epithelium. A layer of inner longitudinal and outer circular smooth muscle in turn covers the epithelial layer, and is responsible for the peristaltic movements necessary to propel urine toward the bladder. The ureters course inferiorly toward the bladder along the psoas muscle. The gonadal vessels cross the ureters anteriorly. As they enter the pelvis, the ureters cross anterior to the bifurcation of the common iliac artery. Upon entering the bladder, they take an oblique course through the bladder wall to form a tunnel, which aids in preventing reflux of urine back to the kidney.

Blood Supply

The blood supply to the ureters varies along their course. The abdominal ureters receive their arterial supply from the renal, gonadal, and common iliac arteries and from the abdominal aorta, all of which run in a medial to lateral direction. After entering the pelvis, the ureters receive their blood supply from branches of the internal iliac artery, including the vesical, uterine, middle rectal, and vaginal arteries, all of which run in a lateral to medial direction. The venous and lymphatic drainage of the ureter parallels the arterial supply.

Nerve Supply

Ureteral peristalsis originates in smooth muscle pacemaker nodes within the collecting system. The autonomic nervous system modulates this input; however, the mechanism is unclear. Pain fibers are stimulated by distention and/or mucosal irritation within the ureter. The pain fibers run with the sympathetic nerves corresponding to the segment of ureter affected. Therefore, the distribution of pain varies, and can be referred to the flank, groin, or labia.

The Bladder

Anatomy

The bladder is a compliant, distensible, hollow viscos, which allows for the storage of urine. When empty, it remains confined to the lesser pelvis, but can distend beyond the pelvic brim into the greater pelvis when full. The most superior segment of the bladder is covered by a reflection of peritoneum, while the rest of the bladder lies inferior to the peritoneum. At its most superior point, the uterus drapes the bladder. Anterior to the bladder is a fat filled potential space, the space of Retzius, which protects the bladder from the pubic symphysis. The base of the bladder rests on the anterior aspect of the cervix and anterior vaginal wall. See Fig. 1.16.

The wall of the bladder is composed of three muscle layers that run in different directions. The outer layer of muscles runs longitudinally, the middle layer's fibers run circularly, and the inner layer's fibers run longitudinally gain. This configuration aids in effective contraction of the bladder wall and full expulsion of urine. Deep to the muscular layers of the bladder wall lies the mucosal layer, which is composed of transitional epithelium which prevents the reabsorption of urine from the bladder into the body. At the posterior–inferior aspect of the bladder is a raised crescent-shaped ribbon of muscle, the trigone, which is bordered on either side by the ureteral orifices.

Blood Supply

The arterial supply to the bladder is derived from the superior and inferior vesicle arteries as well as branches arising from the internal iliac artery. Because the branches are variable, the blood supply is thought of as lateral and posterior pedicles, which run along the cardinal and uterosacral ligaments. The venous drainage of the bladder forms a plexus, which drains into the external iliac vein.

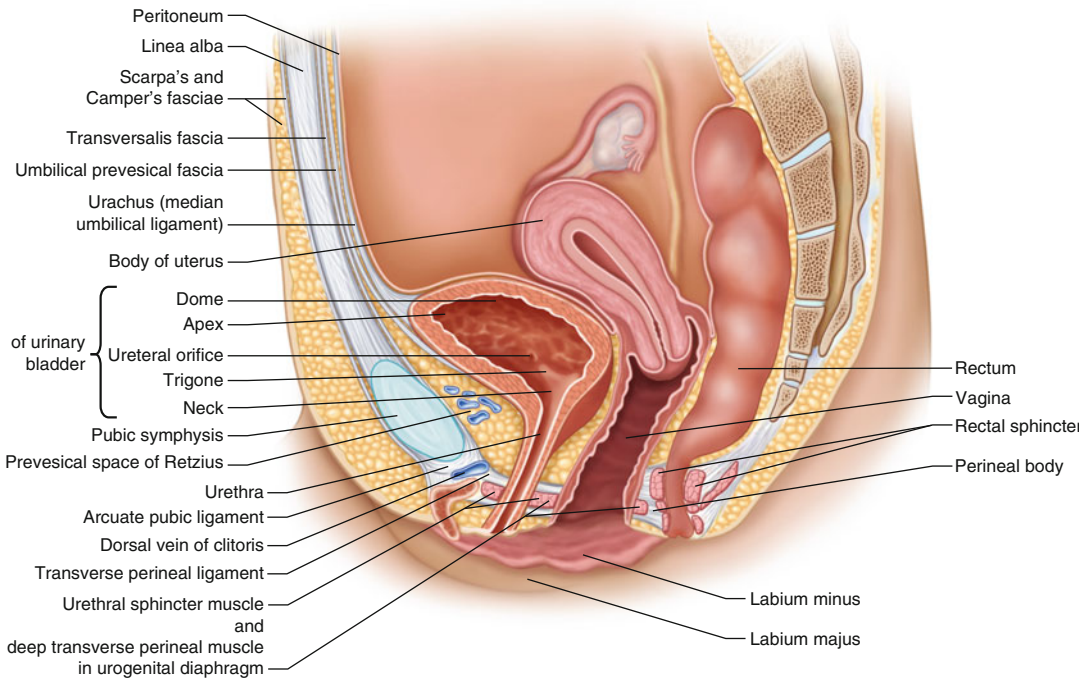


Fig. 1.16 Relationship of bladder to neighboring organs

Nerve Supply

The bladder derives most of its nerve supply from sacral nerves 2–4, with some contribution from the hypogastric plexus. Most of the innervation to the bladder base comes from the parasympathetic nervous system, while the bladder neck and trigone are under sympathetic control from the hypogastric nerve (T10 to L2). Under somatic control from S2, the pudendal nerve innervates the external urinary sphincter.

The Urethra (Fig. 1.17)

The female urethra is 2.5 to 5 cm in length and runs under the pubic bone along the anterior vaginal wall. It is supported by the pubourethral ligaments (also called the urethropelvic ligament), which are contiguous with the pubocervical ligaments (vesicopelvic) that support the bladder. The three walls of the urethra include the mucosa, the submucosa, and the fibromuscular layer, which contains spongy tissue and the cavernous veins. Skene's glands enter into the urethra cephalad to the external urinary sphincter. The distal third of the urethra is lined by squamous epithelium, while transitional cells line the proximal portion. The fibromuscular layer is divided into a circular layer on the outside and a longitudinal layer on the inside. Near the bladder neck, the muscle fibers are interwoven with those of the bladder neck, creating a contiguous structure. In the middle and lower thirds of the urethra, striated fibers of the bulbocavernosus and ischiocavernosus muscles encircle the urethra, creating the external urinary sphincter.

Blood Supply

The urethra shares its blood supply with the anterior vaginal wall and the bladder. The uterine artery gives off the vaginal artery and the artery to the cervix. Branches from both arteries supply the urethra. The artery to the clitoris, from the internal pudendal artery, also gives off branches to the urethra as well.

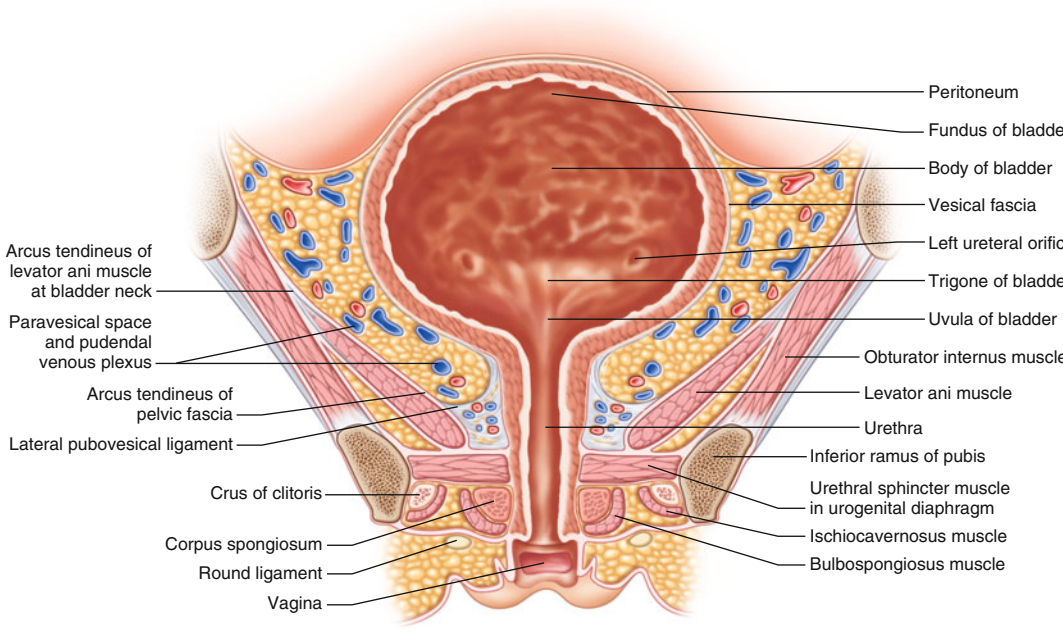


Fig. 1.17 Anatomy of the urethra

Nerve Supply

The parasympathetic and sympathetic nerves to the urethra travel through the hypogastric plexus. The pudendal nerve controls the external urinary sphincter.

The Female Genital Tract

Vulva

Embryology (Fig. 1.18)

Sexual differential of the external genitalia occurs at the eighth week of gestation; however, it isn't until three months that sexual differentiation can be recognized. In the first 4 weeks, a small outgrowth at the external surface of the cloacal membrane appears in front of the genital tubercle. On either side of the membrane are swellings that will eventually form either labia majora or scrotal sacs. By the end of the seventh week, the urogenital sinus forms a separate opening under the genital tubercle. By the eighth week, the genital tubercle begins to form the clitoris. The caudal portion of the urogenital sinus will form the vaginal vestibule and the urethral folds remain separate, becoming the labia minora. The genital swellings merge in front of the anus and form the labia majora.

Anatomy (Fig. 1.19)

The vulva includes the mons pubis, the labia minor and majora, the clitoris, and the glandular structures, including Batholin's glands and Cowper's glands. Pubic hair grows in an inverted triangle pattern, although 25 % of women will have hair that extends upward along the linea alba. The vestibule is the area that is bordered by the labia minora, laterally, the posterior commissure at the perineum, and anteriorly, at the clitoris and urethra. The inferior border is the hymen. Skene's glands open into the vagina from under the urethra. At the 5 and 7o'clock positions Bartholin's glands ducts can be seen as small papillary swellings. During sexual activity, they secrete fluid. By about age 30, they generally involute. The skin of the vestibule is made up of squamous cells.

Analogous to the scrotum, the labia majora are two thick, hair-bearing mounds of tissue that extend from the mons pubis to the perineum. They extend 7–9 cm in length and 2–4 cm in width. Three layers of the labia majora can be identified. The most superficial layer is called the tunica dartos labialis whose fibers run perpendicular to the folds of the skin. The middle layer contains adipose tissue and areolar tissue as well as sweat glands. The deepest layer contains muscle fibers that are contiguous with the round ligament.

The labia minora measure 5 cm in length and only 1 cm in thickness. Fusing with the prepuce of the clitoris, they originate at the clitoris and fuse posteriorly with the labia majora. The skin is smooth, pigmented, and hairless. The glands of the labia minora are analogous to the glands of Littre in the male urethra.

The clitoris contains two cavernous bodies that join at the glans, which contains erectile tissue. The suspensory ligament of the clitoris inserts into the inferior rami of the pubic symphysis. Inferiorly, the labia minora fuse to form a frenulum.

Blood Supply

The anterior branch of the internal iliac artery (hypogastric artery) gives rise to the internal pudendal artery. It courses through the lesser sciatic foramen where it joins the pudendal nerve and dives through Alcock's canal and then branches into the arteries to the gluteal region, the inferior hemorrhoidal artery, the perineal artery, and the artery to the clitoris. The inferior hemorrhoidal artery supplies the anus, the anal canal, and the perineum. The perineal artery perforates the urogenital diaphragm and supplies the ishiocavernosus, bulbocavernosus, and transverse perinei muscles. The termination of the perineal artery is in the labia.

The artery to the clitoris runs along the inferior pubic rami where it branches into four parts: the artery of the bulb, the urethral artery, the deep artery of the clitoris, and the dorsal artery

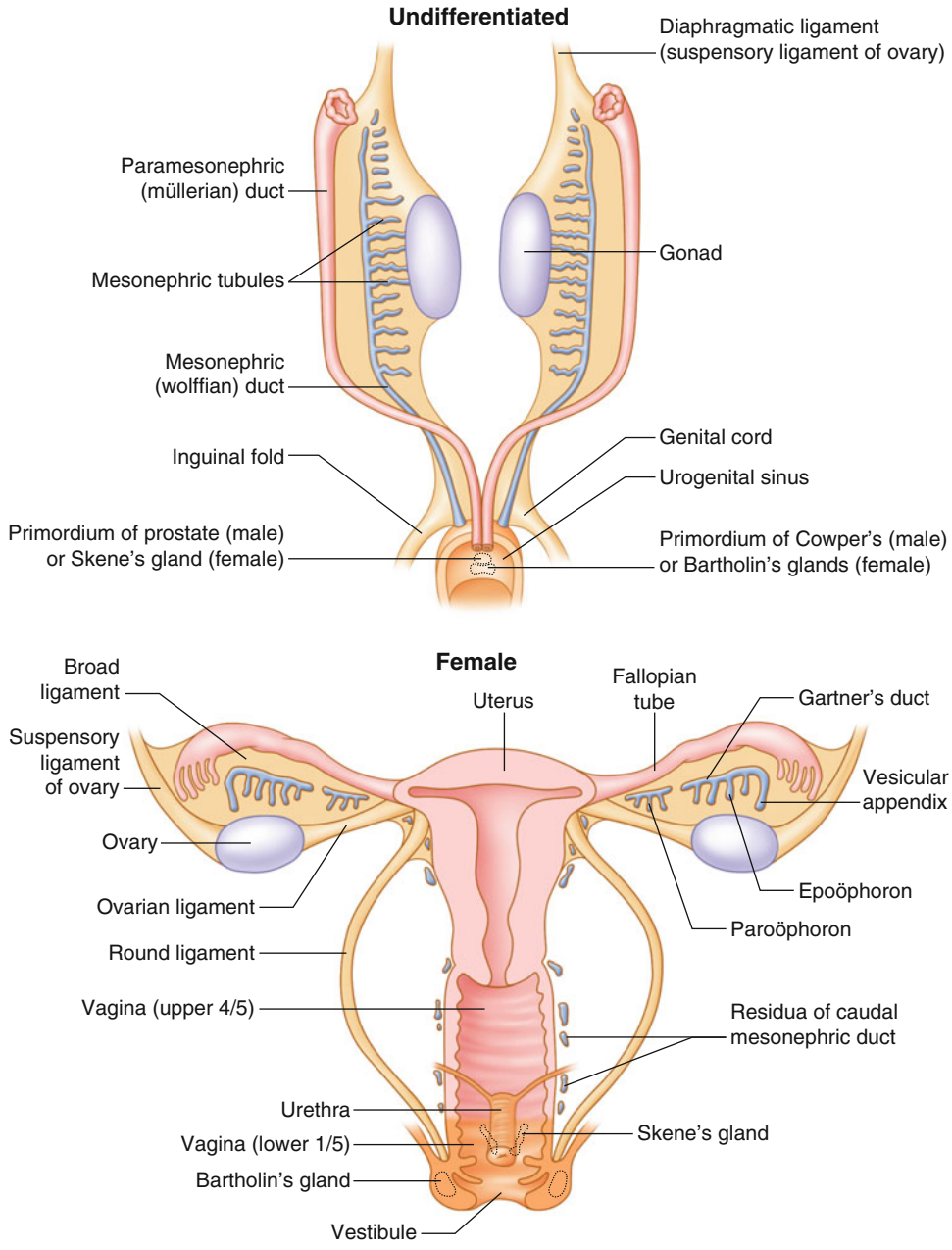


Fig. 1.18 Embryology of the female genital tract

of the clitoris. The artery of the bulb supplies the vestibule and Bartholin's gland. The urethra artery supplies the urethra and anastomoses with the artery of the bulb. The deep artery of the clitoris supplies the corpus cavernosum. The superficial artery supplies the glans.

Nerve Supply

The vulva is highly innervated with nerves, originating from the lumbosacral region of the spinal cord. The main nervous supply to the region comes from the pudendal nerve, which combines branches from S2-4. It divides into three branches

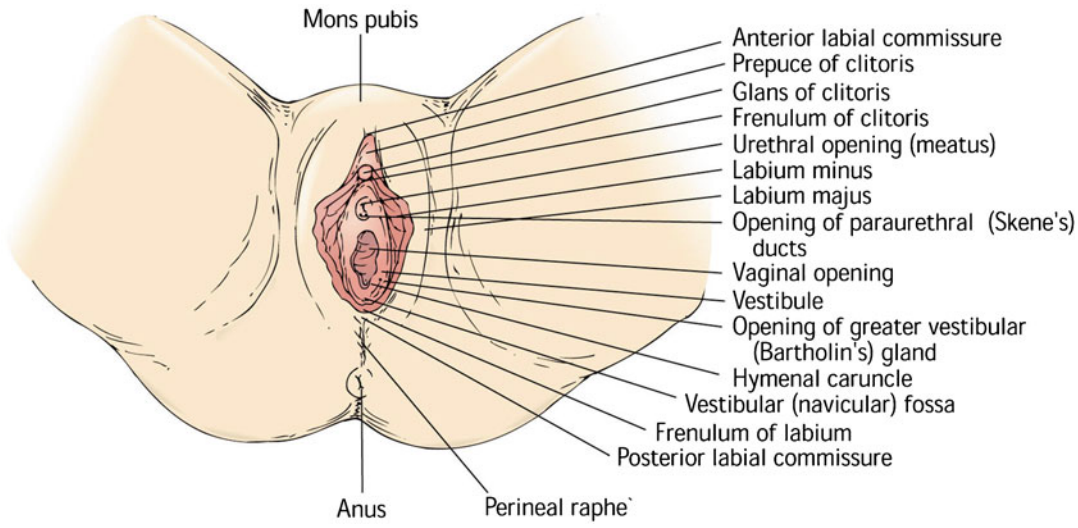


Fig.1.19 Anatomy of the vulva

after it exits Alcock's canal, including the inferior hemorrhoidal nerve, the perineal nerve, and the deep nerve to the clitoris. The inferior hemorrhoidal nerve supplies the external anal sphincter and peri-anal skin. The perineal nerve divides into deep and superficial branches. The deep branch supplies the bulbocarvernosus and ischiocavernosus muscles. The superficial branches supply the labia. The deep nerve to the clitoris supplies the glans.

Many nerves supply the skin of the vulva. Arising from T12 to L1, the iliohypogastric nerve runs between the internal iliac and transversus muscles to the iliac crest, where it divides into an anterior and posterior portion. The anterior hypogastric nerve runs superficially along the skin of the symphysis, and supplies the labia majora and the mons pubis. The posterior hypogastric nerve dives down into the gluteal area. The ilioinguinal nerve originates from L1 and supplies the labia majora as well. Also arising from L1-2, the genitofemoral nerve runs along the psoas muscle and innervates the muscle fibers within the labia majora. The femoral cutaneous nerve originates from posterior branches of S1-2 and the anterior branches of S2-3. It supplies the lateral aspect of the thigh and the labia majora.

Vagina

Embryology (Fig. 1.18)

Early in the embryonic stage, both genders form the Wolffian (mesonephric) ducts and the Mullerian (mesonephric) ducts. The Wolffian ducts eventually develop into the male ducts and the seminal vesicles. The Mullerian ducts become the fallopian tubes, the uterus, and the upper vagina. The muscles and fascia that line these ducts are derived from mesoderm that is adjacent to the ducts. Both sets of ducts terminate in the cloaca, which is later separated by the urogenital ridge.

The upper vagina originates from primordial mesoderm that differentiates into the urogenital ridge, which forms the Mullerian duct. By the eighth week of gestation, the two caudal ends of the Mullerian duct fuse in the midline, and the septum between involutes, creating the uterovaginal canal. The epithelium of the vagina, the vestibular glands, and the hymen, comes from the urogenital sinus, which comes from the cloaca, a derivative of the endoderm.

Anatomy

The vagina is a muscular canal approximately 7–8 cm in length that extends from the vestibule

to the uterus. Laying parallel to the sacrum, it meets the cervix at a 45° angle. The anterior wall is slightly shorter than the posterior wall because of the insertion of the cervix. The apex of the vagina includes the anterior and posterior fornices and the two lateral fornices. The distal end of the vagina traverses the urogenital diaphragm and is surrounded by the bulbocaversnosus muscles and bodies, which can be voluntary contracted during Kegel maneuvers.

Anteriorly, the urethra, ureters, and bladder lie on top of the vagina. The posterior aspect is covered by peritoneum, which forms the pouch of Douglas, or the rectovaginal pouch. Proximally, the vaginal sits on top of the rectum but the organs separate more distally as the rectum dives posteriorly. This muscular space creates the perineal body. The lateral apex of the vaginal canal sits about 1 cm from where the uterine vessels cross the ureter. Distally, the vaginal abuts the levator ani muscles. The vaginal supports include the bulbocavernosus muscles at the introitus, the levator ani muscles (puborectalis muscle) in the distal third, and the cardinal ligaments at the apex. Gartner ducts, vestiges of the Wolffian ducts (mesonephric duct), are sometimes palpable on the lateral aspect of the vagina.

The vagina is lined by stratified squamous epithelium, which is devoid of glands. Vaginal secretions are composed of cervical mucus, desquamated epithelium, and direct transudate. The mucosa is arranged in rugae on the anterior and posterior wall that can distend during parturition and intercourse. The submucosa contains a rich plexus of veins, as well as the lymphatics and nerves. The muscular component of the vagina is arranged in three layers, an outer longitudinal layer, and middle and inner circumferential layers.

Blood Supply

Although it may arise from the hypogastric artery or the superior vesical artery, the main blood supply to the vagina usually comes from the uterine artery, which is a branch of the internal iliac (hypogastric) artery. Passing behind the ureter at the cephaled end of the canal, the vaginal artery anastomoses with branches of the uterine artery, creating a rich blood supply. The posterior aspect of the vaginal canal receives blood from the inferior and middle hemorrhoidal arteries.

Nerve Supply

The vaginal receives somatic innervation from the pudendal nerve, which branches into the perineal nerve as it comes through Alcock's canal. The perineal nerve supplies the bulbocavernosus and ischiocavernosusm muscles that surround the vaginal canal. Both sympathetic and parasympathetic nerves that emanate from the inferior hypogastric plexus supply the vaginal mucosa.

The Uterus

Embryology (Fig. 1.18)

Even in the absence of ovaries, the mullerian ducts differentiate into the uterine tubes, the uterus, and the upper vagina in the female embryo. The intermediate mesoderm evolves into the urogenital ridge, by the fourth week of gestation. The urogenital ridge gives rise to the mesonephric (wolffian) duct and the paramesonephric (mullerian) duct. The paramesonephric ducts become the uterine tubes and the uterovaginal primordia. Between weeks 9 and 16, the uterus and upper part of the vagina form. The uterovaginal primordia join the urogenital sinus as it is separating from the cloaca. The septum between the fused paramesonephric ducts, now the uterovaginal primordia, degenerates, creating a single cavity. The fundus, body, and isthmus of the uterus, including the glands and endometrial epithelium, form from this structure. The endometrial stroma and smooth muscle are derived from splanchnic mesenchyme. Developing over the entire gestation, the form of the uterus evolves in the first trimester, the glands and muscle layers form in the second trimester, and the mucus producing cells of the cervix form during the last trimester.

Anatomy

The uterus is a muscular organ that sits in the pelvis between the bladder and the rectum. Divided

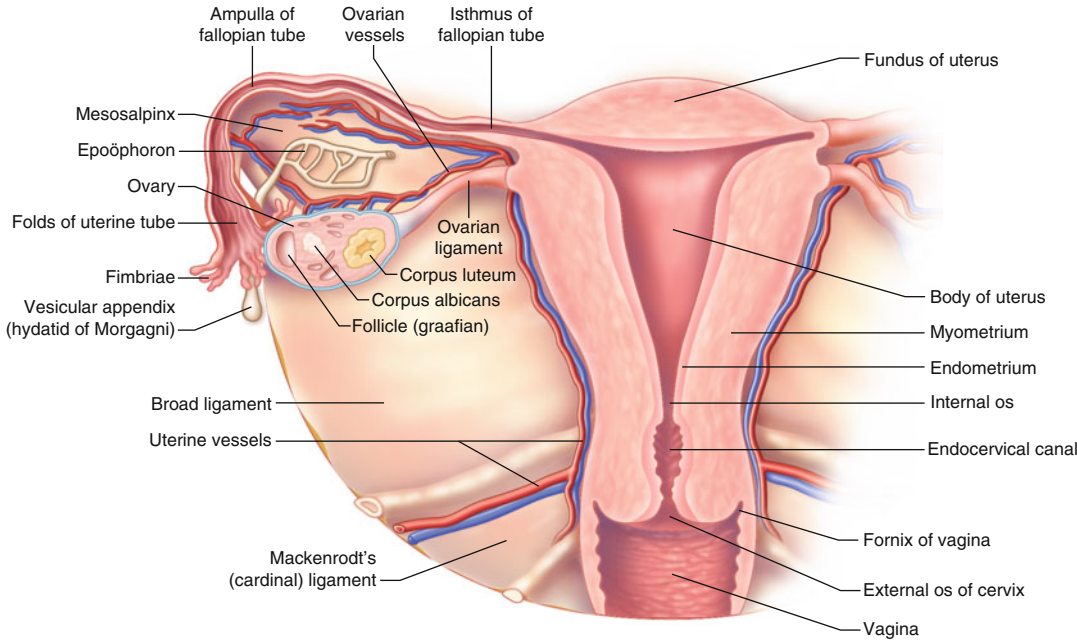


Fig. 1.20 Uterine ligaments and relationships to ovaries and fallopian tubes

into two main portions, the body is connected to the cervix by the isthmus, a transverse constriction. The broad ligament, a fold of peritoneum, covers the body of the uterus. The fundus is the part of the uterus that sits above the axis of the tubes. The communication of the cervix into the body of the uterus is called the internal os and the opening of the cervix into the vagina is called the external os. In nulliparous women the external os is round, whereas it forms a slit in women who have borne children. The uterus changes size with age and parity. In adult women who have not had children, it measures 7 cm in length and 4–5 cm in diameter. It is larger in parous women.

Anteriorly, the bladder sits on the uterus separated by the uterovesical pouch, which is a fold of peritoneum. On the posterior surface, the rectum separates the uterus by a deep fold of peritoneum that extends down to the upper vagina, creating the pouch of Douglas. The small intestine sits on the uterus and can extend into the pouch of Douglas if the walls are not fused. Laterally, the tubes, round ligament, ligament of the ovary, the uterine artery and vein, and the ureter all sit in close proximity. The ureters run parallel to the cervix as they enter the bladder. The uterine vessels cross over the ureter 1.5 cm from the lateral fornix of the vagina (water under the bridge).

The ligaments (Fig. 1.20) that support the uterus include the uterosacral ligaments, the cardinal ligaments (also called the transverse ligaments of the cervix and the ligamentum transversum colli, and the ligament of Mackenrodt), the round ligaments, and the broad ligaments. Although the cervix is fixed, the body and fundus of the uterus move freely with changes in the bladder and with pregnancy. The uterosacral ligaments are folds of peritoneum that coalesce at the cervix. Although they have some involvement in fixing the cervix to the sidewall, they mostly carry sympathetic, parasympathetic, and C fibers from the inferior hypogastric nerve. Supporting the cervix, the cardinal ligaments fan out from the cervix to the lateral sidewall. They contain the uterine artery, vein, and nerves, which perforate the ligament at the internal os. The round ligament is a vestige of the gubernaculum and enters the inguinal ring with the ilioinguinal and the genitofemoral nerves. Providing no support, the broad ligament drapes over most of the uterus.

The uterine wall consists of three layers: serous, muscular, and mucous. The serous layer

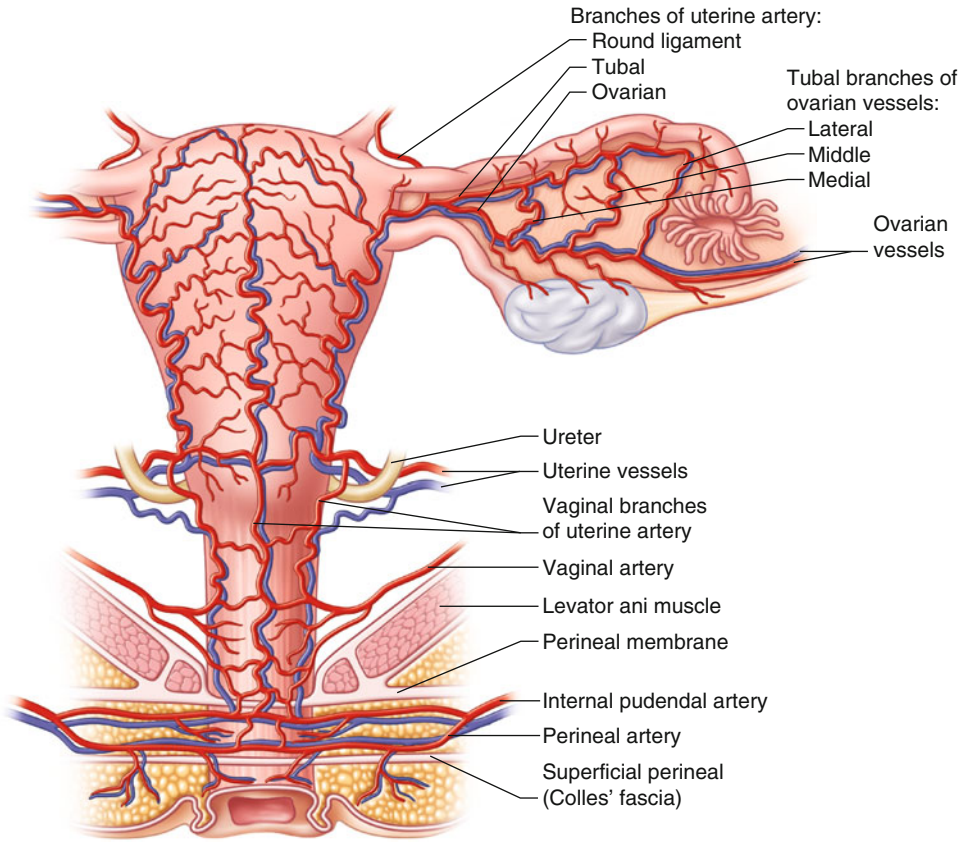


Fig. 1.21 Blood suppply to the uterus

(parametrium) is the peritoneal covering over the uterus. The muscular layer (myometrium) is a thick layer of muscle that is continuous with the vaginal muscle layer, the muscle layer of the tubes, and with the round, ovarian, cardinal, and uterosacral ligaments. While the outer layer of muscle is weak, the inner layer contains strong multidirectional fibers interspersed with a large venous plexus. The cervix has no smooth muscle layer beyond the internal os. The inner layer, the endometrium, is composed of soft, spongy connective tissue containing multiple tubular glands lined by ciliated columnar epithelium.

Blood Supply (Fig. 1.21)

The blood supply to the uterus primarily comes from the ovarian artery, a branch of the aorta, and the uterine artery, a terminal branch of the internal iliac (hypogastric) artery. Giving off branches as it descends, the uterine artery runs laterally along the parametrium to the cervix, where it travels over the ureter about two centimeters lateral to the cervix. Taking a tortuous course through the organ, the uterine artery gives off spiral branches that provide nutrients to the placenta during pregnancy. Branches of the uterine artery encircle the cervix and anastomose with branches of the vaginal artery, which is a terminal branch of the hypogastric or superior vesical artery (a branch of the hypogastric artery as well). Cephalad, the uterine artery, becomes the tubal artery, where it supplies the mesosalpinx and anastomoses with the ovarian artery, a direct branch of the aorta.

Nerve Supply

The visceral organs of the pelvis are mainly innervated by the autonomic nervous system,

which carries both motor and sensory nerve fibers. Divided into three groups, the innervation to the uterus is comprised of the superior hypogastric plexus, the middle hypogastric plexus, and the inferior hypogastric plexus. Located below the inferior mesenteric artery over the middle sacral vessels at the level of L4- L5, the superior hypogastric plexus connects with the inferior hypogastric plexus through intermesenteric nerves, which also receive branches from the lumbar sympathetic ganglia. Not always present, the middle hypogastric plexus sits at the sacral promontory. The inferior mesenteric plexus, also known as the hypogastric nerves, derives innervation from S2-S4 (nervi erigentes—the parasympathetic component of the inferior mesenteric plexus), while it dives into the pelvis through the sacrouterine ligament into the upper vagina, where it becomes the pelvic plexus.

Located lateral to the uterus, the hypogastric ganglia supply nerves to the uterus through the internal os, the isthmus, and the broad ligament, where they enter the body of the uterus. Both myelinated and unmyelinated fibers innervate the uterus and run along the same course as the blood vessels with a higher concentration of fibers at the isthmus and a lower concentration at the fundus. Vader–Pacini corpuscles (corpuscles lamellose) and Dogiel and Krause corpuscles are seen within nerve bundles in the region of the endocervix, the broad ligament, and where the uterine artery meets the uterus. They induce uterine contractions during labor through modulation of the stretch response.

The Oviducts (Fallopian Tubes)

Embryology (Fig. 1.18)

The oviducts arise from the unfused, cranial end of the paramesonephric duct. The ostium of the oviduct is formed from the open end of the same segment. The mesoderm gives rise to the urogenital ridge, which gives rise to the mesonephric (Wolffian ducts) and paramesonephric (Mullerian) ducts. The mesonephric ducts give rise to the male genital ducts and the paramesonephric ducts give rise to the uterine tubes, the uterus, and the upper vagina.

Anatomy

Each oviduct is 7–14 cm long and is divided into three parts: the isthmus, the ampulla, and the infundibulum. Joined to the uterus, the isthmus is straight, with a long intramural segment. The ampulla is the longest part of the tube. It terminates into the infundibulum, which extends into finger-like projections, called fimbriae, the longest of which communicate with the ovary. The mouth of the infundibulum sits inside the peritoneum.

Each tube is covered by peritoneum on three sides, and the mesosalpynx on the fourth side. Blood vessesl and nerves run in the subserous layer of each tube. The muscle layer has smooth muscle fibers arranged in an outer longitudinal and an inner circular layer. The mucosal layer is lined with ciliated columnar epithelium.

Blood Supply

Both the ovarian and the uterine artery supply the tubes. The blood supply coming off of the uterine artery runs on the inferior aspect of the tube up to the fimbria where it meets branches of the ovarian artery.

Nerve Supply

Both sympathetic and parasympathetic nerve fibers originating in the pelvic plexus innervate the fallopian tubes. The ampulla receives innervation from nerves of the ovarian plexus, while the uterovaginal plexus supplies the isthmus. The nerves enter the tubes through the mesosalpinx.

Ovaries

Embryology (Fig. 1.18)

Derived from the mesoderm, the urogenital ridge gives rise to the gonadal ridge, which differentiates into the testis or the ovary at 8 to 10 weeks of gestation. In the absence of the Y chromosome, the embryo does not produce testis-determining factor, anti-mullerian hormone, or testosterone. Without these substances, the gonad will become an ovary by default. Although formation of the ovary forms by default, without two X chromosomes, the ovaries will not develop into functioning organs. Around the fourth week of gestation, the

germ cells migrate from the allantoic region to the urogenital ridge.

From weeks 9 to 10, the germ cells organize in the cortical region of the ovary, with interspersed epithelial and somatic cells. The primordial follicles contain diplotene oocytes. The ovarian medulla is composed of mesenchymal cells, somatic cells, connective tissue, and blood vessels. The mesovarian joins the ovary with the vestigial urogenital ridge.

Anatomy

Located immediately below the pelvic brim, the ovaries are 2.5–5 cm in length and weigh from 4 to 8 g each. The anterior surface of each ovary is tethered to the mesovarian (a peritoneal covering that connects the ovary to the broad ligament) and the posterior aspect is free. The upper pole sits next to the ipsilateral tube and the lower pole abuts the uterus. The ovaries are covered by the small intestine. The blood vessels, lymphatics, and nerves that supply the ovaries enter through the mesovarian at the hilum.

The ligaments that support the ovaries are the mesovarian, the suspensory ligament of the ovary, the ovarian ligament and the infundibulopelvic ligament. The mesovarian is made up of a double layer of peritoneum, containing branches of the uterine and ovarian arteries, a nerve plexus, the pampiniform vein plexus, and the lateral end of the ovarian ligament. Crossing the iliac vessels, the suspensory ligament is a peritoneal attachment at the lateral aspect of the ovary and contains the ovarian vessels. The ovarian ligament attaches the ovary to the medial aspect of the uterus. The infundibulopelvic ligament attaches the ovary to the sidewall. It contains the ovarian artery, veins, and nerves as they pass through the mesovarian into the hilum of the ovary.

The ovary is divided into the cortex and the medulla. The cortex contains the ova, which mature at different rates. Follicles form around the developing ova and project onto the free surface of the ovary. These are called graafian follicles. Fully mature ova within a follicle transform into corpus luteum. Once the ova is released or involutes, the corpus luteum becomes a corpus albicans, or scarred follicle. The medulla contains connective tissue, which supports the blood supply, lymphatics, and nerves to the cortex.

Blood Supply

Emanating from the abdominal aorta, the ovarian blood vessels are the main blood supply to the ovary. The left ovarian artery often comes off of the left renal artery. After crossing the common iliac artery, they turn medially over the ureter and into the pelvis where they are covered by the folds of the suspensory ligament of the ovary and enter the hilum through the mesovarian. The ovarian branch of the uterine artery supplies the ovary from the medial aspect, creating an anastomosis with the ovarian vessels.

Nerve Supply

The nerves run along the same course as the arteries. They originate from the lumbosacral sympathetic chain.

Bibliography

1. DeCherney AH, Nathan L. Current obstretric and gynecologic diagnosis and treatment. 9th ed. New York, NY: McGraw-Hill/Lange Medical Books; 2003.
2. Hoffman B, Schorge J, Schaffer J, Halvorson L, Bradshaw K, Cunningham F. Williams gynecology. 2nd ed. New York, NY: McGraw-Hill Professional; 2012.
3. Moore KL, Agur AMR, Dalley AF. Essential clinical anatomy. Baltimore, MD: Lippincott Williams & Wilkins; 2011.
4. Netter FH. Atlas of human anatomy—Volume 2: reproductive system. CIBA Medical Education Department, East Orange, NJ, CIBA Collection. 1954.
5. Raz S, Rodriquez LV. Female urology. 3rd ed. Philadelphia, PA: Saunders; 2008.
6. Rock JA, Jones HW. TeLinde's operative gynecology. 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.
7. Tenagho E, McAninch J. Smith's urology. 18th ed. New York, NY: Lange Medical Books, McGraw- Hill; 2012.
8. Vasavada SP, Appell RA, Sand P, Raz S. Female urology, urogynecology, and voiding dysfunction. London: Taylor and Francis; 2005.
9. Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA. Campbell-Walsh urology. 9th ed. Philadelphia, PA: Saunders; 2006.

Instrumentation for Female Pelvic Surgery

2

Stephan Seklehner, Melissa A. Laudano, and Richard Lee

Introduction

Vaginal surgery requires proper instrumentation for success. The following instruments represent only a sampling of all that are available but which we have found most useful for female pelvic surgery. A picture as well as a brief description of each instrument has been provided. For a full listing, see Table 2.1.

Instruments Used for Open/Vaginal Surgery

Uterine Tenaculum (Fig. 2.1)

Tenacula are useful instruments for grasping and holding tissue. The piercing hooks of a tenaculum can be anchored onto areas such as the cervix. The term tenaculum is derived from the Latin word "tenere" which means "to hold or grasp." Uterine tenacula can be made of stainless steel, which makes them reusable after sterilization, or of plastic as disposable devices.

S. Seklehner, M.D. • M.A. Laudano
R. Lee, M.D., M.B.A (✉)
Department of Urology, Weill Medical College of Cornell University, New York, NY, USA
e-mail: St.seklehner@gmx.net; melissa.laudano@gmail.com; Ril9010@med.cornell.edu

Allis Forceps (Fig. 2.2)

Allis forceps are instruments designed to grasp and hold structures in atraumatic fashion. Their serrated jaws contain an atraumatic tooth-like structure, which enables the surgeon to hold on to delicate structures. Both self-locking and non-self-locking forceps are available according to the surgeon's preference.

DeBakey Forceps (Fig. 2.3)

DeBakey forceps are atraumatic forceps meant to grasp delicate tissue. The inner aspect of the tips contains microscopic, atraumatic teeth while the outer side of the instrument is normally ridged providing a better grip while handling with delicate structures. DeBakey forceps are normally straight with curved variants available for special utilization. Size can range up to 9.5 in.

Metzenbaum Scissors (Fig. 2.4)

Metzenbaum scissors are named after the American surgeon who first designed the instrument. Mostly known as dissecting scissor, it is an excellent tool to perform fine cuts. Models with tungsten carbide cutting edges are the most precise while cheaper models are made of stainless steel. Regardless if the blades are curved or straight, if small (4.5 in.) or large (14 in.), they

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_2,

Table 2.1 List of instruments and devices for female pelvic surgery

Open surgery	Catheters Diluted indigo carmine solution Yellofin® Stirrups (Allen Medical Systems, Hill-Rom Company, Batesville, IN) Cystoscope Weighted vaginal speculum Uterine tenaculum Allis forceps DeBakey forceps Metzenbaum scissors Mayo scissors Deaver retractor Vaginal handheld retractors Self-retaining retractors Stamey needle Raz double-pronged needle Heaney needle holder Capio device Phaneuf clamp Surgical sutures Vaginal packing
Laparoscopic surgery	Veress needle Laparoscopic trocars Ligasure™ (Covidien, Dublin, Ireland) Harmonic® scalpel (Ethicon Endo-Surgery, Inc., Johnson & Johnson, New Brunswick, NJ) Laparoscopic forceps Laparoscopic scissors
Robotic surgery (Da Vinci® Surgical System) (Intuitive Surgical, Inc., Sunnyvale, CA)	Monopolar scissors Bipolar cautery Dissectors Forceps Needle drivers

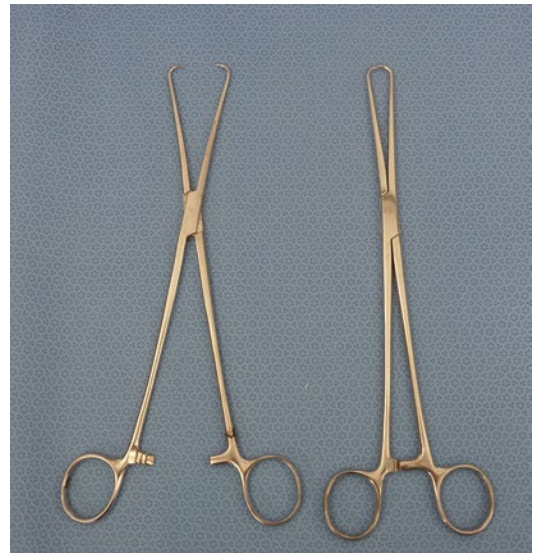


Fig. 2.1 Uterine tenaculum

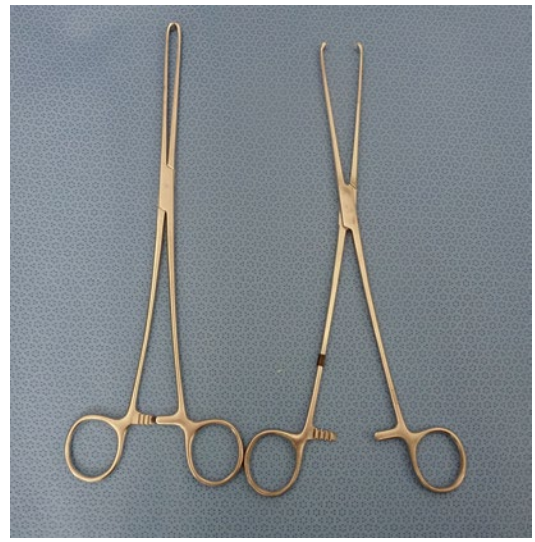


Fig. 2.2 Allis forceps

typically possess a long shank or handle with a blunt-tipped scissor sorter blades. Metzenbaum scissors should not be used to cut regular sutures as they can be dulled easily.

Mayo Scissors (Fig. 2.5)

In contrast to the Metzenbaum scissor, the Mayo scissor, named after the Mayo Clinic where it was invented, is designed to cut tougher structures. It can be used for dissecting but it is not as precise as Metzenbaum scissor. Mayo scissors can have either straight or curved blades, although they typically have semi-blunt ends. Straight-bladed Mayo scissors are also called "suture scissors" as they are amenable to cutting sutures since they do not dull as easily as other types of scissors. Curved Mayo scissors are usually used for cutting deeper thick tissue like

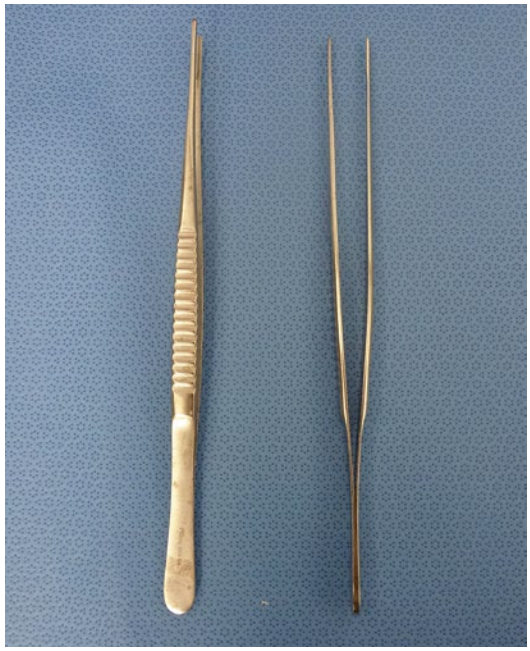


Fig. 2.3 DeBakey forceps

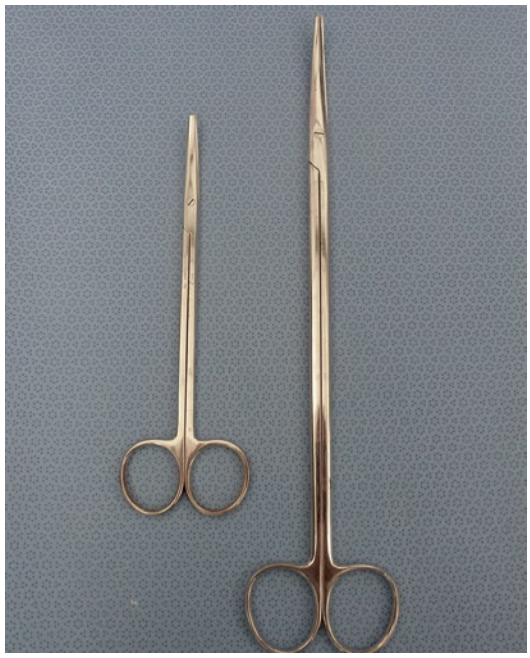


Fig. 2.4 Metzenbaum scissors

the uterus as they allow deeper penetration into the wound. Mayo scissors can be made of titanium or stainless steel and normally range from 6 to 6 ¾ inches.

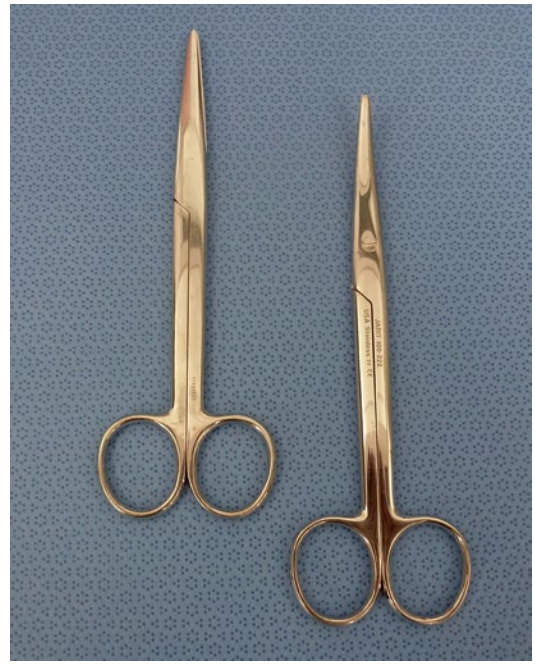


Fig. 2.5 Mayo scissors

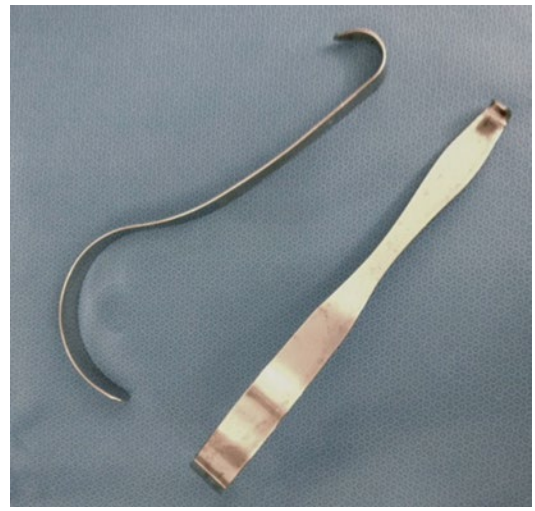


Fig. 2.6 Deaver retractors

Deaver Retractor (Fig. 2.6)

Deaver retractors are flat, thin devices with curved ends. Models can vary according to length, width, and angel of curvature. The edges of the Deaver retractor are smoothed to avoid harm to surrounding tissue.

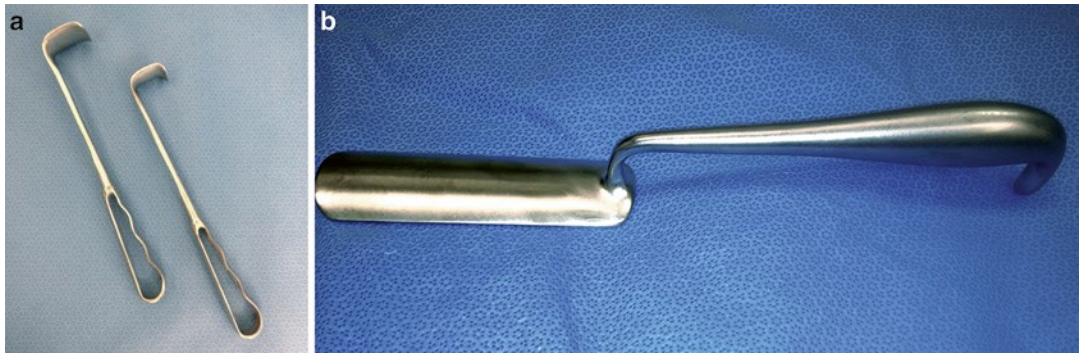


Fig. 2.7 Vaginal handheld retractors

Vaginal Handheld Retractors (Fig. 2.7a, b)

Normally, vaginal retractors consist of a handle and a curved blade with varying lengths, widths, and angles. One of the most commonly used retractors is the Breisky–Navratil retractor, which has a ridged handle and a notch for good retraction and comfortable handling. Lighted vaginal retractors, like the Heaney–Simon or the Miyazaki retractors, have also been proposed for use in the deep pelvic surgery.

Self-Retaining Retractors (Fig. 2.8)

The Scott retractor is a self-retaining retractor commonly used in vaginal surgery. Multiple piercing or non-piercing hooks can be used to optimize exposure of the surgical field. It can be used in tandem with a weighted vaginal speculum to maximize visualization during vaginal surgery.

Fig. 2.8 Self-retaining retractor (Cooper surgical, Trumball, CT)

Weighted Vaginal Speculum (Fig. 2.9)

A weighted vaginal speculum contains a weight at one end, utilizing gravity to help retract the posterior aspect of the vagina. Two main types of weighted specula exist: the traditional and the articulated device. Traditional specula have a

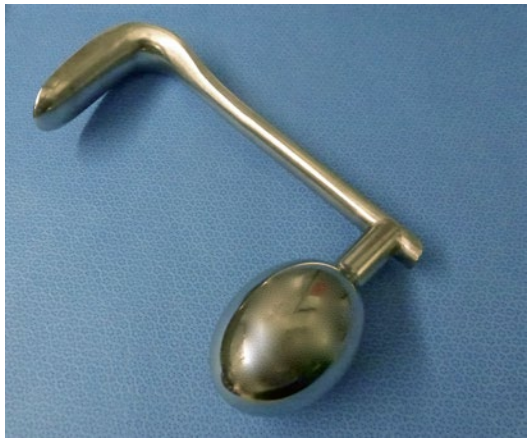


Fig. 2.9 Weighted vaginal speculum

rounded heavy end and a hollow groove which is positioned at a 90° angle to the blade of the retractor. Articulated specula can be adjusted by altering angles and lengths and can be made of plastic as well. There are several types of weighted specula, such as the Hardy-Duddy, Auvard, or Steiner variants.

Stamey Needle

The Stamey needle is a reusable special needle, with which sutures can be pulled from a vaginal incision into the suprapubic area. This needle can be needed for bladder suspension surgery.

Raz Double-Pronged Needle (Fig. 2.10)

This device is a double-pronged ligature carrier, which can be used for bladder neck suspension or sling placement. The tip of the needle is placed under finger guidance to the desired position. The inner segment of the device can be slid over the external support, extending the tip of the needle to place the suture in the desired position.

Fig. 2.10 Raz double-pronged needle

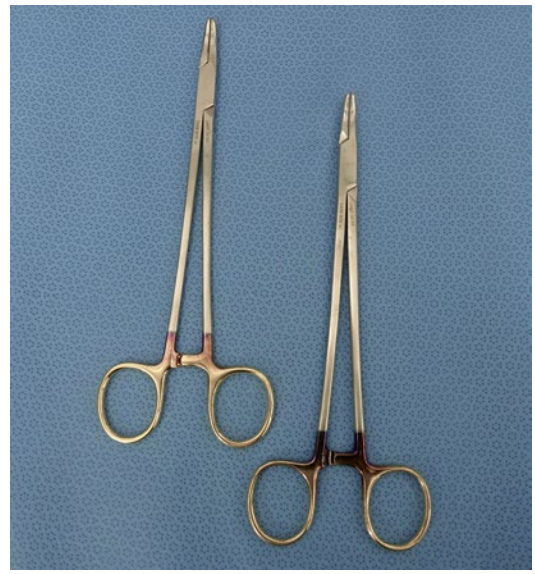


Fig. 2.11 Heaney needle holder

Heaney Needle Holder (Fig. 2.11)

The Heaney needle holder is a curved needle holder, which can be useful in situations where

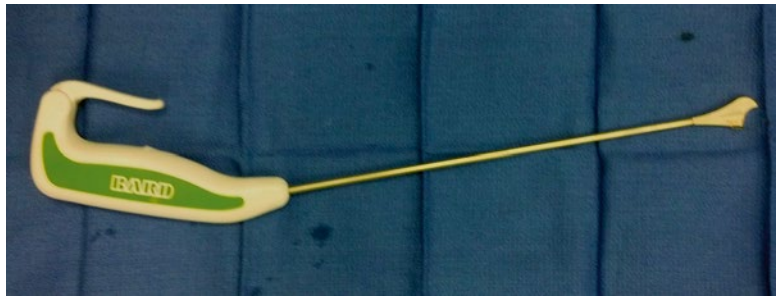


Fig. 2.12 Capio® device (Bard Medical, Covington, GA)

the use of a straight needle holder is difficult due to nearby anatomic structures.

Capio® Device (Fig. 2.12)

The Capio® (Bard Medical, Covingon, GA) device is designed to throw, catch, and retrieve sutures. It is supposed to extend the surgeon's reach into deep areas while ensure precise suture placement in difficult-to-reach areas. It consists of a head that can be angled, a needle driver component, as well as alignment indicator.

Phaneuf Clamp (Fig. 2.13)

The Phaneuf clamp can either be straight or curved-bladed. With single teeth at its blunt tip and serrated inner blades, it represents a solid instrument to clamp bigger and thicker structures like the cardinal, uterosacral, and broad ligaments during hysterectomy.

Cystoscope (Fig. 2.14)

Urethrocystoscopy is an endoscopic procedure of the urethra and the bladder performed with a cystoscope. Cystoscopy is a routinely performed diagnostic as well as therapeutic procedure and can be done with flexible or rigid devices. Most cystoscopes have one or two ports allowing delicate instruments to be inserted like forceps or ureteral stents.

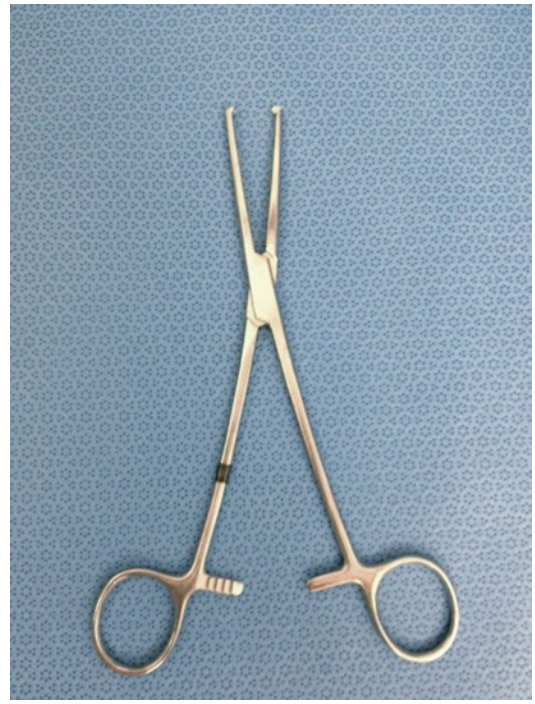


Fig. 2.13 Phaneuf clamp

Catheters (Fig. 2.15)

Indwelling urinary catheters are commonly used during surgery and in the perioperative period in order to guarantee emptying of the urinary bladder. Catheters can be made of many different types of material with silicone being most common. The size of catheters is measured in Charrière (in English speaking countries the term "French" is mostly used), with one Charrière being approximately 0.3 mm.

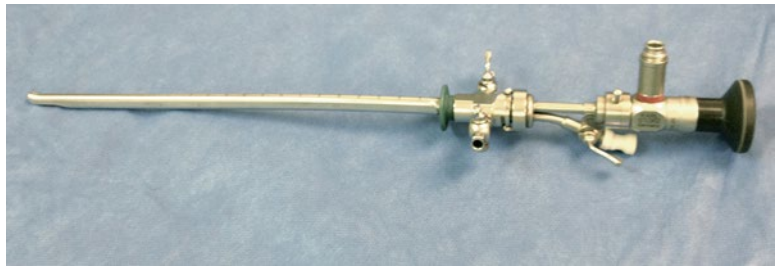


Fig. 2.14 Cystoscope

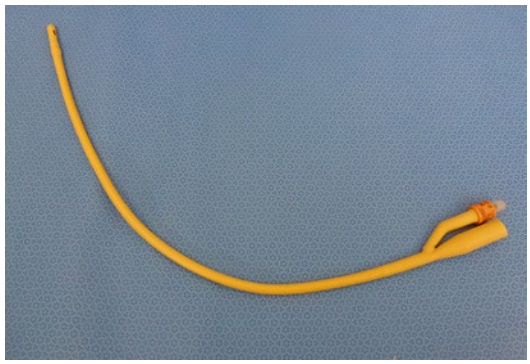


Fig. 2.15 Catheter

Diluted Indigo Carmine Solution

Diluted indigo carmine solution is primarily excreted by the kidneys within a few minutes after intravenous injection. The blue color can be very useful during surgery either to evaluate the exact position of the orifices under cystoscopy or simply to ensure the integrity of the bladder.

Surgical Sutures

Two main types of surgical sutures exist in terms of durability: absorbable and nonabsorbable. Surgical sutures may come in monofilament vs. braided (woven) forms. Suture sizes can range from 11-O to 7 as defined by the United States Pharmacopeia (USP).

Yellofin® Stirrups (Fig. 2.16)

Yellofin® stirrups (Allen Medical Systems, Hill-Rom Company, Batesville, IN) are used to hold legs in position while surgery is performed. The fin design of the boot reduces the likelihood of peroneal nerve injury, which might occur otherwise if the patient is not positioned in a proper way.

Fig. 2.16 Yellofin® stirrup (Allen Medical Systems, Hill-Rom Company, Batesville, IN)

Vaginal Packing

Vaginal packing typically refers to cotton-woven gauze placed into the vagina to absorb bleeding and to provide pressure for tamponade.

Instruments Used for Laparoscopic Procedures

Veress Needle and Laparoscopic Trocars (Fig. 2.17)

Instruments for laparoscopy are introduced into the body via trocars. Although blunt trocars exist, most possess a sharp tip to penetrate tissue. Most trocars have two openings: a primary port for inserting devices into the body and a secondary gas port. Trocars come in different sizes (e.g., 5 French for laparoscopic forceps, 10 or 12 French for cameras and larger instruments) and lengths. Trocars may be reusable or disposable.

It is possible to place the trocars under direct vision (Hassan technique) or blindly. If a blind technique is to be utilized, the desired surgical field should be first insufflated with gas. A Veress needle can be used to first penetrate the skin and underlying tissues to gain access to the desired surgical compartment. Gas is insufflated through the hollow core of the needle, after which the needle is removed and the trocar inserted.

Ligasure™ (Fig. 2.18)

LigaSure™ (Covidien, Dublin, Ireland) is an electrothermal bipolar tissue sealing system, which can be used for both open and laparoscopic procedures. It provides a combination of pressure and energy to seal and cauterize blood vessels and tissue. Vessels up to and including 7 mm in diameter as well as tissue bundles can be fused permanently without dissection or isolation. The seals can withstand up to three times normal systolic blood pressure. In contrast to ultrasonic devices, the LigaSure™ device is able to coagulate larger vessels and tissue structures but at the cost of a larger device size.

Harmonic® Scalpel (Fig. 2.19)

In contrast to the Ligasure™ device, the so-called Harmonic® scalpel (Ethicon Endo-Surgery, Inc., Johnson & Johnson, New Brunswick, NJ) is a cutting and sealing instrument using ultrasound vibratory energy. It is often used in laparoscopic procedures, as it can be directed to either cut tissue

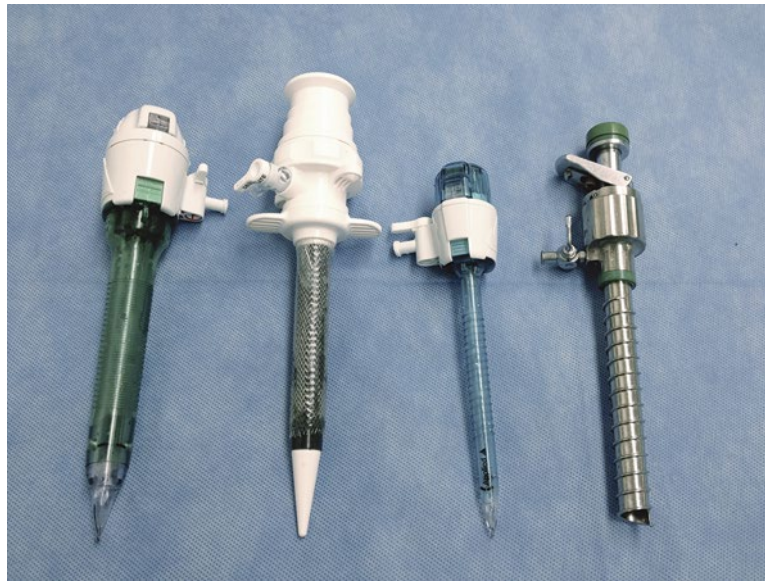


Fig. 2.17 Laparoscopic trocars

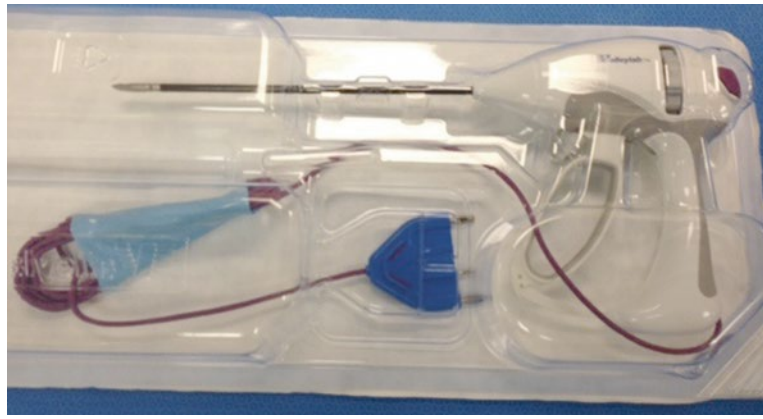


Fig. 2.18 Ligasure™ (Covidien, Dublin, Ireland)

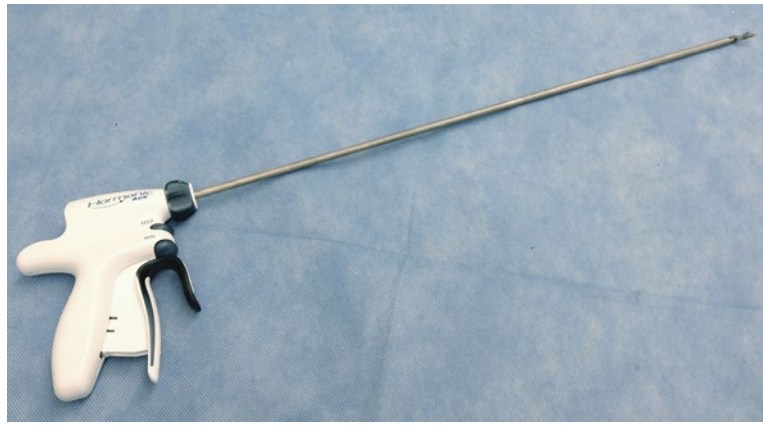


Fig. 2.19 Harmonic® scalpel (Ethicon Endo-Surgery, Inc., Johnson & Johnson, New Brunswick, NJ)

(by vibrating in the range of 55.5 kHz) or to coagulate smaller vessels and tissue by sealing them through protein denaturization.

Laparoscopic Maryland Forceps and Laparoscopic Scissors (Fig. 2.20)

The Maryland forceps has a 10 mm diameter and has a 33 cm length (with up to 45 cm available on request). With is serrated semi-blunt tip, which can be rotated by 360° at the handle, it can be used to grab and secure tissue. Additionally, tissue can be cauterized using electrocautery. As with laparoscopic forceps, the laparoscopic scissors can also be rotated and used to cauterize tissue.

Instruments Used for Robotic Surgery: Da Vinci® Surgical System (Figs. 2.21, 2.22, 2.23, 2.24, and 2.25 a, b)

The da Vinci® Surgical System (Intuitive Surgical, Inc., Sunnyvale, CA) has been approved by the Food and Drug Administration (FDA) for gynecologic laparoscopic surgical procedures. The system consists of a patient-side cart with, depending on the model, three or four robotic arms, and an accompanying command console. While one robotic arm is utilized for the endoscopic camera, which enables full stereoscopic vision for the surgeon, the other arms are used to

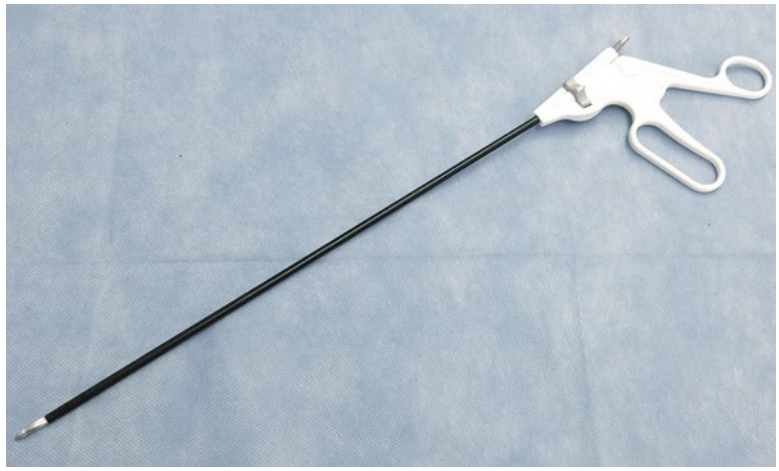


Fig. 2.20 Laparoscopic scissors

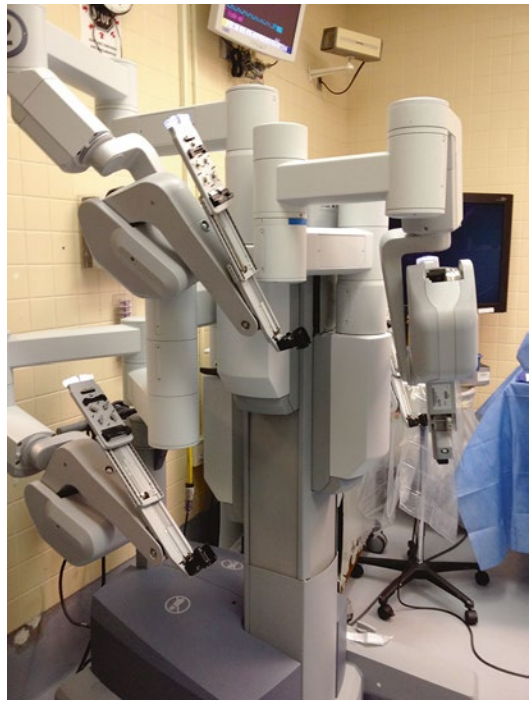


Fig. 2.21 Da Vinci® robot (Intuitive Surgical, Inc., Sunnyvale, CA)

manipulate laparoscopic instruments. The robotic arms can be moved with hand controllers and foot pedals. One of the advantages of the Da Vinci® Surgical System is that it has been designed to maximize flexibility of movements and to minimize human tremor.

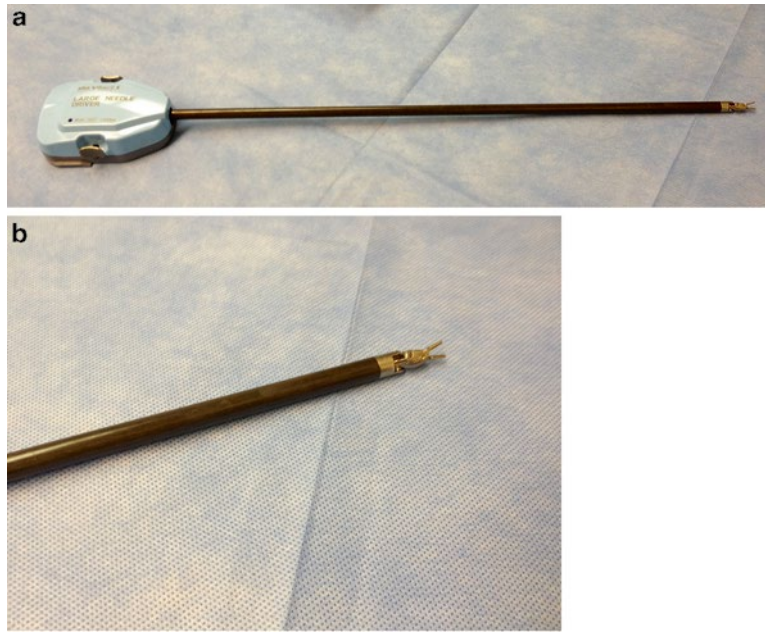


Fig. 2.22 (**a**) Da Vinci® robot needle driver. (**b**) Tip of Da Vinci® robot needle driver (Intuitive Surgical, Inc., Sunnyvale, CA)

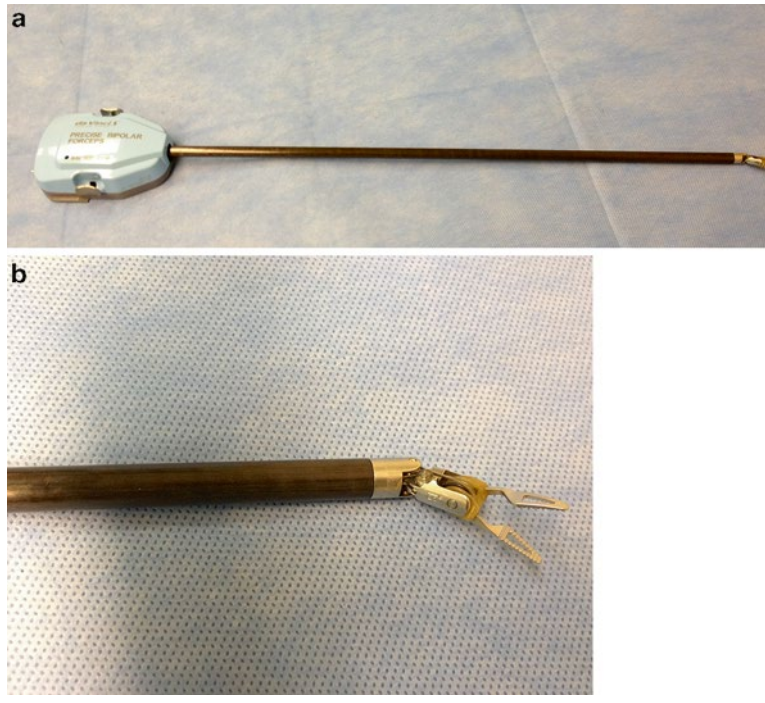


Fig. 2.23 (**a**) Da Vinci® robot precise bipolar forceps. (**b**) Tip of Da Vinci® robot precise bipolar forceps (Intuitive Surgical, Inc., Sunnyvale, CA)

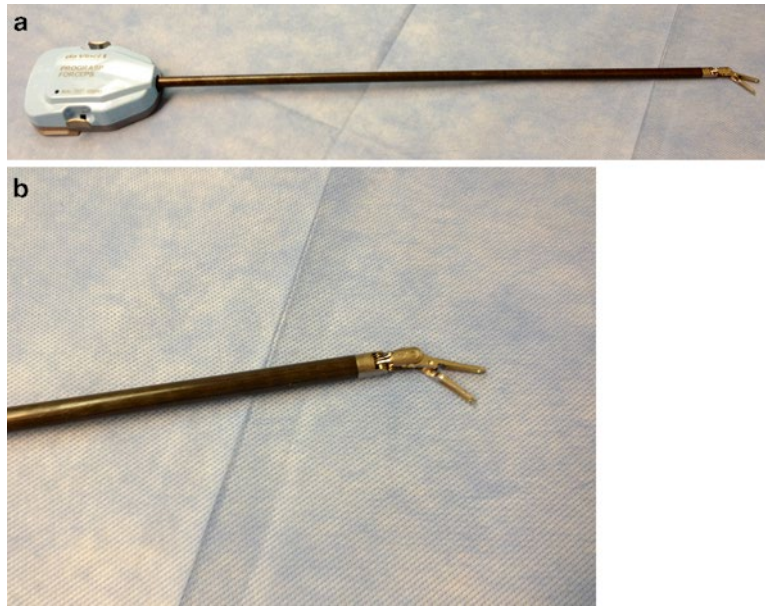


Fig. 2.24 (**a**). Da Vinci® robot prograsp forceps. (**b**) Tip of Da Vinci® robot prograsp forceps (Intuitive Surgical, Inc., Sunnyvale, CA)

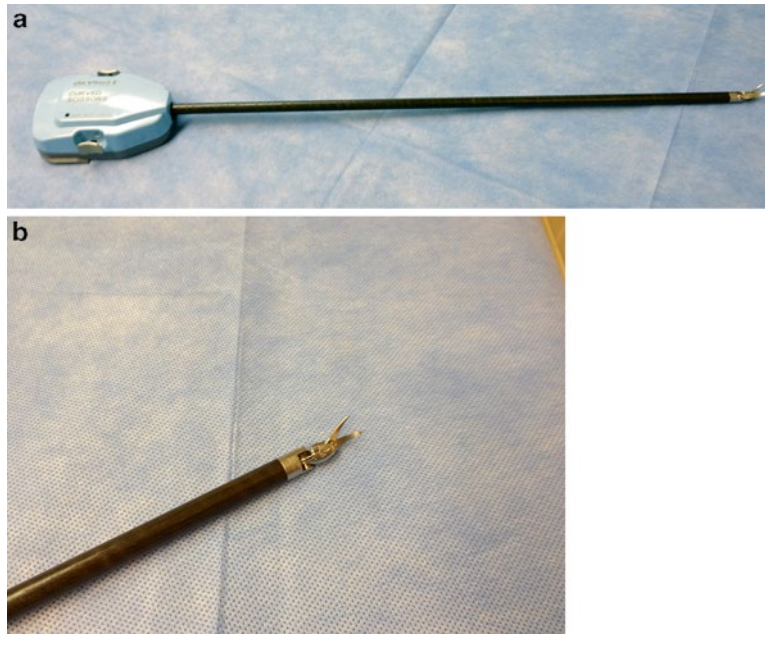


Fig. 2.25 (**a**) Da Vinci® robot curved scissors. (**b**) Tip of Da Vinci® robot curved scissors (Intuitive Surgical, Inc., Sunnyvale, CA)

Surgical Management of Stress Urinary Incontinence

3

Kai-Wen Chuang and Farzeen Firoozi

Introduction

Stress urinary incontinence (SUI) has a reported prevalence between 12.8 and 46 % [1]. There is no doubt that SUI has been clearly shown to negatively impact the everyday quality of life (QOL) of the women who suffer from this dysfunction [2]. The economic burden for the treatment of urinary incontinence have been estimated to be approximately 19 billion annually in the United States [3]. Risk factors for the development of UI include age, obesity, previous pelvic surgery, and childbirth [4].

Surgical management of SUI is the standard of care once conservative options, such as behavioral modification, pelvic floor exercises, fluid modification, and scheduled voiding, have been exhausted [5]. The surgical options have evolved over the last few decades to include the Burch colposuspension, periurethral bulking agents, pubovaginal slings, and the newest multitude of approaches for midurethral synthetic slings [6]. The synthetic slings include retropubic, transobturator, and the newest additions which include the so-called single-incision slings. The aims of this chapter include the evaluation and management of SUI and review each of the surgical techniques currently available to pelvic floor surgeons.

Evaluation

Surgical Options

Burch Colposuspension

In 1961, Burch reported his series of retropubic uretropexies for the management of SUI [7]. The surgery today can be performed via laparotomy, laparoscopy, or Robotic assistance. Although the approaches have become more minimally invasive, the basic surgical tenets remain unchanged.

The surgical selection for the Burch colposuspension includes patients with genuine UI and hypermobility of the urethra. This specifically includes patients who have low leak point pressures with urethral hypermobility as well as those with low urethral closure pressures. Patients with intrinsic sphincteric deficiency (ISD)—defined as SUI despite complete support of the urethra—are not good candidates for the Burch colposuspension. Nowadays, most pelvic surgeons would agree that the Burch colposuspension is a safe and effective surgical option for SUI, largely considered for women undergoing a concomitant

K.-W. Chuang, M.D.
Department of Urology, North Shore—Long Island Jewish Health System, New Hyde Park, NY, USA
e-mail: Kaiwen.chuang@gmail.com

F. Firoozi, M.D., F.A.C.S. (✉)
Hofstra North Shore—LIJ School of Medicine, The Smith Institute for Urology, Lake Success, NY, USA
e-mail: ffiroozi@nshs.edu

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_3,

open or laparoscopic procedure such as pelvic organ prolapse repair.

Surgical Technique

There have been some modifications to the techniques of the Burch colposupension described in the literature since its inception in 1961. The majority of open Burch colposuspensions performed today are closest in technique described by Tanagho [8]. The laparosopic/robotic Burch colposupensions adopt essentially all the same surgical steps but differ in that these are minimally invasive options with equivalent outcomes.

Open

The patient is placed in a dorsal lithomtomy position with legs in stirrups. A 16 Fr. Foley catheter is placed at the beginning of the procedure. A 5 cm transverse suprapubic incision is made to expose the rectus facsia. The rectus fascia is then opened with a similar length incision. The fascia is then mobilized from underlying rectus muscle with electrocautery. The space of Retzius is then entered bluntly and dissected laterally in both directions. A self-retaining retractor (e.g., mini-Bookwalter or Balfour retractor) is placed followed by entry into the dome of the bladder. The surgeon's nondominant hand is placed into the vagina. Two No. 1 nonabsorbable sutures are placed on each side of the urethra. The sutures are thrown with double bites, almost including full thickness of the vaginal wall. The initial suture is at the urethrovesical junction and the second suture is approximately 1 cm caudal. Care is taken to place these sutures at least 4 mm lateral to the urethra. The sutures are then placed through Cooper's ligament (Fig. 3.1). The interior of the bladder is then examined to rule out injury and bilateral efflux from the ureteral orifices is noted. A cotton swab test is performed, after the Foley catheter is removed, demonstrating a 0–10° angle to the horizon by loosening or elevating the sutures. Once this is established, the sutures are tied down. The bladder is then closed with 2-0 absorbable suture in standard 2-layer fashion. A Foley catheter is left indwelling at the end of the procedure.

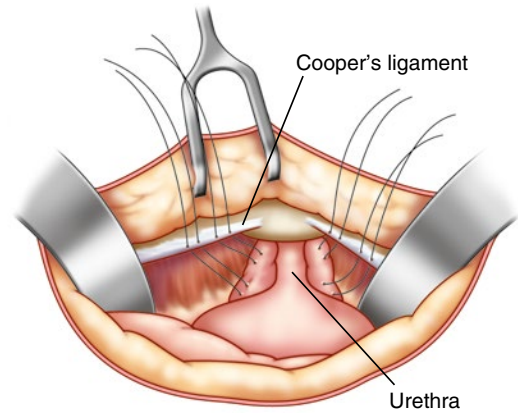


Fig. 3.1 Burch colposuspension: The initial suture is at the urethrovesical junction and the second suture is approximately 1 cm caudal. Care is taken to place these sutures at least 4 mm lateral to the urethra. The sutures are then placed through Cooper's ligament

Laparoscopic/Robotic

The patient is placed in a dorsal lithomtomy position with legs in stirrups. A 16 Fr. Foley catheter is placed at the beginning of the procedure. Monitors, typically two for the surgeon and assistant surgeon, are placed at the patient's feet. The two midline trocars are for both introduction and extraction of curved needles and passage of the laparoscope. The 5 mm trocars are placed laterally at the border of the rectus muscles, at the level of the suprapubic 12 mm trocar (the distance between the pubic symphysis and 5 mm trocars would be no less than 4 fingerbreadths). This distance allows for adequate access to the space of Retzius.

Once any concomitant procedures are completed (e.g., hysterectomy, prolapse repair) the space of Retzius is entered. Blunt and sharp dissection is used to expose the pubic symphysis and Cooper's ligament. Also dissected out and exposed are the lateral pelvic sidewall, obturator neurovascular canal, ischial spine and arcus tendinius, arcus of the levator ani, and the paravaginal fascia. If indicated, a paravaginal repair can be performed if there is a lateral cystocele defect.

At this point, Ethibond No. 0 sutures are placed in the same fashion as described for the open approach. After the sutures are tied down, cystoscopy is performed after indigo carmine is

injected intravenously to check for ureteral efflux, ruling out any obstruction. The 12 mm trocras are closed in standard fashion with 2-0 absorbable suture. The 5 mm trocar facsia does not need to be closed. The patient may undergo a voiding trial in the recovery room according to individual surgeon preference.

Outcomes

The Burch colposuspension has been shown to outperform pharmacotherapy, conservative management, needle suspensions, Marhsall–Marchetti–Krantz procedure, and anterior colporraphy [9]. A recent Cochrane review of open Burch colposuspensions reported an overall success rate of 69–88 %. This same meta-analysis had separately reviewed 12 trials comparing open approach versus laparoscopic approach and found no statistically significant difference in patient-reported incontinence and the 1-year and 5-year follow-up periods.

There have been studies that have evaluated the long-term success rates of the open Burch colposuspension. Sivaslioglu et al. reported an 84 % success rate at 7 years in their series of 262 patients [10]. The Burch colposuspension is a safe and effective surgical option for SUI, largely considered for women undergoing a concomitant open or laparoscopic procedure such as pelvic organ prolapse repair [11].

Complications

As any open or laparoscopic abdominal procedure, there are common risks including bleeding, infection, erosion of materials involving the bladder, injury to abdominal organs, and hernias [12]. The main long-term issues at hand center around voiding dysfunction and pelvic organ prolapse postoperatively. These pelvic floor issues include detrusor overactivity, urinary retention, and formation of enterocele/rectocle.

Detrusor Overactivity

Many studies have reported differing rates of de novo detrusor overactivity. The mechanism of the dysfunction is widely thought to be secondary to increased elevation of the vagina, and ostensibly the bladder trigone at urethropexy. This therapy further emphasizes the importance of stabilization versus elevation as an important factor in the success of the Burch colposuspension. One of the earlier reports that came from Stanton et al., whose group reported postoperative urodynamic-proven de novo detrusor overactivity, demonstrated a rate of de novo detrusor overactivity at 18.5 % [13]. In Langer et al.'s 10-year follow-up study, the incidence of de novo detrusor overactivity was 16.6 %. Voiding dysfunction appeared within the first year in 70.5 % of the patients ultimately diagnosed with de novo detrusor overactivity [14].

Urinary Retention

In reviewing the literature the on incidence of long-term urinary retention, the authors acknowledge that there is not a great deal reported. Alcalay et al. reported four of the 366 patients who underwent the Burch colposuspension required urethrolysis postoperatively [15]. Although Feyeriesl reported a 16 % rate of residual >60 ml in their patient population at 5- to10-year follow-up, the authors do not report on any patients with residuals greater than 150 ml. Suffice it to say, there is a risk of urinary retention in the Burch colposuspension technique, albeit most likely a low risk.

Enterocele/Rectocele Formation

As discussed in the technique section, the goal of Burch colposuspension is stabilization, not elevation. In early series, the risk of enterocele or rectocele formation is widely thought to be secondary to over-elevation of the vaginal wall [16]. Keeping this in mind, more recent series have lower rates of this anatomic sequelae by avoiding excessive elevation.

Periurethral Bulking Agent

The first description of the injection of a periurethral agent for the management of stress urinary incontinence came from Murless in 1938. The substance used was sodium morrhuate. Following that, may others published experiences with a wide variety of injectables, including paraffin wax, sclerosing agents, polytetrafluoroethylene, collagen, autologous fat, silicone, and stem cells.

Despite the significant presence of injectable agents in urologic practice, there have been very few well-designed published studies evaluating the efficacy of this therapy.

The patient selection for this procedure consists of patients with ISD and normal detrusor function. The urodynamic cutoff for Leak Point Pressure (LPP) is typically 60 cm H_2O [17]. The success in ISD patients is thought to be secondary to the mechanism of action which is thought to be a result of increased area and pressure transmission ratio. This would ostensibly prevent the bladder neck or proximal urethra from opening under stress. Patients may also have hypermobility of the urethra, and still have their ISD component addressed with an injectable agent [18]. In this section, we will review the technique and outcomes of this therapeutic modality. In addition to these indications, urethral bulking agents are also indicated in patients who are young and desire more children, poor surgical candidates, persistent SUI after anti-incontinence procedure, and SUI with poor bladder emptying.

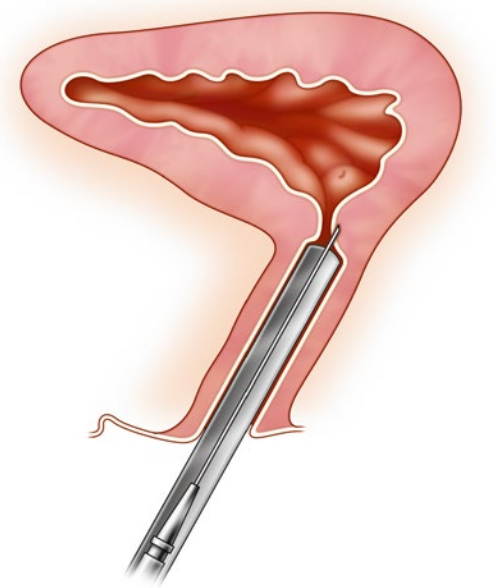


Fig. 3.2 Bulking agent injection for SUI: The agent is injected in the 3 o'clock position on the right, followed by 9 o'clock position on the left. The goal is to create blebs that meet in the midline, akin to prostatic lateral lobes

Surgical Technique

The most common environment for this procedure is under local anesthesia in an outpatient basis. There are two main approaches—transurethral and periurethral. The agent is typically placed submucousally or into the lamina propria. The injectable can be placed at the bladder neck or the proximal urethra. The typical sites of implant are the 3 and 9 o'clock positions. The size of the needle is dependent on the injectable agent. The propose mechanism of action is to achieve coaptation of the urethra during the storage phase, with maintenance of this coaptation when there is an increase in abdominal pressure transmitted to the bladder with a valsalva maneuver.

Periurethral

The patient is placed in dorsal lithotomy position. Local anesthesia is injected in the 3 and 9 o'clock positions 3 mm lateral to the urethral meatus. A 30° cystoscope is introduced after local anesthesia is injected. The periurethral needle is then placed lateral to the urethral meatus (same site as local injection) and advanced to the bladder neck/proximal urethra. The agent is injected in the 3 o'clock position on the right, followed by 9 o'clock position on the left. The goal is to create blebs that meet in the midline, akin to prostatic lateral lobes (Fig. 3.2). If there is any mucosal leakage of the injectable agent from a rent in the mucosa, which can be seen with a transurethral technique, the needle can be repositioned and agent reinjected. At this point, the patient is asked to valsalva to evaluate for SUI. If there is still SUI, more of the injectable agent may be injected. Once completed, the patient is asked to void and residual is checked. If in urinary retention, a small caliber catheter, 8 or 10 Fr., is inserted. A theoretical benefit of the periurethral technique is the avoidance of mucosal leakage and local bleeding that may occur with transurethral needle injection.

Transurethral

The set up is quite similar to the periurethral approach. Local anesthesia may be instilled via the urethra. Needles specific to the injectable agent or generic needles may be used to inject

transurethrally in the same locations described in the above section. A proposed advantage of this technique is better visualization of the injected material compared to the periurethral technique.

Outcomes

There have been many agents that have been used over the years as periurethral bulking materials. For the purposes of remaining current, the authors will review outcomes of bulking agents that are available at the time of publication of this text.

Macroplastique® (Uroplasty Inc, Minneapolis, MN) is a nonbiodegradable hydrogel composed of vulcanized polydimethylsiloxane elastomer suspended in a water-soluble carrier gel (polyvinylpyrrolidone). The agent does not require preadministration testing. The bulking agent can be administered with a 18 gauge endoscopic needle or a proprietary nonendoscopic transurethral injection device called the MIS (Macroplastique® Implantation System, Uroplasty Inc, Minneapolis, MN). The device is a mutichanneled needle positioning tool angled needle entry point with 6, 2, and 10 o'clock position. The typical volumes of injection are 2.5 ml, 1.5 ml, and 1.5 ml, respectively. There have been many studies reporting the success rates of Macroplastique. Most recently, there was a multicenter trial of 247 patients randomized to Macroplastique or Contigen® (collagen) (Bard Medical, Murray Hill, NJ). At 12 months follow-up, improved and dry/cure rates were 61.5 % and 36.9 % in patients injected with Macroplastique versus 48 % and 24.8 % in patients injected with Contigen.

Durasphere® (Boston Scientific, Natick, MA) is made of pyrolytic carbon-coated zirconium beads suspended in a water-based carrier gel composed of 2.8 % glucan. Due to concern for the potential of migration, Durasphere was designed with large-caliber particles (>80 m) in order to obviate this issue. There are 1 ml and 3 ml formulation. That having been said, there have been reports published on local periurethral and local lymphatic migration [19]. The first generation of Durasphere was plagued by issues of difficulty with injection using a proprietary 18 gauge needle with standard endoscopic instruments. Dusrasphere EXP was developed, which included a reformulated carbon bead size and carrier gel to be injected with a customized, side-firing 18-gauge or 20-gauge needle. One of the larger randomized trials of 355 women compared Durasphere to bovine collagen. The study showed no significant difference in outcomes: 80.3 % treated with Durasphere and 69 % treated with collagen were improved by one or more continence grade at 12 months [20].

Coaptite® (Boston Scientific, Natick, MA) is composed of particles of calcium hydroxylapatite ranging in diameter from 75 to 125 μ suspended in an aqueous gel carrier composed of sodium carboxymethylcellulose and glycerin. There is a 1 ml formulation. The injection can be performed with standard endoscopic instruments with a supplied 21-gauge rigid injection needle, available in end-firing and side-firing capability. One of the largest multicenter randomized trials compared Coaptite to cross-linked collagen in 296 women. Patients treated with Coaptite had 63.4 %, versus 57 % of those treated with collagen, rate of improvement of 1 Stamey incontinence grade or more; this was statistically significant. The study also demonstrated that fewer patients treated with Coaptite required repeat injections compared to collagen patients, 62 % versus 74 % [21].

Complications

Complications have been reported in all injection agents currently available in the US market. Macroplastique adverse events have included dysuria (short-lived, self-limited), frequency, and hematuria in many patients. Urinary retention has been reported in 6–10 % of patients injected with Macroplastique [22]. In addition to the common adverse events listed above for Macroplatique, Dursaphsere has been shown to result in noninfectious periuerthral abscess formation and urethral prolapsed [23]. There have also been case reports of urethral prolapsed after Coaptite injection [24].

Pubovaginal Sling

First introduced at the beginning of the twentieth century, the pubovaginal sling procedure has remained an excellent, viable option for the

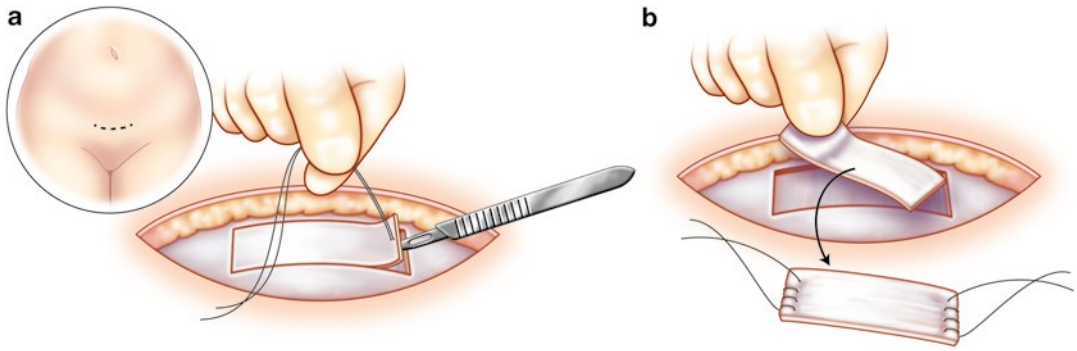


Fig. 3.3 Pubovaginal sling: A 2×8 cm portion of the fascia is marked (**a**). The fascia is then harvested with either sharp or electrocautery dissection (**b**)

management of SUI. The materials used include both synthetic and biologic options. A common synthetic described in the literature is polypropylene. Biologic have included autografts (rectus fascia, fascia lata, and vaginal wall), allografts (fascia, dermis, and dura mater), and xeongrafts (procine or bovine). Although there are many published studies evaluating all of these options, autologous rectus fascia is the most commonly used approach and represents the greatest body of literature (this will be the focus of this section). Before the widespread application of the synthetic midurethral sling, pubovaginal slings were largely considered the gold standard of care for the management of SUI.

Surgical Technique

The patient is placed in a dorsal lithotomy position and the abdomen and vagina are prepped and draped in standard fashion. A transverse lower abdominal incision is made 2 cm above the pubic symphysis approximately 7 cm in length. Dissection is carried down to the rectus fascia, which is cleared of overlying fat. A 2×8 cm portion of the fascia is marked. The fascia is then harvested with either sharp or electrocautery dissection (Fig. 3.3a, b). Once the fascia is harvested, the defect is closed with 0 delayed absorbable suture. With a Foley catheter in place, the bladder neck is identified. A midline, vertical incision is made after the anterior vaginal wall is hydrodissected with a mixture of 1 % lidocaine with 1:200,000 epinephrine solution. A tunnel is then created to the retropubic space using sharp and blunt dissection. The dissection is carried to the level of the posterior rectus abdominis facsia. Pereyra needles are then passed suprapubically 2 cm on either side of the midline into the vaginal incision. A cystoscopy is performed with both 30 and 70° lenses to rule out injury to the urethra or bladder.

The harvested fascia is then prepared for implantation. 0 prolene sutures are placed on either side. The sutures are then placed through the eyelet of the Pereyra needles and brought through the abdominal wall bilaterally (Fig. 3.4a, b, c). The sutures are then tied over the abdominal wall (on top of one finger to avoid overtensioning). The anterior abdominal subcutaneous layer is closed with 2-0 absorbable suture and skin with 4-0 absorbable suture. The vaginal wall is closed with 2-0 absorbable suture. A vaginal packing is placed along with a 16 Fr. Foley catheter. The patient will have a voiding trial in 5–7 days. If there are elevated residuals (>150 ml), the patient will perform intermittent straight catheterization until her residuals return to normal.

Outcomes

The SISTeR trial was the largest randomized control trial reported in the literature evaluating the efficacy of autologous rectus fascia pubovaginal sling. The Urinary Incontinence Treatment Network (UITN) designed and executed this multicenter trial. The study, which consisted of 655 women, compared outcomes of patients

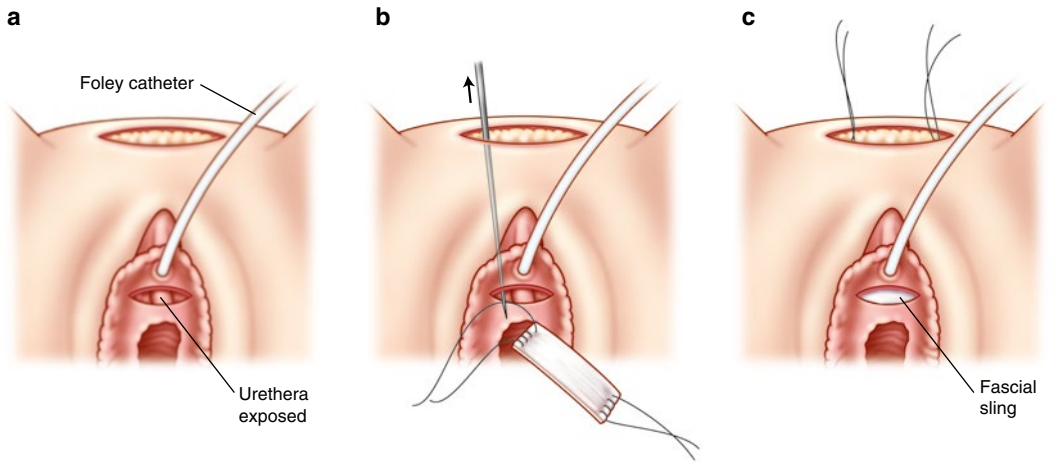


Fig. 3.4 Pubovaginal sling: the anterior vaginal wall dissection is performed (**a**). 0 prolene sutures are placed on either side (**b**). The sutures are then placed through the eylelet of the Pereyra needles and brought through the abdominal wall bilaterally (**c**)

randomized to autologous rectus fascial pubovaginal sling and Burch colposuspension. The success rates, defined as no self-reported symptoms of SUI, was higher in the pubovaginal sling group than the Burch colposuspension group, 66 % and 49 % respectively. This reached statistical significance with a $P<000.1$. The same group went on to publish their 5-year follow-up data on 482 patients. The authors found that there were significant declines in continence in both groups. However, there were higher continence rates in the pubovaginal sling group compared to the Burch colposuspension group, 30.8 % and 24 % respectively ($P=0.002$). Although patient satisfaction decreased for both groups, rates of patient satisfaction were still higher in the pubovaginal sling group compared to the Burch colposuspension group after 5 years, 83 % and 73 % respectively ($P=0.03$) [25].

There are 14 other published RCTs looking at pubovaginal slings, a majority utilizing autologous rectus fascia. All consistently demonstrated the efficacy of the pubovaginal sling in the management of SUI. The pubovaginal sling has also seen another indication in light of recent complications noted with midurethral synthetic slings, namely lower urinary tract erosion [26]. In addition, pubovaginal slings have become the transvaginal anti-incontinence procedure of choice for concomitant repairs of urethral diverticula and urethrovaginal fistulas [27].

Complications

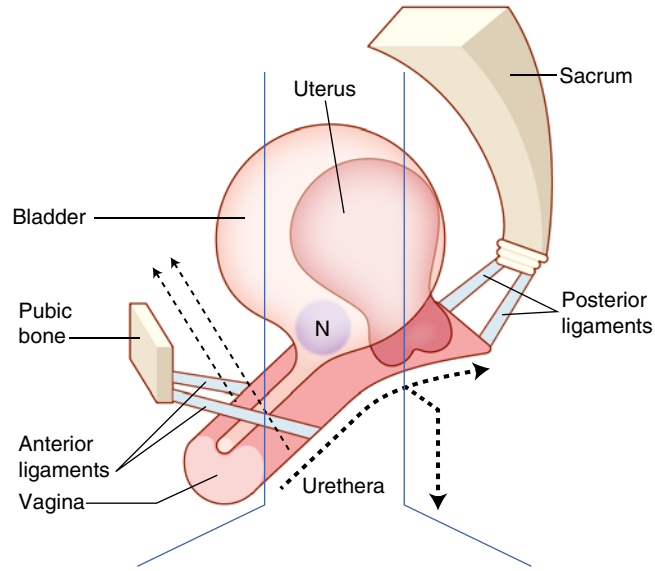
There are known complications related to pubovaginal slings. Some common adverse events include urinary tract infections (UTI) (48 %), voiding dysfunction (14 %), and postoperative urge incontinence requiring treatment (27 %) [25]. Of note, adverse events were more common in studies which used synthetic material for pubovaginal slings.

Midurethral Synthetic Slings

In 1995, Ulmsten first introduced the synthetic midurethral sling procedure [28]. In the last 3 decades, this procedure has become the most commonly employed for the treatment of SUI [29]. Proponents of this surgical option would argue the reason for this overwhelming popularity is due to short learning curve, brevity of the procedure, and low morbidity. In addition, there have been many studies that have demonstrated the excellent long-term durability and success rate of the procedure.

The first iteration of the technique was retropubic placement. This approach was based on the integral theory proposed by Ulmsten and Petros [30]. The theory postulates that there are

Fig. 3.5 Integral theory



three structures (the pubourethral ligament, the suburethral vaginal hammock, and the pubococcygeus muscles) that, as a group, control the opening and closing of the bladder neck and urethra (Fig. 3.5). The goal of the was to retropubic placement of a synthetic sling was to reconstitute the suburethral vaginal support and the pubourethral ligament. A top-down and bottom-up approach has been described, which will be discussed in the next section.

The transobturator approach was introduced in 2001 in an attempt to reduce the risk of bladder, bowel, and vascular injury experienced with the retropubic approach. The mechanism of action of the procedure is based on Delancy's "hammock theory" of SUI. The hammock theory postulates that a combination of urethral support and constriction are necessary for continence. It is the layers of facsia, muscles and vaginal wall that comprise this support and construction, according to the theory (Fig. 3.6). The transobturator synthetic sling placed in a horizontal plane provides the same support for the urethra during moments of increased abdominal pressure, thereby preventing incontinence. An out-in and in-out technique has been designed and utilized for the transobturator synthetic sling, also described in the following section.

Retropubic

Surgical Technique

Top-Down

Some examples of the top-down retropubic slings available in the United States include the Lynx® (Boston Scientific, Natick, MA) and SPARC® (American Medical Systems, Minnetonka, MN). The technique below applies for all top-down approaches, regardless of specific brand.

The patient is placed in a dorsal lithotomy position with legs in stirrups. The abdomen and vagina are prepped and draped in standard fashion. A 16 Fr. Foley catheter is placed and the bladder is drained. The anterior vaginal wall over the midurethral complex is hydrodissected using 1 % lidocaine diluted with 1:200,000 epinephrine. A 2 cm vertical midline incision is made in the anterior vaginal wall, approximately 1 cm from the urethral meatus. Suburethral pockets are created with sharp and blunt dissection, carried to the retropubic space. The trocars are placed bilaterally through stab incisions directly above the pubic symphysis, each one fingerbreadth lateral to the midline. The trocars are advanced into the vaginal incision (Fig. 3.7). The vaginal wall is evaluated for any perforation by the trocar. The Foley catheter is removed and a rigid cystoscopy is performed with 30 and 70° lenses. Once injury is ruled out,

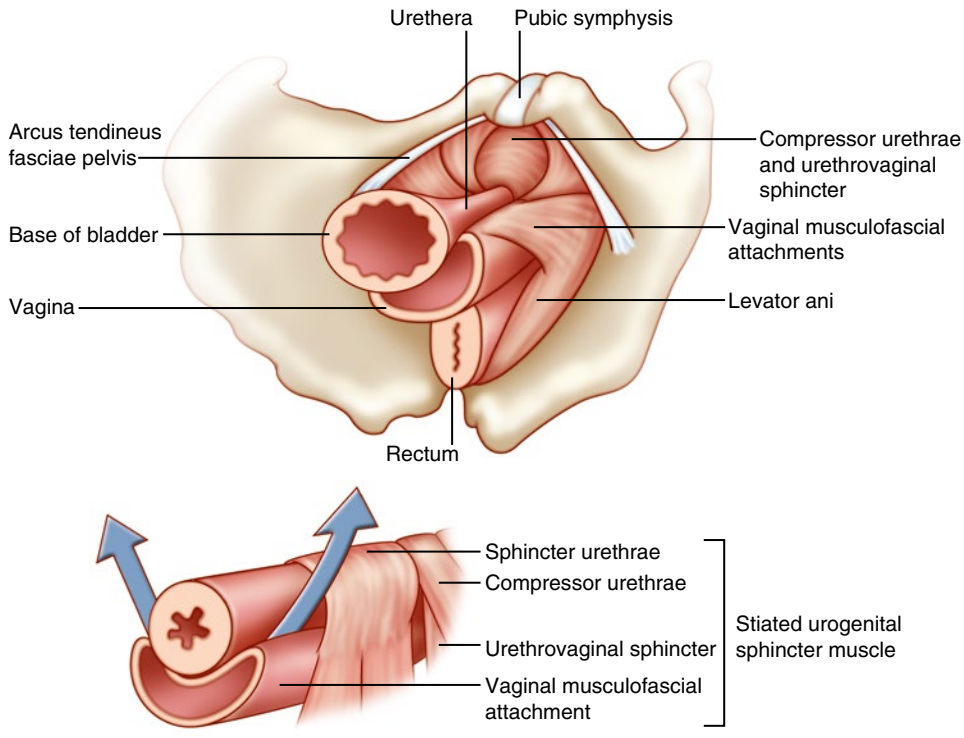


Fig. 3.6 Hammock theory

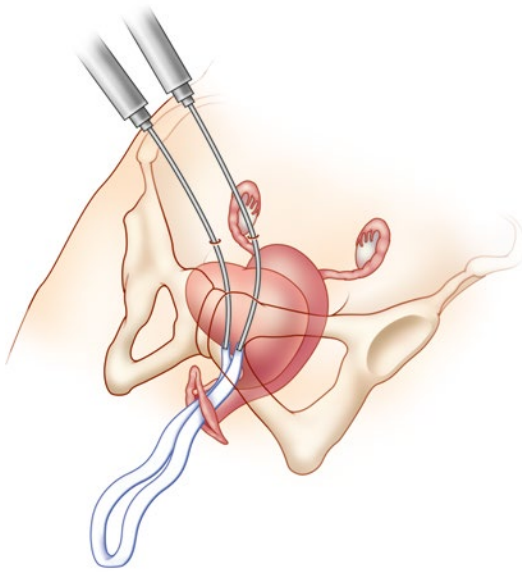


Fig. 3.7 Retropubic sling: top-down

the 16 Fr. Foley catheter is replaced. The mesh is then attached to the trocars and the mesh is placed under the midurethral complex which is covered by a Kelly clamp. This prevents any tension while placing the mesh. Excess mesh is then excised. The stab incisions may be closed with a skin adhesive or 4-0 absorbable suture. The vaginal wall is then closed using 2-0 absorbable suture. A vaginal packing is placed. The patient can undergo a voiding trial in the recovery room.

Bottom-Up

Some examples of the bottom-up retropubic slings available in the US market include the TVT and TVT Advantage (Boston Scientific, Natick, MA). The technique below applies for all bottom-up approaches, regardless of specific brand.

The patient is placed in a dorsal lithotomy position with legs in stirrups. The abdomen and vagina are prepped and draped in standard fashion. A 16 Fr. Foley catheter is placed and the bladder is drained. The anterior vaginal wall over the midurethral complex is hydrodissected using 1 % lidocaine diluted with 1:200,000 epinephrine. A 1 cm vertical midline incision is

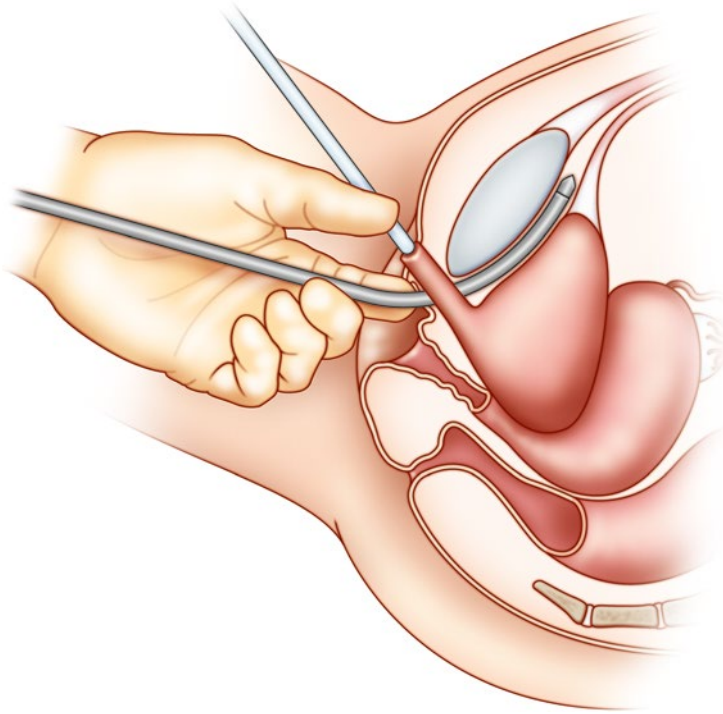


Fig. 3.8 Retropubic sling: bottom-up

made in the anterior vaginal wall, approximately 1 cm from the urethral meatus. Metzenbaum scissors are used to sharply dissect a tract in the direction of the retropubic space. Stab incisions are made 2.5 cm on either side of the midline at the level of the pubic symphysis (some surgeons will choose to hydrodissect through these incisions to aide with trocar placement). The Foley catheter is replaced with the catheter guide in the direction of the contraletral side to where the trocar will be placed. The trocars are placed one at a time through the vaginal incision into the suprapubic stab incision. The trocar is aimed towards the patient's ipsilateral shoulder (Fig. 3.8). The vaginal wall is evaluated for any perforation by the trocar. The Foley catheter is removed and a rigid cystoscopy is performed with 30 and 70° lenses. Once injury is ruled out, the 16 Fr. Foley catheter is replaced. The mesh is then placed under the midurethral complex which is covered by a Kelly clamp. This prevents any tension while placing the mesh. Excess mesh is then excised. The stab incisions may be closed with a skin adhesive or 4-0 absorbable suture. The vaginal wall is then closed using 2-0 absorbable suture. A vaginal packing is placed. The patient can undergo a voiding trial in the recovery room.

Transobturator

Surgical Techniques

Out-In

Some examples of the out-in transobturator slings available in the US market include the Monarc® (American Medical Systems, Minnetonka, MN) and Aris® (Coloplast, Humblebaek, Denmark). The technique below applies for all out-in approaches, regardless of specific brand.

The patient is placed in a dorsal lithotomy position, legs in stirrups. The lower abdomen and vagina are prepped and draped in standard fashion. The anterior vaginal wall over the

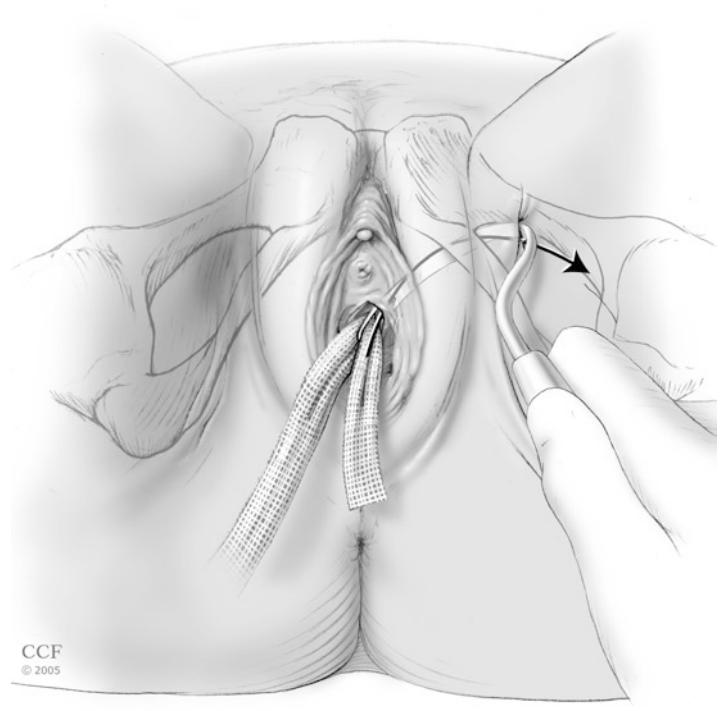


Fig. 3.9 Transobturator sling: out-in (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2005–2014. All Rights Reserved)

midurethral complex is hydrodissected with 1 % lidocaine diluted with 1:200,000 epinephrine. A vertical midline incision is made 1 cm from the urethral meatus 2 cm in length. Suburethral pockets are created with sharp and blunt dissection carried to the level of the obturator internus. Stab incisions are made in the inguinal groin crease at the level of the clitoral hood (two fingerbreadths inferior to the adductor longus tendon). The helical trocars are placed through the stab incision and advanced to the vaginal incision on both sides (Fig. 3.9). The vagina is inspected for any sign of perforation. The Foley catheter is removed and a rigid cystoscopy is performed with 30 and 70° lenses to rule out any injuries. The Foley catheter is then replaced, and the mesh is then attached and placed under the midurethra. A Kelly clamp is placed between the urethra and mesh to prevent tension. Excess mesh is excised. The stab incisions are closed with a skin adhesive or 4-0 absorbable suture. The vaginal wall is closed with 2-0 absorbable suture. A vaginal packing is placed. The patient undergoes a voiding trial in the recovery room.

In-Out

Some examples of the in-out transobturator slings available in the US market include the TVT-O and Abbrevo® (Ethicon, Blue Ash, OH). The technique below applies for all in-out approaches, regardless of specific brand.

The patient is placed in a dorsal lithotomy position, legs in stirrups. The lower abdomen and vagina are prepped and draped in standard fashion. The anterior vaginal wall over the midurethral complex is hydrodissected with 1 % lidocaine diluted with 1:200,000 epinephrine. A vertical midline incision is made 1 cm from the urethral meatus 1 cm in length. Stab incisions are made 2 cm lateral to the inguinal groin crease and 2 cm superior to the level of the clitoral hood. The helical trocars with mesh attached are placed with a guide and advanced to the groin incisions on both sides (Fig. 3.10). The vagina is inspected for any sign of perforation. The Foley catheter is removed and a rigid cystoscopy is performed with 30 and 70° lenses to rule out any injuries. The Foley catheter is then replaced, and the mesh positioned under the midurethra. A Kelly clamp

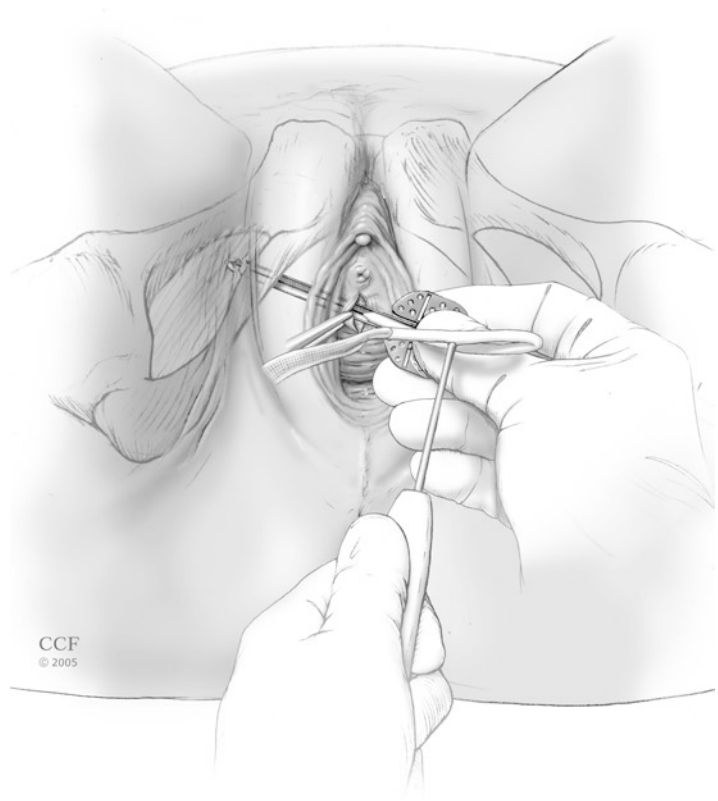


Fig. 3.10 Transobturator sling: in-out (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2005–2014. All Rights Reserved)

is placed between the urethra and mesh to prevent tension. Excess mesh is excised. The stab incisions are closed with a skin adhesive or 4-0 absorbable suture. The vaginal wall is closed with 2-0 absorbable suture. A vaginal packing is placed. The patient undergoes a voiding trial in the recovery room.

Outcomes

There have been many meta-analyses and systematic reviews reported in the literature evaluating outcomes of the different synthetic midurethral slings. In 2010, Novara et al. performed a systematic review and meta-analysis of the comparative data on Burch colpopsuspensions, pubovaginal slings, and midurethral synthetic slings. They found that patients with retropubic slings had higher continence rates compared to those treated with Burch colposuspensions. Retropubic slings and pubovaginal slings were similarly effective. Although objective cure rates were higher in retropubic slings compared to transobturator slings, there was no difference in subjective cure rates between the two [31].

Another meta-analysis conducted by Ogah et al. in 2009 reviewed 62 trials involving 7,101 patients who underwent synthetic midurethral sling for SUI. A subanalysis of eight RCTs comparing pubovaginal slings to synthetic midurethral slings demonstrated an equivalent subjective cure rate at 12 months follow-up (RR 1.03, 095 % CI 0.94–1.13). Six trials evaluated in this meta-analysis compared laparoscopic Burch colposuspensions to synthetic midurethral slings and found no difference in subjective cure rates at 12 months follow-up (80 % vs. 74 %) [4].

In terms of comparing retropubic versus transobturator, there have also been meta-analyses describing the outcomes based on these two different approaches. Novara et al. discovered that

retropubic synthetic slings were associated with a higher objective cure rate when compared to transobturator synthetic slings (OR 0.8, 95 % CI 0.65–0.99) [31]. Although, the same analysis found that the subjective cure rates were equivalent in both approaches. Ogah et al. also compared retropubic and transobturator slings and reported that subjective cure rates in both approaches were 83 %. In addition, even though the objective cure rates were statistically significantly greater in the retropubic versus transobturator groups, 88 % and 84 % respectively, the clinical significance could be argued [4].

There have also been RCTs comparing retropubic synthetic slings and transobturator slings. Richter et al. published their RCT of 587 women randomized to retropubic or transobturator synthetic sling for SUI. The retropubic group did demonstrate higher subjective performance compared to the transobturator group at 12 months follow-up. In addition, the objective success rate for the retropubic group was 80.8 % compared to 77.7 % in the transobturator group (3 % difference, 95 % CI 3.6–9.6).

The author continued this study with a 5-year Longitudinal follow up and found that long-term treatment success and satisfaction with retopublic and transobturator midurethral slings declined over time. They also reported that complications continued to rise. They demonstrated that women who underwent transobturator midurethral slings had more sustained improvement in urinary symptoms, quality of life and sexual function despite lower treatment success rates [32].

Taken in aggregate, the data suggest that retropubic synthetic slings have a slight edge on transobturator synthetic slings in terms of success rates. Of course, the complications do differ as will be discussed in the next section.

Complications

Complications stemming from placement of synthetic midurethral slings range from minor to major. Similar to vaginal mesh for prolapsed complications, these issues are typically codified to timing of complication: intraopertaive, early postoperative, or delayed postoperative.

Fig. 3.11 Single-incision sling: Trocar with sling (MiniArc™ ; Used with permission of American Medical Systems, Minnetonka, MN, USA)

Intraoperative complications include injury to the urethra, bladder, bowel, vascular structures, or vagina. Urethral injuries have been shown to be equivalent in retropubic and transobturator synthetic slings, 0.88 % and 1.09 % respectively [33]. Bladder injuries have been reported as more common in retropubic synthetic slings compared to transobturator synthetic slings. In terms of bowel and vascular injury, no differences have been reported between retropubic and transobturator slings [4].

Early postoperative complications include voiding dysfunction, UTI, groin pain, and urinary retention. Retropubic synthetic slings have been shown to have higher rates of voding dysfunction and urinary retention compared to transobturator synthetic slings. Voiding dysfunction requiring surgical intervention has also been reported greater in retropubic versus transobturator synthetic slings, 2.7 % and 0 %, respectively. Groin pain is a complication more common in transobturator synthetic slings, reported as high as 8.2 % in some studies. Although suprapubic pain is more common in retropubic synthetic slings, the rates are low (1.7 %) [4].

Late postoperative complications include de novo voiding dysfunction and mesh extrusion/

erosion. Similar to early voiding dysfunction, late de novo voiding dysfunction has also been reported to be more common in retropubic synthetic slings. Mesh extrusion/erosion rates have been equivalent for retropubic and synthetic slings [31].

Single Incision

Single-incision slings, or sometimes referred to as mini-slings, were introduced into the market with the purpose of minimizing morbidity and anesthetic requirements. There has been a paucity of literature on outcomes of this procedure. Some of the initial publications reported on devices that are no longer available in the market [34]. The basic tenet of the procedure involves use of no external trocar, with the sling deployed with transvaginal trocar (Fig. 3.11). This section will discuss the current available devices and outcomes published on these currently practiced procedures.

Surgical Technique

Some examples of the SISs available in the US market include the MiniArc™ (American Medical Systems, Minnetonka, MN), Altis® (Coloplast, Humlebaek, Denmark), and Ajust® (Bard Medical, Covington, GA). The technique below applies for all SIS approaches, regardless of specific brand.

The patient is placed in a dorsal lithotomy position, legs in stirrups. The lower abdomen and vagina are prepped and draped in standard fashion. The anterior vaginal wall over the midurethral complex is hydrodissected with 1 % lidocaine diluted with 1:200,000 epinephrine. A vertical midline incision is made 1 cm from the urethral meatus 1.5 cm in length. Suburethral pockets are created with sharp dissection carried to the level of the obturator internus. Push the first slip tip onto the curved needle. Advance the needle at a 45° angle on one side. Advance the needle into the obturator internus until the midline of the sling is over the midline of the urethra. Release the sling tip and remove the needle. Perform the same maneuver to place the other end of the sling into the contralateral obturator internus. Tension appropriately. (Some devices do differ with respect to method and device-specific aspects for this portion of the procedure.) Check both vaginal sulci to rule out mesh perforation. The Foley catheter is removed and a rigid cystoscopy is performed with 30 and 70° lenses to rule out any injuries. The vaginal wall is then closed with 2-0 absorbable suture.

Outcomes

There have been varying results reported for single-incision slings. Although the TVT-Secure (Gynecare) were among the first published, due to the fact that it is no longer available in the market, we will not discuss outcomes. The MiniArc™ (American Medical Systems, Minnetonka, MN) has had success rates reported between 77.8 % and 94 % at 12 months follow-up. A recent meta-analysis published by Abdel-Fattah in 2011 compared SIS to standard synthetic slings. A total of nine RCTs with 758 women were reviewed. In this review, although operative times and pain scores were significantly lower in the SIS group, the subjective and objective success rates were significantly lower when compared to standard synthetic slings (RR 0.83, 95 % CI 0.70–0.99 and RR 0.85, 95 % CI 0.74–0.97, respectively). There were also significantly higher rate of repeat continence surgery in the SIS group (RR 6.72, 95 % CI 2.39–18.89). Even though the SIS were found safe and somewhat efficacious, the conclusion was that they were inferior to standard synthetic slings.

Complications

The same approach to evaluation of complications that applies to standard slings applies to SIS. Adverse events include UTI (4.3 %), urinary retention (3.2 %), dyspareunia (2.1 %), and vaginal extrusion (1.6 %) [35]. Despite the fact that complication rates for SIS have been comparable to standard slings, the FDA has requested postmarketing surveillance data from manufacturers of these devises to ensure safety and efficacy.

Conclusion

The surgical management of SUI options has evolved over the past few decades. The modern era of SUI management started with Burch colposuspensions and have slowly followed a path of less invasive options including periurethral bulking agents, pubovaginal slings, and the newest multitude of approaches for midurethral synthetic slings. The synthetic slings include retropubic, transobturator, and the newest additions which include so-called single-incision slings. As a result of this evolution, there are now many surgical procedures in the armamentarium of pelvic floor surgeons for treating SUI.

References

1. Botlero R, Urquhart DM, Davis SR, Bell RJ. Prevalence and incidence of urinary incontinence in women: review of the literature and investigation of methodological issues. Int J Urol. 2008;15(3):230–4.
2. Coyne KS, Zhou Z, Thompson C, Versi E. The impact on health-related quality of life of stress, urge and mixed urinary incontinence. BJU Int. 2003;92(7):731–5.
3. Hu TW, Wagner TH, Bentkover JD, Leblanc K, Zhou SZ, Hunt T. Costs of urinary incontinence and overactive bladder in the United States: a comparative study. Urology. 2004;63(3):461–5.
4. Ogah J, Cody JD, Rogerson L. Minimally invasive synthetic suburethral sling operations for stress urinary incontinence in women. Cochrane Database Syst Rev. 2009;4, CD006375.
5. Abrams P, Andersson KE, Birder L, et al. Fourth international consultation on incontinence recommendations of the international scientific committee: evaluation and treatment of urinary incontinence, pelvic organ prolapse, and fecal incontinence. Neurourol Urodyn. 2010;29(1):213–40.
6. Leach GE, Dmochowski RR, Appell RA, et al. Female stress urinary incontinence clinical guidelines panel summary report on surgical management of female stress urinary incontinence. The American Urological Association. J Urol. 1997;158(3 Pt 1):875–80.
7. BURCH JC. Urethrovaginal fixation to cooper's ligament for correction of stress incontinence, cystocele, and prolapse. Am J Obstet Gynecol. 1961;81: 281–90.
8. Tanagho EA. Colpocystourethropexy: the way we do it. J Urol. 1976;116(6):751–3.
9. Lapitan MC, Cody JD. Open retropubic colposuspension for urinary incontinence in women. Cochrane Database Syst Rev. 2012;6, CD002912.
10. Sivaslioglu AA, Unlubilgin E, Keskin HL, Gelisen O, Dolen I. The management of recurrent cases after the burch colposuspension: 7 years experience. Arch Gynecol Obstet. 2011;283(4):787–90.
11. Walter AJ, Morse AN, Hammer RA, et al. Laparoscopic versus open burch retropubic urethropexy: comparison of morbidity and costs when performed with concurrent vaginal prolapse repairs. Am J Obstet Gynecol. 2002;186(4):723–8.
12. Demirci F, Petri E. Perioperative complications of Burch colposuspension. Int Urogynecol J Pelvic Floor Dysfunct. 2000;11(3):170–5.
13. Stanton SL, Cardozo LD. Results of the colposuspension operation for incontinence and prolapse. Br J Obstet Gynaecol. 1979;86(9):693–7.
14. Langer R, Lipshitz Y, Halperin R, Pansky M, Bukovsky I, Sherman D. Long-term (10-15 years) follow-up after Burch colposuspension for urinary stress incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2001;12(5):323–6. discussion 326-7.
15. Alcalay M, Monga A, Stanton SL. Burch colposuspension: a 10-20 year follow up. Br J Obstet Gynaecol. 1995;102(9):740–5.
16. Wiskind AK, Creighton SM, Stanton SL. The incidence of genital prolapse after the Burch colposuspension. Am J Obstet Gynecol. 1992;167(2):399–404. discussion 404-5.
17. McGuire EJ, Cespedes RD, O'Connell HE. Leak-point pressures. Urol Clin North Am. 1996;23(2):253–62.
18. Rovner ES, Ginsberg DA, Raz S. Why anti-incontinence surgery succeeds or fails. Clin Obstet Gynecol. 1998;41(3):719–34.
19. Pannek J, Brands FH, Senge T. Particle migration after transurethral injection of carbon coated beads for stress urinary incontinence. J Urol. 2001;166(4):1350–3.
20. Lightner D, Calvosa C, Andersen R, et al. A new injectable bulking agent for treatment of stress urinary incontinence: results of a multicenter, randomized, controlled, double-blind study of durasphere. Urology. 2001;58(1):12–5.
21. Mayer R, Lightfoot M, Jung I. Preliminary evaluation of calcium hydroxylapatite as a transurethral bulking agent for stress urinary incontinence. Urology. 2001;57(3):434–8.
22. ter Meulen PH, Berghmans LC, van Kerrebroeck PE. Systematic review: efficacy of silicone microimplants (Macroplastique) therapy for stress urinary incontinence in adult women. Eur Urol. 2003;44(5):573–82.
23. Madjar S, Sharma AK, Waltzer WC, Frischer Z, Secrest CL. Periurethral mass formations following bulking agent injection for the treatment of urinary incontinence. J Urol. 2006;175(4):1408–10.
24. Lai HH, Hurtado EA, Appell RA. Large urethral prolapse formation after calcium hydroxylapatite (coaptite) injection. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(9):1315–7.
25. Albo ME, Litman HJ, Richter HE, et al. Treatment success of retropubic and transobturator mid urethral slings at 24 months. J Urol. 2012;188(6): 2281–7.

26. Welk BK, Herschorn S. The autologous fascia pubovaginal sling for complicated female stress incontinence. Can Urol Assoc J. 2012;6(1): 36–40.
27. Gomelsky A, Dmochowski RR. Bladder neck pubovaginal slings. Expert Rev Med Devices. 2005;2(3):327–40.
28. Ulmsten U, Henriksson L, Johnson P, Varhos G. An ambulatory surgical procedure under local anesthesia for treatment of female urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 1996;7(2):81–5. discussion 85-6.
29. Fong ED, Nitti VW. Review article: Mid-urethral synthetic slings for female stress urinary incontinence. BJU Int. 2010;106(5):596–608.
30. Petros PP, Ulmsten U. An anatomical classification–a new paradigm for management of female lower urinary tract dysfunction. Eur J Obstet Gynecol Reprod Biol. 1998;80(1):87–94.
31. Novara G, Artibani W, Barber MD, et al. Updated systematic review and meta-analysis of the comparative data on colposuspensions, pubovaginal slings, and midurethral tapes in the surgical treatment of female stress urinary incontinence. Eur Urol. 2010;58(2): 218–38.
32. Kenton K, Stoddard AM, Zyczynski H et al. 5-year Logitudinal follow-up after retropbubic and transobturator midurethral slings. J Urol. 2014 Aug 23 (Epub ahead of print).
33. Morton HC, Hilton P. Urethral injury associated with minimally invasive mid-urethral sling procedures for the treatment of stress urinary incontinence: A case series and systematic literature search. BJOG. 2009;116(8):1120–6.
34. Cornu JN, Lizee D, Sebe P, et al. TVT SECUR single-incision sling after 5 years of follow-up: the promises made and the promises broken. Eur Urol. 2012;62(4):737–8.
35. Kennelly MJ, Moore R, Nguyen JN, Lukban JC, Siegel S. Prospective evaluation of a single incision sling for stress urinary incontinence. J Urol. 2010; 184(2):604–9.

Sacral Neuromodulation

4

Kimberly L. Burgess and Steven W. Siegel

Background

Millions of men and women throughout the world are bothered by urinary incontinence and voiding dysfunction. A recent economic study estimated the disease specific costs of overactive bladder (OAB) in the United States to be $24.9 billion affecting approximately 18.6 % or 42.2 million adults [1]. First and second line treatments include behavioral therapies, dietary changes, pelvic floor muscle training, and antimuscarinic medications. A significant number of patients discontinue medical therapy secondary to side effects or costs. In a large study of the California Medicaid population, Yu et al. found that only 10 % of patients continued taking a prescription medication for management of their OAB after 1 year, with the median time to discontinuation of 50 days [2]. After initial therapies have failed, either secondary to suboptimal control of symptoms, or poor toleration by patients, other treatment options such as sacral neuromodulation (SNM) should be considered in appropriate patients as recommended in the AUA guidelines [3]. This chapter describes indications for use, patient evaluation, surgical techniques, and outcomes for sacral neuromodulation and percutaneous tibial nerve stimulation (PTNS).

Sacral Neuromodulation Mechanism of Action

SNM is proposed to exert its affects through different mechanisms of action. Neural pathways exist that result in amplification, coordination, and timing that are needed for normal micturition [4]. The micturition reflex is initiated through signaling to the brain that the bladder is full from bladder afferent nerves, which are composed of myelinated Aδ fibers and unmyelinated C fibers [4]. The brain then sends signals through spinal pathways to turn off the guarding reflex, relax the external sphincter, and contract the detrusor, resulting in voluntary voiding of urine [5]. Bladder overactivity and voiding dysfunction are likely the result of an imbalance of the reflexes between the bladder, pelvic floor, and urethral sphincter. SNM appears to restore this balance that is needed for normal voiding function. One possible explanation is that SNM exerts its effect by stimulating afferent axons in the spinal roots

K.L. Burgess, M.D.
Division of Urology, Department of Surgery, Greenville Health System, Greenville, SC, USA
e-mail: Kburgess2@ghs.org

S.W. Siegel, M.D. (✉)
Metro Urology, Center for Female Urology and Continence Care, Woodbury, MN, USA
e-mail: pnedoc@comcast.net; siegel.steven@gmail.com

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_4,

modulating the reflex pathways. Areas where SNM may affect micturition include:

- Inhibition of post-ganglionic nerve terminals
- Presynaptic inhibition of primary afferents
- Affecting pudendal afferents that transmit somatic and visceral neurochemical signaling
- Inhibition of spinal tract neurons involved in micturition reflex
- Suppression of guarding reflexes through turning off bladder afferent input to internal sphincter or external sphincter interneurons
- Activation of bladder efferents to stimulate voiding while simultaneously turning off excitatory pathway to urethra [5].

In the case of OAB, bladder preganglionic neurons or interneuronal transmission may be inhibited in the afferent micturition reflex. In the case of incomplete emptying, SNM may improve non-obstructive urinary retention through suppression of guarding reflexes [4, 5].

Initial work demonstrating the possible clinical value of SNM was conducted by Tanagho and Schmidt. The therapy, commercially marketed as InterStim™ (Medtronic, Inc., Minneapolis, MN), was first approved by the US Food and Drug Administration (FDA) for treatment of urinary urge incontinence in 1997 followed by approval for treatment of urinary urgency, urinary frequency, and non-obstructive urinary retention in 1999. More recently in 2011, SNM was also approved for fecal incontinence [6].

Over time, SNM has evolved with technological improvements, including the development of the percutaneous tined lead, decreased size of the implantable neurostimulator (INS), and use of fluoroscopy during office stimulation procedures (percutaneous nerve evaluation or PNE). Initial non-tined leads required fascial or bone anchoring, resulting in the need for larger incisions and general anesthesia for placement. In 2002, the tined lead, initially described by Spinelli et al. [7], was FDA approved allowing for placement under conscious sedation combined with local anesthesia, which increased the ability to use patient sensory information during chronic lead placement [8]. Anchoring within the surrounding subcutaneous tissue and muscle is accomplished with four sets of tines proximal to the electrodes decreasing the possibility of lead migration [7]. A recent publication reporting long-term outcomes for SNM found the revision rate decreased from 50 to 31 % using the tined lead [9]. The rate of revision noted in the InSite Study was 12.9 % in the first year after implantation of the device [10]. In 2006, the InterStim II INS was introduced, which is 50 % smaller than the previous model, resulting in less discomfort and improved patient acceptance. However, this change was made at the expense of a shorter average battery life (3–5 years compared to 7–10 years) [8]. The use of fluoroscopy during PNE allows for improved temporary lead placement, potentially resulting in a greater sensitivity of the office trial, decreasing the need for a staged procedure performed in the operating room [7].

Evaluation/Work-Up

Indications and Patient Selection

Overactive Bladder

The International Continence Society defines OAB as "urgency with or without urge incontinence usually with frequency and nocturia in the absence of an underlying metabolic or pathological condition [11]." Patients should be evaluated with a thorough history and physical exam to evaluate their voiding complaints. In history taking, one may identify that voiding symptoms developed after an inciting event such as pelvic surgery, trauma, or back injury. During the history and physical exam, patients should be evaluated for possible neurologic causes of their symptoms. During pelvic exam, the patient should be evaluated for evidence of pelvic floor muscle dysfunction as a cause of their symptoms, which can be managed successfully with pelvic floor physical therapy. It is important to evaluate for incomplete bladder emptying by obtaining a post-void residual, and also to rule out urinary tract infection as the source of symptoms with a urinalysis and possible urine culture. A cystoscopy should be considered if patient has risk factors for bladder pathology such as, but not limited to, hematuria, tobacco use, or prior pelvic surgery.

Urodynamic evaluation (UDS) should be considered, especially in patients with mixed incontinence, evidence of incomplete bladder emptying, or in patients with possible neurologic causes of their voiding complaints. A UDS may or may not show uninhibited detrusor contractions [5]. A voiding diary documenting number of daily voids, episodes of urgency, episodes of incontinence, voided volume, and fluid intake should be obtained. This information is paramount in not only demonstrating severity of voiding dysfunction but also gives a baseline to compare to the posttreatment voiding diary to assess for objective improvement. Patients with symptoms of an overactive bladder that have not had successful management with conservative therapies are candidates for SNM. Success of SNM in patients with OAB is reported at 59–85 % with average follow-up ranging from 29 months to 5 years [9, 12–14].

Non-obstructive Urinary Retention

Patients with urinary retention or incomplete bladder emptying must be evaluated for obstructive causes, which can be evaluated with a UDS. A voiding diary should be completed prior to test stimulation that includes daily number of catheterizations, voided volume, and catheterized volume. A repeat voiding diary should then be obtained during the test stimulation phase to determine success. SNM is not indicated in patients with evidence of an obstructive etiology of their voiding complaints. Success (defined as minimum of 50 % decrease in catheterized volumes and number of daily catheterizations) at 18 months is reported at 70–83 % [14, 15] with longer follow-up ranging from mean of 51 months to 5 years showing success in 71–88 % of patients [9, 13].

Pelvic Pain/Bladder Pain Syndrome

Pelvic pain and bladder pain syndrome are currently not FDA approved indications for SNM; however, patients with these conditions often have coexisting voiding complaints such as urinary urgency or frequency, which would make them candidates for this therapy. As discussed above, it is important to evaluate for pelvic floor muscle dysfunction on physical exam as pelvic floor physical therapy should be considered as an initial therapy when appropriate. In patients where conservative therapies have failed, a trial of SNM is an excellent potential option. Studies have shown the therapy to provide some benefit in patients with bladder pain syndrome and pelvic pain. However, it is important to educate the patient that their pain may not improve with SNM. One of the treatment's potential benefits is that a minimal risk trial is conducted to assess how the therapy will affect their symptoms. Multiple studies have shown that SNM results in improvement of pain parameters, with long-term success ranging from 60 to 77 % with median follow-up of 19–61.5 months [16–18]. Gajewski and Al-Zahrani found that the presence of urgency at baseline was a positive predictor of long-term success [17]. One study found that patients with bladder pain syndrome required more programming visits [19] following implant, while another group found that the rate of explant was highest in the bladder pain syndrome patients compared to those with OAB or urinary retention [9]. A study of patients with refractory interstitial cystitis showed a 36 % reduction in the mean dose of daily opiate use after implant compared to baseline, with 22 % of patients able to stop their use completely [20].

Other Indications

Fecal incontinence can be a devastating symptom, affecting 1–2 % of the population [21]. Initial management includes dietary changes, biofeedback, and medical therapy. The FDA has also approved the use of SNM for patients with chronic fecal incontinence. One-, two-, and three-year therapeutic success is reported at 83 %, 85 %, and 86 %, respectively, with complete continence in 40 % of patients at 3-year follow-up. Success was defined as at least a 50 % reduction of incontinent episodes per week compared with baseline and to be included in the study; patients were required to have more than two incontinent episodes per week on average and symptoms present greater than 6 months [21, 22]. This specific indication for the use of SNM will be discussed further in Chap. 16.

Another indication for SNM, although not FDA approved, is childhood dysfunctional

elimination syndrome that is refractory to medical management and behavioral therapies. At median follow-up of 27 months, one group reported improvement or resolution of incontinence, urgency, frequency, nocturnal enuresis, and constipation in 88 %, 69 %, 89 %, 69 %, and 71 %, respectively [23].

Procedures

Two different types of trials exist for patients to undergo test stimulation with SNM, including the PNE and staged lead implantation. A PNE involves placement of a temporary monopolar lead that is removed at the end of a 3–10 day trial period. Advantages to the PNE are that it can be performed in the office with local anesthesia, and there is easy removal of the temporary leads. A disadvantage is the potential for migration of the leads, thereby decreasing the accuracy of the trial in determining whether a patient will benefit from the therapy. During the staged trial, a quadripolar tined lead is positioned as the first stage with placement of the INS as a second stage if successful. The advantages of a staged trial are decreased risk of lead migration, increased programming options during the trial due to the lead configuration, and ability for a longer test period. A disadvantage of the staged procedure is the need for two procedures using monitored anesthesia care (MAC) in the operating room. One's choice to perform a PNE versus a staged procedure may depend on the patient and the surgeon's access to equipment. A PNE may be performed using fluoroscopy, which is the author's preference, or using anatomic landmarks to determine placement of the temporary leads. One may prefer to perform staged procedures if access to fluoroscopy is not available in the office. The author uses both types of test stimulation based on the patient's preference and condition, and both techniques will be described. Since "frequent" fecal incontinence may only happen once per week, a staged trial has been thought to be more likely to capture the benefit, though we have often noted changes during a PNE trial.

Percutaneous Nerve Evaluation

Patient Positioning and Preparation

Prior to the procedure, the patient should complete a baseline voiding diary that documents their specific voiding complaints and any incontinence if present. Diaries should include the intake volume, frequency of urination, volume voided, degree of urgency, number and severity of incontinent episodes, and type and number of pads used. If the patient establishes their baseline diaries using anticholinergic mediations, the trial is also conducted using the medication, since the intention is to measure the effect of only one variable. If the patient has urinary retention, they should also record the number of daily catheterizations and catheterized volumes. If the trial is being conducted for fecal incontinence, details of these episodes along with consistency of stool and number of daily bowel movements should also be recorded. It is important to discuss the procedure with the patient, so they know what to expect during the procedure and help reduce patient anxiety since it will be performed without sedation.

The patient is placed on a table that will accommodate the fluoroscopic c-arm in the prone position with pillows placed under the patient's head, hips, and shins. It is important to insure that the patient's hips are positioned where the c-arm unit can be passed under the table to allow for viewing of the sacrum during the procedure. The buttocks and feet need to be uncovered to watch for motor responses during the procedure. The patient's lower back and buttocks are prepped with an antiseptic solution and the operative field is draped with towels and/or small adhesive drapes provided in the Test Stimulation Lead Kit (Medtronic, Minneapolis, MN) (Fig. 4.1a, b). A grounding pad is placed on the patient's heel or foot if there is deep callous, and the red connector of the Test Stimulation Cable is connected to the grounding pad. The black connector of the Test Stimulation Cable is attached to the Patient Cable. The Test Stimulation Cable is attached to the test stimulation box (Fig. 4.2).

Fig. 4.1 PNE components. (**a**) Prep kit which includes drapes and prepping materials. (**b**) Test stimulation kit: foramen needles, syringe, and needle for local anesthetic, test stimulation lead, marking pen, hemostat, and forceps

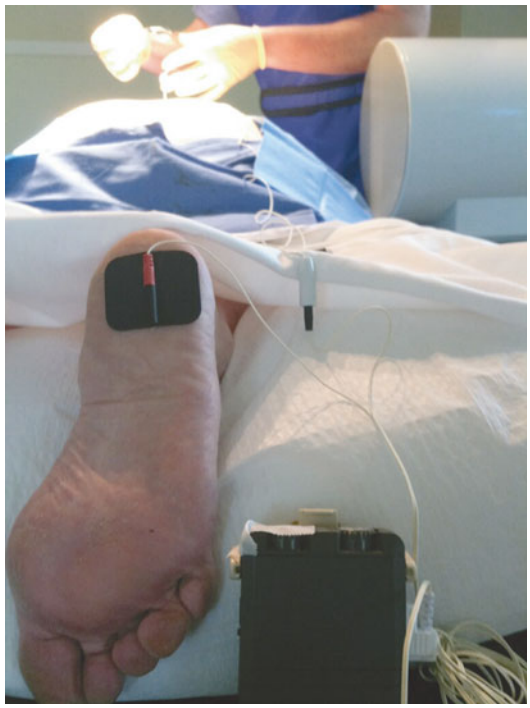


Fig. 4.2 The grounding pad is attached to the patient's foot with the red connector of the Test Stimulation Cable attached. The black connector of the Test Stimulation Cable is attached to the Patient Cable and the Test Stimulation Cable is attached to the test stimulation box as shown

Selecting the Appropriate Foramen Using Fluoroscopy

S3 is the ideal target for lead placement and we attempt to place bilateral leads at this level during the PNE. Rarely, S4 is adequate or preferable, but S2 is not appropriate for chronic stimulation due to the activation of lower extremity motor efferents. Fluoroscopy is used to visualize the sacrum in an anteroposterior (AP) view to identify the medial edge of the sacral foramen on one side, which is marked on the skin with a marking pen (Fig. 4.3a, b). The contralateral foramen location can be approximated by moving two fingerbreadths over and once verified with fluoroscopy, the skin is marked. The skin may be marked or one can visualize an imaginary vertical line parallel to the spine at the medial edge of the foramen to help stay lined up when accessing the foramen from a superior point on the skin. A cross-table fluoroscopic view is then obtained to locate the S3 foramen laterally. Only a few minutes total is spent using the AP projection, with the major portion of the procedure performed in the lateral view. S2 is readily identified as the point where the sacroiliac joint fuses, forming a characteristic shadow. The first anterior protrusion or "hillock" from the anterior surface of the sacrum below this shadow is typically S3. In order to determine the skin entry point of the foramen needle, the skin is marked with a hemostat while looking at skin surface and the sacrum together in a lateral plane (Fig. 4.4). The point of entry is found along the line marking the medial edge of the sacral foramen (previously made while in the AP view), with a line drawn in the mind's eye starting at the skin mark, paralleling

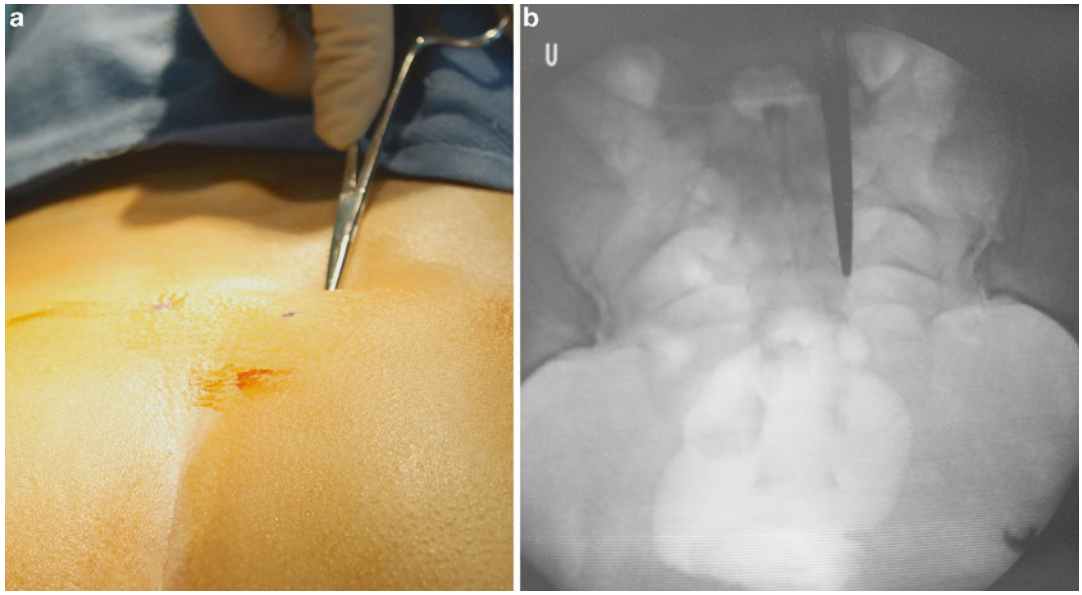


Fig. 4.3 Identifying the medial edge of the sacral foramina using fluoroscopy with (**a**) showing the clamp on the patient's skin and (**b**) showing the AP X-ray

Fig. 4.4 Identifying S3 on lateral view [24]

the fusion plain between the sacral vertebra, and targeting a point approximately 1 cm above the S3 hillock. This line is in a cephalocaudal direction at an approximately 60-degree angle. The use of fluoroscopy allows the correct angle to be determined for any body habitus.

Positioning the Needle in the S3 Foramen and Placement of the Temporary Lead

The location of the needle entry point on the skin should be anesthetized with 0.25 % bupivacaine, 1 % lidocaine, or a combination to create a dime-sized wheal. The subcutaneous tissue does not need to be infiltrated with the local anesthetic but the sacral periosteum may require some of the anesthetic if the patient experiences discomfort as the foramen needle touches the bone. Once the bone is touched, a spot lateral fluoroscopy view is obtained to determine if the insertion point is correct, and whether the needle must be aimed in more cephalic or caudal angle. Once the orientation is correct, the needle is "walked" up or down along the line of the medial edge of the foramina until the needle drops off into the foramen (Fig. 4.5). It is important to pull the needle back before changing positions while trying to locate the foramen to avoid torqueing or bending the needle. For larger patients, a 5 in. needle is available. We typically refer to this as "the other" needle in order to minimize patient anxiety.

Since the patient is not sedated during the PNE, it can cause discomfort to reposition the

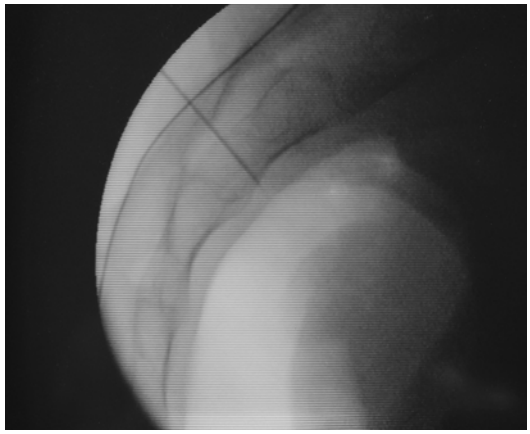


Fig. 4.5 Foramen needle placed superior to the hillock of S3

Table 4.1 Sacral root responses

Sacral level	Motor response	Sensory response
S2	Bellows, clamp, dorsiflexion foot, heel rotation, calf cramping	Genital
S3	Bellows, dorsiflexion great toe, dorsiflexion foot	Genital, perineal, anal
S4	Bellows	Anal

needle during the procedure, and we often will keep the initial foramen placement if responses are consistent with positioning along S3. During placement of the tined chronic lead, one should re-direct the foramen needle as needed to get the best positioning along the course of the S3 nerve root. The needle is then stimulated using the patient cable that is attached to the test stimulation device and the patient is observed for sacral root responses (Table 4.1). Responses that correspond to simulation of S3 are bellows (levator contraction resulting in rolling inward and deepening of the intergluteal fold), dorsiflexion of the ipsilateral great toe, and sensation of the stimulation in the genital, perineal, or anal region.

If responses are not consistent with S3 and the patient will tolerate further needle manipulation, the skin entry point is re-approximated using lateral fluoroscopy to aim for the next foramen, either above or below the initial point of stimulation. Once appropriate responses are elicited confirming position in S3, the needle stylet is removed and the Test Stimulation Lead (Medtronic) is passed through the needle to the marking on the lead that corresponds to the needle size used (3 or 5 in.). A fluoroscopic image is obtained to gauge the depth of the lead. It is better to err on being slightly deeper rather than too shallow as the lead will likely migrate during the testing period. The foramen needle is removed along with the lead, while maintaining its depth and keeping the stylet in place, and then it is stimulated to assess responses. The PNE lead can be pulled back under fluoroscopy and/or while being stimulated, to gain better positioning. Finally, the lead stylet is removed leaving only the test lead in place. The procedure is then repeated on the contralateral side.

To decrease the potential for lead migration during the testing phase, the author prefers to tunnel the leads using a spinal needle (Fig. 4.6a, b). The leads are then secured in place using a gauze and Tegaderm dressing. Lastly, the leads are attached to Test Stimulation Cables, which are in turn attached to the external stimulation device during sub-chronic stimulation. Patients are then instructed on device use and will complete voiding diaries during a 3–10 day length of trial to evaluate the therapy. During the testing period, they will trial both leads individually. At the end of the trial period, the leads are extracted in the office by removing the dressing and simply pulling gently on each lead. Success is determined through comparing the voiding diary obtained during test stimulation to the baseline diaries, and also by asking the patient subjectively how they feel their symptoms were managed during the trial. If trial is successful, defined as >50 % improvement in symptoms, the patient is offered implantation with placement of the tined lead and INS during a single procedure. If therapy did not result in at least a 50 % improvement in symptoms, the patient can choose to undergo a staged trial, especially if there were any concerns regarding lead migration or lead placement that may have affected the outcome.

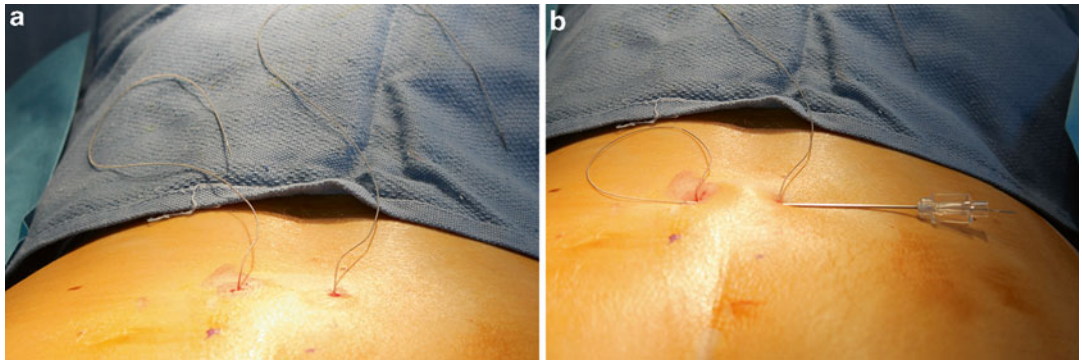


Fig. 4.6 (**a**) PNE leads after placement. (**b**) Tunneling of the leads to decrease migration during the trial

Staged Lead Implantation as Initial Trial

Patient Positioning and Preparation

A staged trial involves placement of the tined quadripolar lead connected to a lead extension that is externalized for a prolonged test stimulation trial. As with the PNE and discussed above, the patient must complete a baseline voiding diary prior to the procedure. The procedure is performed with a combination of conscious sedation or MAC with local anesthesia and therefore the patient does require medical clearance to undergo anesthesia. It is preferable to perform the procedure under MAC rather than general anesthesia to allow the patient to give sensory feedback during lead placement. On rare occasion, a patient is not an appropriate candidate for MAC based on other comorbidities and requires general anesthesia. It is important in these situations that the surgeon has good communication with the anesthesia team about the medications given as only a short-acting paralyzing agent should be used to avoid blocking pelvic floor muscle responses during intraoperative stimulation. A preoperative dose of an antibiotic with coverage against skin flora such as cephazolin (Ancef) or vancomycin, if there is concern for MRSA, is administered prior to the procedure. A Hibiclens wash the night before, and morning of surgery, and pre-op skin wipe down with a sage cloth are also measures, which may reduce the potential for infection.

The patient is placed in prone position on a table that will accommodate fluoroscopy with the hips positioned on the table where the c-arm unit will pass beneath the table allowing the surgeon to view the sacrum on both AP and lateral views. Two chest rolls are placed to assist with respiration and pillows are placed under the hips and shins to pad pressure points. The feet remain uncovered to allow observation of motor responses during intraoperative stimulation. The lower back and buttocks are first wiped with alcohol, and then, after drying, prepped with an antiseptic solution such as Dura Prep (3M) solution. After the prepping solution has dried, towels are placed at the bottom of the buttocks, on the sides lateral to where an INS would be positioned, and above the estimated location of L5–S1. An Ioban sheet (3M) is placed smoothly over the entire remaining area of exposed, prepped skin, and the buttocks are gently pulled apart and then released to allow the Ioban to attach/insert itself into the intergluteal fold. Taping apart of the buttocks is not needed in order to see the bellows response, and is discouraged due to the potential for creating patient anxiety and muscle tension. The components of the lead kit are shown in Fig. 4.7. As with the PNE discussed previously, a grounding pad is placed and Test Stimulation Cable and Patient Cable are connected to the external stimulation device (Fig. 4.2).

Selecting the Appropriate Foramen and Positioning the Needle

The process of using fluoroscopy to identify the medial edge of the S3 foramen and identifying S3 on lateral views is the same as described

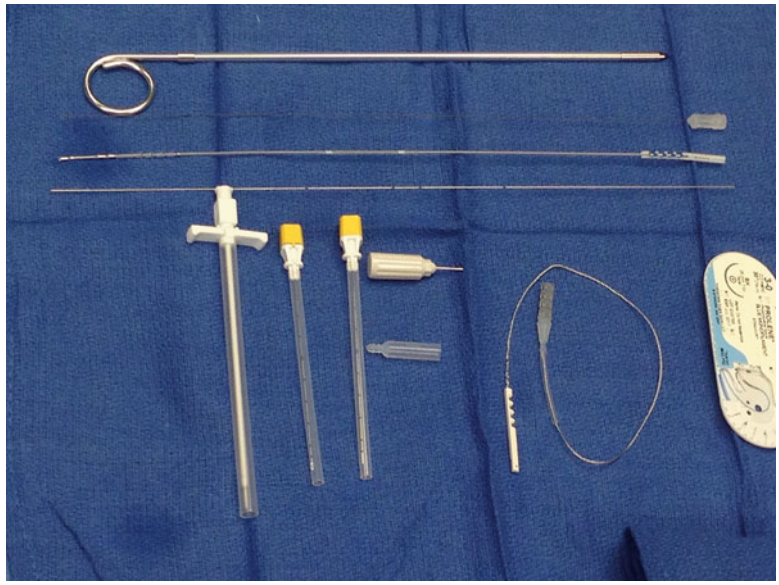


Fig. 4.7 Components of lead introducer and lead kits: tunneling device, straight stylet, lead with ball tip stylet in place, directional wire guide, introducer and sheath, two 3 in. foramen needles, torque wrench, plastic covering/boot, and lead extension wire

for the PNE. One difference in placement of the tined lead is that only a single lead is placed rather than performing bilateral placement. Either side can be chosen for placement but if the patient has a lateralizing pain component, it is preferred to place the lead on that side. Identifying the skin entry point, administration of local anesthesia to the skin, and placement of the foramen needle is also the same as the PNE. The foramen needle should follow a cephalocaudal path, paralleling the fusion plane between the sacral vertebrae, passing through the sacrum approximately 1 cm superior to the tip of the hillock. The foramen needle is tested only with the tip just anterior to the anterior surface of the sacrum. It is at this point where the "opening threshold" is measured. As opposed to the PNE, it is vital to reposition the needle as necessary to get the most robust responses. We usually aim for this threshold to be at or below 1 V. If it is higher than 2 V, reorientation of the foramen needle is needed, making sure that it is as medial as possible related to the edge of the bone, and also as high as possible related to the hillock. For example, if the tip of the foramen needle is at or below the hillock just as it enters the pelvis, it is nearly always incorrectly placed, and does not need to be tested before repositioning. Also, it is important to note the order in which motor responses are seen at this point. Bellows should come first, and then toe flexion. An opening threshold at or below 1.5 V, with the appropriate pattern of motor and sensory responses, should be obtained before attempting to deploy the tined lead.

Placement of the Introducer Sheath and the Tined Lead

Once the needle has been positioned, a small skin nick is made alongside the needle making sure the needle moves freely within the small incision. The skin nick must be through the skin into fat, and long enough (0.5–1.0 cm) to allow eventual tined lead placement without causing it to be trapped superficially. The inner stylet of the needle is removed and the directional wire guide is passed to the mark corresponding to the needle length (3 or 5 in.). The foramen needle is removed leaving the directional wire guide in place. The lead introducer sheath is then passed over the directional guide under fluoroscopy until the radiopaque marker present at the tip of the sheath is located approximately 1/2–2/3 through the

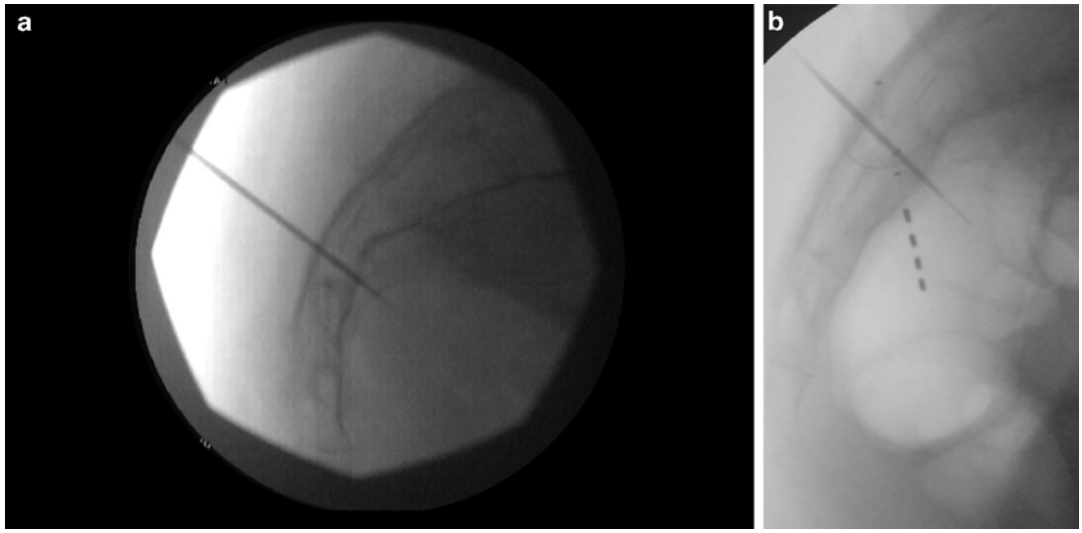


Fig. 4.8 (**a**) Lead introducer and sheath in place. (**b**) Lead introducer in place during a lead revision. Note the different angles of entry and position with respect to the S3 hillock

bone table (Fig. 4.8a, b). This step is emphasized as it is critical to the reproducibly successful positioning of the tined lead. Once the sheath has been placed at the proper depth, the introducer stylet and directional guide are removed.

Prior to passing the lead, the authors suggest exchanging the straight and stiffer stylet, the lead comes packaged with, to the ball-tipped (curved) stylet, which is provided in the lead kit. This is because it is softer than the straight stylet, and allows for attachment to the lead. The lead/stylet assembly becomes more flexible and steerable, allowing it to more precisely approximate the course of the sacral nerve as it enters the pelvis. The lead is passed through the introducer under fluoroscopy with care not to deploy the lead. Accidental deployment can be avoided easily by keeping the introducer tip 1/2–2/3 of the way through the bone table, and making sure that contact points 2 and 3 of the tined lead straddle the bone table. The lead has two marks on the upper half of the lead and if the most proximal (upper) marking passes into the sheath, the lead will be deployed as the tines will be located outside of the sheath. The lead should point slightly caudally and laterally ("down and out") as it passes anterior to the sacrum. When viewing on a lateral X-ray, one can assess for whether the lead curves outward or laterally by looking at the distance between the lead contact points. The distal contact points (0 and 1) will appear to be spaced closer together in the lateral view if the distal lead points away from its initial entry point anterior to the sacrum (Fig. 4.9a, b). On the AP view (usually taken after final deployment) the proximal contact spacing (between points 3 and 2) should appear closer than the distal (1 and 0) since the lead is coming towards and then moving away from the viewer. If the lead is not pointing in a "down and out" position, it should be repositioned by pulling the lead back under fluoroscopy until the distal tip is within the introducer sheath before adjusting/turning and re-advancing to help pass it along a different path, which can be confirmed on fluoroscopy.

Once positioned, the lead is tested by stimulating at each of the four contact points (0–3) starting at the most distal contact (0) and observing for the threshold at which appropriate responses are first visualized or sensed by the patient. If the response is elicited at a low threshold, this supports that the lead is parallel to the nerve. Ideally, low thresholds should be obtained with stimulation at all four of the contact points, which will allow for longer battery life and maximal programming options. Also, the pattern of

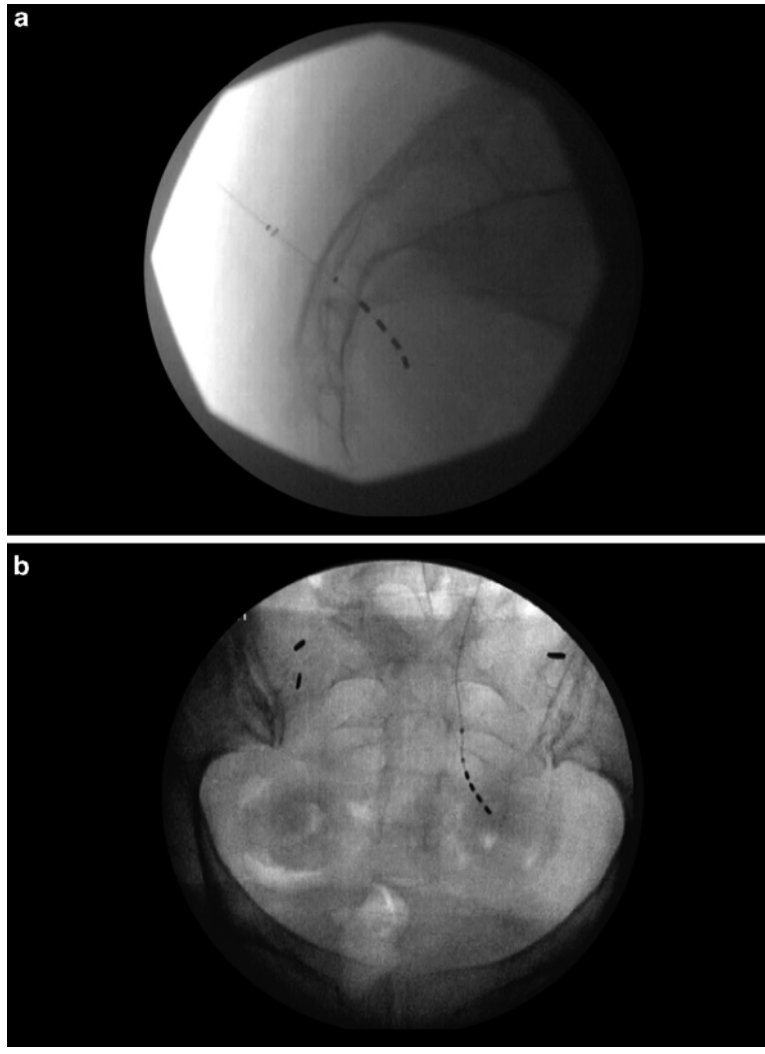


Fig. 4.9 (**a**) Deployed lead on lateral view. (**b**) Deployed lead on AP view

responses will help to indicate which way the lead trajectory must be altered. For example, if there is mostly foot response, or foot first and bellows second (favoring S2), the lead course needs to be more downward. If there is all bellows and no toe (favoring S4), the lead must be made to course more upward. If appropriate responses are not seen at a low threshold at each point, the lead should be repositioned as described above. Moving the introducer sheath deeper or shallower within the boney canal changes the "launch point" of the lead, which can help adjust the path the lead takes if needed. The launch point can be retracted simply by pulling back the introducer sheath under fluoroscopic control, but it should only be advanced while the sheath is cannulated by the lead or the introducer stylet and directional guide. It should never be advanced beyond the anterior sacral surface. Even if this happens by mistake, it can interfere with the ideal course of placement due to creating a "false passage." If after several attempts to reposition the lead, ideal responses are not elicited, it may be necessary to start again with the foramen needle to obtain a different path for the lead. Also, one should consider switching to the contralateral side if unable

to get appropriate positioning of the lead. Since the vast majority of patients undergoing lead placement are anatomically and neurologically normal, the main cause for not getting all four contacts yielding appropriate responses at similarly low thresholds is related to technique or lack of persistence, and not patient factors. Once the ideal position is obtained, the tines are deployed by pulling back the introducer sheath under fluoroscopy. During this step, since all four responses are nearly equal, indicating the lead is placed along the nerve, we will slightly advance the lead to the point where contact 3 is just anterior to the bone table. This allows more flexibility in using contact 3 in programming. After final retesting to confirm positioning, the lead stylet should be removed.

Connecting the Lead to the Lead Extension

If the patient is undergoing a full implant following a successful PNE with placement of the lead and INS during a single procedure, this section can be skipped. The authors prefer to place the lead connection at the future site of the INS. To identify this site, the lateral edge of the sacrum is palpated and marked along with the posterior superior iliac crest. The site of the future INS should be marked in a slight diagonal plane overlying an area of fat away from the marked bony landmarks in the upper buttock. Before making the incision, make sure the lead will easily reach this area once tunneled. After administering the local anesthetic to the lateral aspect of the marked incision, a 1–2 cm incision is made through the lateral portion of the marked area extending through the skin into the subcutaneous layer of fat.

The length of the plastic sheath surrounding the provided tunneling device can be used to measure the furthest possible site on the contralateral side of the patient's back where the lead connection will be externalized. The local anesthetic should be applied to the skin in that area in a quarter sized wheel. The tunneling device is then passed from the incision, through the subcutaneous fat, to the anesthetized area on the contralateral side. It is important that the path of the tunneling device is superior to the sacral incision where the lead was placed. The sharp end of the tunneler is externalized and the metal portion is removed leaving the plastic sheath in place. The distal end of the lead extension is passed through the sheath until it exits the skin and the plastic sheath is then removed. If there is a hang-up while passing the lead extension, the directional guide can be used to help push the extension completely through the tunneling sheath.

Two methods can be used to pass the lead from the sacral incision to the incision in the buttock where it will be connected to the lead extension wire. One method that can be used if the distance is short is using the lead introducer sheath. This technique is more often useful when placing the lead and INS at the same time. The other method is using the tunneling device. The tunneling device may be curved if needed to decrease the amount of excess lead present within the buttock incision. It is important to pass whichever device is chosen deep within the fat so that it does not lie superficially beneath the skin. It is also important that the pre-sacral incision is big enough to allow the tunneling device to be passed without damaging the lead with the tunneling device, or leaving the lead trapped superficially within the incision as mentioned earlier. The lead is in turn connected to the lead extension wire and the four screws are tightened using the provided torque wrench, turning a quarter turn past the click. The plastic covering or boot is placed over the connection hub and secured in place with a prolene tie. A straight clamp is used to grasp gently the plastic sleeve covering the proximal aspect of the lead extension wire, with care taken to avoid damage to the wires, and used to pass the connection along the path of the lead extension until the connection hub lies within the subcutaneous fat just medial to the incision. This allows easy identification of this connection at the time of the completion procedure or during removal. If there is redundant lead present, this should be looped in the subcutaneous fat inferior to the incision so it will not bunch up within the small incision preventing optimal healing and be out of the way when the incision is reopened for the next stage. The wounds are irrigated with

antibiotic solution and the subcutaneous fat anterior to the lead and lead extension is closed with 2-0 vicryl suture. The skin of both the buttock incision and pre-sacral incision are closed with a subcuticular 4-0 vicryl suture. Skin glue is placed over both incisions after closure.

Finally, the externalized portion of the lead extension is connected to the lead extension cable, which in turn is to be attached to the test stimulation device for the trial. The connection between the extension and cable is secured to the patient's back with a Telfa and Tegaderm dressing.

Implantation of the INS (Stage 2)

A completion phase procedure is scheduled 2–4 weeks after the first stage where the INS will be implanted if the trial is successful, or removal of the lead and lead extension if unsuccessful. If there has been significant improvement (>50 %) in the target symptoms based on voiding diary and subjective changes noted by the patient, implantation of the permanent INS is supported. This step is also performed using a combination of MAC and local anesthesia.

Perioperative anesthesia and skin preparation are the same as for the lead implant. The area of the buttock where the prior connecting incision was made is prepped and draped leaving the area where the lead extension wire is externalized outside of the prepped field to decrease contamination. The buttock incision from the first stage is marked and extended medially in the direction of the tunneled lead so that it will be just long enough to accommodate the INS. The skin in this area is infiltrated with the local anesthetic and after incision is made, electrocautery is used to maintain hemostasis. The subcutaneous fat is opened overlying the connection hub. The lead extension wire is cut distal to the plastic sleeve covering adjacent to the connection. The hub is usually left on the lead to protect it until creation of the pocket is complete. The remaining lead extension wire is removed by pulling on the externalized portion, which should be performed by someone outside of the sterile field, usually after the case has been completed and the drapes are being removed.

A subcutaneous pocket is then created using cautery and blunt dissection extending 2–3 cm beneath and parallel to the skin staying anterior to fascia of the muscle. This should be just large enough to allow the INS to be placed assuring that it lies flat and parallel to the skin without excess room to avoid the device "flipping" within the pocket. The prolene tie and plastic boot over the lead connection are cut and removed. The lead is separated from the remaining portion of the lead extension and the wound is irrigated with antibiotic solution. The lead is attached to the INS, which is then placed in the pocket tucking any excess length posterior to the device to prevent lead injury during any future revision surgery that may be needed. The overlying fat should come together without tension if the pocket size is optimal. The subcutaneous tissue is closed with absorbable suture, the skin is closed with a subcuticular absorbable stitch such as Vicryl, and skin glue is placed over the incision.

Initial Device Programming

Following placement of the tined lead, the device is programmed in the postoperative recovery area. It is important to discuss the use of the brown box (placement of lead only) or ICon Patient Programmer (INS placement) prior to the procedure with the patient as sedation may affect their retention of the information given postoperatively. If possible, it is useful to also have a family member or friend present during education for device use.

When only the tined lead is placed, the brown box is set using the Electrode Select switches with one contact set as the negative electrode and one contact set as the positive electrode. The box is then turned on and amplitude is increased until the patient feels the stimulation. If stimulation is not felt comfortably in an appropriate area or the amplitude reaches 10 V, a different combination of positive and negative electrodes should be used. The initial program tried is typically contact 0 set as the negative electrode and contact 3 set as the positive electrode. During the trial, the patient is contacted periodically to assess symptom control and change the program as needed.

If both the tined lead and the INS are implanted, programming is done using the N'Vision Clinician Programmer and the ICon Patient Programmer. An impedance check is performed to assess the lead and the connection of the lead to the INS using the N'Vision Clinician Programmer. As with the brown box, one contact point is set as the negative electrode while a second contact point is set as the positive electrode. Four programs can be set in the ICon Patient Programmer with initial programming set as:

Program 1 Negative 0, Positive 3
Program 2 Negative 1, Positive 3
Program 3 Negative 2, Positive 0
Program 4 Negative 3, Positive 0

The patient is able to adjust the amplitude and switch between the programs as needed using the ICon patient programmer.

Removal of the Tined Lead

If during the trial the target symptoms are not sufficiently controlled, the tined lead should be removed along with the lead extension. This can be performed in the office using local anesthesia or in the operating room using sedation (MAC) and local anesthesia. After prepping the skin, the local anesthetic is administered to the area of the buttock incision overlying the lead connection. The incision and subcutaneous tissue are opened, exposing the connection hub medially. The hub is pulled up into the incision, and the lead extension wire is cut distal to the plastic sleeve. Later, the remaining extension wire is removed from the skin. Traction is then applied to the tined lead until it is removed. If there is significant resistance and the lead begins to elongate, it may fracture during removal. This would rarely if ever occur after only a staged trial. If this is the case, consideration should be given to opening the sacral incision to remove the lead from that location. The lead can usually be removed without opening the sacral incision after the trial period but when the lead has been in place more permanently, the site of the sacral incision usually needs to be opened. Once removed, the wound is irrigated and closed with absorbable suture.

Complications

Potential complications include wound infection, pain related to stimulation or at implantation site, loss of efficacy, or need for magnetic resonance imaging (MRI). If a wound infection develops, all components of the device must be removed. The authors recommend also removing the capsule that forms around the INS. The wound should be irrigated copiously and should usually be left to close by secondary intention by packing the wound. It is important that sufficient time between wound healing and reimplantation occur to try to prevent recurrent infection. The rate of infection noted in a recent trial (InSite) involving implantation in 272 patients was 3.3 % [10]. The infection rate noted in two studies with long-term follow-up as 5 % [9, 25]. In the case of painful stimulation, the first step should be reprogramming to try to get a more comfortable stimulation setting. Pain at the INS site can often be limited by using a bipolar setting. If pain is associated with palpation over the lead site in the pre-sacral area or over the INS, a revision is sometimes necessary. If the lead is too superficial under the skin, it can be uncomfortable and require revision surgery to pass the lead deeper in the subcutaneous tissue. If too small of a pre-sacral incision has been made during lead placement, it can lead to a palpable knuckle of the lead in the pre-sacral skin, or to anterior migration of the lead. This is particularly true in thin patients. Initially making a larger incision, or using an absorbable suture to lay the lead flat against the fascia, can be helpful to prevent this. One group reported pain at the lead site in 5.4 % and pain at the INS site in 15.3 % of patients with long-term follow-up [14]. Another study reported pain with any of the device components in 6.8 % of patients with use of the tined lead [25]. The INS can cause discomfort if placed too superficially, not parallel to the skin, or in too large of a pocket, causing it to move or "flip" within the pocket. It can also cause pain if it sits against a bony prominence. In these instances, the site may need to be revised or the INS may need to be moved to a different location. If the INS is moved to a new incision

site, the capsule should be removed from the abandoned implantation site. If it is to be placed deeper or more parallel within the same pocket, the old capsule can serve as a sub-layer, under which the new pocket may be created, and then the capsule can be largely obliterated by using it as a separate closure layer over the newly positioned device.

If the lead is placed ideally along the course of the nerve, allowing all four contact points to have appropriate responses at low stimulation thresholds, it should increase the flexibility in programming by allowing for more potential options. It should also increase the battery life. In general, the average length of battery life for an implanter is a good indication of the quality of lead placements. We frequently have InterStim 2 devices lasting beyond the 5 year mark. When a battery depletes on the short end of its life expectancy, a problem related to lead position is clearly implied. If the patient loses benefit with the therapy, the first step is to evaluate the device in terms of confirming the device is turned on, that the patient is using it properly, estimating remaining battery life, and performing an impedance check. If all of these are normal, the device should be reprogrammed. After reprogramming fails to manage the patient's symptoms sufficiently, consideration may be given to performing a lead revision, especially if the patient previously had good success with the therapy. During a lead revision, a new lead should be placed with removal of the old lead. With development of the tined lead, the rate of lead revision surgery has decreased as discussed earlier. The decision for simultaneous replacement of the INS depends on the estimated remaining battery life.

There are occasions where an MRI may be warranted in a patient with an implanted device. It has been shown that MRI of the head using an open 1.5 T machine with RF transmit/receive head coil only can be safely obtained in patients with the InterStim device (Medtronic, Minneapolis, MN) [26]. The device should be turned off during the study and the patient should not be sedated, so they can signal any discomfort that may develop during the scan. In patients where an MRI of other areas such as the pelvis or back is required and no other imaging studies are adequate for evaluation of the patient's condition, the device would need to be removed completely with care taken to remove the entire lead. It is important to avoid implantation initially in patients with a medical condition that will require the routine use of MRI.

Percutaneous Tibial Nerve Stimulation

PTNS is another form of neuromodulation that can be offered to patients with OAB. Neuromodulation is achieved by accessing the posterior tibial nerve above the ankle sending stimulation to the sacral plexus (S2–S4) modulating bladder innervation. Urgent PC is an FDA approved neuromodulation system providing this retrograde stimulation as an office based therapy. The induction therapy consists of 30 min sessions given weekly for 12 weeks and for those that benefit; therapy is maintained with a session every 3–4 weeks thereafter. The Overactive Bladder Innovative Therapy (OrBIT) trial was a randomized control trial performed in patients with urinary frequency comparing PTNS to tolterodine. The patients' subjective improvement with the initial 12 weeks of therapy was 79.5 % in PTNS group compared to 54.8 % in tolterodine group with objective measures similarly improved in the two groups [27]. A second phase of the trial was performed evaluating the durability of PTNS by extending therapy to 1 year for a group of patients. Sustained improvement was noted in those that initially received benefit from the therapy at 6 and 12 months in 94 % and 96 %, respectively [28].

Procedure

With the patient seated or supine in a comfortable position, the needle electrode insertion site is identified three fingerbreadths superior to the medial malleolus and one fingerbreadth posterior to the tibia. An alcohol pad is used to prep the skin at the insertion site, and the sterile needle

electrode (34 Gauge) is placed at a 60° angle to the skin with the tip pointed cephalad. The stop plug is removed and the needle electrode is tapped to pierce the skin and then the guide tube around the needle is removed. The needle electrode is advanced using a twisting motion until approximately 2 cm is inserted. Once the needle electrode has been placed, the grounding pad or surface electrode is attached to medial aspect of the ipsilateral foot and the attached hook is connected to the needle. The lead wire is connected to the stimulator, which is then used to administer 30 min of stimulation. After 30 min, the needle electrode is removed along with the grounding pad/surface electrode. This procedure is repeated for each therapy session. All of the described components are available in the Urgent PC Lead Set (Uroplasty) [29].

Conclusion

OAB affects millions of people worldwide and when conservative therapies have failed to provide adequate management of voiding symptoms, SNM should be considered as the next step in the treatment algorithm. Neuromodulation can be offered in the form of SNS or PTNS. Other indications where SNS is an appropriate treatment option are non-obstructive urinary retention, chronic pelvic pain, fecal incontinence, and childhood dysfunctional elimination syndrome. For SNS, there are two trial methods that may be offered to patients: (1) PNE, (2) Staged Implantation. On multiple long-term follow-up studies, SNS has been shown to be effective and safe. Technological advances such as the tined lead and smaller INS have improved the therapy over time.

References

1. Onunkwugha E, Zuckerman IH, McNally D, et al. The total economic burden of overactive bladder in the Unites States: a disease-specific approach. Am J Manag Care. 2009;15(4 Suppl):S90–7.
2. Yu YF, Nichol MB, Yu AP, et al. Persistence and adherence of medications for chronic overactive bladder/urinary incontinence in the California Medicaid program. Value Health. 2005;8:495.
3. Gormley EA, Lightner DJ, Burgio KL, et al. Diagnosis and treatment of overactive bladder (non-neurogenic) in adults: AUA/SUFU guideline. J Urol. 2012;188: 2455–63.
4. Leng WW, Chancellor MB. How sacral nerve simulation neuromodulation works. Urol Clin N Am. 2005;32:11.
5. Siegel SW, Moeller SE. Chapter 22: Sacral neuromodulation for the treatment of overactive bladder. In: Raz S, Rodriquez L, editors. Female urology. 3rd ed. Philadelphia, PA: WB Sanders; 2008. p. 266.
6. U.S. Food and Drug Administration. www.fda.gov/MedicalDevices/productsandmedicalprocedures/deviceapprovalsandclearances. Accessed September 2013.
7. Spinelli M, Giardiello G, Gerber M, et al. New sacral neuromodulation lead for percutaneous implantation using local anesthesia: description and first experience. J Urol. 2003;170:1905.
8. Thompson JH, Sutherland SE, Siegel SW. Sacral neuromodulation: therapy evolution. Indian J Urol. 2010;26(3):379.
9. Al-zahrani AA, Elzayat EA, Gajewski JB. Long-term outcome and surgical interventions after sacral neuromodulation implant for lower urinary tract symptoms: 14 year experience at 1 center. J Urol. 2011;185:981.
10. Siegel SW. Results of a prospective, multicenter study evaluating the safety and efficacy of sacral neuromodulation through 12-month follow-up in subjects with milder symptoms of overactive bladder. Abstract SUFU Winter meeting; 2013.
11. International Continence Society. www.ics.org. 4th international consultation on incontinence; 2009.
12. Bosch JLHR, Groen J. Sacral (S3) segmental nerve stimulation as a treatment for urge incontinence in patients with detrusor instability: results of chronic electrical stimulation using an implantable neural prosthesis. J Urol. 1995;154:504.
13. van Kerrebroeck PEV, van Voskuilen AC, Hessakkers JPFA, et al. Results of sacral neuromodulation therapy for urinary voiding dysfunction: outcomes of a prospective worldwide clinical study. J Urol. 2007; 178:2029.
14. Siegel SW, Catanzaro F, Dijkema HE, et al. Long-term results of a multicenter study on sacral nerve stimulation for treatment of urinary urge incontinence, urgency-frequency, and retention. Urology. 2000;56:87.
15. Jonas U, Fowler CJ, Channcellor MB, et al. Efficacy of sacral nerve stimulation for urinary retention: results 18 months after implantation. J Urol. 2001;165:15.
16. Siegel S, Paszkiewicz E, Kirkpatrick C, et al. Sacral nerve stimulation in patients with chronic intractable pelvic pain. J Urol. 2001;166:1742.
17. Gajewski JB, Al-Zahrani AA. The long term efficacy of sacral neuromodulation in the management of

intractable cases of bladder pain syndrome: 14 years of experience in one centre. BJU Int. 2010;107:1258.
18. Marcelissen T, Jacobs R, van Kerrebroeck P, et al. Sacral neuromodulation as a treatment for chronic pelvic pain. J Urol. 2011;186:387.
19. Ghazwani YQ, Elkelini MS, Hassouna MM. Efficacy of sacral neuormodulation in treatment of bladder pain syndrome: long term follow-up. Neurourol Urodyn. 2011;30:1271.
20. Peters KM, Konstandt D. Sacral neuromodulation decreases narcotic requirements in refractory interstitial cystitis. BJU Int. 2004;93:777.
21. Wexner SD, Coller JA, Devroede G, et al. Sacral nerve stimulation for fecal incontinence. Ann Surg. 2010;251:441.
22. Mellgren A, Wexner SD, Coller JA, et al. Long-term efficacy and safety of sacral nerve stimulation for fecal incontinence. Dis Colon Rectum. 2011;54:1065.
23. Roth TJ, Vandersteen DR, Hollatz P, et al. Sacral neuromodulation for the dysfunctional elimination syndrome: a single center experience with 20 children. J Urol. 2008;180:306.
24. Bullock TL, Siegel SW. Chapter 94: Neuromodulation. In: Smith Jr JA, Howards SS, Preminger GM, editors. Hinman's Atlas of urologic surgery. 3rd ed. Philadelphia, PA: Elsevier/Saunders; 2012.
25. Sutherland SE, Lavers A, Carlson A, et al. Sacral nerve stimulation for voiding dysfunction: one institution's 11-year experience. Neurourol Urodyn. 2007;26:19.
26. Medtronic: MRI guidelines for InterStim therapy neurostimulation systems. www.medtronic.com. Accessed April 2013.
27. Peters KM, MacDiarmid SA, Wooldridge LS, et al. Randomized trial of percutaneous tibial nerve stimulation versus extended-release tolterodine: results from the overactive bladder innovative therapy trial. J Urol. 2009;182:1055.
28. MacDiarmid SA, Peters KM, Shobeiri SA, et al. Long-term durability of percutaneous tibial nerve stimulation for the treatment of overactive bladder. J Urol. 2010;183:234.
29. Urgent PC Neuromodulation System. www.uroplasty.com/healthcare/urgetpc

Transvaginal Prolapse Repair

5

Nitya E. Abraham and Howard B. Goldman

Introduction

Transvaginal pelvic floor reconstruction can be divided by compartment: anterior repair, posterior repair, and apical repair. Approaches can be restorative, compensatory, and obliterative. Restorative repairs utilize native tissue to fix defects, while compensatory repairs utilize biologic or synthetic graft material to fix defects. It is critical to recognize that the presence of prolapse alone is not an indication for treatment. Up to half of women who have had a vaginal delivery will have prolapse to the hymen. Many, especially as they get older, will have prolapse beyond the hymen [1]. For a significant proportion of these patients the prolapse is asymptomatic and does not require intervention. *Only symptomatic patients should be treated.*

Anterior Repair

Background

Anterior compartment prolapse is herniation of pelvic organs into the anterior vaginal wall, including urethrocele (herniation of the urethra), cystocele (herniation of the bladder), and anterior enterocele (herniation of the small bowel) [2]. The prevalence and incidence of anterior compartment prolapse is not well described. In the Women's Health Initiative, the prevalence of cystocele was 34 % in women age 50–79 years [3]. Risk factors for anterior compartment prolapse include increasing age, body mass index, and number of vaginal deliveries. Other possible risk factors include pregnancy, forceps delivery, young age at first delivery, prolonged labor, high infant birth weight, smoking, elevated intra-abdominal pressure (due to constipation, chronic cough, or occupations requiring heavy lifting), estrogen deficiency, previous hysterectomy, connective tissue disorders (e.g., Ehlers–Danlos syndrome, Marfan's syndrome), muscular disorders (e.g., multiple sclerosis, muscular dystrophy), low socioeconomic status, ethnicity, family history, and history of prior prolapse repair [2–4]. The strongest risk factor is vaginal delivery with one study noting a 2.2 (1.8–2.7) times increased risk of cystocele after a single childbirth compared to nulliparity [3].

Evaluation

The first and most important step in evaluation of anterior compartment prolapse is a history and physical examination. Women with symptomatic anterior compartment prolapse may complain of a sensation of a vaginal bulge, pressure, or

N.E. Abraham, M.D. • H.B. Goldman, M.D. (✉)
Female Pelvic Medicine and Reconstructive Surgery, Glickman Urology and Kidney Institute, Cleveland Clinic Foundation, Cleveland, OH, USA
e-mail: nitya.abraham@gmail.com; goldmah@ccf.org

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_5,

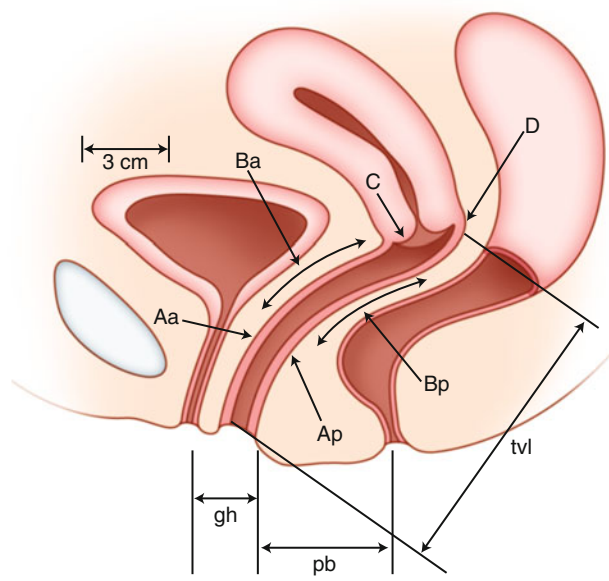


Fig. 5.1 POP-Q exam. *Aa* point A anterior, *Ap* point A posterior, *Ba* point B anterior, *Bp* point B posterior, *C* cervix or vaginal cuff, *D* posterior fornix (if cervix is present), *gh* genital hiatus, *pb* perineal body, *tvl* total vaginal length

heaviness. They may visualize the protrusion. Urinary symptoms may include incontinence, frequency, urgency, obstructive voiding symptoms, or the need to manually reduce the prolapse to void. Patients may also complain of dyspareunia [3]. General symptoms can include low back pain, generalized pelvic pain, or bloody discharge due to ulceration of the prolapsed vaginal skin [2]. The history should thus elucidate the presence of risk factors and symptoms as listed above.

Physical examination is standardized by utilizing the Pelvic Organ Prolapse Quantification (POPQ) system, which involves the measurement of 9 points (Fig. 5.1):

Aa—anterior vaginal wall 3 cm proximal to the hymen

Ba—most distal position of the remaining upper anterior vaginal wall

C—most distal edge of cervix or vaginal cuff scar

D—posterior fornix (not applicable if post-hysterectomy)

Ap—posterior vaginal wall 3 cm proximal to the hymen

Bp—most distal position of the remaining upper posterior vaginal wall

Genital hiatus (gh)—middle of external urethral meatus to posterior midline hymen

Perineal body (pb)—posterior margin of gh to middle of anus

Total vaginal length (tvl)—depth of vagina with prolapse reduced

The POPQ can be categorized into stages:

Stage 0—No prolapse

Stage 1—The most distal point of the prolapse is at least 1 cm above the level of the hymen

Stage 2—The most distal point of the prolapse is between 1 cm proximal and 1 cm distal to the level of the hymen

Stage 3—The most distal point of the prolapse is between 1 cm distal to the level of the hymen and 2 cm less than the tvl

Stage 4—The most distal point of the prolapse is equal to or beyond 2 cm less than the tvl, from the level of the hymen

Some clinicians simplify the POPQ exam and do not routinely measure Aa or Ap.

Other staging systems generally utilize the relationship of the leading edge of the prolapse to the hymenal ring or introitus.

Imaging is not routinely used in the evaluation of pelvic organ prolapse. However, it has been argued that clinical examination assesses surface anatomy and is more limited in assessing

structural abnormalities [5]. Underestimation or misdiagnosis of the compartment that is prolapsed can occur in 45–90 % of cases [6]. What appears to be a cystocele could in rare cases be a urethral diverticulum, Gartner duct cyst, or anterior enterocele [5]. Thus, in ambiguous cases, translabial ultrasound or dynamic MR imaging can be utilized. However, this is not routine, requires trained personnel to interpret the imaging, and may be cost-prohibitive.

Surgical Repair

The patient is placed in the dorsal lithotomy position. The external genitalia is prepped and draped in the usual sterile fashion. Some choose to shave the perineum but it is not required. One dose of intravenous antibiotics is administered for prophylaxis prior to incision. As per the 2011 American Urological Association Guidelines on Antibiotic Prophylaxis, the antibiotics of choice for vaginal surgery are a first/second generation Cephalosporin or an Aminoglycoside (Aztreonam if the patient has renal insufficiency) plus Metronidazole or Clindamycin. Alternative antibiotics are Ampicillin/Sulbactam or Flouroquinolone [7]. A foley catheter is placed to drain the bladder. A weighted speculum is placed in the vagina. A Scott retractor or translabial sutures are used to retract and expose the prolapse. The anterior compartment prolapse repair is then performed using one of the following techniques: traditional anterior colporrhaphy, mesh-augmented colporrhaphy, or paravaginal defect repair.

Traditional Anterior Colporrhaphy

1. An Allis clamp is placed 1 cm distal to the vaginal cuff or cervix. A second Allis clamp is placed just proximal to the bladder neck.
2. While pulling the anterior vaginal wall outward with the Allis clamps, the vaginal wall is infiltrated superficially with a dilute solution of lidocaine mixed with epinephrine (Fig. 5.2).
3. A midline incision is made between the two Allis clamps (Fig. 5.3).
4. Allis clamps are placed on the edges of the vaginal skin on both sides of the incision.

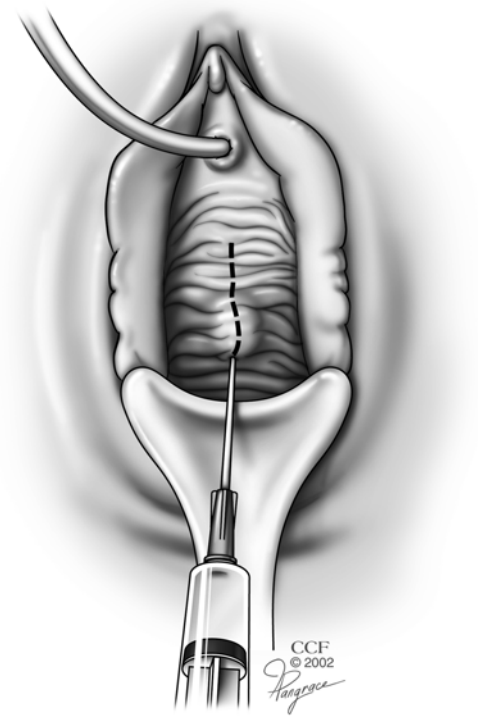


Fig. 5.2 Hydrodissection with a dilute solution of lidocaine mixed with epinephrine. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

5. While retracting the Allis clamp outward, the assistant should provide countertraction on the pubocervical fascia thus delineating the plane between the vaginal skin and pubocervical fascia. (Note, the more appropriate term is vaginal muscularis and not pubocervical fascia, since there is no actual fascial layer.) A combination of sharp and blunt dissection is used to dissect the vaginal skin off the underlying pubocervical fascia (Fig. 5.4).
6. The pubocervical fascia is then plicated in the midline with 2-0 absorbable interrupted sutures (Fig. 5.5).
7. Many surgeons perform a cystoscopy at this point to evaluate for ureteral efflux ensuring that they have not caused ureteral obstruction, and to verify there are no sutures in the bladder.
8. Excess vaginal skin is excised and the incision is closed with a running, locking absorbable

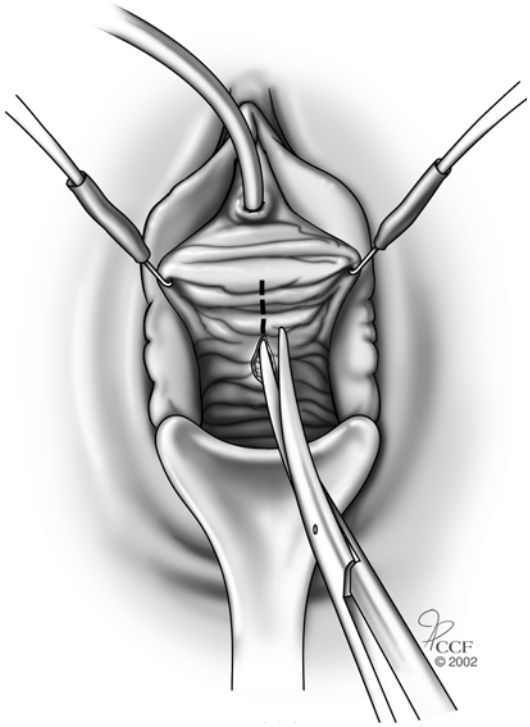


Fig. 5.3 Midline incision for anterior colporrhaphy. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

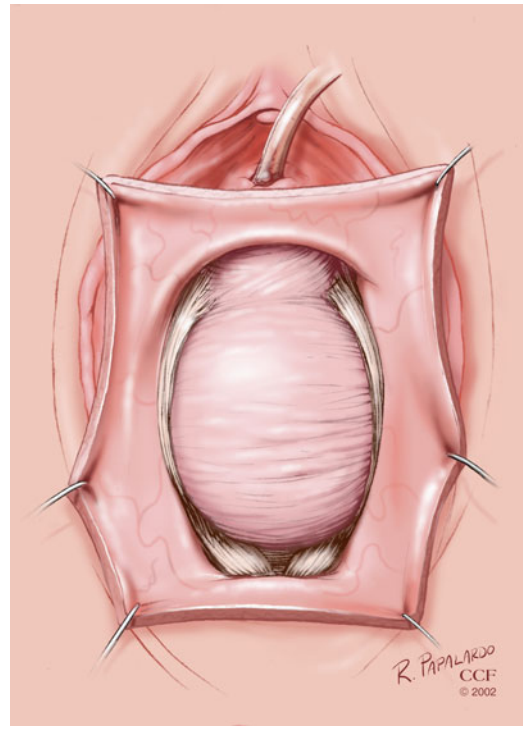


Fig. 5.4 Dissection between vaginal skin and pubocervical fascia. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

suture—it is important not to overtrim the vaginal skin.

9. If a concomitant mid-urethral sling is being placed, it should be done after the cystocele repair through a separate more distal incision.

Mesh-Augmented Colporrhaphy

Mesh placement for mesh-augmented anterior colporrhaphy can be performed using self-tailored biologic or synthetic mesh, a transobturator and/or transgluteal trocar-guided synthetic mesh kit, or a non-trocar synthetic mesh kit. (The objective outcome data is better for macroporous polypropylene synthetic mesh compared to biologic mesh. See section on Recent Randomized Trials on Outcomes)

1. An Allis clamp is placed 1 cm distal to the vaginal cuff or cervix. A second Allis clamp is placed 1–2 cm proximal to the bladder neck.
2. While pulling the anterior vaginal wall outward with the Allis clamps, the vaginal wall is infiltrated *deeply* and hydrodissected with a dilute solution of lidocaine mixed with epinephrine, thus developing a plane between the pubocervical fascia and the bladder adventitia.
3. A midline incision is made between the two Allis clamps.
4. Allis clamps are placed on the edges of the vaginal skin on both sides of the incision.
5. A combination of sharp and blunt dissection is used to dissect the vaginal skin with the underlying pubocervical fascia off the bladder adventitia, thus developing the vesicovaginal space. The dissection to achieve this plane is very different from the traditional colporrhaphy dissection. The correct plane for mesh placement is critical to ensure that mesh extrusion does not occur.
6. The mesh is placed loosely to allow for possible scarring and tightening of the mesh.
7. The mesh is secured to the arcus tendineus fascia pelvis (ATFP), iliococcygeus muscle, or sacrospinous ligament depending on the technique used (Fig. 5.6).

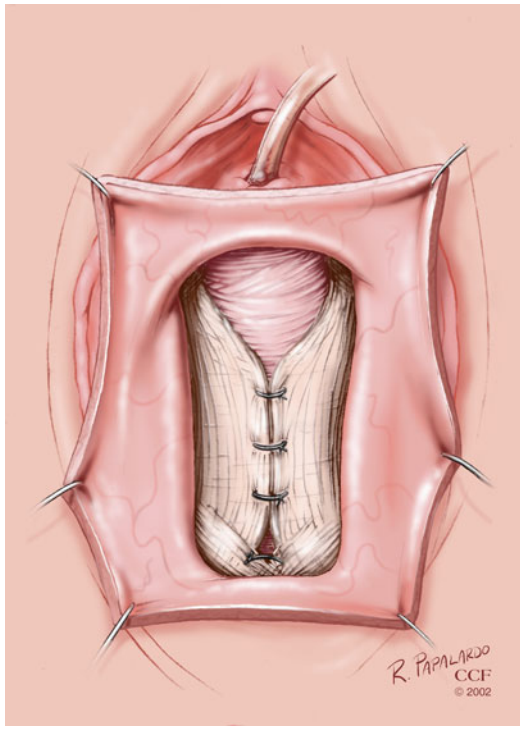


Fig. 5.5 Plication of pubocervical fascia for anterior colporrhaphy. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

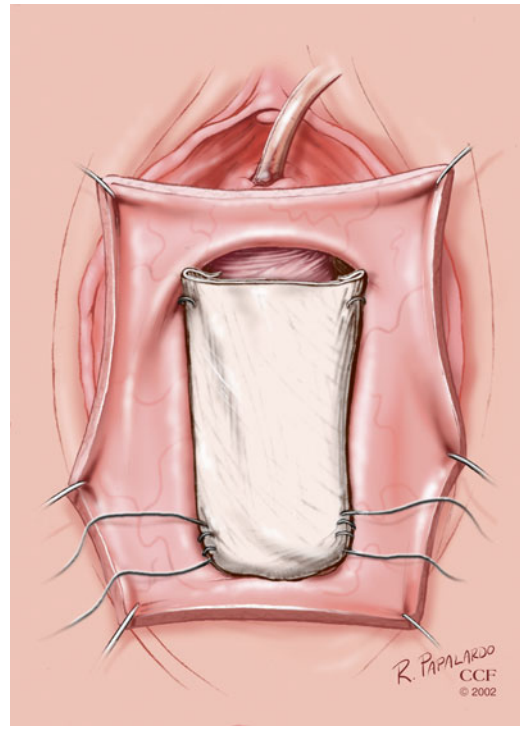


Fig. 5.6 Biologic mesh-augmented anterior colporrhaphy. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

8. Cystoscopy is performed to verify there are no sutures or mesh in the bladder.
9. The vaginal skin is not trimmed and is closed with a running absorbable suture [8].
10. A vaginal pack is left in place overnight

Paravaginal Defect Repair

1. The pubocervical fascia is exposed in the same manner as detailed above for traditional anterior colporrhaphy. However, the dissection is extended further so that the anterior border of the developed space is the ischiopubic rami, the medial border is the pubic symphysis, and the lateral border is the ischial spine.
2. The pubocervical fascia is then plicated in the midline with 2-0 absorbable interrupted sutures.
3. The lateral defect is then repaired by placing nonabsorbable suture through the avulsed lateral edge of the pubocervical fascia, ATFP, and muscularis of the lateral vaginal wall. Transvaginally the exposure and suture placement on the arcus tendineous can be challenging but is made easier by use of a device that allows for suture placement based on palpation (Capio®, Boston Scientific Company). A series of four to six stitches are placed along the ATFP from the ischial spine toward the level of the urethrovesical junction.
4. The same is performed on the contralateral side.
5. The sutures are tied sequentially from one side to the other starting from the urethrovesical junction heading toward the ischial spine. This technique can also be performed transabdominally. Transabdominal paravaginal defect repair is currently often performed with a minimally invasive laparoscopic or robotic approach.

6. Cystoscopy is performed to evaluate for ureteral efflux and to verify there are no sutures in the bladder.
7. Excess vaginal skin is excised and the incision is then closed with a running, locking absorbable suture.

Recent Randomized Trials on Outcomes [9]

Several prospective trials have compared outcomes between these approaches. The first randomized trial comparing anterior colporrhaphy techniques was published by Weber et al. in 2001 [10]. Chmielewski et al. reanalyzed the data using a more clinically relevant definition of success, which included no prolapse beyond the hymen, absence of prolapse symptoms, and absence of retreatment. One hundred fourteen women were randomized to standard anterior colporrhaphy, ultralateral colporrhaphy, or anterior colporrhaphy with (absorbable) mesh, and were followed at 6 months, 1 year, and 2 years after repair. Eighty-eight percent of women with sufficient follow-up data at 1 year met the definition of surgical success. There was no difference between repair groups. The authors concluded that standard anterior colporrhaphy is appropriate for primary cystocele repair at 2 years follow-up [11]. Some have criticized this study as a reanalysis years after the original with changing definitions and relatively short follow-up given the length of time from the original study [12].

Several more recent randomized, controlled trials have compared anterior colporrhaphy with mesh-augmented colporrhaphy. Anatomical success rates for anterior colporrhaphy were 41–72 % versus 81–91 % for self-tailored synthetic mesh-augmented colporrhaphy and 87–91 % for synthetic mesh-kit-augmented colporrhaphy at 1 year follow-up. Vaginal mesh extrusion rates were 4–7 %. Rates of de novo dyspareunia were not significantly different between groups [13–16].

The randomized, controlled study with the longest follow-up, 3 years, reported 59 % anatomical success in the traditional colporrhaphy group versus 87 % in the synthetic mesh-augmented colporrhaphy group ($p<0.0001$). Symptomatic outcomes and rates of de novo dyspareunia were similar in both groups. The mesh extrusion rate was 19 % [17].

The largest randomized controlled trial compared 389 women who underwent trocar-guided, transvaginal polypropylene-mesh repair (Gynecare Prolift™ Anterior Pelvic Floor Repair System kit, Ethicon) to women who underwent traditional colporrhaphy. The primary outcome was a composite of anatomic and symptomatic success. At 1-year follow-up, 60.8 % of women treated with transvaginal mesh had no prolapse or vaginal bulge symptoms compared to 34.5 % of women who underwent traditional colporrhaphy ($p<0.001$). The rate of reoperation for mesh exposure was 3.2 %. The mesh repair group also experienced more surgical complications such as longer operating time, increased blood loss, bladder injury, and mesh-related complications [18].

A criticism of several studies has been that anterior colporrhaphy has had worse anatomic outcomes compared to mesh-augmented colporrhaphy due to not addressing concomitant apical prolapse. Nguyen et al. performed a randomized trial comparing anterior colporrhaphy with synthetic mesh-augmented colporrhaphy. The majority of these women underwent uterosacral vault suspension. At 1-year follow-up, anatomic success (no stage 2 or greater anterior prolapse) was 55 % and 87 % in the traditional and synthetic mesh-augmented groups, thus demonstrating the superiority of mesh-augmented repairs even after addressing apical prolapse in both groups [15].

Given the risk of extrusion with synthetic mesh, three randomized trials have compared traditional colporrhaphy with biologic graft-augmented colporrhaphy. Meschia et al. utilized Pelvicol in their study of women with primary stage 2 or greater cystocele. At 2 years follow-up, the failure rate in the traditional group was 23 % versus 11 % in the Pelvicol group (RR 2.08, 95 % CI 1.08–4.01). One woman underwent graft removal due to "rejection." Subjective outcomes at 2 years were not reported [19]. Dahlgren et al. performed a similar study utilizing Pelvicol® (Bard Medical) for women with recurrent

cystocele. Recurrence rates at 3 years follow-up were 57 % without versus 62 % with Pelvicol (not significant). Symptomatic improvement and sexual function were similar in both groups [20]. The third randomized trial utilized cadaveric fascial lata (Tutoplast®, Davol) in women with primary or recurrent cystocele. At 13 months follow-up, there was no difference in objective and subjective outcomes [19]. Thus, it seems there is no functional or symptomatic advantage with the use of a biologic graft for colporrhaphy.

A randomized controlled trial comparing traditional colporrhaphy, xenograft-augmented, and synthetic mesh-augmented colporrhaphy has been conducted with 2 year follow-up. Anatomic failure rate was 58 %, 46 %, and 18 % in each respective group ($p<0.05$). Symptomatic failure rates were not statistically different between groups. The mesh extrusion rate was 14 %. The authors concluded that synthetic mesh-augmented repair had the best anatomic outcome but symptomatic outcomes were similar between all groups [21].

Finally, a recent Cochrane review has noted that there are better outcomes for anterior repair with mesh augmentation than anterior colporrhaphy alone and that synthetic mesh does better than biologic mesh [19].

See Table 5.1.

Table 5.1 Success rates for pelvic organ prolapse repair

	Objective success (%)	Subjective success (%)[a]
Anterior repair		
Traditional	41–89	81–100
Paravaginal	83	93
Synthetic mesh augmented	81–96	81–91
Biologic mesh augmented	54–82	
Posterior repair		
Traditional	75–91	80–93
Site-specific	78	88
Synthetic mesh augmented	78–96	79–96
Biologic mesh augmented	54–88	97
Apical repair		
USVS	86–97	94
SSLF	69	91
Transvaginal mesh augmented	43	79
Open ASC	76–94	94
Laparoscopic ASC	77–91	87
Robotic ASC	88–94	
Uterine sparing ASC	68	
Hysterectomy and A–P repair	87	
Colpocleisis	85–100	

[a]Subjective outcomes not reported as a percentage for robotic ASC, uterine sparing ASC, hysterectomy and A–P repair, and colpocleisis, and thus not listed

Summary

The randomized controlled trials comparing colporrhaphy with and without mesh are heterogeneous. However, the consensus seems to be that mesh repair provides superior anatomic outcome but equivalent symptomatic outcome. While subjective outcomes appear similar in the short run, one cannot know at this point if that will remain so in the long run. Specifically, will those patients with asymptomatic anatomic recurrences now become those that have symptomatic recurrences in the future? Rates of de novo dyspareunia are not significantly increased with mesh repair, but mesh exposure rates and the increased surgical complication rates are not negligible. Given the risk of mesh extrusion, the FDA safety update on transvaginal mesh for pelvic organ prolapse [22], and after a recent $5.5 million settlement against the manufacturers of Avaulta mesh, surgeons must use caution when utilizing transvaginal mesh [23]. Does that mean there is no role for the use of synthetic mesh in transvaginal cystocele repair? The reality is that a percentage of women will fail primary traditional colporrhaphy and women with Stage 3 or higher prolapse are more likely to recur [24]. We thus recommend a pragmatic approach to the use of transvaginal mesh for prolapse repair [25], by carefully selecting women at high risk for recurrence and having an informed discussion of the postoperative risks and complications of synthetic mesh-augmented colporrhaphy. In addition, some women with significant apical prolapse with concomitant anterior vaginal wall prolapse may do well with an abdominal sacrocolpopexy. This is discussed further in the section on apical prolapse.

Key Points

- Anatomic cure after traditional colporrhaphy is as high as 72 % compared to as high as 91 % after synthetic mesh-augmented colporrhaphy, even after addressing apical prolapse.
- Biologic graft-augmented colporrhaphy is not superior to traditional colporrhaphy.
- Early subjective outcomes and rates of de novo dyspareunia are similar for all types of colporrhaphy.
- Synthetic mesh extrusion rates are 4–19 %.
- Long-term anatomic durability and subjective outcomes are unknown.
- Women with recurrent cystocele or Stage 3 or higher prolapse may benefit from a synthetic mesh-augmented repair if appropriately counseled.

Posterior Repair and Perineorrhaphy

Background

Posterior compartment prolapse is herniation of pelvic organs into the posterior vaginal wall, including rectocele (herniation of the rectum) and posterior enterocele (herniation of the small bowel) [2]. The prevalence and incidence of posterior compartment prolapse is also not well described. In the Women's Health Initiative, the prevalence of rectocele was 19 % in women age 50–79 years [3]. Risk factors for posterior compartment prolapse are similar to those for anterior compartment prolapse (see above). The strongest risk factor is vaginal delivery with one study noting a 1.9 (1.7–2.2) times increased risk of rectocele after a single childbirth compared to nulliparity [3].

Evaluation

The first step in evaluation of posterior compartment prolapse is a history and physical examination. Women with symptomatic posterior compartment prolapse may complain of a sensation of a vaginal bulge, pressure, or heaviness. They may visualize the protrusion. Bowel symptoms may include incontinence of flatus or stool, feeling of incomplete emptying, straining to defecate, fecal urgency, digital evacuation of stool from rectum, splinting or manual reduction of prolapse to defecate, and feeling of obstruction during defecation. Patients may also complain of dyspareunia [3]. General symptoms can include low back pain, generalized pelvic pain, or bloody discharge due to ulceration of the prolapsed vaginal skin [2]. The history should thus elucidate the presence of risk factors and symptoms as previously described.

Physical examination is standardized by utilizing the POPQ system as detailed in the previous section.

Imaging is not routinely used in the evaluation of pelvic organ prolapse. However, transperineal ultrasound or MR defecography can differentiate a rectocele from an enterocele.

Surgical Repair

The patient is placed in the dorsal lithotomy position. The external genitalia is prepped and draped in the usual sterile fashion. One dose of intravenous antibiotics is administered for prophylaxis prior to incision. As per the 2011 American Urological Association Guidelines on Antibiotic Prophylaxis, the antibiotics of choice for vaginal surgery are a first/second generation Cephalosporin or an Aminoglycoside (Aztreonam if the patient has renal insufficiency) plus Metronidazole or Clindamycin. Alternative antibiotics are Ampicillin/Sulbactam or Flouroquinolone [7]. A foley catheter is placed to drain the bladder. A weighted speculum is placed in the vagina. A Scott retractor or translabial sutures are used to retract and expose the prolapse. The posterior compartment prolapse repair is then performed using one of the following techniques: traditional posterior colporrhaphy, site-specific posterior repair, or graft-augmented posterior colporrhaphy. Perineorrhaphy is often performed at the same

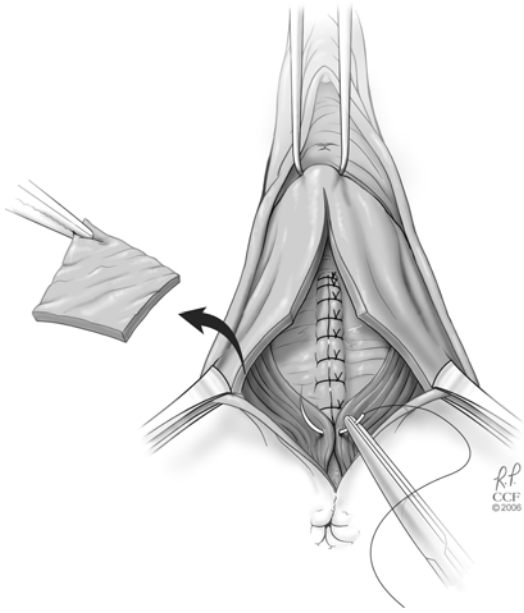


Fig. 5.7 Plication of rectovaginal fascia for posterior colporrhaphy. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

time. The goals of posterior compartment prolapse repair are plication of the prerectal and pararectal fascia, narrowing of the levator hiatus, and repair of the perineal body [26].

Traditional Posterior Colporrhaphy

1. The posterior vaginal wall is infiltrated superficially with a dilute solution of lidocaine and epinephrine.
2. A transverse incision is made at the hymen and a midline incision made extending towards the cervix, creating an inverted T (some prefer a triangle instead).
3. The vaginal skin is carefully dissected off the rectovaginal fascia using sharp and blunt dissection. (Note, the more appropriate term is vaginal muscularis and not rectovaginal fascia, since there is no actual fascial layer.)
4. The lateral rectovaginal fascia is plicated together in the midline with interrupted 2-0 absorbable stitches, starting proximally and progressing distally towards the perineal body (Fig. 5.7).
5. A perineorrhaphy is typically performed by placing deep plication sutures in the puborectalis muscle.
6. Excess vaginal skin is excised after which the posterior vaginal wall is closed with running locking absorbable suture. It is important to not over plicate the perineum and to leave adequate skin so the incisions are not closed under tension. Too much narrowing of the vagina can lead to dyspareunia. It is the author's practice to leave the introitus large enough to accommodate three fingers (Fig. 5.8).

Site-Specific Posterior Colporrhaphy

1. The posterior vaginal wall is not hydrodissected in order to better identify defects in the rectovaginal septum.
2. A transverse incision is made at the hymen and a midline incision made extending towards the cervix, creating an inverted T (some prefer a triangle instead).
3. The vaginal skin is carefully dissected off the rectovaginal fascia using sharp and blunt dissection.
4. A finger from the non-dominant hand is placed in the rectum and pushed upward to identify fascial defects, which are then repaired with interrupted stitches (Fig. 5.9).
5. Excess vaginal skin is excised, a perineorrhaphy is performed if needed, and the vaginal skin is then closed.

Graft-Augmented Posterior Colporrhaphy

1. A plane deep to the rectovaginal fascia is developed (usually after copious hydrodissection).
2. The mesh is sutured proximally to the cervix, uterosacral ligaments, or sacrospinous ligaments, distally to the perineal body or distal rectovaginal septum, and in some techniques laterally to the pelvic sidewall. Alternatively, if a smaller patch of the mesh is used, it is sutured laterally to the pararectal fascia and proximally and distally to the rectovaginal fascia.
3. The vaginal skin is not trimmed and is subsequently closed over the mesh.

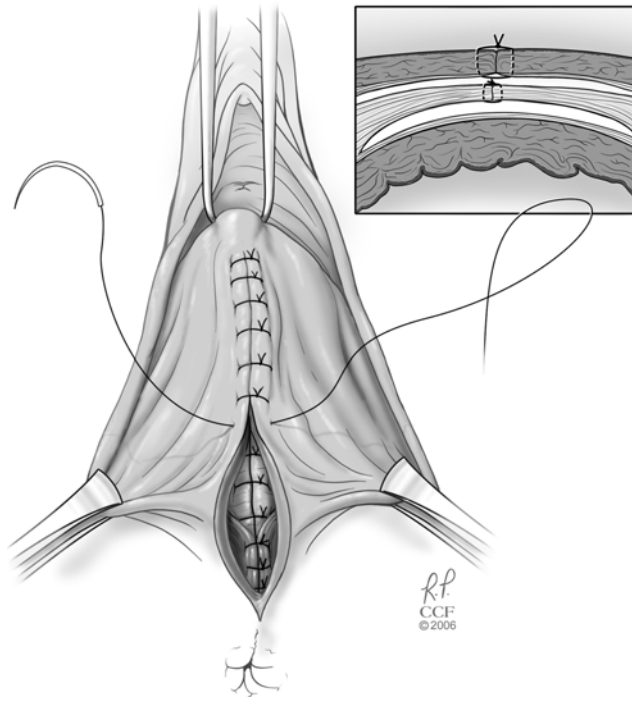


Fig. 5.8 Closure for posterior colporrhaphy. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

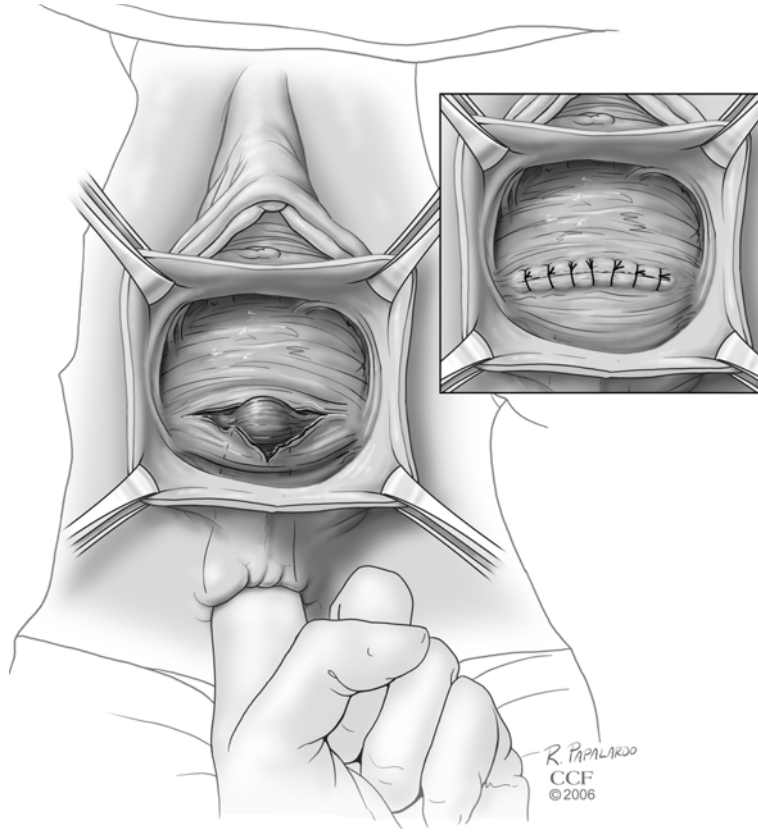


Fig. 5.9 Site-specific posterior colporrhaphy. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

Recent Randomized Trials on Outcomes [9]

There are two randomized controlled trials specifically studying transvaginal rectocele repair [27, 28]. Paraiso et al. randomized women to posterior colporrhaphy ($n=37$), site-specific rectocele repair ($n=37$), or site-specific rectocele repair augmented with a porcine 4×8 cm Fortagen graft (Organogenesis Inc, Canton, MA; $n=32$). Anatomic cure was defined as POP-Q point Bp less than or equal to −2 at 12 months follow-up. Patients also completed validated questionnaires to assess subjective outcomes. At 1-year follow-up, the rate of anatomic cure was 86 % in the posterior colporrhaphy group, 78 % in the site-specific group, and 54 % in the porcine graft-augmented group. There was no significant difference in subjective outcomes or rates of de novo dyspareunia between the three groups. The authors concluded that porcine graft did not improve anatomic outcomes for posterior colporrhaphy and that all three methods provided symptomatic and quality of life improvement [28]. One criticism of this study is that the graft used may not be as durable as other commonly used biologic grafts, and thus may not have functioned as well. However, clearly the traditional colporrhaphy group did very well.

Sung et al. conducted a randomized controlled trial comparing native tissue rectocele repair and porcine subintestinal submucosal graft-augmented rectocele repair in 160 women with stage 2 or greater rectocele. Women in the control group underwent either midline plication of the rectovaginal connective tissue or site-specific repair at the discretion of the surgeon, whereas women in the study group similarly underwent midline plication or site-specific repair, augmented with a 4×7 cm porcine graft. Anatomic failure was defined as points Ap or Bp greater than or equal to −1 on POP-Q. Subjective symptoms were based on items taken from the Pelvic Floor Distress Inventory questionnaire. At 12 months follow-up, the rate of anatomic failure was 8.6 % in the native tissue repair group and 12 % in the graft-augmented repair group. There was no statistically significant difference between the groups regarding subjective symptoms or dyspareunia. The authors concluded that augmentation of posterior colporrhaphy with porcine graft did not provide any objective or subjective benefit over native tissue repair [27].

There are no randomized controlled studies solely comparing traditional posterior colporrhaphy with synthetic-mesh-augmented posterior repair. Two trials included outcomes on the posterior compartment [29, 30]. Sokol et al. compared traditional anterior–posterior repair and uterosacral ligament suspension with vaginal colpopexy using the Prolift® (Ethicon, Somerville, NJ) synthetic mesh. Overall objective recurrence was 69.7 % in the traditional repair group versus 62.5 % in the mesh repair group. Posterior compartment recurrence was 18.2 % in the traditional repair group versus 21.9 % in the mesh repair group (not significant). The mesh extrusion rate was 15.6 % [30]. This study has been criticized for the low number of patients enrolled, the few procedures performed per surgeon, and the short follow-up rate. Withagen et al. compared traditional vaginal prolapse repair with trocar-guided mesh repair. The posterior compartment recurrence rate was 24.5 % in the traditional repair group versus 4.1 % in the mesh repair group ($p=0.003$). The mesh extrusion rate was 16.9 % [29].

There are two randomized trials comparing transvaginal and transanal rectocele repair[19]. Transvaginal repair was superior to transanal repair subjectively (RR 0.36, 95 % CI 0.13–1) and objectively (RR 0.24, 95 % CI 0.09–0.64) [19].

See Table 5.1.

Summary

Transvaginal repair is preferred over transanal repair of rectocele. Given the risk of mesh extrusion and the overall similar objective and subjective outcomes for mesh-augmented versus traditional repair noted in most studies, posterior compartment repair with native tissue is currently the gold standard [31].

Key Point

- The gold standard for rectocele repair is transvaginal traditional posterior colporrhaphy.

Apical Repair

Background

Apical compartment prolapse is herniation of the cervix, uterus, or vaginal cuff into the apical vaginal wall [2]. In the Women's Health Initiative, the prevalence of uterine prolapse was 14 % in women age 50–79 years [3]. Risk factors for apical compartment prolapsed are similar to those for anterior compartment prolapse (see above). The strongest risk factor is vaginal delivery with one study noting a 2.1 (1.7–2.7) times increased risk of uterine prolapse after a single childbirth compared to nulliparity [3].

Evaluation

The first step in evaluation of apical compartment prolapse is a history and physical examination. Women with symptomatic apical compartment prolapse may complain of a sensation of a vaginal bulge, pressure, or heaviness. They may visualize the protrusion. They may report urinary and bowel symptoms as detailed previously. Patients may also complain of dyspareunia [3]. General symptoms can include low back pain, generalized pelvic pain, or bloody discharge due to ulceration of the prolapsed vaginal skin [2]. The history should thus elucidate the presence of risk factors and symptoms previously described.

Physical examination is standardized by utilizing the POPQ system as detailed previously.

Imaging is not routinely used in the evaluation of pelvic organ prolapse. Transperineal ultrasound is least useful in the apical compartment [5, 6].

Surgical Repair

The options for transvaginal apical repair can be divided into restorative, compensatory, and obliterative procedures. The restorative techniques include transvaginal sacrospinous ligament fixation (SSLF), transvaginal iliococcygeus suspension, or transvaginal uterosacral suspension.

The patient is placed in the dorsal lithotomy position. The external genitalia is prepped and draped in the usual sterile fashion. One dose of intravenous antibiotics is administered for prophylaxis prior to incision. As per the 2011 American Urological Association Guidelines on Antibiotic Prophylaxis, the antibiotics of choice for vaginal surgery are a first/second generation Cephalosporin or an Aminoglycoside (Aztreonam if the patient has renal insufficiency) plus Metronidazole or Clindamycin. Alternative antibiotics are Ampicillin/Sulbactam or Flouroquinolone [7]. A foley catheter is placed to drain the bladder. A weighted speculum is placed in the vagina. A Scott retractor or translabial sutures are used to retract and expose the prolapse.

Restorative Apical Prolapse Repair

Transvaginal Sacrospinous Ligament Fixation

SSLF can be performed unilaterally or bilaterally, using an anterior or posterior approach. SSLF is preferred if there is coexistent posterior prolapse. However, anterior prolapse can be exacerbated by SSLF.

Posterior Approach

1. If the patient has previously undergone hysterectomy, the location on the apex of the vagina that will be secured to the sacrospinous ligament is marked with a stitch. Otherwise the cervix is secured to the sacrospinous ligament.
2. The posterior vaginal wall is infiltrated with a dilute solution of lidocaine and epinephrine.
3. A midline incision is made extending towards the apex/cervix.
4. The right pararectal space is bluntly dissected to reach the right sacrospinous ligament (Fig. 5.10).
5. Three Breisky–Navratil retractors are used to expose the sacrospinous ligament, one retracting the rectum medially, another retracting the vaginal wall upward, and the third retracting laterally thus exposing the ligament. The full extent of the ligament is exposed such that the ischial spine

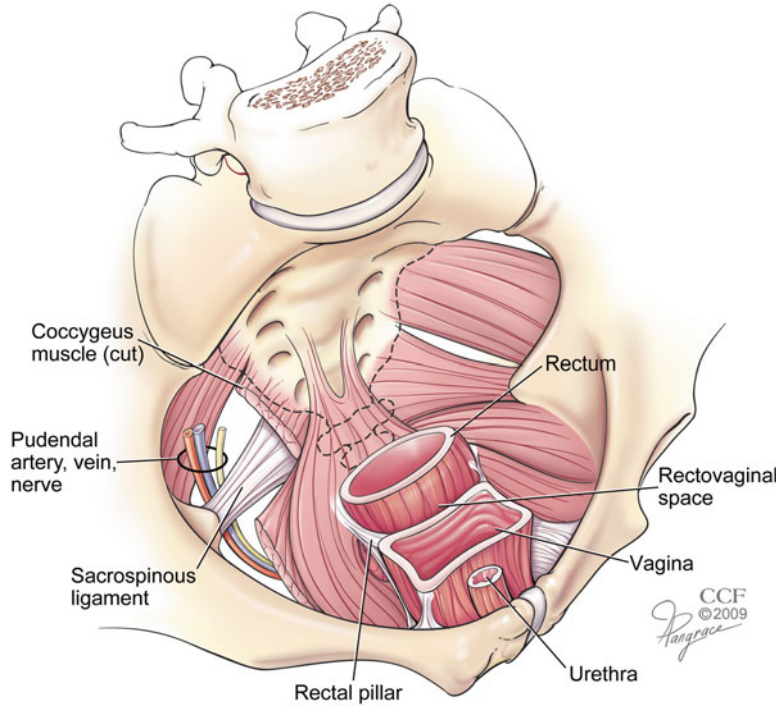


Fig. 5.10 Anatomical location of sacrospinous ligament. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

and sacrum are palpable. A Kittner can be used to bluntly expose and clean off any tissue overlying the sacrospinous ligament.

6. Two adjacent stitches, using permanent or delayed absorbable suture, are placed through the sacrospinous ligament under direct visualization using the Deschamps needle driver 2 cm medial to the ischial spine to avoid the pudendal neurovascular complex. Alterna-tively, a Miya hook or automatic suture-capturing device like the Capio® device (Boston Scientific, Natick, MA) can be used to pass the suture through the ligament. A 1×1 cm segment of macroporous polypropylene mesh can be placed through the suture and placed on top of the ligament to aid in apical scarring (Fig. 5.11).
7. The posterior vaginal wall is then closed.
8. If the sacrospinous ligament is attenuated or if the vagina is foreshortened and the apex cannot reach the sacrospinous ligament, the sutures can be placed through the right iliococcygeus muscle. When performing an iliococcygeus suspension bilateral sutures are often placed.

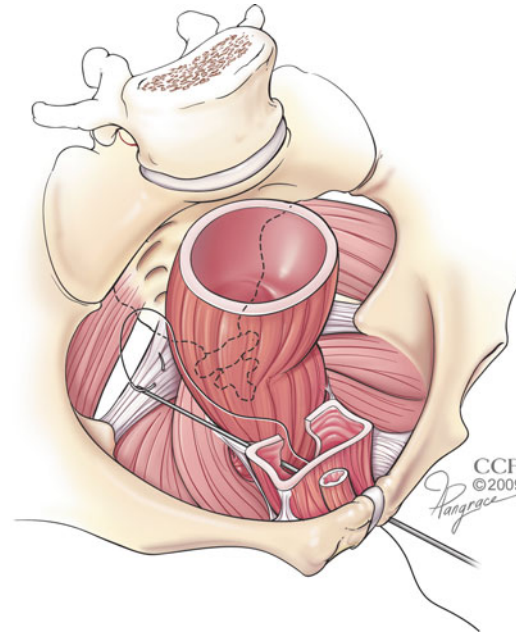


Fig. 5.11 Pathway for placement of suture in transvaginal sacrospinous ligament fixation. (Reprinted with permission, Cleveland Clinic Center for Medical Art and Photography © 2002–2013. All Rights Reserved.)

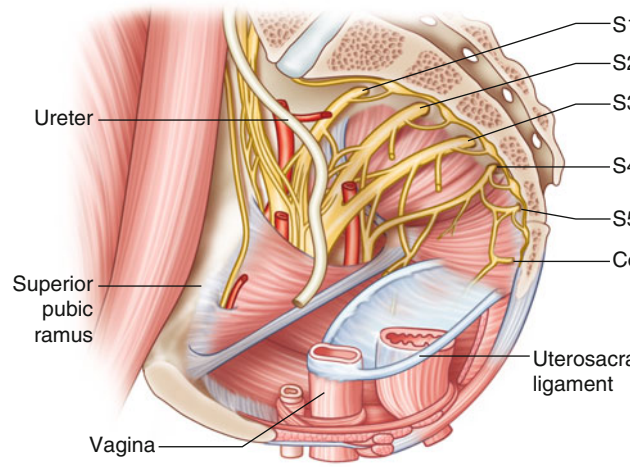


Fig. 5.12 Anatomical location of uterosacral ligament and proximity of sacral nerves

Transvaginal Uterosacral Ligament Suspension

Transvaginal uterosacral ligament suspension is preferred if concomitant hysterectomy is being performed and the vaginal cuff is open since uterosacral ligament suspension is best performed intraperitoneally. This may also be the preferred technique post-hysterectomy in the presence of a significant enterocele.

1. After the hysterectomy is performed, pack the bowel away with a moistened laparotomy sponge and lift the bowel upward with a Deaver, Heaney, or Breisky–Navratil retractor to expose the uterosacral ligaments (USL). In the post-hysterectomy patient it is helpful to place a suture at each USL dimple at the apex after which the peritoneum can be entered via a diamond-shaped incision and with tension on the sutures the USL can be palpated.
2. Palpate the ischial spines and sacrospinous ligaments bilaterally. An Allis clamp is placed on the distal uterosacral ligament and placed on traction to allow palpation of the uterosacral ligament. The USL approaches the sacrospinous ligament and then takes a medial course to insert into the sacrum.
3. The more cephalad the sutures are placed on the USL, the more medial the sutures will be thus minimizing ureteral injury. Two or three permanent or delayed absorbable sutures are placed through the proximal uterosacral ligaments and vaginal apex bilaterally. Note the suture through the USL should not be too deep so as to avoid injury to sacral nerve roots (Fig. 5.12).
4. If an anterior colporrhaphy is required, it should be performed at this time. The anterior vaginal wall incision and vaginal cuff should be closed.
5. Indigo carmine should be administered intravenously. It is the authors' practice to place the uterosacral ligament fixation sutures on traction and then perform cystoscopy to ensure efflux from both ureteral orifices, thus confirming patency. The sutures are tied down and cystoscopy is performed again. If there is no efflux all the sutures on that side are removed and replaced.

Compensatory Apical Prolapse Repair

Compensatory techniques for apical repair entail the utilization of mesh to augment the repair. Mesh can be used via a transvaginal or transabdominal approach. Hysterectomy can be performed concomitantly or the procedure can be uterine-sparing.

There are several transvaginal mesh kits for apical repair including: Gynecare Anterior Prolift® (Ethicon, Inc. Somerville, NJ), the American Medical Systems Perigree (Minnetonka, MN),

Boston Scientific Pinnacle and Uphold, AMS Elevate, and Ethicon Prosima. The mesh kits utilize delivery of mesh arms via transobturator trocars (Prolift, Perigee), fixation to SSL and/or ATFP with a Capio® (Boston Scientific, Natick, MA) needle driver (Uphold™, Pinnacle), with another anchoring system (Elevate®, AMS), or fixation utilizing a vaginal support device (Prosima™, Ethicon) [32]. For a number of reasons the Prolift and Pinnacle devices are no longer being manufactured or marketed.

Sacral colpopexy is performed transabdominally in women who have undergone hysterectomy. Two strips of polypropylene mesh or a preformed Y-shaped mesh is secured to the anterior and posterior vaginal wall with permanent or delayed absorbable sutures. The cranial ends are secured to the anterior spinous ligament on the sacral promontory. If uterine sparing is desired, only a posterior mesh strip is utilized or a Y-shaped strip of mesh is tunneled through the broad ligament bilaterally and secured anterior to the uterus. Abdominal sacral colpopexy (ASC) or hysteropexy can be performed open or more commonly today via a laparoscopic or robotic approach. These procedures are described in further detail in Chaps. 7 and 8.

Obliterative Apical Prolapse Repair

For women who are not sexually active or who have multiple comorbidities warranting a less complicated procedure, colpocleisis is a simple obliterative procedure that can be utilized for significant apical prolapse (anterior and posterior prolapse is needed as well to technically perform colpocleisis). Success rates are 85–100 % [26]. These procedures will be discussed in Chap. 6.

Recent Randomized Trials on Outcomes [9] (Table 5.1)

Transvaginal Repair with Versus Without Mesh

There is only one randomized controlled trial comparing transvaginal native tissue apical repair with mesh-augmented apical repair. Lopes et al. randomized 32 women to SSLF or mesh repair with Nazca R, Promedon (Cordoba, Argentina). After 1-year follow-up the rate of anatomic cure and quality of life measures were similar in both groups. However, there were five cases of mesh extrusion (35.7 %). This study is limited by its small sample size and use of a transvaginal mesh kit that is not commonly available [33].

Transvaginal Versus Transabdominal Approach

There are several randomized controlled trials comparing transvaginal to transabdominal approaches for apical repair. Maher et al. published an updated summary version Cochrane review of all the available studies up to 2010 [19]. The rate of recurrent vault prolapse was lower for ASC compared to vaginal SSLF (RR 0.23, 95 % CI 0.07–0.77). The risk of postoperative dyspareunia was also lower for ASC (RR 0.39, 95 % CI 0.18–0.86). However, operative time was longer for ASC (mean difference 21 min, 95 % CI 12–30), and ASC was more expensive (mean difference US$1,334, 95 % CI 1,027–1,641).

Of the studies included in the Cochrane review, the most recent was published in 2004 [34]. Ninety-five women were randomized to ASC or vaginal sacrospinous colpopexy and followed for 2 years. The subjective success rates were 94 % and 91 % ($p=0.19$), objective success rates 76 % and 69 % ($p=0.48$), and satisfaction rates 85 % and 81 % ($p=0.78$) for ASC and sacrospinous colpopexy, respectively. There was no difference in sexual function and similar improvement in bladder and quality of life scales between the two groups. Postoperative anterior vaginal wall and vault prolapse (grade 2 or more) were significantly less (13 % versus 45 %) with the abdominal approach ($p=0.01$). However, operative time and return to routine activities was longer for the abdominal approach. The authors concluded that abdominal sacrocolpopexy and sacrospinous colpopexy were equally effective for pelvic organ prolapse. However women with predominantly anterior and vault prolapse may benefit more from an abdominal sacrocolpopexy, whereas women with predominantly posterior

and vault prolapse may benefit from vaginal sacrospinous colpopexy repair [34].

Maher et al. also performed a randomized trial comparing LSC versus total vaginal mesh (TVM) using Total Prolift® (Gynecare, Ethicon). 108 women were randomized with the primary outcome being objective success using POP-Q measurements. Secondary outcomes were perioperative outcomes, patient satisfaction, quality of life, complications, and reoperations. At 2 years follow-up the objective success rate (no vaginal prolapse ≥ grade 2 at any site) was 77 % for LSC versus 43 % for TVM ($p<0.001$). Operative time was longer in the LSC group, but inpatient stay and time to return to activities was shorter. The reoperation rate was 22 % in the TVM group compared to 5 % in the LSC group ($p=0.006$). There was no difference in symptomatic prolapse between the groups; however, patient satisfaction was higher in the LSC group ($p=0.002$). Maher et al. concluded that LSC was superior to TVM based on higher objective success and satisfaction, and lower perioperative morbidity and rate of reoperation [35]. In the authors' experience many of the mesh procedures that are designed to repair anterior vaginal wall prolapse are not as successful at reducing apical prolapse and when apical prolapse is present a concomitant sacrospinous or uterosacral suspension may be important to prevent apical recurrence.

Summary

There are many options for apical repair including restorative, compensatory, or obliterative, transvaginal versus transabdominal, concomitant hysterectomy versus uterine sparing, and open versus endoscopic repairs. The literature demonstrates that abdominal sacrocolpopexy has better anatomic results than vaginal sacrospinous ligament fixation. However, subjective success rates are similar. Robotic and laparoscopic ASC is gaining popularity as patients seek minimally invasive surgery. Women with predominantly anterior and vault prolapse may benefit more from an abdominal sacrocolpopexy, whereas women with predominantly posterior and vault prolapse may benefit from vaginal sacrospinous or uterosacral colpopexy. For elderly women who no longer desire to be sexually active, colpocleisis is a suitable repair option. There is no gold standard repair; the surgical approach must be customized to each patient.

Key Points

- The procedures for apical repair are restorative, compensatory, or obliterative.
- Rate of recurrent anatomic vault prolapse is lower for abdominal sacrocolpopexy compared to vaginal sacrospinous ligament fixation, but symptomatic outcomes are similar.
- Colpocleisis is the least invasive repair option and has an 85–100 % cure rate, but is only feasible for women who no longer desire to be sexually active and have very significant prolapse.

Conclusion

Transvaginal prolapse repair should be tailored to the individual patient, making sure to factor in the degree of prolapse, risk for recurrence, and patient expectations. More prospective, randomized trials with longer follow-up are needed to determine the gold standard of care.

References

1. Swift SE. The distribution of pelvic organ support in a population of female subjects seen for routine gynecologic health care. Am J Obstet Gynecol. 2000;183(2):277–85.
2. Marchese K. Improving evidence-based practice: use of the POP-Q system for the assessment of pelvic organ prolapse. Urol Nurs. 2009;29(4):216–23.
3. Jelovsek JE, Maher C, Barber MD. Pelvic organ prolapse. Lancet. 2007;369(9566):1027–38.
4. Machin SE, Mukhopadhyay S. Pelvic organ prolapse: review of the aetiology, presentation, diagnosis and management. Menopause Int. 2011;17(4):132–6.
5. Dietz HP. Why pelvic floor surgeons should utilize ultrasound imaging. Ultrasound Obstet Gynecol. 2006;28(5):629–34.
6. Colaiacomo MC, Masselli G, Polettini E, Lanciotti S, Casciani E, Bertini L, et al. Dynamic MR imaging of the pelvic floor: a pictorial review. Radiographics. 2009;29(3):e35.

7. Wolf Jr JS, Bennett CJ, Dmochowski RR, Hollenbeck BK, Pearle MS, Schaeffer AJ. Best practice policy statement on urologic surgery antimicrobial prophylaxis. J Urol. 2008;179(4):1379–90.
8. M Muffly T, Barber MD. Insertion and removal of vaginal mesh for pelvic organ prolapse. Clin Obstet Gynecol. 2010;53(1):99–114.
9. Abraham N, Goldman HB. Surgical techniques for pelvic floor reconstruction: review of the recent literature. Minerva Ginecol. 2013;65(1):29–39.
10. Weber AM, Walters MD, Piedmonte MR, Ballard LA. Anterior colporrhaphy: a randomized trial of three surgical techniques. Am J Obstet Gynecol. 2001;185(6):1299–304. discussion 304–6.
11. Chmielewski L, Walters MD, Weber AM, Barber MD. Reanalysis of a randomized trial of 3 techniques of anterior colporrhaphy using clinically relevant definitions of success. Am J Obstet Gynecol. 2011;205(1): 69 e1–8.
12. Jacquetin B. Traditional native tissue vs mesh-augmented pelvic organ prolapse repairs: providing an accurate interpretation of current literature. Comment. Int Urogynecol J. 2013;24(1):181–2.
13. Carey M, Higgs P, Goh J, Lim J, Leong A, Krause H, et al. Vaginal repair with mesh versus colporrhaphy for prolapse: a randomised controlled trial. BJOG. 2009;116(10):1380–6.
14. Sivaslioglu AA, Unlubilgin E, Dolen I. A randomized comparison of polypropylene mesh surgery with site-specific surgery in the treatment of cystocoele. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(4): 467–71.
15. Nguyen JN, Burchette RJ. Outcome after anterior vaginal prolapse repair: a randomized controlled trial. Obstet Gynecol. 2008;111(4):891–8.
16. Vollebregt A, Fischer K, Gietelink D, van der Vaart CH. Primary surgical repair of anterior vaginal prolapse: a randomised trial comparing anatomical and functional outcome between anterior colporrhaphy and trocar-guided transobturator anterior mesh. BJOG. 2011;118(12):1518–27.
17. Nieminen K, Hiltunen R, Takala T, Heiskanen E, Merikari M, Niemi K, et al. Outcomes after anterior vaginal wall repair with mesh: a randomized, controlled trial with a 3 year follow-up. Am J Obstet Gynecol. 2007;203(3):235 e1–8.
18. Altman D, Vayrynen T, Engh ME, Axelsen S, Falconer C. Anterior colporrhaphy versus transvaginal mesh for pelvic-organ prolapse. N Engl J Med. 2011; 364(19):1826–36.
19. Maher CM, Feiner B, Baessler K, Glazener CM. Surgical management of pelvic organ prolapse in women: the updated summary version Cochrane review. Int Urogynecol J. 2011;22(11):1445–57.
20. Dahlgren E, Kjolhede P. Long-term outcome of porcine skin graft in surgical treatment of recurrent pelvic organ prolapse. An open randomized controlled multicenter study. Acta Obstet Gynecol Scand. 2011;90(12):1393–401.
21. Menefee S, Dyer K, Lukacz E, et al. Colporrhaphy compared with mesh or graft-reinforced vaginal paravaginal repair for anterior vaginal wall prolapse: a randomized controlled trial. Obstet Gynecol. 2011;118(6):1337–44.
22. FDA. FDA Safety Communication: UPDATE on serious complications associated with transvaginal placement of surgical mesh for pelvic organ prolapse; 2011.
23. Mirando K. Couple wins landmark vaginal mesh lawsuit settlement. Read more at http://www.topclassactions.com/lawsuit-settlements/prescription/2173-couple-wins-landmark-vaginal-mesh-lawsuit-settlement#2646hbzG03bldsfY.99; July 24, 2012.
24. Salvatore S, Athanasiou S, Digesu GA, Soligo M, Sotiropoulou M, Serati M, et al. Identification of risk factors for genital prolapse recurrence. Neurourol Urodyn. 2009;28(4):301–4.
25. Goldman HB, Fitzgerald MP. Transvaginal mesh for cystocele repair. J Urol. 2010;183(2):430–2.
26. Goldman H, Vasavada S, editors. Female urology: a practical clinical guide. New York: Humana Press; 2007.
27. Sung VW, Rardin CR, Raker CA, Lasala CA, Myers DL. Porcine subintestinal submucosal graft augmentation for rectocele repair: a randomized controlled trial. Obstet Gynecol. 2012;119(1):125–33.
28. Paraiso MF, Barber MD, Muir TW, Walters MD. Rectocele repair: a randomized trial of three surgical techniques including graft augmentation. Am J Obstet Gynecol. 2006;195(6):1762–71.
29. Withagen MI, Milani AL, den Boon J, Vervest HA, Vierhout ME. Trocar-guided mesh compared with conventional vaginal repair in recurrent prolapse: a randomized controlled trial. Obstet Gynecol. 2011;117(2 Pt 1):242–50.
30. Sokol AI, Iglesia CB, Kudish BI, Gutman RE, Shveiky D, Bercik R, et al. One-year objective and functional outcomes of a randomized clinical trial of vaginal mesh for prolapse. Am J Obstet Gynecol. 2012;206(1):86 e1–9.
31. Marks BK, Goldman HB. What is the gold standard for posterior vaginal wall prolapse repair: mesh or native tissue? Curr Urol Rep. 2012;13(3):216–21.
32. Soules K, Winters JC, Chermansky CJ. Central compartment and apical defect repair using synthetic mesh. Curr Urol Rep. 2012;13(3):222–30.
33. Lopes ED, Lemos NL, Carramao Sda S, Lunardelli JL, Ruano JM, Aoki T, et al. Transvaginal polypropylene mesh versus sacrospinous ligament fixation for the treatment of uterine prolapse: 1-year follow-up of a randomized controlled trial. Int Urogynecol J. 2010;21(4):389–94.
34. Maher CF, Qatawneh AM, Dwyer PL, Carey MP, Cornish A, Schluter PJ. Abdominal sacral colpopexy or vaginal sacrospinous colpopexy for vaginal vault prolapse: a prospective randomized study. Am J Obstet Gynecol. 2004;190(1):20–6.
35. Maher CF, Feiner B, DeCuyper EM, Nichlos CJ, Hickey KV, O'Rourke P. Laparoscopic sacral colpopexy versus total vaginal mesh for vaginal vault prolapse: a randomized trial. Am J Obstet Gynecol. 2011;204(4):360 e1–7.

Obliterative Vaginal Procedures

6

Karen Soules, Ryan M. Krlin, and Jack Winters

Background

Pelvic organ prolapse (POP) is a common condition among women, and its prevalence increases with age [1–3]. Approximately 4.1 % of women age 80 years or older have symptomatic POP [3], and an estimated 11.1 % of women will undergo at least one surgery for POP repair or stress urinary incontinence by 80 years of age [4].

As the population of older women expands, there will be increasing numbers of patients suffering from and seeking care for POP. U.S. Census Bureau estimates indicate that starting in 2056, the population, age 65 and over, will outnumber the population under age 18 [5]. Additionally, Census calculations project that the population age 65 and older will double between 2012 and 2060, from 43.1 million to 92.0 million [5]. Using population projections and age-specific prevalence of POP, Wu et al. [6] estimated that between 2010 and 2050 the number of women with POP will increase 46 % from 3.3 to 4.9 million. In a second study, Wu et al. predict that between 2010 and 2050 there will be a 47 % increase in women undergoing procedures for POP (166,000 in 2010 and 245,970 in 2050) [7].

K. Soules, M.D. • R.M. Krlin, M.D.
J. Winters, M.D. (✉)
Department of Urology, Louisiana State University Health Sciences Center, New Orleans, LA, USA
e-mail: karensoules@gmail.com; Rkrli1@lsuhsc.edu; cwinte@lsuhsc.edu

Surgical repair of POP is challenging and has been fraught with a high reoperation rate of up to 29 % [4]. Pelvic tissues that are either weakened or damaged are thought to predispose some women to failure. The mean time to first reoperation for recurrent prolapse after primary surgical correction has been reported to be between 3 and 4 years [8, 9]. Each additional repair appears to be less successful, with the time between surgeries decreasing with each successive repair [4]. Johnson et al. looked at patient reported outcomes and found a high rate of early recurrence with 35.4 % of patients experiencing recurrent prolapse within 3 months of a primary surgical repair. Furthermore, they found a much higher overall recurrence rate of 64.6 %, with 30 % of patients not reporting recurrences to their primary surgeon [10]. This low reporting rate could account for an underestimation of failure rates in any given physician's practice.

Colpocleisis is simply a closure of the vagina with reduction of the prolapse back into the pelvis. Replacement of the pelvic organs into their anatomic position allows for the relief of the symptoms caused by POP. Closure of the vagina is permanent and it precludes future vaginal intercourse, a point which should be stressed to the patient. Obliteration may be performed in the setting of a prior hysterectomy or with the uterus left intact. The LeFort modification of the procedure is utilized when leaving the uterus in situ. Additionally, hysterectomy may be performed concurrently with colpocleisis in those patients

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_6,

that require removal of the uterus and/or cervix. Removal of the vaginal epithelium followed by apposition of the anterior and posterior fibromuscularis layers achieves obliteration of the vaginal space.

Colpocleisis is an effective and durable procedure for the treatment of POP. Anatomical success rates range from 97 to 100 % in most series [11–18]. Patients with symptomatic POP commonly experience other pelvic floor symptoms including lower urinary tract symptoms, incomplete bladder emptying, and various bowel complaints. Significant improvements have been seen in these additional domains in several studies.

Hullfish et al. looked at symptom relief via postsurgical attainment of patient goals that were set preoperatively. In this format, 91 % of patients reported improvement of urinary urgency and frequency following colpocleisis [19]. In a series of 324 women who underwent colpocleisis, Zebede et al. reported preoperative urgency symptoms in 54 % of patients. Following surgical repair, there was a statistically significant reduction in these urgency symptoms by 50 % ($p < 0.001$) [18].

Again, looking at postoperative attainment of patient goals, Hullfish et al. found a 76.4 % subjective improvement in bladder emptying following colpocleisis [19]. In a cohort of women with POP and a postvoid residual (PVR) greater than 100 cm^3, Fitzgerald et al. reported 89 % resolution of incomplete bladder emptying after surgical prolapse repair [20]. Similarly, in a series of 64 women who underwent colpocleisis, 36 % had an elevated preoperative PVR volumes all of which normalized postoperatively [15].

The resolution of bowel symptoms is equally encouraging. In a prospective study by Gutman et al, bothersome bowel symptoms resolved in the majority of patients after colpocleisis. Specifically, all obstructive symptoms (digital assistance, straining, incomplete emptying) and the majority of incontinence symptoms [anal (fecal) incontinence with stress and urge, anal incontinence of flatus and liquid stool] were significantly decreased 1 year after surgery [21]. Likewise, in their large case series, Zebede et al. found a significant resolution of bowel symptoms including: constipation, obstructed defecation, and fecal incontinence [18].

Patients report a high rate of satisfaction after colpocleisis ranging from 90.3 to 100 % [12, 14, 17, 18, 22–24]. Barber et al. reported that patients had significant improvements in multiple quality of life measures including: bodily pain, vitality, social functioning, and mental health measures [22]. Also, in this study of women 65 or older with stage 3 or 4 prolapse, there were no differences found between the reconstructive and obliterative groups as both demonstrated significant improvements in health-related quality of life [22]. Correspondingly, Murphy et al. also found that quality of life and patient satisfaction was similar between groups of women who had reconstructive versus obliterative prolapse repairs [25].

Rates of regret following colpocleisis are low typically ranging from 3 to 9 % [19, 26, 27]. In a series by von Pechmann et al. a higher rate of regret (12.9 %) was reported; however, half of those patients also stated that they would have the surgery again. There are concerns that closure of the vagina may negatively affect a patient's body image, but most patients report improved body image following surgery [17, 19, 28]. In their series of 40 patients with self-created goals, Hullfish et al. found a 96.9 % improvement in self-image after surgery [19]. Utilizing questions regarding body image and perception, Koski et al. found that 50 % of patients felt their body looked better after colpocleisis and 82 % reported their body felt better after the procedure [17].

In the carefully selected patient, these results demonstrate that an obliterative procedure remains a particularly good option following a thorough informed discussion.

Evaluation/Work-Up

Preoperative evaluation for colpocleisis should include a thorough history of the prolapse complaint including prior reconstructive procedures and associated pelvic floor symptoms. Details should be obtained regarding pain and pressure symptoms, urinary incontinence, voiding dysfunction, fecal incontinence, and defecatory

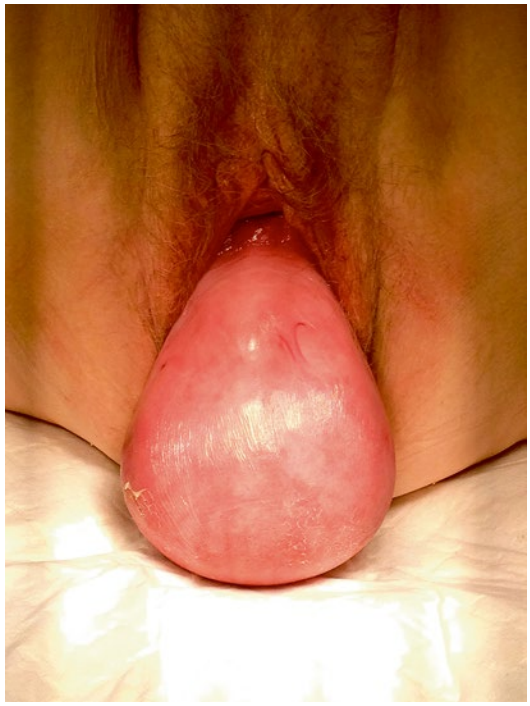


Fig. 6.1 Stage 4 vaginal vault prolapse

dysfunction. A detailed vaginal exam, both bimanual and speculum, is required with evaluation of all compartments. A quantitative scoring of the prolapse, assessment of uterine size when applicable, measurement of postvoid residual, and assessment of urine for infection and hematuria should be included. Colpocleisis is most easily completed in patients with Stage 3 or greater prolapse (Fig. 6.1). In patients with less severe support defects the dissection required may be more challenging.

Some type of preoperative evaluation for stress urinary incontinence (SUI), even in patients that report continence, is recommended due to the high rate of occult SUI in women with POP. The rate of occult stress urinary incontinence in the setting of Stage 2 prolapse or greater ranges from 33.5 to 67.9 % [18, 29–32]. A simple cough stress test with a full bladder and the prolapse reduced is often sufficient in patients with uncomplicated, demonstrable SUI. Patients with voiding dysfunction, mixed incontinence, incomplete bladder emptying, or prior urologic surgery should undergo a more thorough investigation with urodynamics.

The data is varied and the true predictive value of preoperative urodynamics remains unclear. Reena et al. studied women both before and after they underwent prolapse repairs without anti-incontinence procedures and found that 64.2 % of patients with documented occult SUI also demonstrated SUI postop [30]. In a small series of patients, Chaikin et al. reported that no patients with negative preoperative testing developed postoperative SUI [29]. Similarly, Hafidh et al. found a very low rate of postoperative SUI (4 %) in patients with no SUI demonstrated on preoperative urodynamics [33]. In contrast, studies by Wei and Al-Mandeel found a high incidence of postoperative SUI, 38 % and 42 % respectively, in patients with preoperative testing that was negative for SUI [32, 34]. What is clear, however, is that it is reasonable to place a midurethral sling at the time of prolapse repair in women with clinical SUI or documented occult SUI. In a 100 women with occult SUI who underwent TVT, Croutz et al. report a 83 % success rate for absence of postoperative SUI and only 2 % of patients with persistent SUI were symptomatic [35]. Meschia et al. also reported high rates of postoperative continence (objective 92 %, subjective 96 %) in patients who underwent TVT placement for occult SUI [36].

It remains controversial whether to place a sling in patients without clinical SUI or documented occult stress incontinence. The large, randomized CARE trial looked at women who were stress continent preoperatively and found decreased rates of postoperative SUI in women who underwent prophylactic Burch procedure at the time of sacrocolpopexy versus those who did not (32 % versus 45.2 %) [37]. However, midurethral slings are the most common anti-incontinence procedures performed, and it is unclear if this data can be extrapolated to colpocleisis and midurethral slings. In another large, randomized trial Wei et al. also looked at stress continent women undergoing prolapse repair and randomized patients to sling versus no sling. They also found a significantly lower rate of urinary incontinence in the sling patients

(27.3 % versus 43 %), but at the expense of increased adverse advents including bladder perforation, urinary tract infection, major bleeding complications, and incomplete bladder emptying [32]. Another argument for prophylactic sling placement at the time of colpocleisis is the issue of access to the suburethral area. Successful colpocleisis is dependent on aggressive closure of the genital hiatus with levator placation [43]. Depending on the degree of closure, this can make it very challenging to access the midurethra for future placement of a sling.

Management of the Uterus

In women with a uterus it is prudent to confirm that there is no cervical or endometrial pathology which would be a contraindication to leaving the uterus in situ. Closure of the vagina will severely limit the ability to perform future surveillance via the traditional routes (pap smear, endometrial biopsy). A complete history should be taken regarding any history of abnormal pap smears as well as any episodes of postmenopausal bleeding. Benign cervical cytology should be documented in a patient with a history of any abnormal pap smears or a previous treatment for cervical intraepithelial neoplasia (CIN). The most recent guidelines from the American College of Obstetricians and Gynecologists (ACOG) recommend that women with a history of CIN2, CIN3, or adenocarcinoma in situ should have 20 years of negative screening following treatment prior to discontinuation of cervical cancer screening [38]. Therefore, it is recommended that any woman who would need continued surveillance based on her history should have a hysterectomy at the time of colpocleisis.

Women with a history of endometrial hyperplasia or any episodes of postmenopausal bleeding should have a preoperative assessment of the endometrium. This can be accomplished with endometrial sampling via endometrial biopsy or dilation and curettage of the uterus. Alternatively, the least invasive approach is to evaluate the endometrial thickness via transvaginal ultrasound. In women with postmenopausal bleeding, endometrial sampling is not required if an endometrial thickness of less than or equal to 4 mm is found on transvaginal ultrasound [39]. The decision to screen asymptomatic women with transvaginal ultrasound for assessment of the endometrial thickness may be left to the discretion of the surgeon. As reported by ACOG, the significance of an endometrial thickness greater than 4 mm in a postmenopausal woman without bleeding has not been established and does not routinely need evaluation in the absence of risk factors [39]. Concurrent hysterectomy is recommended for women with the finding of endometrial hyperplasia. Patients with the diagnosis of atypical endometrial hyperplasia should be referred to a gynecologic oncologist for surgical management due to the high rate (42.6 % [40]) of concurrent carcinoma.

It is important to note that hysterectomy with concurrent colpocleisis does not improve success rates over colpocleisis alone [14, 41], and the combination of procedures may significantly increased blood loss and patient transfusion requirements [14]. Due to this increased morbidity, exceptions to the above recommendations may be reasonable in patients who are of advanced age or debilitated and should be a joint decision between the patient and the surgeon.

Because colpocleisis eliminates the possibility of future vaginal intercourse, preoperative counseling is extremely important and patient selection is key. There is no identified minimum age requirement for consideration of the procedure. With colpocleisis, as in all cases of prolapse repair and reconstruction, the treatment plan must be individualized for each patient. Preoperative counseling should be specific and thorough including information on potential pessary management, alternative options for repair, possibility of postoperative urinary incontinence, and recurrence risk.

The option for concurrent midurethral sling placement should also be discussed with patients. Specifically, in the situation of demonstrated SUI in the setting of incomplete bladder emptying as well as patients with no preoperative urinary incontinence. The addition of a midurethral sling does not appear to cause a high risk of urinary retention and preoperative incomplete bladder emptying seems to resolve in most patients [31, 42].

In a series of 38 women who underwent colpocleisis and midurethral sling placement, Abbasy et al. reported a 2 % rate of elevated PVR postoperatively. Additionally, they saw a 90 % postoperative resolution of preoperative incomplete bladder emptying (defined as PVR greater than 100 ml) [42]. In a much larger series of 210 women, Smith et al. found a de novo voiding dysfunction rate of 1.9 % in women who underwent colpocleisis and midurethral sling. Similarly, they found a 91 % resolution of preoperative incomplete emptying [31]. An alternative, nonpermanent approach is to offer periurethral bulking injections to patients for whom the risk of retention is thought to be particularly high.

The decision whether to offer a midurethral sling to continent patients at the time of colpocleisis remains controversial. As detailed previously, the risk for de novo SUI may be quite significant; however, midurethral slings are not without complications or sequela. A large randomized controlled trial by Wei et al. specifically addressed this question by randomizing women without SUI who were undergoing vaginal prolapse repair to either have a midurethral sling or sham sling incisions. The sling group had significantly decreased rates of urinary incontinence at both 3 [23.6 % versus 49.4 % ($p<0.001$)] and 12 months [27.3 % versus 43.0 % ($p=0.002$)] [32]. However, the sling group did have significantly higher rates of complications including: bladder perforation, urinary tract infection, major bleeding complications, and incomplete bladder emptying for up to 6 weeks following surgery. Also of note, 5 % of patients in the sham group had a sling placement within the first year after surgery, but only 2.4 % of patients in the sling group required sling revision for voiding dysfunction. A detailed discussion of all the possible risks and benefits should be carried out with patients when making the determination of whether to place a sling in this population.

Surgical Procedures

All patients receive a preoperative prophylactic broad-spectrum antibiotic. Additionally, all patients have DVT prophylaxis; our standard is to use compression stockings and sequential compression devices on the lower extremities. Table 6.1

For a patient in whom the uterus is to remain in situ, a LeFort colpocleisis is performed (Fig. 6.2a–e). To begin, outward traction is placed on the cervix using a tenaculum or Allis clamp. Two rectangles (anterior and posterior) are outlined with a surgical marker starting approximately 2 cm distal to the cervix and extending to the bladder neck anteriorally and mirroring this posteriorly. This will aid in maintaining orientation during removal of the vaginal epithelium. Laterally, there should be at least 2 cm of epithelium separating anterior from posterior rectangles in order to allow adequate tissue for creation of the drainage channels. Starting with the posterior wall 1 % lidocaine with a 1:200,000 dilution of epinephrine is infiltrated in to the subepithelial space to aid in hemostasis and hydrodissection. The demarcated areas are circumscribed with knife and sharp dissection is performed to start the removal of the vaginal epithelium from the underlying fibromuscularis layer. We use a number 10 blade to make the initial incisions. Dissection is initiated with tenotomy scissors for precision in finding the appropriate plane and then is completed with curved mayo scissors which are safer for combined blunt and sharp dissection. It can be helpful to refrain from making all incisions initially but rather to proceed in a systematic fashion (posterior to anterior) in order to decrease blood loss and improve visualization during dissection. Typically, a combination of sharp and blunt finger dissection with a sponge can be employed to facilitate removal of the epithelium once the appropriate plane is achieved. Hemostasis is maintained with meticulous use of monopolar cautery throughout the dissection. With the LeFort procedure only the areas of anterior and posterior rectangles are denuded.

To continue the LeFort procedure, channels are created after the removal of the epithelium and prior to starting closure of the vagina. Absorbable suture is used to tubularize the lateral strips of epithelium by suturing the epithelial edges together, superior to inferior. This may be done with an interrupted or running stitch. Our preference is to use 2-0 polyglycolic acid suture on a CT2 needle

Table 6.1 Risk classification for venous thromboembolism

Level of risk	Definition	Prevention strategies
Low	Surgery less than 30 min in patients younger than 40 years with no additional risk factors	No specific prophylaxis, early mobilization
Moderate	Surgery lasting less than 30 min in patients with additional risk factors Surgery lasting less than 30 min in patients aged 40–60 years with no additional risk factors Major surgery in patients younger than 40 years with no additional risk factors	Low-dose unfractionated heparin: (5,000 units every 12 h) OR Low molecular weight heparin: (2,500 units dalteparin or 40 mg enoxaparin daily) OR Graduated compression stockings OR Intermittent pneumatic compression device
High	Surgery lasting less than 30 min in patients older than 60 years or with additional risk factors Major surgery in patients older than 40 years or with additional risk factors	Low-dose unfractionated heparin: (5,000 units every 8 h) OR Low molecular weight heparin: (5,000 units dalteparin or 40 mg enoxaparin daily) OR Intermittent pneumatic compression device
Highest	Major surgery in patients older than 60 years plus prior venous thromboembolism, cancer, or molecular hypercoagulable state	Low-dose unfractionated heparin: (5,000 units every 8 h) OR Low molecular weight heparin: (5,000 units dalteparin or 40 mg enoxaparin daily) OR Intermittent pneumatic compression device/graduated compression stockings + low-dose unfractionated heparin or low molecular weight heparin Consider continuing prophylaxis for 2–4 weeks postop

Data from: American College of Obstetricians and Gynecologists Committee on Practice Bulletins—Gynecology. ACOG Practice Bulletin Number 84: Prevention of Deep Vein Thrombosis and Pulmonary Embolism. Obstet Gynecol. 2007 Aug;110(2Pt1):429–40; and from Geerts WH, Pineo GR, Heit JA, et al. Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004;126(suppl):338 s–400 s

and run this closure towards the cervix, thus allowing the surgeon to sew towards him/herself. These channels will allow the drainage of cervical and uterine secretions. Care should be taken to continue to identify the location of the channels throughout the rest of the procedure in order to avoid inadvertently suturing them closed.

Following creation of the channels, imbricating sutures using 2-0 polyglycolic acid on CT-2 needle are placed in the fibromuscularis to begin reduction of the prolapse. Successive anterior to posterior imbricating sutures (Fig. 6.3a, b) in either an interrupted or figure-of-eight fashion are the most effective when reducing the epithelialized cervix. Once the cervix has been fully reduced, it is usually most straightforward to continue with anterior to posterior imbrication until the prolapse has been reduced to the level of the levator plate. Cystoscopy is then carried out following administration of Indigo Carmine to ensure ureteral efflux. From this point onward the procedure is completed with a levator plication and perineorrhaphy, using 2-0 and 3-0 polyglycolic acid, respectively, in the same fashion as a complete colpocleisis is performed without the uterus in situ.

As addressed already, concomitant hysterectomy should be performed for patients with a contraindication to retention of the uterus. This combined procedure can have increased morbidity due to inherent risk of entry into the peritoneal cavity, increased operative time, and increased blood loss. Following vaginal hysterectomy, the cuff should be closed to protect the intraperitoneal structures at which point removal of the epithelium is then started.

In the patient with a prior hysterectomy, a complete colpocleisis requires removal of the

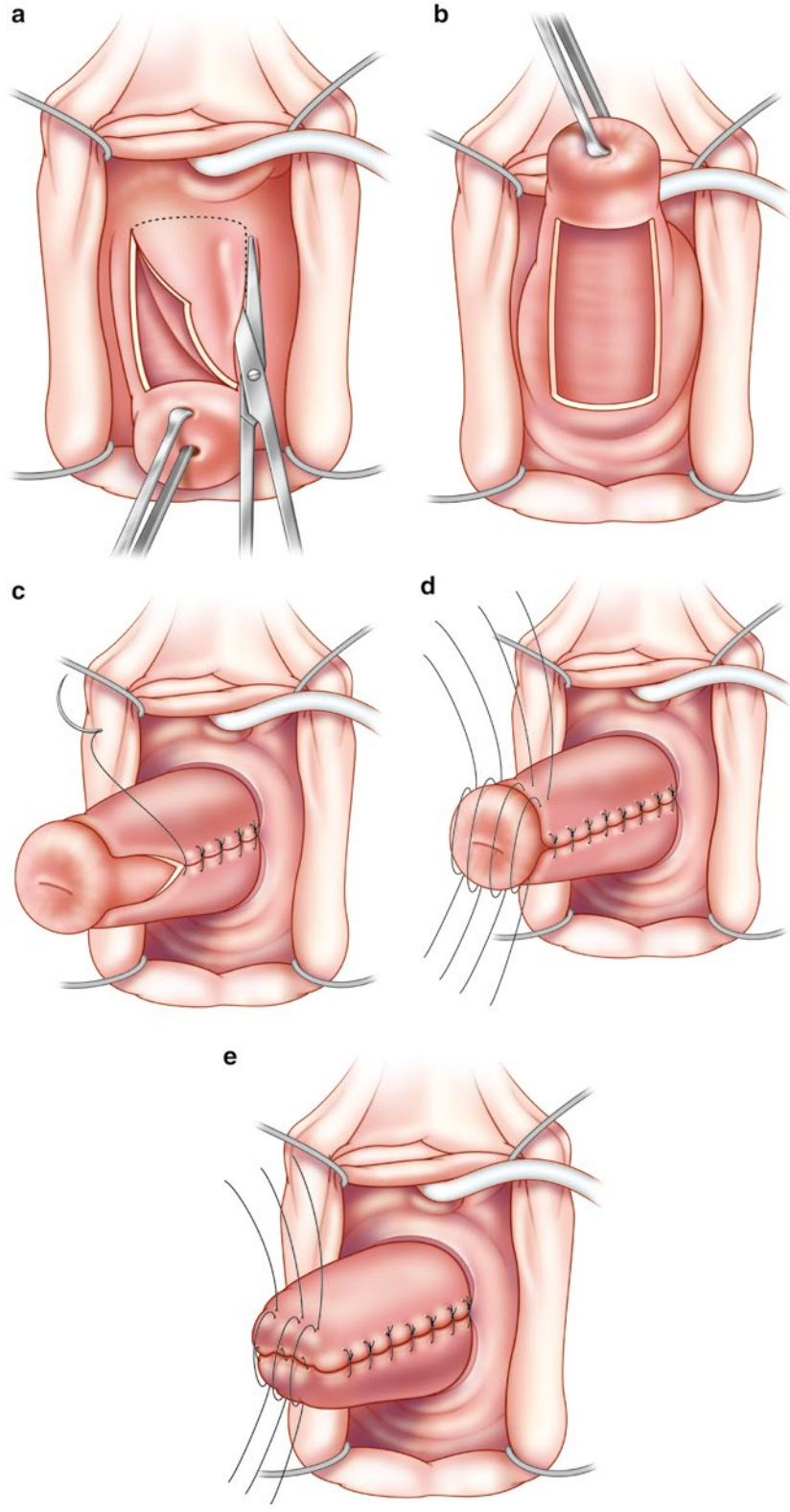


Fig. 6.2 LeFort colpocleisis. (**a**) Removal of the anterior rectangle of vaginal epithelium. (**b**) After removal of the posterior rectangle at least 2 cm of epithelium should remain laterally separating the denuded anterior and posterior rectangles. (**c**) Creation of the drainage channels: an interrupted or running stitch is used to tubularize the remaining, lateral strips of epithelium. (**d**) Anterior to posterior imbricating sutures to reduce the epithelialized cervix. (**e**) Further reduction of the uterus with continued anterior to posterior imbrications

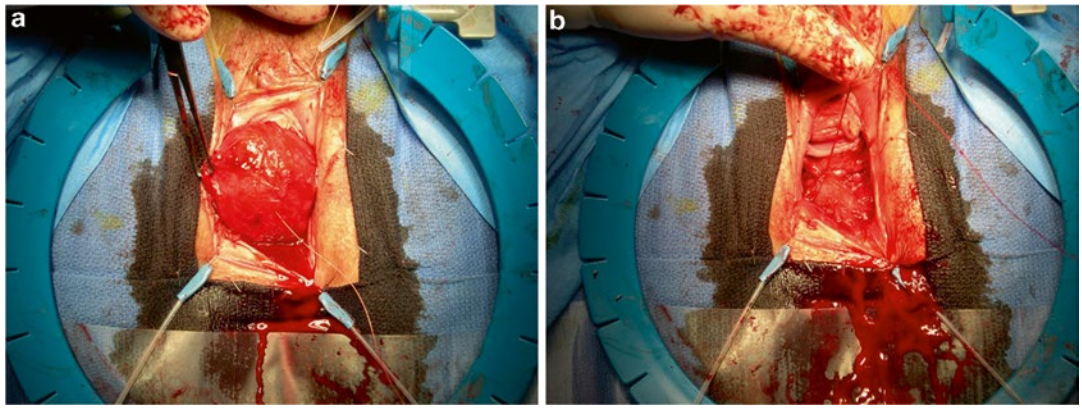


Fig. 6.3 (**a**) Placement of stitch for anterior to posterior imbrications. (**b**) Reduction of the prolapse after performing several anterior to posterior imbricating sutures

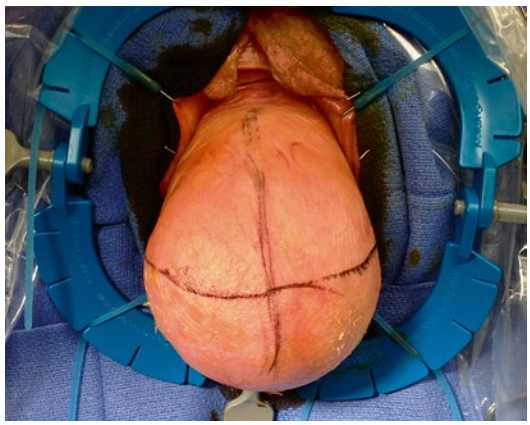


Fig. 6.4 Demarcated quadrants for dissection

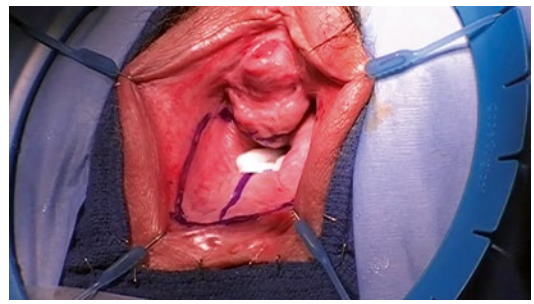


Fig. 6.5 Boundaries of perineal dissection

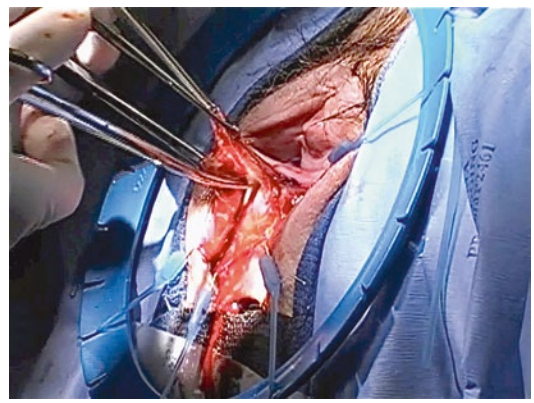


Fig. 6.6 Posterior dissection of vaginal epithelium from fibromuscularis

entire vaginal epithelium. A surgical marker is used to outline the lateral borders of dissection along the perineum, vaginal sidewalls, and anterior vaginal wall proximal to the urethra. This may be further demarcated into quadrants in order to aid in maintaining orientation, which can be easily lost, during dissection with severe POP (Figs. 6.4 and 6.5). Injection of 1 % lidocaine with a 1:200,000 dilution of epinephrine into the subepithelial space may be utilized to aid in hemostasis and hydrodissection. The demarcated areas are circumscribed with a knife and sharp dissection is used to initiate removal of the vaginal epithelium from the underlying fibromuscularis. Similar to the Lefort, it is best to proceed in a systematic fashion in order to maintain orientation, decrease blood loss, and preserve visualization. The authors would recommend posterior to anterior (Fig. 6.6). Again, once the appropriate plane is entered, a combination of sharp and blunt

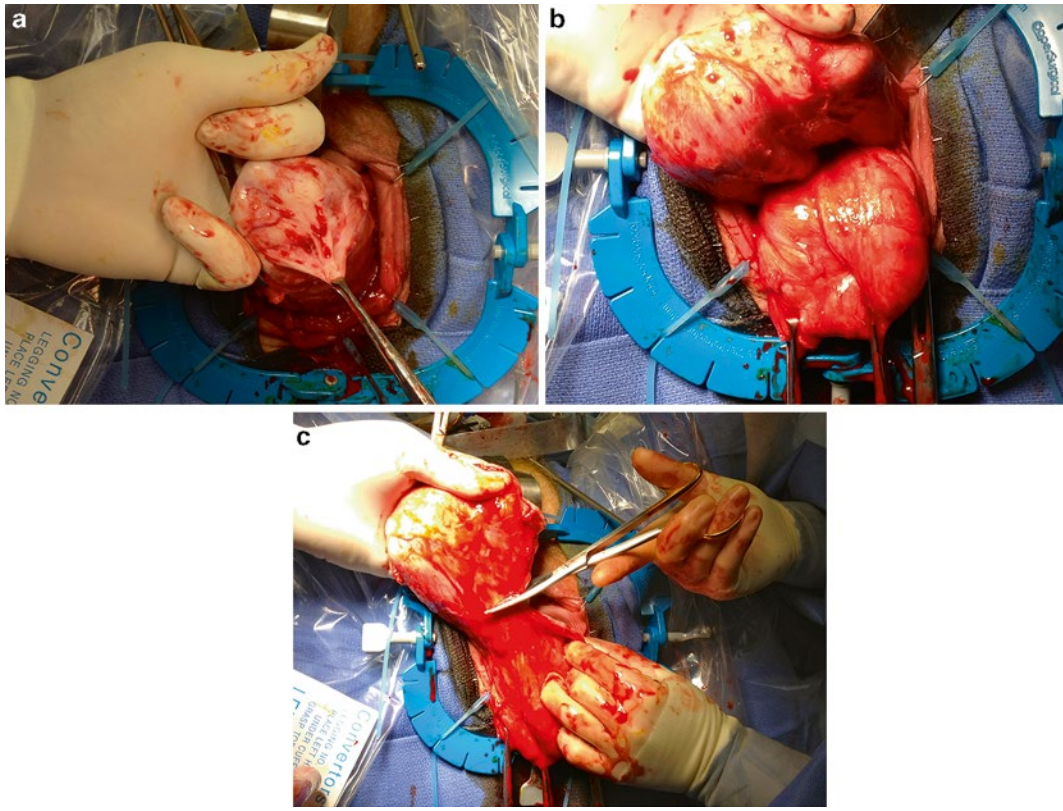


Fig. 6.7 (**a**–**c**) Dissection and removal of vaginal epithelium

dissection can be used to separate the epithelium from the fibromuscularis layer (Fig. 6.7a–c). Attention should be given to maintaining hemostasis throughout the dissection with judicious use of the monopolar cautery. Significant blood loss can be encountered when performing extensive dissection on severe POP, so all efforts towards hemostasis will help to decrease the need for transfusion.

It is not uncommon to encounter an enterocele during removal of the vaginal epithelium. An attempt should be made to avoid entering the enterocele. However, these dissections can be challenging and with some severe defects there may be peritoneum directly abutting vaginal epithelium. If an enterocele is entered, the sac should be meticulously mobilized circumferentially from the surrounding tissue with special care taken to avoid small bowel injury. The enterocele sac should then be tied off using 3-0 polyglycolic acid on a SH needle and a circular purse-string stitch. For large defects in the peritoneum, 2–3 full purse-string sutures are required to ensure adequate closure. If there is an excessive amount of redundant, prolapsing enterocele sac, the peritoneum can be trimmed circumferentially for a more proximal and effective closure.

Following removal of the vaginal epithelium, reduction of the prolapse can be performed with one of two techniques or a combination of both. One option is to use successive anterior to posterior imbricating sutures in either an interrupted or figure-of-eight fashion. Alternatively, sequential, circular purse-string stitches are an effective technique for reduction of the prolapse (Fig. 6.8). The authors favor using 2-0 polyglycolic acid suture on a CT2.

Several centimeters of vaginal epithelium should be retained on the distal, anterior vaginal wall underneath the urethra. This is recommended

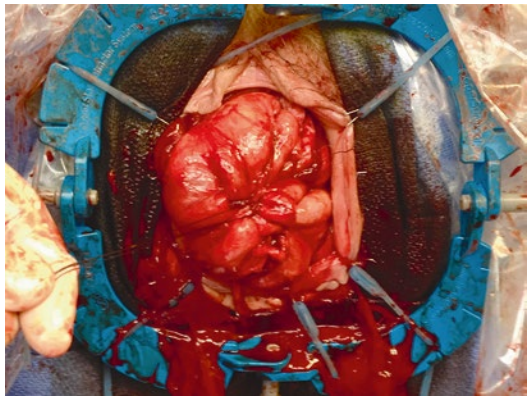


Fig. 6.8 Purse-string reduction of the prolapse

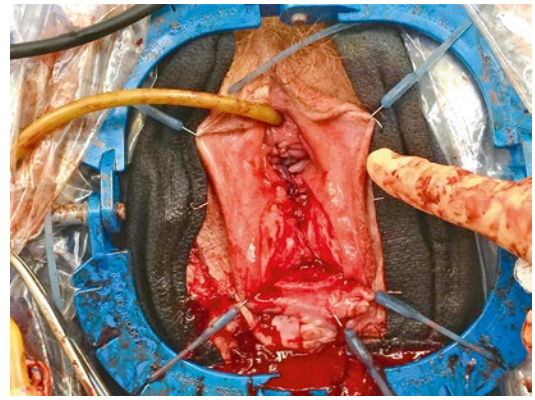


Fig. 6.9 Completed levator placation

for all patients whether they are having a concomitant sling placement or not. Maintenance of this distal epithelium prevents excessive traction on the urethra, which can predispose the patient to postoperative incontinence and leaves room for immediate or future sling placement. Placement of a midurethral sling is most easily achieved after the prolapse has been reduced to or above the levator plate and before levator plication.

Cystoscopy with IV indigo carmine administration is performed at this point to rule out bladder injury and ureteral obstruction. If ureteral obstruction is diagnosed on cystoscopy, a prudent first step is to remove the anterolateral sutures as this is often the location where the ureters are encountered. Cystoscopy is then repeated.

Next a levator plication is performed close to the genital hiatus and buttress the repair. Using 2-0 polyglycolic acid suture on a CT 2, interrupted or figure-of-eight sutures are performed pulling the muscles together in the midline (Fig. 6.9). Initially, excessively lateral bites of tissue can cause undue tension and may make it difficult to achieve approximation in the midline. Following levator plication, the perineorrhaphy should include reapproximation of the transverse perineal and bulbocavernosus muscles at the introitus. This is also most easily achieved using 2-0 polyglycolic acid on CT-2 needle so that large, secure bites can be taken. Finally, the vaginal epithelium is reapproximated with 3-0 polyglycolic acid on a SH needle in 1–2 layers with a subcutaneous and a subcuticular stitch or a running through-and-through stitch (Fig. 6.10).

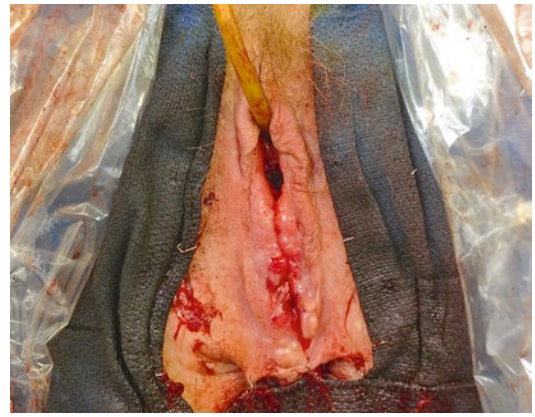


Fig. 6.10 Completion of perineorrhaphy

Summary

Colpocleisis is a successful operation with few complications and little regret for patients postoperatively [43]. SUI should be evaluated preoperatively, but may warrant postoperative reassessment based on patient symptoms. Urgency urinary incontinence after these surgeries can be problematic and may require additional medical treatment. Overall, the procedure is a very attractive option in the properly selected patient.

References

1. Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford Family Planning Association study. Br J Obstet Gynaecol. 1997;104(5):579–85.

2. Hendrix SL, Clark A, Nygaard I, Aragaki A, Barnabei V, McTiernan A. Pelvic organ prolapse in the women's health initiative: gravity and gravidity. Am J Obstet Gynecol. 2002;186(6):1160–6.
3. Nygaard I, Barber MD, Burgio KL, et al. Prevalence of symptomatic pelvic floor disorders in US women. JAMA. 2008;300(11):1311–6.
4. Olsen AL, Smith VJ, Bergstrom JO, et al. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol. 1997;89(4):501–6.
5. U.S. Census Bureau Projections Show a Slower Growing, Older, More Diverse Nation a Half Century from Now. Available at: http://www.census.gov/newsroom/releases/archives/population/cb12-243.html. Accessed 6 April 2013.
6. Wu JM, Hundley AF, Fulton RG, Myers ER. Forecasting the prevalence of pelvic floor disorders in U.S. Women: 2010 to 2050. Obstet Gynecol. 2009;114(6):1278–83.
7. Wu JM, Kawasaki A, Hundley AF, et al. Predicting the number of women who will undergo incontinence and prolapse surgery, 2010 to 2050. Am J Obstet Gynecol. 2011;205(3):230.e1–5.
8. Price N, Slack A, Jwarah E, Jackson S. The incidence of reoperation for surgically treated pelvic organ prolapse: an 11-year experience. Menopause Int. 2008; 14(4):145–8.
9. Fialkow MF, Newton KM, Weiss NS. Incidence of recurrent pelvic organ prolapse 10 years following primary surgical management: a retrospective cohort study. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(11):1483–7.
10. Johnson P, Larson KA, Hsu Y, et al. Self-reported natural history of recurrent prolapse among women presenting to a tertiary care center. Int J Gynaecol Obstet. 2013;120(1):53–6.
11. DeLancey JO, Morley GW. Total colpocleisis for vaginal eversion. Am J Obstet Gynecol. 1997;176(6): 1228–32.
12. Cespedes RD, Winters JC, Ferguson KH. Colpocleisis for the treatment of vaginal vault prolapse. Tech Urol. 2001;7(2):152–60.
13. Harmanli OH, Dandolu V, Chatwani AJ, Grody MT. Total colpocleisis for severe pelvic organ prolapse. J Reprod Med. 2003;48(9):703–6.
14. von Pechmann WS, Mutone M, Fyffe J, Hale DS. Total colpocleisis with high levator plication for the treatment of advanced pelvic organ prolapse. Am J Obstet Gynecol. 2003;189(1):121–6.
15. FitzGerald MP, Brubaker L. Colpocleisis and urinary incontinence. Am J Obstet Gynecol. 2003;189(5): 1241–4.
16. Misrai V, Gosseine PN, Costa P, et al. Colpocleisis: indications, technique and results. Prog Urol. 2009; 19(13):1031–6.
17. Koski ME, Chow D, Bedestani A, et al. Colpocleisis for advanced pelvic organ prolapse. Urology. 2012; 80(3):542–6.
18. Zebede S, Smith AL, Plowright LN, et al. Obliterative LeFort colpocleisis in a large group of elderly women. Obstet Gynecol. 2013;121(2 Pt 1): 279–84.
19. Hullfish KL, Bovbjerg VE, Steers WD. Colpocleisis for pelvic organ prolapse: patient goals, quality of life, and satisfaction. Obstet Gynecol. 2007;110(2 Pt 1):341–5.
20. Fitzgerald MP, Kulkarni N, Fenner D. Postoperative resolution of urinary retention in patients with advanced pelvic organ prolapse. Am J Obstet Gynecol. 2000;183(6):1361–3. discussion 1363–4.
21. Gutman RE, Bradley CS, Ye W, et al. Effects of colpocleisis on bowel symptoms among women with severe pelvic organ prolapse. Int Urogynecol J. 2010;21(4):461–6.
22. Barber MD, Amundsen CL, Paraiso MF, et al. Quality of life after surgery for genital prolapse in elderly women: obliterative and reconstructive surgery. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18(7):799–806.
23. Fitzgerald MP, Richter HE, Bradley CS, et al. Pelvic support, pelvic symptoms, and patient satisfaction after colpocleisis. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(12):1603–9.
24. Lu YX, Hu ML, Wang WY, et al. Colpocleisis in elderly patients with severe pelvic organ prolapse. Zhonghua Fu Chan Ke Za Zhi. 2010;45(5):331–7.
25. Murphy M, Sternschuss G, Haff R, et al. Quality of life and surgical satisfaction after vaginal reconstructive vs obliterative surgery for the treatment of advanced pelvic organ prolapse. Am J Obstet Gynecol. 2008;198(5):573.e1–7.
26. FitzGerald MP, Richter HE, Siddique S, et al. Colpocleisis: a review. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17(3):261–71.
27. Wheeler 2nd TL, Richter HE, Burgio KL, et al. Regret, satisfaction, and symptom improvement: analysis of the impact of partial colpocleisis for the management of severe pelvic organ prolapse. Am J Obstet Gynecol. 2005;193(6):2067–70.
28. Zhang YH, Lu YX, Liu X, et al. Impact of colpocleisis on body image in women with severe pelvic organ prolapse. Zhonghua Fu Chan Ke Za Zhi. 2011;46(6):431–4.
29. Chaikin DC, Groutz A, Blaivas JG. Predicting the need for anti-incontinence surgery in continent women undergoing repair of severe urogenital prolapse. J Urol. 2000;163(2):531–4.
30. Reena C, Kekre AN, Kekre N. Occult stress incontinence in women with pelvic organ prolapse. Int J Gynaecol Obstet. 2007;97(1):31–4.
31. Smith AL, Karp DR, Lefevre R, et al. LeFort colpocleisis and stress incontinence: weighing the risk of voiding dysfunction with sling placement. Int Urogynecol J. 2011;22(11):1357–62.
32. Wei JT, Nygaard I, Richter HE, et al. A midurethral sling to reduce incontinence after vaginal prolapse repair. N Engl J Med. 2012;366(25):2358–67.

33. Hafidh BA, Chou Q, Khalil MM, et al. De novo stress urinary incontinence after vaginal repair for pelvic organ prolapse: one-year follow-up. Eur J Obstet Gynecol Reprod Biol. 2013;168:227–30. pii: S0301-2115(13)00011-0.
34. Al-Mandeel H, Ross S, Robert M, et al. Incidence of stress urinary incontinence following vaginal repair of pelvic organ prolapse in objectively continent women. Neurourol Urodyn. 2011;30(3): 390–4.
35. Groutz A, Gold R, Pauzner D, et al. Tension-free vaginal tape (TVT) for the treatment of occult stress urinary incontinence in women undergoing prolapse repair: a prospective study of 100 consecutive cases. Neurourol Urodyn. 2004;23(7):632–5.
36. Meschia M, Pifarotti P, Spennacchio M, et al. A randomized comparison of tension-free vaginal tape and endopelvic fascia plication in women with genital prolapse and occult stress urinary incontinence. Am J Obstet Gynecol. 2004;190(3):609–13.
37. Brubaker L, Nygaard I, Richter HE, et al. Two-year outcomes after sacrocolpopexy with and without burch to prevent stress urinary incontinence. Obstet Gynecol. 2008;112(1):49–55.
38. American College of Obstetricians and Gynecologists Committee on Practice Bulletins—Gynecology. ACOG Practice Bulletin Number 131: Screening for cervical cancer. Obstet Gynecol. 2012;120(5):1222–38.
39. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 426: the role of transvaginal ultrasonography in the evaluation of postmenopausal bleeding. Obstet Gynecol. 2009;113 (2 Pt 1):462–4.
40. Trimble CL, Kauderer J, Zaino R, et al. Concurrent endometrial carcinoma in women with a biopsy diagnosis of atypical endometrial hyperplasia: a Gynecologic Oncology Group study. Cancer. 2006;106(4):812–9.
41. Hoffman MS, Cardosi RJ, Lockhart J, et al. Vaginectomy with pelvic herniorrhaphy for prolapse. Am J Obstet Gynecol. 2003;189(2):364–70.
42. Abbasy S, Lowenstein L, Pham T, et al. Urinary retention is uncommon after colpocleisis with concomitant mid-urethral sling. Int Urogynecol J Pelvic Floor Dysfunct. 2009;20(2):213–6.
43. Stepp KJ, Barber MD, Yoo EH, et al. Incidence of perioperative complications of urogynecologic surgery in elderly women. Am J Obstet Gynecol. 2005; 192(5):1630–6.

Open Transabdominal Sacrocolpopexy

7

Christopher F. Tenggardjaja
and Sandip P. Vasavada

Introduction

Pelvic organ prolapse (POP) is a common condition with an estimated 19 % lifetime risk for undergoing a surgical procedure for treatment. Although transvaginal and minimally invasive techniques have been developed, transabdominal sacrocolpopexy (ASC) is the gold standard for treatment of apical prolapse. Surgical preparation for ASC begins with a thorough history and physical examination culminating in an earnest discussion between the patient and surgeon regarding treatment options and goals of therapy. Open ASC requires the pelvic surgeon to have technical prowess in abdominal surgery, a familiarity with pelvic anatomy, and synthetic graft materials. Long-term surgical outcomes for ASC are durable and efficacious with acceptably low rates of complications and recurrence. Although ASC has been around since the 1960s in its modern form, the technique has continued to evolve and controversies such as concomitant anti-incontinence procedures are addressed.

Background

POP affects nearly 50 % of parous women and is a common finding on pelvic examination [1, 2]. Despite its high anatomic prevalence, a 3 % symptomatic prevalence was noted in the 2005–2006 National Health and Nutrition Examination Survey and other studied populations demonstrate an 11–19 % lifetime risk of undergoing surgery for treatment of POP [3–5]. Traditionally, management of the patient with POP depends on several different factors including the patient's preferences, comorbidities, and the surgeon's expertise. Treatment options vary from pelvic floor physical therapy to pessary usage to surgical correction. Surgical treatment options depend on the compartment which has prolapsed. Regarding apical prolapse, surgical treatment consists of either an obliterative or restorative approach. Patients who are no longer sexually active are candidates for an obliterative approach with a colpocleisis. For those who prefer a restorative approach, the gold standard for apical POP after hysterectomy is transabdominal sacrocolpopexy (ASC) [6].

History

Treatment of POP has been described since the time of antiquity. Succession was described by Hippocrates with the intent of reducing prolapse

C.F. Tenggardjaja, M.D. • S.P. Vasavada, M.D. (✉)
Female Pelvic Medicine and Reconstructive Surgery, Cleveland Clinic, Glickman Urologic and Kidney Institute, Cleveland, OH, USA
e-mail: ctenggardjaja@gmail.com; vasavas@ccf.org

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_7,

by hanging women upside down [7]. Pessary usage has been described since the middle ages with dipping linen and other materials into different concoctions [7]. While there are various techniques for the surgical management of apical prolapse, the gold standard of ASC was developed as a counterpart to the transvaginal techniques for addressing apical vault prolapse. Huguier and Scalin in 1958 and Lane in 1962 described using a graft to attach the vaginal cuff to the sacrum [8–10]. The S3–S4 graft placement was subsequently described by Birnbaum but was later revised to the S1–S2 level by Sutton after hemorrhage [11, 12]. Although different biologic and artificial grafts have been used in surgical correction/augmentation of POP repair, ASC continues to be the gold standard.

Patient Evaluation

All patients referred for POP undergo a complete history and physical examination at their initial visit. Quality of life and baseline symptoms are documented with various questionnaires such as the Urinary Distress Inventory (UDI) and Incontinence Impact Questionnaire (IIQ). A focused history of present illness can elicit symptoms of prolapse including obstructive emptying, bulging sensation towards the end of the day versus vaginal soreness/bleeding, and splinting with voiding or defecation. A thorough review of systems also evaluates for any urinary incontinence related to urgency or stress, defecatory issues, and/or comorbidities (such as neurologic disease) that affect continence and bladder function. Prolapse surgery remains an elective surgery that has the ability to greatly improve a patient's quality of life. A careful evaluation of medical and surgical history may change the approach or rule out surgery as an option for those patients with multiple exclusionary comorbid conditions. A vaginal approach for prolapse correction may be associated with less morbidity in a patient who has had multiple abdominal surgeries and is at risk for adhesive disease. Parity, method of delivery, and family history are taken into account as these are risk factors for POP [13–15]. For all patients, we believe it is paramount to address the patient's goals at the first visit. If they are not interested in sexual intercourse, obliterative procedures such as colpocleisis become a viable surgical option for prolapse. For those who wish to spare their uterus, the discussion may include sacrospinous hysteropexy or sacrouteropexy.

A routine pelvic examination is performed in the office with a half speculum. Visual examination can assess for vaginal atrophy, abnormal discharge, rashes, or masses. Urethral tip angulation suggesting hypermobility is assessed with a cotton tip applicator in the urethra. Measurements are obtained and recorded using the POP-Q classification [16] (Fig. 7.1). Stress urinary incontinence (SUI) is elicited with a supine stress test and occult SUI is tested for with reduction of the prolapse.

Given the results of the Colpopexy and Urinary Reduction Efforts (CARE) and Outcomes Following Vaginal Prolapse Repair and Midurethral Sling (OPUS) trials, all our patients are counseled on the probability of a concomitant anti-incontinence procedure at the time of POP surgery [17, 18].

Subsequently, all patients that have comorbidities such as coronary artery disease, obstructive sleep apnea, uncontrolled hypertension, diabetes, or symptoms suggesting non-diagnosed medical problems are referred to anesthesia for preoperative clearance. Prior to surgery, all patients are told to refrain from NSAIDs/blood thinners for up to 1 week prior to surgery date, a bowel preparation to decrease stool content in the pelvis region, and nothing by mouth after midnight. For patients who routinely smoke, we advise them to stop smoking to aid with recovery, wound healing, and also improve their general overall health. An informed consent is performed in conjunction with the patient and operating surgeon. Given the recent FDA announcements regarding transvaginal mesh, numerous questions can be expected given that ASC is most successfully performed with artificial synthetic graft material. It is important to note that the FDA

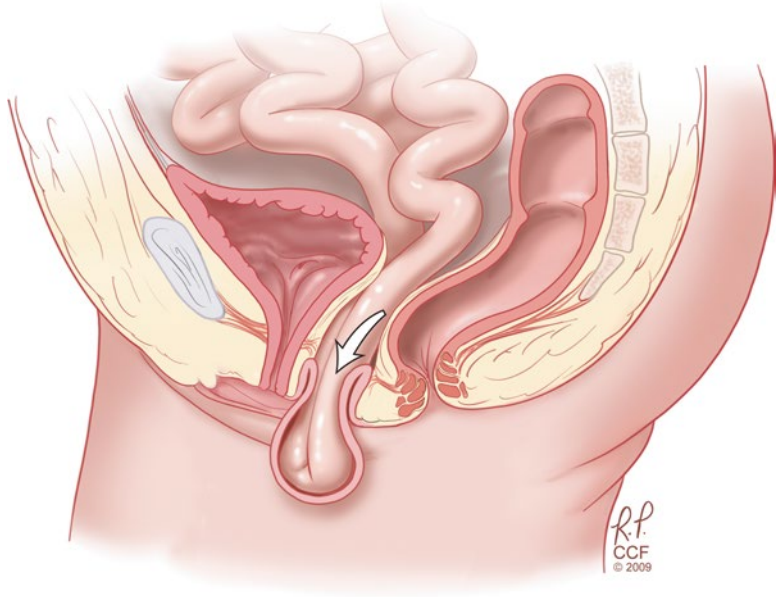


Fig. 7.1 Sagittal view of enterocele in post-hysterectomy patient. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2009–2013. All Rights Reserved)

announcement focuses on transvaginal mesh placement and addresses the need for further studies regarding transvaginal mesh placement for POP (Table 7.1). We do not routinely correct anterior/posterior compartment defects at the time of ASC but this should be individualized to each patient depending on goals and symptoms. Concomitant anti-incontinence procedures are typically performed with a mid-urethral sling utilizing synthetic macroporous polypropylene mesh, with efficacy and safety that have been demonstrated in long-term studies [19, 20].

Complications discussed with all patients include the risk of infection (UTI 10.9 %, wound infection 4.6 %) hemorrhage/transfusion 4.4 %, bladder/bowel or ureteral injury 1–3 %, DVT or PE 3.3 % [21]. Ileus and small bowel obstruction requiring reoperation are quoted at 6.9 % and 1.2 % respectively [22]. Extrusion rates with polypropylene mesh from 0.5 % to 10.5 % are quoted [21, 23, 24]. Subjective improvement based on global assessment is quoted upwards of 85 % [25]. Rates of reoperation for POP are expected to be less than 5 % in modern series but can be as high as 29 % [3, 21, 25].

Technique

The day of surgery, patients arrive in the preoperative care unit where an intravenous line is started by anesthesia. Perioperative antibiotics are administered within 60 min of the surgical incision. Given the intra-abdominal nature of the case, we prefer using cefazolin or clindamycin and gentamycin in patients who have a severe penicillin allergy or allergy to cephalosporins [26]. Subsequently the patient is positioned in the lithotomy position with a slight amount of flex to open the pelvis. We routinely utilize yellow fin stirrups for the legs. All pressure points are padded. Sequential compression devices are placed. The patient's vagina and abdomen are prepped. Preoperatively a dose of prophylactic subcutaneous 5,000 units of heparin or 40 mg enoxaparin is administered.

The patient is then prepped and draped. A 16 fr Foley catheter is placed to empty the bladder. One may choose to make either a Pfannensteil or lower midline incision. Camper's and Scarpa's fascia are dissected through with electrocautery.

Table 7.1 FDA safety communication

FDA safety communication: serious complications associated with transvaginal placement of surgical mesh for pelvic organ prolapse
The FDA first released a notice in 2008 regarding the complications of transvaginal mesh placement for POP. In July 2011, an update was provided regarding transvaginal mesh usage in POP. Given the additional 2,874 reports of complications received from January 1, 2008 to December 31, 2010, they concluded that serious complications are not rare. While open ASC utilizes artificial synthetic mesh, this FDA notification does not apply specifically to transabdominal mesh placement for pelvic organ prolapse (see below). Although not specific to transabdominal mesh placement, this notice highlights the need for patient education and a thorough informed discussion process between the surgeon and patient regarding the realistic goals of treatment and the complications stemming from any surgery. The following is a summary of the recommendations for healthcare providers:
• Obtain specialized training for each technique; be aware of the risks. • Be vigilant for potential adverse effects. • Watch for complications associated with tools used for mesh placement. • Inform patients of the permanency of mesh and that some complications may need additional surgery. • Inform patients that complications can affect their quality of life due to dyspareunia, scarring, and narrowing of the vagina. • Provide patients with a patient labeling from the mesh manufacturer. • POP can be treated without mesh. • Choose mesh after weighing the risks and benefits of all alternative options. • Consider the following before placing mesh: – Mesh is permanent making further surgery difficult. – Mesh may put the patient at risk for further surgery and the development of new complications. – Removal of mesh is difficult and may require multiple surgeries and poorer quality of life due to complications. – Mesh placed abdominally for POP repair may result in lower rates of mesh complications compared to transvaginal mesh. • Inform the patient about all options for POP including nonsurgical and non-mesh including the likely success of the alternatives. • Notify the patient if mesh will be used and what specific type. • Ensure the patient understands the risks and complications including the limited long-term data.
From FDA safety communication: UPDATE on serious complications associated with transvaginal placement of surgical mesh for pelvic organ prolapse. http://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm262435.htm. Last updated: November 20, 2012. Accessed April 18, 2013

The rectus is split in the midline. The peritoneum is opened close to the umbilicus. Any adhesions encountered are taken down sharply with metzenbaum scissors. Pelvic exposure is improved by using a self-retaining Bookwalter retractor and also packing the rectum to the patient's left side. The anterior plane of the vagina is dissected away from the bladder. We find that utilizing an end-to-end anastamotic (EEA) sizer or sponge stick in the vagina helps in exposing the vagina and aiding with dissection. Only in extreme cases of scarring do we find it appropriate to backfill the catheter to find the bladder. Once the bladder has been dissected free from the vagina, the posterior vagina is addressed. The vagina is dissected free from the rectum. Again in conditions of extreme adhesion or uncertainty, do we find an additional EEA sizer useful for rectal delineation.

Once the anterior and posterior walls of the vagina are free, dissection of the anterior longitudinal ligament is performed. Care is taken to incise the peritoneum overlying the sacral promontory at the midline in a longitudinal fashion and avoid the iliac vessels. Bleeding in this area can be attributed to any number of vessels in the area including the middle sacral vessels and superior and inferior hypogastric plexus. In cadaveric studies, on average the left common iliac vein was the closest major vessel (2.2–2.7 cm) to the mid-sacral promontory while the middle sacral artery and vein were closer at less than a centimeter (Fig. 7.2) [27]. After the sacral promontory at the level of S1 is cleared off, two pieces of 3 cm × 15 cm macroporous synthetic polypropylene mesh are used for grafting. While many different biologic (fascia lata, rectus fascia, porcine dermis) and artificial synthetic grafts (polytetrafluoroethylene, polyester, polyethylene, silicone coated) have been used, we prefer to use polypropylene mesh given its efficacy and decreased rate of exposure/erosion (Fig. 7.3a, b) [28–32]. The polypropylene mesh is attached to the anterior and posterior vaginal wall using non-braided delayed absorbable suture such as polydioxanone. Alternatively, several permanent monofilament sutures can be utilized away from the anterior bladder dissection. The mesh is fixated at approximately 5 points along both the posterior

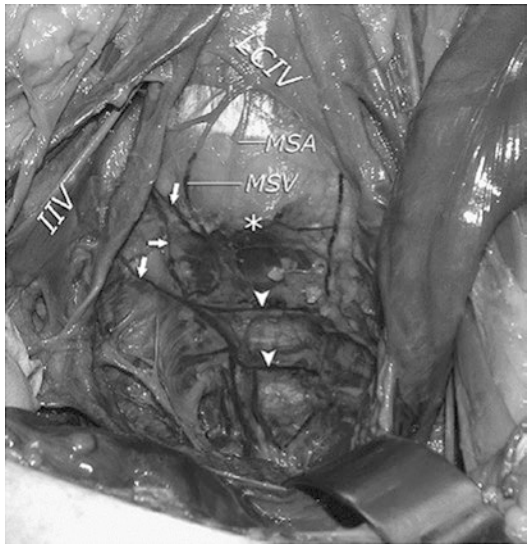


Fig. 7.2 Cadaver pelvic vascular anatomy dissection. Sacral venous plexus. Left common iliac vein (LCIV), internal iliac vein (IIV), middle sacral artery (MSA), middle sacral vein (MSV), midsacral promontory (*asterisk*), lateral sacral veins (*arrows*), and sacral venous plexus anastomoses (*arrowheads*). (Used with permission from Wieslander CK, Rahn DD, McIntire DD, Marinis SI, Wai CY, Schaffer JI et al. Vascular anatomy of the presacral space in unembalmed female cadavers. American journal of obstetrics and gynecology 2006 Dec;195(6): 1736–41)

and anterior vaginal walls. These are preferentially tied down with multiple knots given the location deep in the pelvis (Fig. 7.4). We have also utilized nonabsorbable braided suture for graft fixation, but patients may occasionally complain about continued vaginal discharge from suture exposure. Multiple studies have suggested a higher rate of exposure/extrusion correlated with the use of braided suture material but they are limited by their small sample sizes, heterogeneous use of graft material, and retrospective nature. No prospective trial exists evaluating the risk associated with monofilament absorbable suture and braided suture on polypropylene mesh extrusion/erosion in ASC [29, 33, 34].

With an EEA sizer in the vagina to reduce the prolapse, the mesh tails are tensioned appropriately and fixated to the anterior longitudinal ligament. Tensioning should be done to assure at least mobility to the bladder neck and avoid undue tension so as to keep the vaginal axis straight, avoiding upwards deviation. The excess mesh is then trimmed. Our suture of choice for fixation to the sacral promontory is a non-braided permanent suture (Fig. 7.5a, b). Two sutures are placed in a horizontal fashion on the anterior longitudinal ligament. In a cadaveric study utilizing female non-embalmed specimens, horizontal versus vertical suture placement was not found to be statistically significant in regard to pull out strength in sutures placed at or 1 cm above the level of S1 [35]. Care should also be taken to place the suture in the anterior longitudinal ligament and not through the disc space which could lead to a potential space for infection/abscess. Risk can also be minimized by ensuring that the vaginal fixation sutures are not through the vaginal epithelium [36, 37].

At this time, the peritoneum is reapproximated over the mesh. Although retroperitonealization of the mesh does not necessarily lead to fewer complications, reapproximation of the peritoneum adds little time and morbidity to the surgery [38]. If a large defect in the posterior cul-de-sac is seen, culdoplasty can be performed at this time. Anecdotally, given the advent of minimally invasive sacrocolpopexy, there has been a decrease in concomitant culdoplasty with minimal change seen in objective results. From below, the apical prolapse is reassessed to ensure the defect has been corrected. The anterior and posterior compartments are reassessed after ASC. Any anterior or posterior vaginal repairs are performed at this time. We do not routinely offer a posterior colporrhaphy to all prolapse patients and concomitant posterior colporrhaphy is based on the patient's preferences and symptoms (Fig. 7.6).

If an anti-incontinence procedure is to be performed, an assistant can begin with the vaginal dissection and exposure for a retropubic midurethral sling while the abdomen is being closed. Hemostasis is confirmed by visualization. The pelvis is irrigated with body temperature saline. All surgical counts are verified. The abdominal closure is done in a sequential fashion using #1 looped PDS for fascial closure. In obese patients, we prefer to re-approximate Scarpa's fascia to avoid dead space. The skin is addressed with a

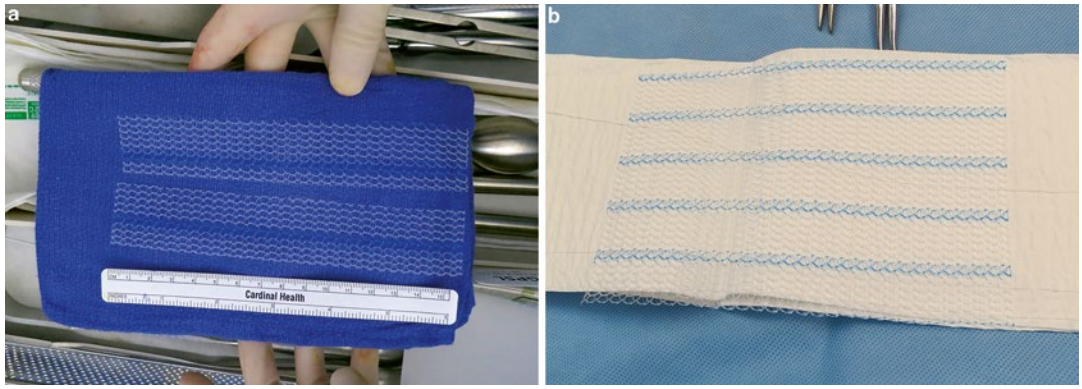


Fig. 7.3 Macroporous polypropylene synthetic mesh used for sacrocolpopexy. (**a**) Cut mesh. (**b**) Whole mesh

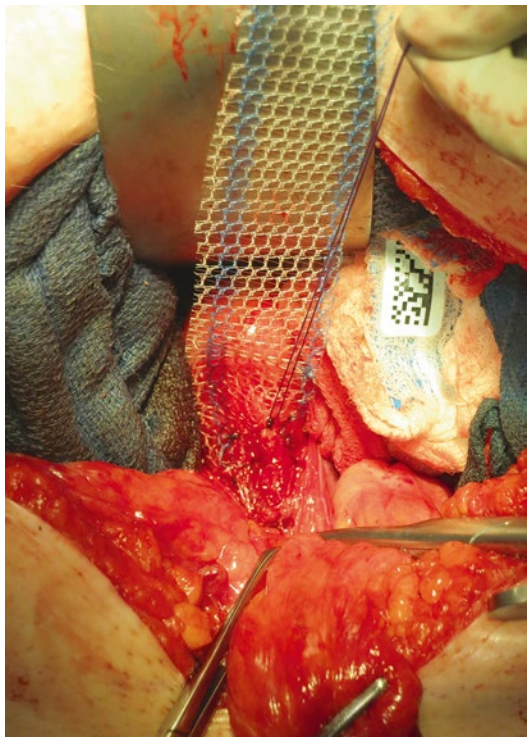


Fig. 7.4 Placement of mesh over the anterior portion of the vagina. We prefer to suture the mesh onto the anterior vagina with absorbable monofilament suture with multiple knots to secure the mesh deep in the pelvis

running subcuticular stitch using polyglactin suture. Cyanoacrylate skin adhesive is used for the skin. The patient is woken up from anesthesia and monitored in the post-anesthesia recovery unit. All patients then transition to an acute surgical floor. Intravenous fluids are continued at maintenance rates until the patient is tolerating a diet. All patients are started on a prophylactic deep vein thrombosis regimen including early ambulation and subcutaneous heparin. On postoperative day 1, clears are started, the vaginal pack is removed and a trial of void is performed. Postoperative labs are not routinely checked after surgery unless bleeding occurred or a patient is symptomatic [39]. The patient is transitioned to oral pain medication when they are tolerating a diet. All patients receiving narcotics receive a stool softener to reduce the incidence of constipation. Postoperative length of stay is usually 2–3 days.

Patients are discharged from the hospital with postoperative instructions. All patients are told to refrain from heavy lifting greater than 10 lbs during this time period, avoid strenuous activity (although avoidance of any activity such as walking is contraindicated), avoid vaginal instrumentation/sexual intercourse. Follow-up is scheduled at 6 weeks. At the patient's follow-up visit, we routinely perform a physical examination to assess for POP recurrence and graft exposure/extrusion.

Outcomes

Definition of Success

Success of ASC encompasses a heterogeneous definition of outcomes. Depending on whether success is defined by objective POP-Q postoperative evaluation versus patient satisfaction, effectiveness can range anywhere from 78 % to

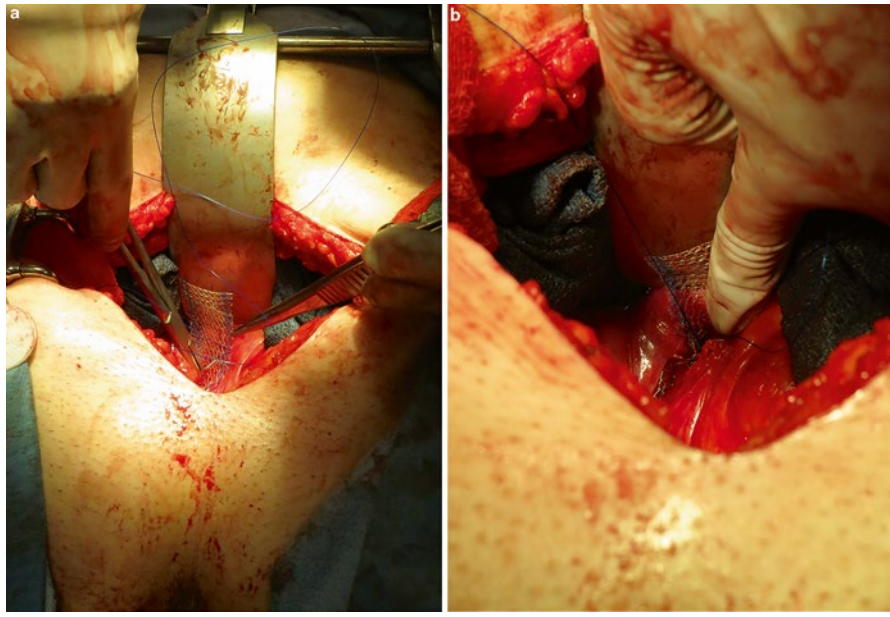


Fig. 7.5 The tails of the mesh are sutured to the anterior longitudinal ligament using nonabsorbable monofilament suture

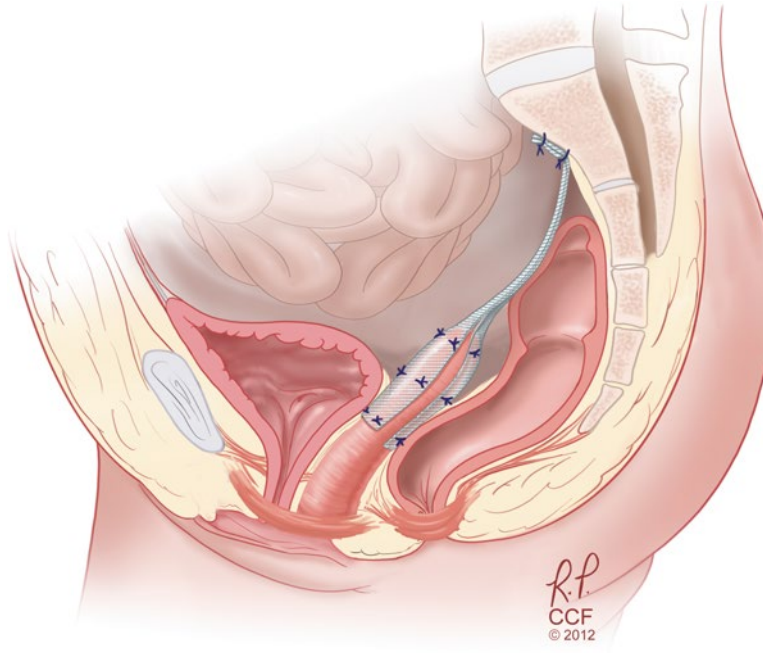


Fig. 7.6 Sagittal view of ASC repair with synthetic mesh. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2009–2013. All Rights Reserved)

100 % for apical prolapse versus 85–100 % for patient satisfaction [21]. In a randomized controlled study evaluating ASC to vaginal sacrospinous colpopexy, subjective success based on prolapse symptoms and satisfaction of ASC based on visual analog scale were 94 % and 85 %, respectively, at an average of 2 years [40]. In one of the longest follow-ups at a mean of 13.7 years, Hilger et al. demonstrated a 74 % success rate with ASC. Success in this study was defined by either no reoperation for POP or a negative answer to question 5 on the Duke Pelvic Floor Distress Inventory ("Do you usually have a bulge or something falling out that you can see or feel in the vaginal area?") Although significantly long in follow-up, only 12 of the original 47 women included in the study were available for examination. Of those 12 women who were examined, 6 patients had failed by their criteria and none of the 12 had greater than stage II prolapse on examination [41]. At the time of writing this chapter, the latest update from the CARE trial with 7 year follow-up demonstrated an estimated anatomic failure rate of 27 % in the urethropexy arm versus an estimated symptomatic POP failure rate of 29 % in that same group. Anatomic failure was defined as reoperation or pessary for POP where the vaginal apex descends below the upper third of the vagina or the anterior/posterior vaginal wall descends past the hymen. Symptomatic failure was defined as a positive response to one or more questions on the POP distress inventory referring to seeing or feeling a bulge or reoperation or pessary for POP [24].

Attempting to address this obtuse definition of success in ASC patients, Barber et al. evaluated the data from the CARE trial and applied 18 different surgical success definitions. Among their objectives was to describe how using different definitions affect estimates of treatment success and compare different definitions of surgical success by examining their relationship to patient's subjective assessments of improvement. At 2 years, 94 % of patients achieved surgical success when it was defined by absence of prolapse beyond the hymen. When applying National Institutes of Health definitions of outcomes such as optimal (POP-Q stage 0) or satisfactory (support higher than 1 cm proximal to hymen), the rates of success were lower at 19 % and 57 % [25].

Rates of reoperation for prolapse in the original CARE trial were low at 2.8 % over 2 years which rose to 5.1 % over the course of 7 years [24, 25]. This is comparable to the 4.4 % (0–18.2 %) median reoperation rate observed in summarized published studies. The most common reason for reoperation was for prolapse of the anterior or posterior compartment [21]. The longest follow-up was noted to be 3 years. Hilger et al. evaluating results at a mean of 13.7 years found a 10.5 % rate of reoperation for recurrence [41].

Genitourinary/Gastrointestinal/ Sexual Function Outcomes

In regard to system specific genitourinary, gastrointestinal, and sexual function after ASC, most studies in the past did not evaluate complaints with standardized validated questionnaires or in prospective fashion thereby making a generalization on outcomes difficult to assess. In a case control study evaluating women who had undergone ASC versus women who had solely undergone hysterectomy, patients were evaluated using a bowel function questionnaire and the Cleveland Clinical Incontinence Score (CCIS). While those undergoing ASC had more significant obstructive defecatory symptoms (splinting, incomplete evacuation, use of enemas), fecal incontinence rates were not different. Incontinence was noted to be higher in patients who had obstetric anal injury. Unfortunately, results from this study are difficult to extrapolate without the context of preoperative symptom scores. On average, time from surgery to questionnaire was 8.1 years for the ASC group [42]. Evaluating 1 year bladder symptoms based on UDI changes in patients who participated in the CARE trial, de novo irritative voiding was reported in 12/131 (9.2 %) women. For those with obstructive voiding symptoms before surgery, improvement was noted in 85.1 %. A statistically significant mean reduction of PVR of 31 mL was observed postoperatively [43].

One year follow-up was also evaluated in regard to sexual function in patients who participated in the CARE trial. Using the Pelvic Organ Prolapse/Urinary Incontinence Sexual Functioning Questionnaire (PISQ-12), patients who had a sexual partner before and after surgery were evaluated at 1 year for effects of surgery on sexual function. There was a statistically significant rise in the amount of women who were sexually active compared to prior to surgery (76.3 % vs. 66.1 %, $p<0.001$). Fewer women after ASC avoided sexual activity due to pelvic or vaginal symptoms, fear of incontinence, bulge in the vagina, or being limited by pain. It was noted that 11/148 (7.4 %) women became sexually inactive after surgery. There was a higher proportion limited by pain but this was not statistically significant (26 % vs. 22 %, $p=1.0$). The authors did note that there was a higher incidence of infrequent sexual desire amongst those who were inactive after surgery (70 % vs. 22.1 %, $p<0.001$) [44].

Open ASC Versus Laparoscopic/Robot Assisted ASC

Although ASC has been recognized as the gold standard surgery for apical POP repair, increased hospital stay, blood loss, and length of recovery have all been listed as drawbacks of open ASC compared to other approaches [45]. Minimally invasive surgery and robot assisted laparoscopic surgery decrease the convalescence associated with transabdominal surgery. Siddiqui et al. evaluated robotic ASC outcomes at 1 year compared to patients in the CARE trial and found no significant difference in surgical failures as defined by bothersome vaginal symptoms or repeat surgery for prolapse (8 % vs. 4 %, $p=0.16$). Operative characteristics that were significantly different between robotic vs. open ASC include estimated blood loss (90 mL vs. 228 mL, $p<0.01$), concomitant hysterectomy (49 % vs. 28 %, $p<0.01$), and posterior repair at time of ASC (8 % vs. 22 %, $p<0.01$). Complications that were significantly different included wound disruption (0 % vs. 4.3 %, $p=0.01$), febrile morbidity (4.8 % vs. 10.9 %, $p=0.04$), and ileus (5.6 % vs. 11.6 %, $p=0.05$) [46]. Rozet et al. similarly found laparoscopic sacral colpopexy to be efficacious in treating POP. The retrospective review evaluated 363 patients who underwent a laparoscopic sacral colpopexy. 25 % of patients had undergone a previous hysterectomy and only 4 % had a concomitant hysterectomy. Complications were low with 2 % requiring open conversion. Average hospital stay was noted to be 3.7 days. On average follow-up for 14.6 months, anatomic cure rate, which was not defined on postoperative visit, was noted to be 96 % with a similar 96 % satisfaction rate [47]. These rates are similar to a recent review article regarding laparoscopic sacrocolpopexy [48]. A retrospective cohort study evaluating laparoscopic and ASC found that although mean operating time (269 min vs. 218 min, $p<0.0001$) was longer in the laparoscopy cohort, mean hospital stay was significantly shorter (1.8 days vs. 4 days, $p<0.0001$). Clinical efficacy was difficult to assess given that not all patients had preoperative and postoperative POP-Q standardized scores [49]. To date no prospective randomized trial have been done to evaluate robotic ASC to open ASC [50].

Controversial Topics in Open ASC

Open ASC has been constantly evolving since being first described in the 1960s. The subjects of uterine sparing, concomitant hysterectomy at time of ASC, and concomitant anti-incontinence procedures are briefly discussed here but the reader is directed to the myriad information for further discussion.

Uterine Sparing

The surgical approach of ASC assumes that POP has occurred in the setting of the post-hysterectomy patient. For those with apical prolapse and an intact uterus, there continues to be a debate on whether to preserve the uterus. Proponents of uterine sparing would argue that

keeping the uterus intact preserves sexual function, decreases the morbidity associated with hysterectomy, and maintains the body as a whole [51]. Detractors from uterine sparing point out that after parturition, the uterus no longer serves a useful function and that sexual function is not diminished after hysterectomy [52]. In a long-term randomized controlled study evaluating sacrocolpopexy with uterus preservation versus vaginal hysterectomy with colporrhaphy for the correction of prolapse, 8 year outcomes were not statistically different between the two groups in regard to reoperation rate and Incontinence Impact Questionnaire (IIQ) or POP-Q (Pelvic Organ Prolapse Quantification) scores [53]. Dietz et al. performed a multicenter randomized controlled study evaluating sacrospinous hysteropexy versus vaginal hysterectomy with uterosacral vault suspension and found increased rates of apical recurrence at 1 year after sacrospinous hysteropexy but no significant difference in IIQ or other functional outcomes and quality of life questionnaires [54].

Concomitant Hysterectomy

After counseling a patient on the options, our preference in the patient with apical POP and an intact uterus is to perform a transvaginal hysterectomy with vault suspension at the time of the procedure to address the prolapse along with a possible anterior or posterior repair if needed. Given the theoretical risk of cuff infection and mesh extrusion, we do not routinely perform ASC in the setting of a hysterectomy. Mattox et al. in a retrospective study found a higher rate of mesh infection in patients who underwent hysterectomy versus those that did not (27 % vs. 1.3 %) [55]. Markinkovic evaluated abdominal hysterectomy at the time of abdominal sacrocolpopexy. In a retrospective review of 67 patients who underwent total abdominal hysterectomy and sacrocolpopexy with two pieces of polypropylene mesh, no exposures/extrusions were noted at a median of 26 months follow-up [56]. This is in contrast to other series which included patients who had concomitant hysterectomies and erosion rates from 1.5 % to 27 % thought to be related to mesh type versus how the cuff was addressed during time of hysterectomy [57–59]. Siddiqui et al. noted no mesh erosions at 1 year follow up in any patients who underwent robotic ASC with supracervical hysterectomy [46]. Thus, the ideal patient for sacrocolpopexy is probably a patient who may have a remote history of hysterectomy and now has vaginal vault descent who desires a reconstructive operation.

Concomitant Anti-incontinence Procedure

During the patient's initial visit and assessment of POP, her symptoms may or may not include stress urinary incontinence. Given provocative testing maneuvers, SUI and occult SUI may not be unmasked. Regardless, all patients are counseled of the probability of a concomitant anti-incontinence procedure at the time of ASC.

The CARE trial was a multicenter randomized controlled trial that randomized continent women undergoing ASC to receive a Burch colposuspension at the same time of surgery. This was designed to evaluate whether postoperative SUI symptoms were reduced by concomitant Burch colposuspension during ASC in continent women. Continence was defined as answering "never" or "rarely" to the SUI portion of the Medical, Epidemiological, and Social Aspect of Aging (MESA) questionnaire. The trial was stopped after the first interim analysis as there was a significant difference between postsurgical SUI symptoms in patients undergoing concomitant Burch colposuspension versus those that did not (23.8 % vs. 44.1 % $p<0.001$). The difference was also significant when evaluating those without evidence of SUI on preoperative UDS (reduction of postoperative SUI from 38.2 % to 20.8 %, $p=0.007$) [17].

Though these results are compelling to offer an anti-incontinence procedure to all our patients regardless of preoperative symptoms of SUI, we counsel patients on possible mid-urethral sling but ultimately give the patient the option in making the final decision. Studies have advocated

a more conservative approach of offering anti-incontinence procedures to patients with occult SUI or symptomatic SUI [60, 61]. Also, our standard anti-incontinence procedure is a mid-urethral sling performed at the time of ASC. The CARE trial evaluated Burch colposuspension as their anti-incontinence procedure which may suggest their results are not wholly applicable to our patient population. The Outcomes Following Vaginal Prolapse Repair and Midurethral Sling (OPUS) trial evaluated stress urinary incontinence at 3 months and 12 months after prolapse surgery with either concomitant mid-urethral sling placement or a sham procedure. The rates of urinary incontinence/treatment were significantly higher in the sham group at 3 months (49.4 % vs. 23.6 %, $p<0.001$) and 12 months (43 % and 27.3 %, $p=0.002$). Also incontinence demonstrated by cough stress test at 12 months was significantly higher in the sham group (20.5 % vs. 3.5 %, $p<0.001$). Occult SUI was observed in preoperative testing in 33.5 % of the women in the study even though women included in this study did not report SUI symptoms [18]. These results are similar to the CARE trial and suggest that any anti-incontinence procedure at the time of prolapse surgery will significantly reduce SUI symptoms afterwards. Whereas the CARE trial involved an anti-incontinence procedure which we do not typically perform, the OPUS trial had a patient population who underwent transvaginal prolapse correction; thus, these results also may not be applicable to patients who undergo ASC with a concomitant retropubic mid-urethral synthetic sling placement.

Borstad et al. evaluated concomitant anti-incontinence surgery in women with SUI at the time of prolapse surgery versus reassessing at 3 months. Utilizing tension-free vaginal tape (TVT) as their anti-incontinence procedure, they found no significant difference in SUI cure rate between either group. They did note that of those randomized to wait 3 months postoperative for their TVT procedure, only 53 out of the original 94 patients required an additional surgery [62]. At the time of writing this chapter, the CUPIDO trial is currently accruing patients who have both SUI and prolapse. The primary outcome of this multicenter randomized control trial is to evaluate absence of SUI 12 months after surgery in patients with SUI or occult SUI are randomized to prolapse surgery combined with anti-incontinence surgery versus prolapse surgery alone. While a significant amount of heterogeneity will be introduced by the type of prolapse surgery performed (vaginal hysterectomy, sacrospinous fixation, Manchester Fothergill operation, anterior colporrhaphy or mesh implantation, posterior colporrhaphy or mesh implantation, and enterocele repair) and type of anti-incontinence surgery (retropubic or transobturator mid-urethral sling), the results should be interesting to compare [63].

Summary

Given the aging population, the number of women who will develop POP will substantially increase in the next 40 years [64]. Although POP can be approached transvaginally or with a minimally invasive approach with robot assistance, transabdominal ASC remains the gold standard operation for apical POP repair. Patients should be evaluated thoroughly including the use of standardized symptom questionnaires on their initial visit as well as POP-Q measurements to objectively evaluate their POP. Comorbid conditions should be fully medically optimized prior to surgery. Most important of all, the patient's goals should be identified in the beginning given that the majority of women with POP are not symptomatic nor do they require an invasive procedure.

For those undergoing a corrective operative procedure, an informed discussion regarding the use of synthetic mesh graft is paramount. Patient's expectations are aligned with the surgeon's goals of surgery. The expected postoperative course is discussed with the patient to minimize any chance of misunderstanding. During surgery, techniques that optimize success include perioperative antibiotics and prophylactic DVT prevention, obtaining exposure, and recognizing pelvic anatomic landmarks to allow for precise dissection and avoidance of neurovascular structures.

We advocate the use of a macroporous polypropylene synthetic graft and non-braided suture.

Successful transabdominal ASC outcomes are high but depend on the definition used which is still not standardized in current literature. While minimally invasive approaches to ASC have been developed and enjoy acceptable rates of success with decreased morbidity, no randomized study exists between the two modalities to dispute transabdominal ASC as the gold standard for POP.

References

1. Beck RP, McCormick S, Nordstrom L. A 25-year experience with 519 anterior colporrhaphy procedures. Obstet Gynecol. 1991;78(6):1011–8.
2. Swift S, Woodman P, O'Boyle A, Kahn M, Valley M, Bland D, et al. Pelvic Organ Support Study (POSST): the distribution, clinical definition, and epidemiologic condition of pelvic organ support defects. Am J Obstet Gynecol. 2005;192(3):795–806.
3. Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol. 1997;89(4):501–6.
4. Smith FJ, Holman CD, Moorin RE, Tsokos N. Lifetime risk of undergoing surgery for pelvic organ prolapse. Obstet Gynecol. 2010;116(5):1096–100.
5. Nygaard I, Barber MD, Burgio KL, Kenton K, Meikle S, Schaffer J, et al. Prevalence of symptomatic pelvic floor disorders in US women. JAMA. 2008;300(11):1311–6.
6. Maher CM, Feiner B, Baessler K, Glazener CM. Surgical management of pelvic organ prolapse in women: the updated summary version Cochrane review. Int Urogynecol J. 2011;22(11):1445–57.
7. Shah SM, Sultan AH, Thakar R. The history and evolution of pessaries for pelvic organ prolapse. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17(2):170–5.
8. Barbalat Y, Tunuguntla HS. Surgery for pelvic organ prolapse: a historical perspective. Curr Urol Rep. 2012;13(3):256–61.
9. McDermott CD, Hale DS. Abdominal, laparoscopic, and robotic surgery for pelvic organ prolapse. Obstet Gynecol Clin North Am. 2009;36(3):585–614.
10. Lane FE. Repair of posthysterectomy vaginal-vault prolapse. Obstet Gynecol. 1962;20:72–7.
11. Birnbaum SJ. Rational therapy for the prolapsed vagina. Am J Obstet Gynecol. 1973;115(3):411–9.
12. Sutton GP, Addison WA, Livengood III CH, Hammond CB. Life-threatening hemorrhage complicating sacral colpopexy. Am J Obstet Gynecol. 1981;140(7):836–7.
13. Gyhagen M, Bullarbo M, Nielsen T, Milsom I. Prevalence and risk factors for pelvic organ prolapse 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. BJOG. 2013;120(2):152–60.
14. Hendrix SL, Clark A, Nygaard I, Aragaki A, Barnabei V, McTiernan A. Pelvic organ prolapse in the Women's Health Initiative: gravity and gravidity. Am J Obstet Gynecol. 2002;186(6):1160–6.
15. Jelovsek JE, Maher C, Barber MD. Pelvic organ prolapse. Lancet. 2007;369(9566):1027–38.
16. Bump RC, Mattiasson A, Bo K, Brubaker LP, DeLancey JO, Klarskov P, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol. 1996;175(1):10–7.
17. Brubaker L, Cundiff GW, Fine P, Nygaard I, Richter HE, Visco AG, et al. Abdominal sacrocolpopexy with Burch colposuspension to reduce urinary stress incontinence. New Engl J Med. 2006;354(15):1557–66.
18. Wei JT, Nygaard I, Richter HE, Nager CW, Barber MD, Kenton K, et al. A midurethral sling to reduce incontinence after vaginal prolapse repair. N Engl J Med. 2012;366(25):2358–67.
19. Svenningsen R, Staff AC, Schiotz HA, Western K, Kulseng-Hanssen S. Long-term follow-up of the retropubic tension-free vaginal tape procedure. Int Urogynecol J. 2013;24(8):1271–8.
20. Tincello DG, Botha T, Grier D, Jones P, Subramanian D, Urquhart C, et al. The TVT Worldwide Observational Registry for long-term data: safety and efficacy of suburethral sling insertion approaches for stress urinary incontinence in women. J Urol. 2011;186(6):2310–5.
21. Nygaard IE, McCreery R, Brubaker L, Connolly A, Cundiff G, Weber AM, et al. Abdominal sacrocolpopexy: a comprehensive review. Obstet Gynecol. 2004;104(4):805–23.
22. Whitehead WE, Bradley CS, Brown MB, Brubaker L, Gutman RE, Varner RE, et al. Gastrointestinal complications following abdominal sacrocolpopexy for advanced pelvic organ prolapse. Am J Obstet Gynecol. 2007;197(1):78.e1–7.
23. Brubaker L, Nygaard I, Richter HE, Visco A, Weber AM, Cundiff GW, et al. Two-year outcomes after sacrocolpopexy with and without burch to prevent stress urinary incontinence. Obstet Gynecol. 2008;112(1):49–55.
24. Nygaard I, Brubaker L, Zyczynski HM, Cundiff G, Richter H, Gantz M, et al. Long-term outcomes following abdominal sacrocolpopexy for pelvic organ prolapse. JAMA. 2013;309(19):2016–24.
25. Barber MD, Brubaker L, Nygaard I, Wheeler II TL, Schaffer J, Chen Z, et al. Defining success after surgery for pelvic organ prolapse. Obstet Gynecol. 2009;114(3):600–9.
26. Wolf Jr JS, Bennett CJ, Dmochowski RR, Hollenbeck BK, Pearle MS, Schaeffer AJ, et al. Best practice

policy statement on urologic surgery antimicrobial prophylaxis. J Urol. 2008;179(4):1379–90.
27. Wieslander CK, Rahn DD, McIntire DD, Marinis SI, Wai CY, Schaffer JI, et al. Vascular anatomy of the presacral space in unembalmed female cadavers. Am J Obstet Gynecol. 2006;195(6):1736–41.
28. Begley JS, Kupferman SP, Kuznetsov DD, Kobashi KC, Govier FE, McGonigle KF, et al. Incidence and management of abdominal sacrocolpopexy mesh erosions. Am J Obstet Gynecol. 2005; 192(6):1956–62.
29. Kohli N, Walsh PM, Roat TW, Karram MM. Mesh erosion after abdominal sacrocolpopexy. Obstet Gynecol. 1998;92(6):999–1004.
30. Tate SB, Blackwell L, Lorenz DJ, Steptoe MM, Culligan PJ. Randomized trial of fascia lata and polypropylene mesh for abdominal sacrocolpopexy: 5-year follow-up. Int Urogynecol J. 2011;22(2): 137–43.
31. Gregory WT, Otto LN, Bergstrom JO, Clark AL. Surgical outcome of abdominal sacrocolpopexy with synthetic mesh versus abdominal sacrocolpopexy with cadaveric fascia lata. Int Urogynecol J Pelvic Floor Dysfunct. 2005;16(5):369–74.
32. Govier FE, Kobashi KC, Kozlowski PM, Kuznetsov DD, Begley SJ, McGonigle KF, et al. High complication rate identified in sacrocolpopexy patients attributed to silicone mesh. Urology. 2005;65(6): 1099–103.
33. Shepherd JP, Higdon III HL, Stanford EJ, Mattox TF. Effect of suture selection on the rate of suture or mesh erosion and surgery failure in abdominal sacrocolpopexy. Female Pelvic Med Reconstr Surg. 2010;16(4):229–33.
34. Kammerer-Doak DN, Rogers RG, Bellar B. Vaginal erosion of cadaveric fascia lata following abdominal sacrocolpopexy and suburethral sling urethropexy. Int Urogynecol J Pelvic Floor Dysfunct. 2002;13(2):106–9. discussion 9.
35. White AB, Carrick KS, Corton MM, McIntire DD, Word RA, Rahn DD, et al. Optimal location and orientation of suture placement in abdominal sacrocolpopexy. Obstet Gynecol. 2009;113(5):1098–103.
36. Muffly TM, Diwadkar GB, Paraiso MF. Lumbosacral osteomyelitis after robot-assisted total laparoscopic hysterectomy and sacral colpopexy. Int Urogynecol J. 2010;21(12):1569–71.
37. Downing KT. Vertebral osteomyelitis and epidural abscess after laparoscopic uterus-preserving cervicosacropexy. J Minim Invasive Gynecol. 2008;15(3): 370–2.
38. Elneil S, Cutner AS, Remy M, Leather AT, Toozs-Hobson P, Wise B. Abdominal sacrocolpopexy for vault prolapse without burial of mesh: a case series. BJOG. 2005;112(4):486–9.
39. Murphy AM, Tunitsky-Bitton E, Krlin RM, Barber MD, Goldman HB. Utility of postoperative laboratory studies after female pelvic reconstructive surgery. Am J Obstet Gynecol. 2013;209(4):363.e1–5.
40. Maher CF, Qatawneh AM, Dwyer PL, Carey MP, Cornish A, Schluter PJ. Abdominal sacral colpopexy or vaginal sacrospinous colpopexy for vaginal vault prolapse: a prospective randomized study. Am J Obstet Gynecol. 2004;190(1):20–6.
41. Hilger WS, Poulson M, Norton PA. Long-term results of abdominal sacrocolpopexy. Am J Obstet Gynecol. 2003;189(6):1606–10. discussion 10-1.
42. Forsgren C, Zetterstrom J, Zhang A, Iliadou A, Lopez A, Altman D. Anal incontinence and bowel dysfunction after sacrocolpopexy for vaginal vault prolapse. Int Urogynecol J. 2010;21(9):1079–84.
43. Burgio KL, Nygaard IE, Richter HE, Brubaker L, Gutman RE, Leng W, et al. Bladder symptoms 1 year after abdominal sacrocolpopexy with and without Burch colposuspension in women without preoperative stress incontinence symptoms. Am J Obstet Gynecol. 2007;197(6):647.e1–6.
44. Handa VL, Zyczynski HM, Brubaker L, Nygaard I, Janz NK, Richter HE, et al. Sexual function before and after sacrocolpopexy for pelvic organ prolapse. Am J Obstet Gynecol. 2007;197(6):629.e1–6.
45. Maher C, Feiner B, Baessler K, Adams EJ, Hagen S, Glazener CM. Surgical management of pelvic organ prolapse in women. Cochrane Database Syst Rev. 2010;4, CD004014.
46. Siddiqui NY, Geller EJ, Visco AG. Symptomatic and anatomic 1-year outcomes after robotic and abdominal sacrocolpopexy. Am J Obstet Gynecol. 2012; 206(5):435.e1–5.
47. Rozet F, Mandron E, Arroyo C, Andrews H, Cathelineau X, Mombet A, et al. Laparoscopic sacral colpopexy approach for genito-urinary prolapse: experience with 363 cases. Eur Urol. 2005;47(2): 230–6.
48. Ganatra AM, Rozet F, Sanchez-Salas R, Barret E, Galiano M, Cathelineau X, et al. The current status of laparoscopic sacrocolpopexy: a review. Eur Urol. 2009;55(5):1089–103.
49. Paraiso MF, Walters MD, Rackley RR, Melek S, Hugney C. Laparoscopic and abdominal sacral colpopexies: a comparative cohort study. Am J Obstet Gynecol. 2005;192(5):1752–8.
50. Kim JH, Anger JT. Is robotic sacrocolpopexy a marketing gimmick or a technological advancement? Curr Opin Urol. 2010;20(4):280–4.
51. Costantini E, Porena M, Lazzeri M, Mearini L, Bini V, Zucchi A. Changes in female sexual function after pelvic organ prolapse repair: role of hysterectomy. Int Urogynecol J. 2013;24(9):1481–7.
52. Diwan A, Rardin CR, Kohli N. Uterine preservation during surgery for uterovaginal prolapse: a review. Int Urogynecol J Pelvic Floor Dysfunct. 2004;15(4): 286–92.
53. Roovers J, Bleijenberg E, Schagen van Leeuwen J, Scholten P, van der Haart H. Long term follow-up of a randomized controlled trial comparing abdominal and vaginal surgical correction of uterine prolapse. Int Urogynecol J. 2008;19 Suppl 1:91–2.

54. Dietz V, van der Vaart CH, van der Graaf Y, Heintz P, Schraffordt Koops SE. One-year follow-up after sacrospinous hysteropexy and vaginal hysterectomy for uterine descent: a randomized study. Int Urogynecol J. 2010;21(2):209–16.
55. Mattox TF, Stanford EJ, Varner E. Infected abdominal sacrocolpopexies: diagnosis and treatment. Int Urogynecol J Pelvic Floor Dysfunct. 2004;15(5): 319–23.
56. Marinkovic SP. Will hysterectomy at the time of sacrocolpopexy increase the rate of polypropylene mesh erosion? Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(2):199–203.
57. Imparato E, Aspesi G, Rovetta E, Presti M. Surgical management and prevention of vaginal vault prolapse. Surg Gynecol Obstet. 1992;175(3):233–7.
58. Culligan PJ, Murphy M, Blackwell L, Hammons G, Graham C, Heit MH. Long-term success of abdominal sacral colpopexy using synthetic mesh. Am J Obstet Gynecol. 2002;187(6):1473–80. discussion 81-2.
59. Brizzolara S, Pillai-Allen A. Risk of mesh erosion with sacral colpopexy and concurrent hysterectomy. Obstet Gynecol. 2003;102(2):306–10.
60. Ballert KN, Biggs GY, Isenalumhe Jr A, Rosenblum N, Nitti VW. Managing the urethra at transvaginal pelvic organ prolapse repair: a urodynamic approach. J Urol. 2009;181(2):679–84.
61. Chermansky CJ, Krlin RM, Winters JC. Selective management of the urethra at time of pelvic organ prolapse repair: an assessment of postoperative incontinence and patient satisfaction. J Urol. 2012; 187(6):2144–8.
62. Borstad E, Abdelnoor M, Staff AC, Kulseng-Hanssen S. Surgical strategies for women with pelvic organ prolapse and urinary stress incontinence. Int Urogynecol J. 2010;21(2):179–86.
63. van der Steen A, van der Ploeg M, Dijkgraaf MG, van der Vaart H, Roovers JP. Protocol for the CUPIDO trials; multicenter randomized controlled trials to assess the value of combining prolapse surgery and incontinence surgery in patients with genital prolapse and evident stress incontinence (CUPIDO I) and in patients with genital prolapse and occult stress incontinence (CUPIDO II). BMC Women's Health. 2010;10:16.
64. Wu JM, Kawasaki A, Hundley AF, Dieter AA, Myers ER, Sung VW. Predicting the number of women who will undergo incontinence and prolapse surgery, 2010 to 2050. Am J Obstet Gynecol. 2011;205(3): 230.e1–5.

Laparoscopic and Robotic Assisted Laparoscopic Sacrocolpopexy

8

Courtenay K. Moore

Introduction

Many studies have shown the prevalence of female pelvic floor disorders, including pelvic organ prolapse (POP), increases with age. Given the anticipated increase in the aging population, it is estimated that the number of women with POP will increase from 3.3 million in 2010 to 4.9 million in 2050, creating a need for safe, durable, and cost-effective procedures.

Over the last 15 years there has been a movement away from invasive, open surgery towards more minimally invasive laparoscopic and more recently robotic procedures in many urologic specialties including female pelvic reconstruction. Robotic assisted laparoscopy has allowed surgeons to offer the "gold standard" treatment for post-hysterectomy vault prolapse, abdominal sacrocolpopexy (ASC), with a less invasive, less morbid approach with similar anatomical outcomes. This chapter will review in detail the technique, outcomes, and complications of laparoscopic sacrocolpopexy (LSC) and robotic assisted laparoscopic sacrocolpopexy (RASC).

C.K. Moore, M.D. (✉)
Lerner College of Medicine, Cleveland Clinic, Cleveland, OH, USA

Cleveland Clinic, Glickman Urological and Kidney Institute, Cleveland, OH, USA
e-mail: Moorec6@ccf.org

Background

Many studies have shown the prevalence of female pelvic floor disorders, including POP, increases with age [1]. It is estimated that the number of women with POP will increase by 46 %, from 3.3 million in 2010 to 4.9 million in 2050, creating a need for safe, durable, and cost-effective procedures [1]. Based on level 1 evidence, the ASC has long been considered the gold standard for vaginal vault prolapse given its long term anatomic success rates [2]. While ASC has demonstrated higher success rates than vaginal approaches, it is more invasive, has a longer recovery time, and has a higher complication rate.

With the advent of laparoscopic techniques in the 1990s, interest in laparoscopic approaches to prolapse repairs surged. Since its introduction by Nezhat in 1994, LSC has undergone several modifications [3]. In the original report, a single piece of gortex mesh was attached to the posterior vaginal apex and sutured or stapled to the anterior longitudinal ligaments of the sacrum. Since its inception, modifications to the procedure have been made including the use of anterior and posterior pieces of synthetic polypropylene mesh. In addition the procedure has been modified for patients desirous of preserving the uterus using a single piece of synthetic polypropylene mesh posteriorly or an anterior Y-shaped piece of synthetic mesh and a

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_8,

posterior synthetic mesh. The laparoscopic approach offers the success, versatility, and durability of the traditional abdominal repairs with a minimally invasive approach and shortened recovery.

However, widespread application of laparoscopic techniques has been limited by a steep learning curve and specialty training requirements. As technology has continued to advance, robotic surgery has been integrated into the treatment of female pelvic floor disorders. The RASC was first described in 2004 and subsequently in 2005 the Food and Drug Administration (FDA) approved Da Vinci® (Intuitive Surgical, Sunnyvale, CA) robotic technology for gynecologic applications. The advantages of robot assisted laparoscopy are that it provides the advantages of laparoscopic surgery while easing the technical challenges of traditional laparoscopic surgery. For instance, the 7 degrees of freedom provided by Da Vinci technology mimic the motions of the human hand and greatly facilitate surgical challenges such as intracorporeal suturing. Therefore, use of robotic technology may allow non-laparoscopic trained surgeons to bring the advantages of laparoscopic surgery to their patients.

In this chapter, we review laparoscopic and robotic approaches to the management of apical and uterine POP.

Evaluation and Work-Up

The management of POP depends on symptom bother, the goals of the patient, and comorbidities. If a patient is older or does not wish to preserve sexual function, observation, a pessary or an obliterative procedure may be more appropriate. In women that have symptomatic vaginal vault POP and desire surgical correction, the surgeon should discuss the various operative repairs, techniques, complications, and recovery time.

Indications for RASC include recurrent apical prolapse, failed previous transvaginal repairs, and apical prolapse in women with foreshortened vaginal length. Additional factors that should be taken into account are the patient's previous abdominal surgeries, which can lead to abdomino-pelvic adhesions, the patient's pulmonary status, and ability to tolerate steep trendelenburg.

Patients should also be assessed for stress urinary incontinence (SUI) to determine if a concomitant anti-incontinence procedure should be performed. The role of anti-incontinence in patients without documented pre-operative SUI remains controversial.

In 2006, the Colpopexy and Urinary Reduction Efforts (CARE) trial showed the addition of a Burch colposuspension in women without preoperative stress undergoing an ASC significantly reduced the risk of postoperative stress urinary incontinence (23.8 %, vs. 44.1 % in the control group, $P<0.001$) [2].

Recently, JAMA published the long term outcomes of the CARE trial, which shows by year 7 women who underwent prophylactic Burch at the time of ASC had a longer time to recurrence of SUI than women who did not undergo a Burch. The estimated probabilities of developing SUI by year 7 were 0.62 in the Burch and 0.77 in the no Burch arm (treatment difference of −0.153; 95 % CI, −0.268 to 0.030) [4].

Many pelvic surgeons have extrapolated the data from the CARE trial data to justify prophylactic concomitant mid-urethral slings in all continent women undergoing ASC or LSC/RASC. Recently, the Outcomes Following Vaginal Prolapse Repair and Midurethral Sling (OPUS), a randomized, multi-center trial involving women with stage 2 or greater anterior vaginal wall POP without symptoms of SUI undergoing vaginal prolapse surgery found urinary incontinence present in 27.3 % and 43.0 % of patients in the sling and sham groups, respectively ($P=0.002$), at 12 month follow-up [5]. The number needed to treat with a sling to prevent one case of urinary incontinence at 12 months was 6.3 [5]. In summary, a prophylactic midurethral sling inserted during vaginal prolapse surgery resulted in a lower rate of urinary incontinence at 12 months but an overall higher rate of adverse events. It is important to remember that only vaginal surgeries were included in this study, not ASC or LSC/RASC, and therefore these conclusions must be applied to these procedures with care.

Surgical Procedure

Laparoscopic Sacrocolpopexy

The initial steps involving patient positioning are the same for both LSC/RASC. The patient is placed in the dorsal lithotomy position using Yellowfin® (Allen Medical Systems, Acton, MA) stirrups. The arms are tucked at the side. All pressure points are padded. The patient's breasts should be padded and the patient secured to the table using surgical tape (Fig. 8.1). The patient's abdomen and vagina should be prepped and draped in standard surgical fashion. Based on surgeon preference, four or five ports are used (Fig. 8.2a, b). An intra- or infra-umbilical incision can be made. Access can be obtained using either the Hassan open technique or a Veress needle. If the Hassan technique is used, a 10–12 mm port is placed into the abdominal cavity. The port or Veress needle should be connected to the CO_2 insufflation tubing and the abdomen insufflated. Intra-abdominal pressures should be monitored, if they exceed 8 mm Hg; the port or needle should be adjusted to ensure that it is not adherent to bowel or omentum and not outside the peritoneal cavity.

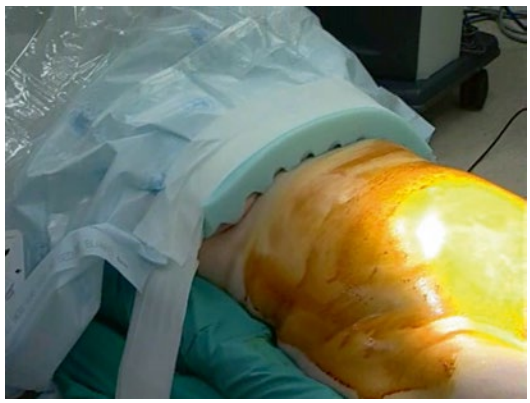


Fig. 8.1 Patient positioning for both LSC/RASC: the patient is placed in the dorsal lithotomy position using Yellowfin® (Allen Medical Systems, Acton, MA) stirrups. The arms are tucked at the side. All pressure points are padded. The patient's breasts should be padded and the patient secured to the table using surgical tape

Two additional 10–12 mm ports are placed lateral to the rectus muscle. One or two additional 5 mm ports are placed 2–3 cm cephalad and 2–3 cm medial to the anterior superior iliac spines, avoiding ilioinguinal and iliohypogastric nerve injury or entrapment. The left lower port is utilized to retract the sigmoid colon to the left and cephalad. This port can be eliminated by using a suture to retract the sigmoid. A 1-0 monofilament suture on a large (CT-X) needle can be passed into the left lower quadrant, through an epiploic appendage of the sigmoid colon, back out through the left lower quadrant, and clamped at the skin level to retract the sigmoid colon (Fig. 8.3).

After the sigmoid is retracted, the sacral promontory, right common iliac artery, and right ureter are identified. The posterior peritoneum over the sacral promontory is incised longitudinally to the level of the vaginal apex. An endoanal sizer (Fig. 8.4) is placed in the vagina, thereby reducing the prolapse and elevating the vagina for exposure (Figs. 8.5 and 8.6). The peritoneum over the vaginal apex is then incised, and this dissection is continued anteriorly along the vaginal wall in an attempt to dissect the plane between the bladder and vagina (Figs. 8.7, 8.8, and 8.9). The bladder can be filled to help demarcate this plane. This can also be accomplished with the introduction of a cystoscope light in the bladder. This plane is dissected at least 3 cm distal to the vaginal apex to allow space for placement of the anchoring sutures. The lack of direct tactile feedback makes this dissection challenging; in a recent study of this technique, cystotomy or sutures thrown into the bladder were noted in 10.7 % of cases [6]. Similar dissection is performed on the posterior vaginal wall to deperitonealize this area and separate the vagina from the rectum posteriorly. The mesh, either in two separate strips (size varies depending on surgeon preference: 2–4 cm × 12–15 cm) or prefashioned in a Y-configuration, is passed into the field and sutured with nonabsorbable suture to the posterior and then the anterior vaginal wall (Fig. 8.10). At least four sutures are required on either side to fully anchor the mesh (Fig. 8.11).

The next step involves suturing the mesh to the longitudinal ligament of the sacral promontory.

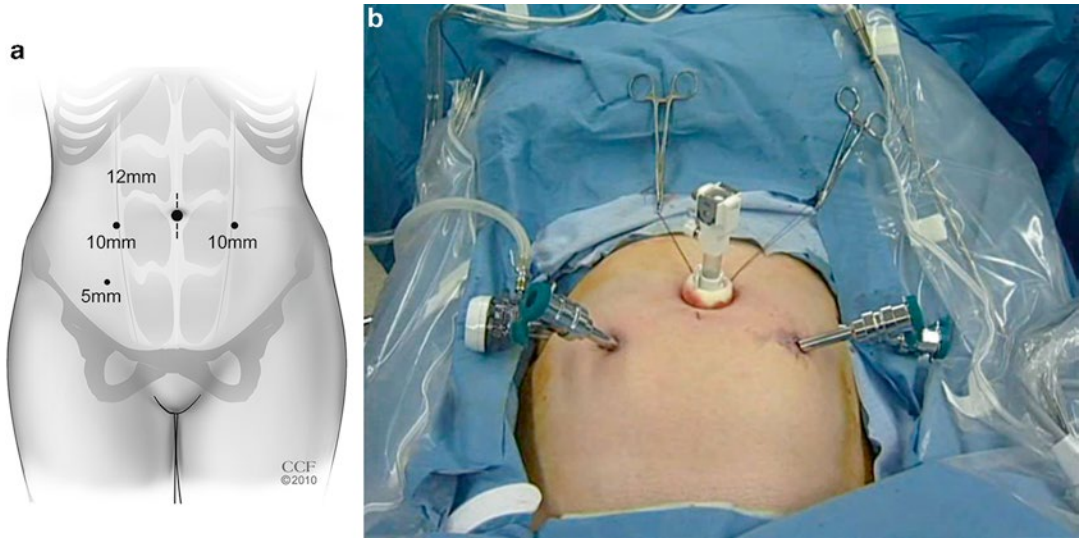


Fig. 8.2 (**a**) Port placements for laparoscopic pelvic organ prolapse surgery. A fifth port (5 mm) may be placed in the left lower quadrant, or a suture may be used to retract the sigmoid colon. (**b**) Laparoscopic port placement for robotic pelvic organ prolapse surgery. (**a**: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2006–2014. All Rights Reserved)

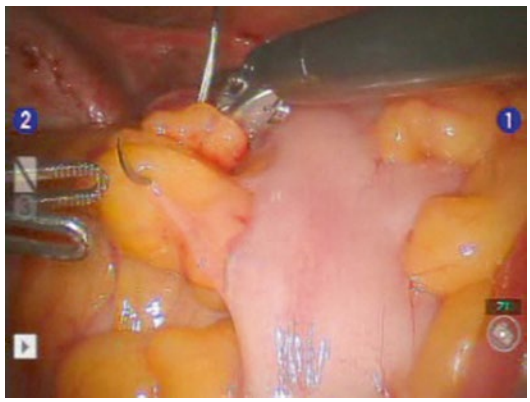


Fig. 8.3 Retraction of the sigmoid colon with a CT-X needle through an epiploic appendage. The needle can be placed though the abdominal wall and clamped at the skin level to retract the sigmoid colon allowing exposure of the sacral promontory

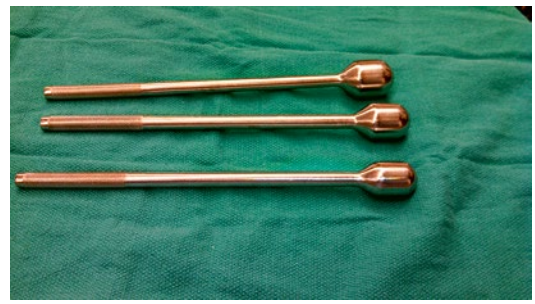


Fig. 8.4 Endoanal sizers (EEA)

Using the third robotic arm the sigmoid colon is retracted to the left pelvic side wall using a bowel grasper or Prograsp™ (Intuitive Surgical, Sunnyvale, CA) exposing the sacral promontory. The peritoneum overlying the promontory is incised using monopolars scissors or hook. Care should be taken to identify the right ureter, iliac bifurcation, and presacral vessels. With careful blunt dissection the fat overlying the promontory is cleared identifying the anterior longitudinal ligament at the S1 or S2 level (Fig. 8.12). This maneuver is frequently done with a laparoscopic Kittner introduced through the 12 mm accessory port which allows tactile feedback of the bone (Fig. 8.13). Should bleeding occur from the presacral space, one can increase insufflation and intrababominal pressure while introducing lap pads to apply direct pressure. In addition sutures, surgical clips, and hemostatic agents can be used. If conversion to open is required, orthopedic thumbtacks can also be used.

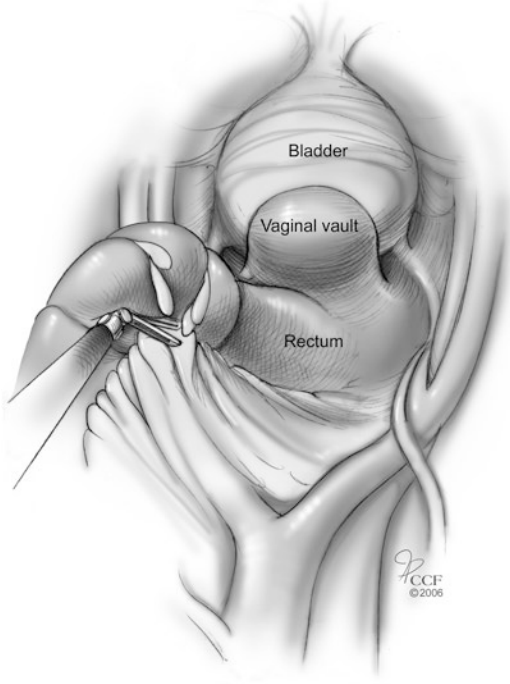


Fig. 8.5 Retraction of the small bowel and sigmoid colon allowing exposure of the sacral promontory. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2006–2014. All Rights Reserved)

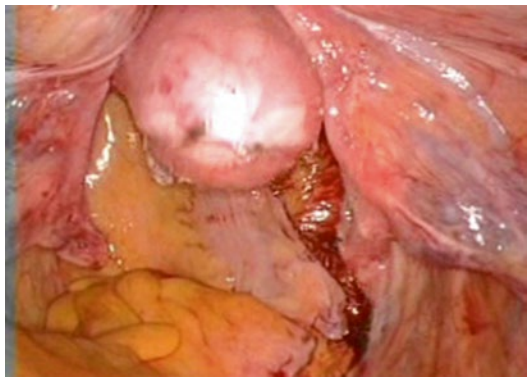


Fig. 8.6 Placement of EEA sizer in the vagina reducing the prolapse and elevating the vagina for exposure

With the prolapse reduced using the endoanal sizer, the proximal end of the mesh is anchored to the anterior longitudinal ligament of the sacrum with two No. 0 nonabsorbable sutures (Figs. 8.14 and 8.15). The excess mesh is trimmed and the posterior peritoneum is then closed over the mesh (Fig. 8.16). Cystoscopy should be performed at the end of procedure to ensure ureteral patency and that none of the sutures have passed into the bladder.

In patients undergoing sacrouteropexy, the uterus can be suspended with a laparoscopic tenaculum or with a Keith needle placed though the fundus (Fig. 8.17). The posterior peritoneum is incised from the level of the sacral promontory, caudally to the level of the posterior vaginal cuff and cervix (Fig. 8.18). Depending on surgeon preference, a single piece of posterior mesh (3–5 cm × 12–15 cm) or an anterior Y-shaped piece of mesh and a posterior mesh are used. In cases using a single piece of mesh, the mesh is sutured to the posterior vaginal cuff and posterior cervix using 0-nonabsorbale sutures. When using two pieces of mesh the posterior mesh is placed as described, while each arm of the anterior Y-shaped mesh is passed through the broad ligament. The mesh/meshes are fixed to the sacrum as described above.

Robotic Laparoscopic Sacrocolpopexy and Sacrouteropexy

This modification of the LSC utilizes the robotic system to facilitate three-dimensional visualization of the operative field, placement of sutures, and tying of the sutures, thereby simplifying the execution of maneuvers and shortening the laparoscopic learning curve. Five ports are typically utilized: an umbilical port (intra-, infra-, or supraumbilical) 12 mm camera port; and two 8-mm robotic ports placed at the lateral edge of the rectus abdominal muscles (8–10 cm lateral to the camera port) at the level of the umbilicus or 1–2 cm caudal; traditionally two additional ports a 5 mm and a 10 mm or two 10 mm ports are placed bilaterally, 3 cm medial and cephalad to the anterior superior iliac spine to allow an assistant to retract the sigmoid colon and small bowel (Fig. 8.19). At least one of these ports should be 10 mm to allow passage of the mesh strips and needles as needed. In women with small pelvises, we place the accessory 10 mm port 8 cm lateral and 2–3 cm cephalad to the umbilical port.

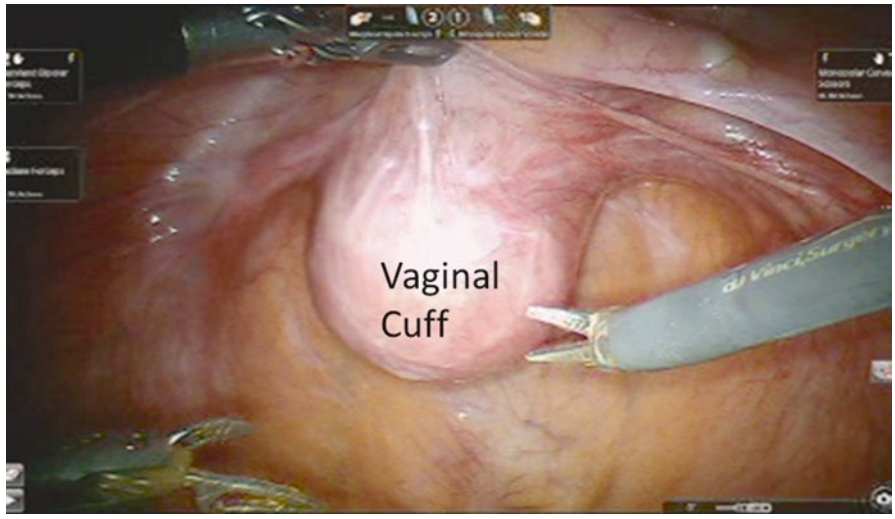


Fig. 8.7 The peritoneum over the vagina apex is incised longitudinally to the level of the sacral promontory. Using the third robotic arm to retract the bladder anteriorly, the peritoneum over the vaginal cuff is exposed. Note the grasper in the left hand and the endoshears in the right hand. (Used with permission of Springer Business + Business Media from McAchran S, Moore C. Robotic Abdominal Sacrocolpopexy. In Best SL, Nakada SY (eds): Minimally Invasive Urology: An Essential Clinical Guide to Endourology, Laparoscopy (LESS), and Robotics. New York: Springer Science + Business Media; 2014)

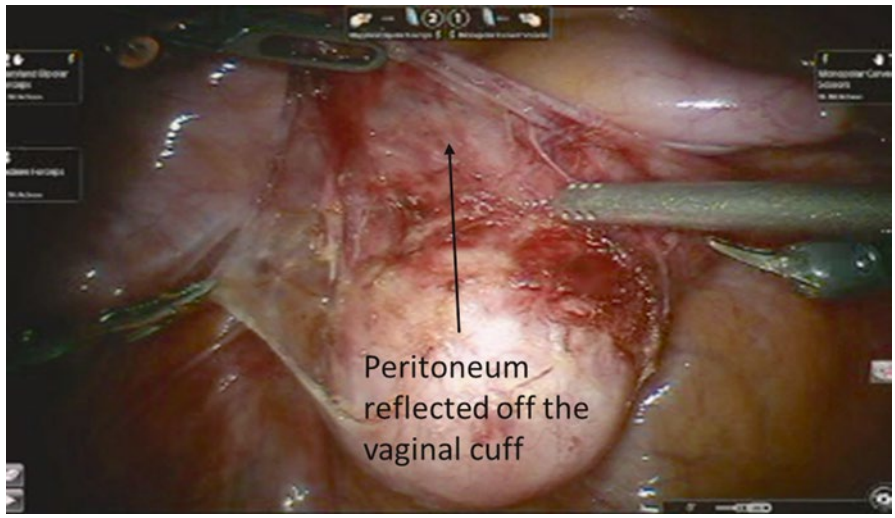


Fig. 8.8 The peritoneum overlying the anterior vagina has been dissected allowing a place to develop between the vagina and the bladder. This will be site for the anterior mesh attachment. (Used with permission of Springer Business + Business Media from McAchran S, Moore C. Robotic Abdominal Sacrocolpopexy. In Best SL, Nakada SY (eds): Minimally Invasive Urology: An Essential Clinical Guide to Endourology, Laparoscopy (LESS), and Robotics. New York: Springer Science + Business Media; 2014)

Once access is obtained the patient is placed in steep Trendelenburg position. The robot can be docked between the legs or side docked (Figs. 8.20 and 8.21). The technique is identical to that described for laparoscopic sacrocolpopexy. As in the laparoscopic procedure the uterus can be spared by performing a robotic laparoscopic sacrouteropexy as described above.

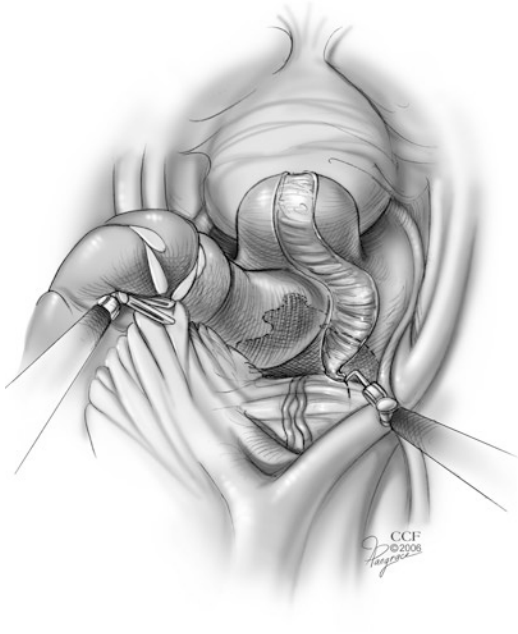


Fig. 8.9 The peritoneum over the vagina apex is incised longitudinally to the level of the sacral promontory. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2006–2014. All Rights Reserved)

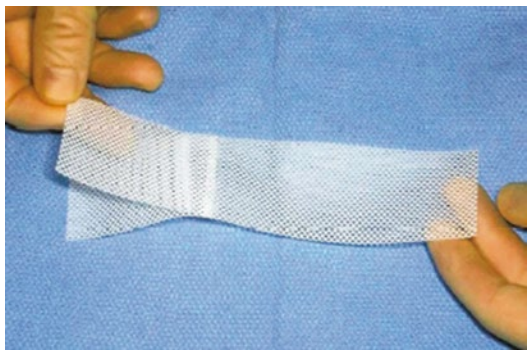


Fig. 8.10 Y-shaped mesh

Complications

In a comprehensive review of the published literature on ASC, Nygaard and colleagues from the Pelvic Floor Disorders Network reported the success rate, defined as lack of apical prolapse postoperatively, ranged from 78 % to 100 % and when defined as no postoperative prolapse, from 58 % to 100 % [7]. Median reoperation rates for POP and for stress urinary incontinence were 4.4 % (range 0–18.2 %) and 4.9 % (range 1.2–30.9 %), respectively [7]. The rate of mesh erosion was 3.4 % [6]. In the recently published JAMA article on long term complications of ASC, the estimated probabilities of treatment failure for anatomic POP was 0.27 in the urethropexy group and 0.22 in the no urethropexy group (treatment difference of 0.050; 95 % CI, −0.161 to 0.271), and 0.29 and 0.24, respectively for symptomatic POP (treatment difference of 0.049; 95 % CI, −0.060 to 0.162) [4]. The probability of mesh erosion was 10.5 % [4].

The Pelvic Floor Disorders Network found intraoperative complications of ASC to include cystotomy in 3.1 %, enterotomy or proctotomy in 1.6 %, ureteral injury in 1.0 %, and hemorrhage or transfusion or both occurred in 4.4 % [6]. Among postoperative complications urinary tract infections were the most common complication with a median range of 10.9 % (2.5–25.9 %) followed by wound infection, hematoma, or superficial separation ranging with a median of 4.6 % (0.4–19.8 %) [6]. Ileus was reported in 3.6 %, deep venous thrombus or pulmonary embolus 3.3 %, 1.1 % required reoperation for small bowel obstruction, and 5.0 % underwent incisional hernia repair [7].

Both the incidence of intraoperative and postoperative complications that occur after LSC and RASC are similar to ASC. Interestingly, the rate of cystotomy or sutures in the bladder is much higher, 10.7 % in RASC and LSC compared to ASC, which was 3.1 % [6, 8].

Outcomes

LSC appears to successfully recapitulate the open technique that has demonstrated durable results for several decades. Several studies have demonstrated the laparoscopic approach to be successful, 90–96 % cure rate, with a low mesh erosion rate, ranging from 1 % to 8 % [9]. The largest series of LSC is a retrospective cohort of 165 patients followed for a mean of 43 months [10]. Of the 165 patients, 27 were lost to

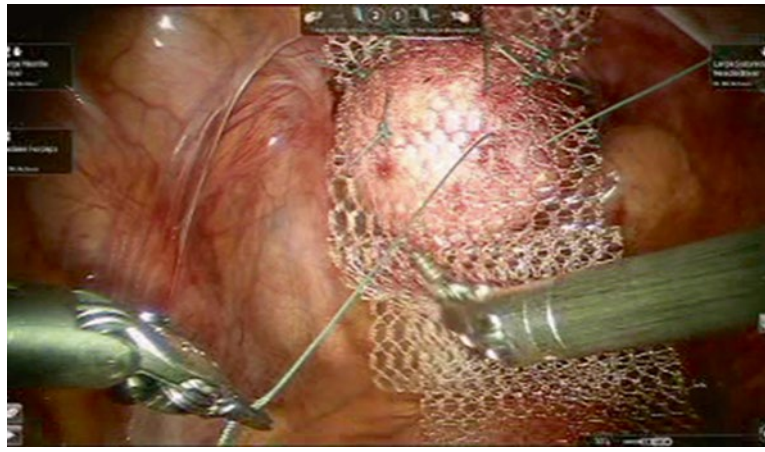


Fig. 8.11 With an EEA sizer in the vagina, the mesh is sutured to the anterior vaginal wall

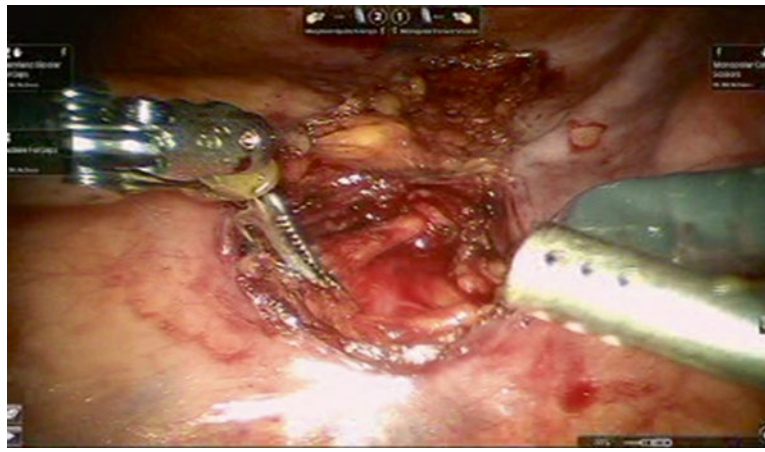


Fig. 8.12 Sacral dissection with the posterior peritoneum incised exposing the anterior longitudinal ligament. (Used with permission of Springer Business + Business Media from McAchran S, Moore C. Robotic Abdominal Sacrocolpopexy. In Best SL, Nakada SY (eds): Minimally Invasive Urology: An Essential Clinical Guide to Endourology, Laparoscopy (LESS), and Robotics. New York: Springer Science + Business Media; 2014)

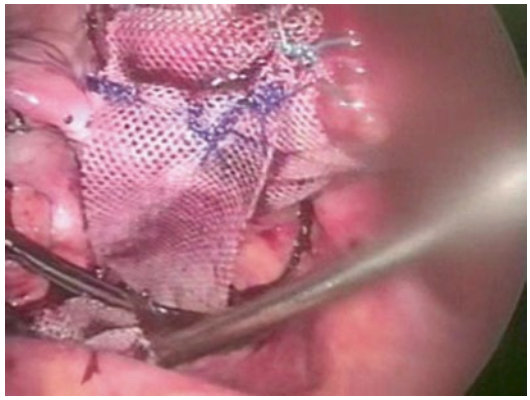


Fig. 8.13 Fat overlying sacral promontory blunted dissected using laparoscopic Kittner exposing the bone

follow-up. Success rate at mean follow-up was 94.9 %, with 5.07 % patients reporting recurrent vaginal vault prolapse. Only 3.62 % of patients reported a recurrent cystocele.

We reported a comparative cohort study from our institution of 61 patients treated with ASC and 56 treated with LSC with a mean follow-up of 16 and 14 months, respectively. The mean total operative time was longer for the laparoscopic group (269 min vs. 218 min), but hospital stay was shorter in the laparoscopic group (1.8 days vs. 4.0 days) [11]. Reoperation rates (11 % laparoscopic vs. 5 % open) and clinical outcomes rates were similar. The sample size was not

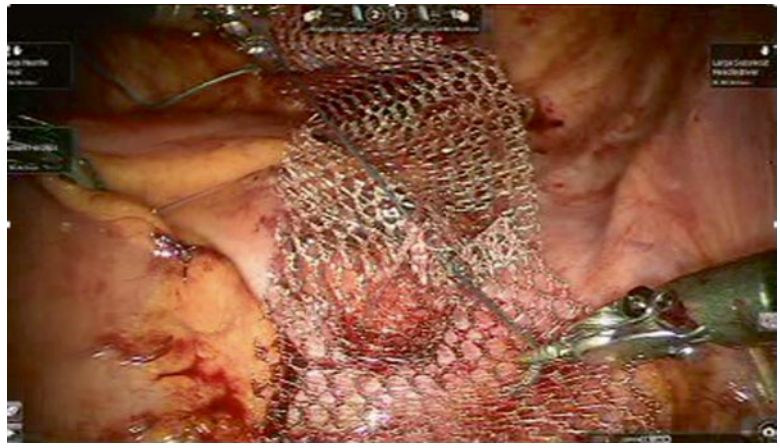


Fig. 8.14 The anchoring suture for the sacral portion of the mesh is placed through the anterior longitudinal ligament. (Used with permission of Springer Business+Business Media from McAchran S, Moore C. Robotic Abdominal Sacrocolpopexy. In Best SL, Nakada SY (eds): Minimally Invasive Urology: An Essential Clinical Guide to Endourology, Laparoscopy (LESS), and Robotics. New York: Springer Science+Business Media; 2014)

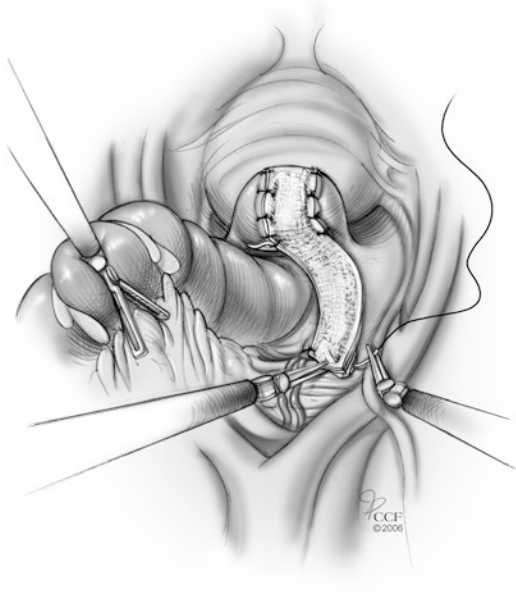


Fig. 8.15 Mesh is introduced and sutured to the vaginal apex anteriorly and posteriorly and sutured to the sacral promontory. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2006–2014. All Rights Reserved)

powered adequately to detect differences in complication rates.

Many studies have shown RASC to have anatomical cure rates similar to ASC and LSC [12]. Two more recent larger series by Akl et al. and Shariati et al. who followed 80 and 77 patients undergoing RASC reported recurrent prolapse in 3.7 % and 1.29 % patients, respectively [13].

In a retrospective study comparing 73 patients who underwent RASC with 10 patients who had ASC 6 weeks postoperatively, Geller et al. found the robotic group to have slightly better POP-Q "C" points, −9 compared with −8 ($P = .008$) [13]. All other anatomic outcomes were similar. Robotic procedures had statistically less blood loss (103 ± 96 mL vs. 255 ± 155 mL, $P < .001$), longer total operative time (328 ± 55 min vs. 225 ± 61 min, $P < .001$), shorter length of stay (1.3 ± 0.8 days vs. 2.7 ± 1.4 days, $P < .001$), and a higher incidence of postoperative fever (4.1 % compared vs. 0.0 %, $P = .04$). Recently the same group published their long term data. At a mean follow-up of 44.2 ± 6.4 months, there was still significant improvement in all POP-Q measurements from baseline. Cure rates for the Apex were 100 % while cure rates for the anterior and posterior compartments were lower 78.9 % for both. Secondary outcomes assessed using PFDI_20, PFIQ-&, PISQ-12 all showed significant improvement in pelvic floor function. At follow-up, there were two cases of mesh erosion in both open (7 %) and robotic group (8 %) [14].

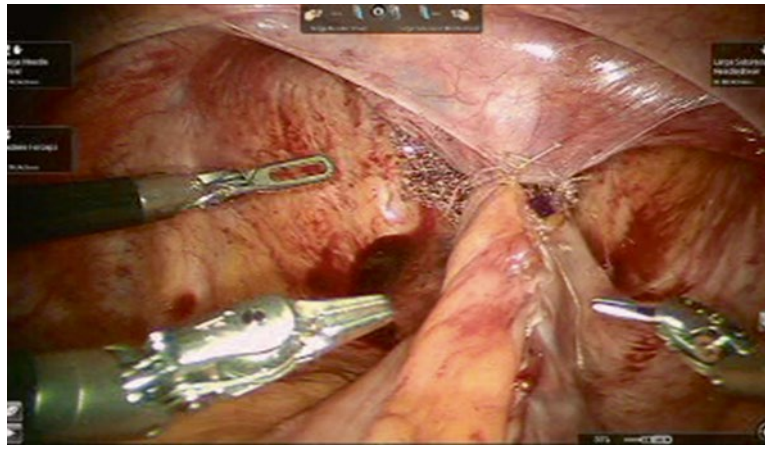


Fig. 8.16 The mesh is covered by the posterior peritoneum. The vagina is suspended to the sacral promontory, recreating normal vaginal anatomy. (Used with permission of Springer Business+Business Media from McAchran S, Moore C. Robotic Abdominal Sacrocolpopexy. In Best SL, Nakada SY (eds): Minimally Invasive Urology: An Essential Clinical Guide to Endourology, Laparoscopy (LESS), and Robotics. New York: Springer Science+Business Media; 2014)

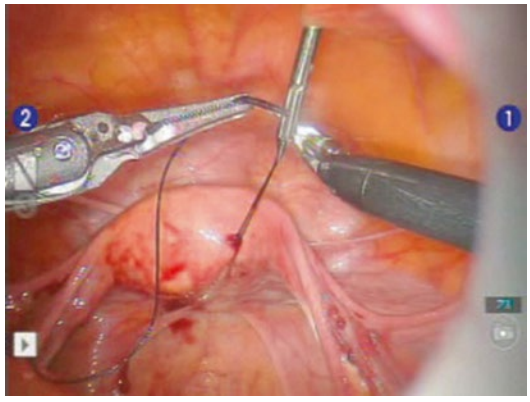


Fig. 8.17 In a sacrouteropexy, the uterus is retracted using a Keith needle

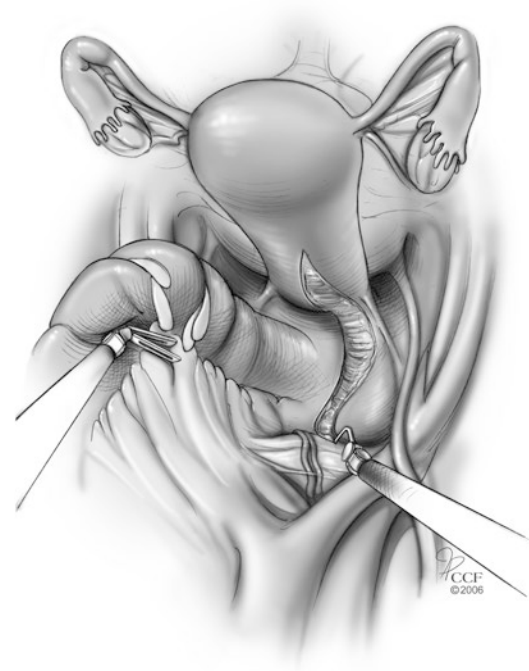


Fig. 8.18 In sacrouteropexy, the posterior peritoneum overlying the posterior vaginal cuff and cervix is incised longitudinally to the level of the sacral promontory. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2006–2014. All Rights Reserved)

Until recently, there were no randomized controlled trials comparing RASC to ASC or LSC. Paraiso et al. conducted a single center randomized trial comparing RASC to LSC [8]. In the RASC group, anesthesia time, total time in the operating room, total sacrocolpopexy time, and total suturing time were all significantly longer. Patients in the robotic group also had significantly higher pain at rest and with activity during weeks 3 through 5 after surgery and required longer use of nonsteroidal anti-inflammatory drugs.

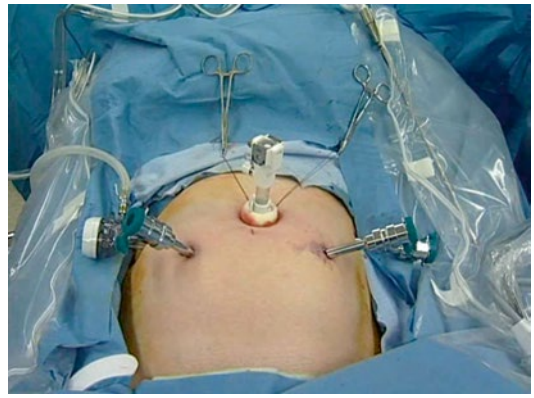


Fig. 8.19 Robotic port placement in W configuration

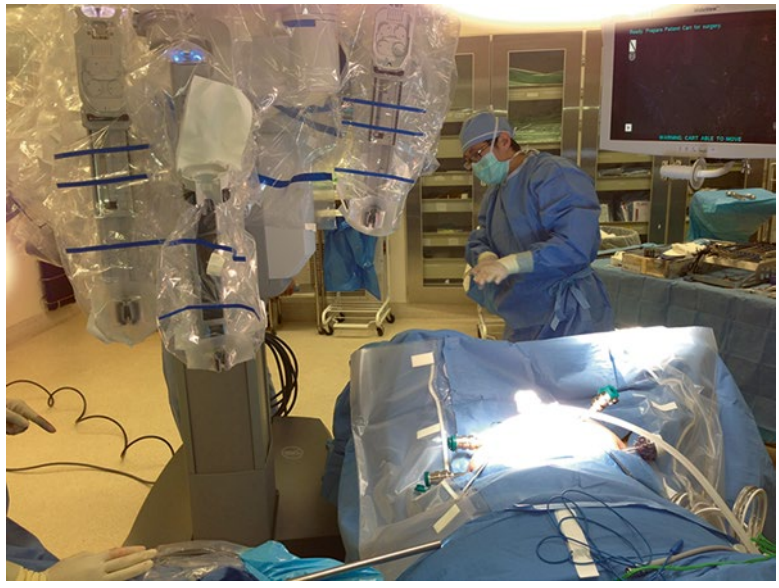


Fig. 8.20 Robot is docked laterally, abutting the base of the operating room table

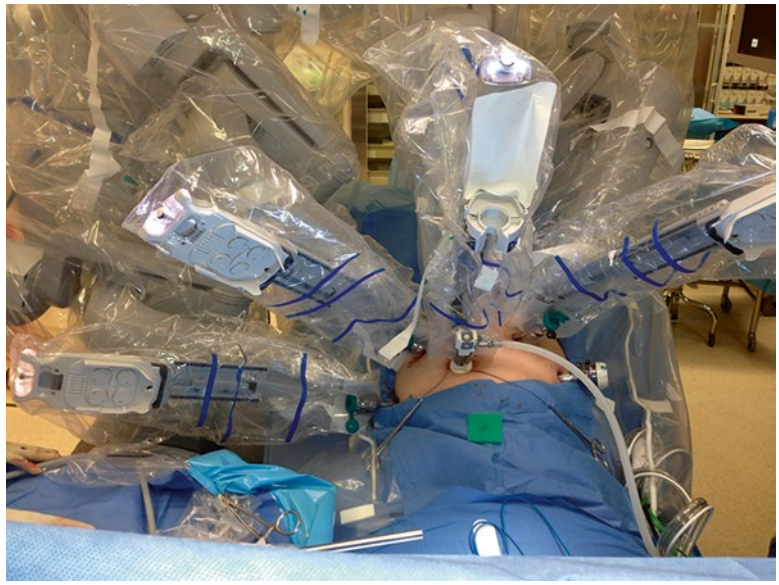


Fig. 8.21 The patient is placed in steep trendelenburg with lateral docking of the robotic ports

In addition, the robotic surgery was significantly more expensive than laparoscopy (mean difference +$1,936; $P = .008$). At 1 year there was no difference in improvement in vaginal support or functional outcomes between groups.

Summary

Over the last decade new minimally invasive laparoscopic and robotic techniques have evolved for the correction POP. All of these techniques aim to achieve the same durable success of the traditional open abdominal techniques while minimizing recovery, pain, blood loss, and hospital stays. The limited, available data suggests that laparoscopic and robotic outcomes are comparable to the open ASC but more expensive.

References

1. Wu JM, Hundley AF, Fulton RG, et al. Forecasting the prevalence of pelvic floor disorders in U.S. Women: 2010 to 2050. Obstet Gynecol. 2009;114:1278.
2. Brubaker L, Cundiff GW, Fine P, et al. Abdominal sacrocolpopexy with Burch colposuspension to reduce urinary stress incontinence. N Engl J Med. 2006;354:1557.
3. Nezhat CH, Nezhat F, Nezhat C. Laparoscopic sacral colpopexy for vaginal vault prolapse. Obstet Gynecol. 1994;84:885.
4. Nygaard I, Brubaker L, Zyczynski HM, et al. Long-term outcomes following abdominal sacrocolpopexy for pelvic organ prolapse. JAMA. 2013;309:2016.
5. Wei JT, Nygaard I, Richter HE, et al. A midurethral sling to reduce incontinence after vaginal prolapse repair. N Engl J Med. 2012;366:2358.
6. Ganatra AM, Rozet F, Sanchez-Salas R, et al. The current status of laparoscopic sacrocolpopexy: a review. Eur Urol. 2009;55:1089.
7. Nygaard IE, McCreery R, Brubaker L, et al. Abdominal sacrocolpopexy: a comprehensive review. Obstet Gynecol. 2004;104:805.
8. Paraiso MF, Jelovsek JE, Frick A, et al. Laparoscopic compared with robotic sacrocolpopexy for vaginal prolapse: a randomized controlled trial. Obstet Gynecol. 2011;118:1005.
9. McDermott CD, Hale DS. Abdominal, laparoscopic, and robotic surgery for pelvic organ prolapse. Obstet Gynecol Clin North Am. 2009;36:585.
10. Granese R, Candiani M, Perino A, et al. Laparoscopic sacrocolpopexy in the treatment of vaginal vault prolapse: 8 years experience. Eur J Obstet Gynecol Reprod Biol. 2009;146:227.
11. Paraiso MF, Walters MD, Rackley RR, et al. Laparoscopic and abdominal sacral colpopexies: a comparative cohort study. Am J Obstet Gynecol. 2005;192:1752.

12. Elliott DS, Frank I, Dimarco DS, et al. Gynecologic use of robotically assisted laparoscopy: sacrocolpopexy for the treatment of high-grade vaginal vault prolapse. Am J Surg. 2004;188:52S.
13. Geller EJ, Siddiqui NY, Wu JM, et al. Short-term outcomes of robotic sacrocolpopexy compared with abdominal sacrocolpopexy. Obstet Gynecol. 2008; 112:1201.
14. Geller EJ, Parnell BA, Dunivan GC. Robotic vs abdominal sacrocolpopexy: 44-month pelvic floor outcomes. Urology. 2012;79:532.

Transvaginal Urethrolysis for Urethral Obstruction

9

Melissa A. Laudano, James M. Weinberger, Rajveer S. Purohit, and Jerry G. Blaivas

Background

Bladder outlet obstruction (BOO) in women is rare, with an incidence ranging from 2.7 % to 8.3 % [1, 2]. However, these rates may underestimate the true incidence because diagnostic parameters and normative values to diagnose female BOO remain controversial. In addition, the presentation of female BOO can range widely from obstructive symptoms associated with voiding to irritative voiding symptoms. Given the variability in presentation and diagnosis, urologists must have a high suspicion for obstruction when managing patients with voiding dysfunction following surgical interventions for stress urinary incontinence (SUI).

M.A. Laudano, M.D. • R.S. Purohit, M.D., M.P.H.
Department of Urology, Weill Medical College of Cornell University, New York Presbyterian Hospital, New York, NY, USA
e-mail: Melissa.laudano@gmail.com; rajpu@yahoo.com

J.M. Weinberger
Institute for Bladder and Prostate Research, New York, NY, USA
e-mail: jmweinberger@mednet.ucla.edu

J.G. Blaivas, M.D. (✉)
Department of Urology, Weill Medical College of Cornell University, New York Presbyterian Hospital, New York, NY, USA

SUNY Downstate College of Medicine, New York, NY, USA
e-mail: jgblvs@gmail.com

Presentation

The difficulty in diagnosing female BOO is, in part, due to the variety of presenting symptoms. While it makes sense that women with urethral obstruction would present with voiding difficulties (hesitancy, decreased stream strength, incomplete emptying, or complete urinary retention), the fact remains that most women present with overactive bladder symptoms [3]. In the immediate, postoperative period many of these symptoms are expected but typically resolve within 4 weeks of surgery [3].

Etiology

We classify the etiology of female BOO into anatomic and functional causes. Anatomic causes may be extrinsic (prolapse, prior surgery, urethral diverticulum, tumor) or intrinsic (stricture, tumor). Functional causes include primary bladder neck obstruction, neurogenic detrusor-external sphincter dyssynergia, and acquired voiding dysfunction [4, 5].

Carr and Webster classified obstruction based on its location. They found that the most common cause of proximal urethral obstruction was extrinsic compression or an abnormal urethral angle [1]. Distal obstruction was typically a functional problem related to the inadequate relaxation of the muscles of the pelvic floor.

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_9,

Extrinsic compression can occur from nearby structures, such as the uterus, vagina, bladder, ureter; abnormal angulation due to an anti-incontinence procedure; or can be luminal due to stricture, neoplasm, foreign body, or urethral valve [1]. Iatrogenic obstruction following anti-incontinence surgery has been cited as the most common etiology of BOO with rates ranging from 2.5 % to 27 % [6–9].

Synthetic Mid-urethral Sling

A meta-analysis conducted by the American Urological Association Stress Urinary Incontinence (AUA SUI) Panel assessed outcomes following surgical management of female SUI. Rates of de novo urge incontinence (UI) at 12–23 months following surgical intervention for SUI in 323 females undergoing synthetic midurethral slings was 6 % [10]. The rate of retention, defined as retention lasting longer than 1 month or requiring intervention, was 3 %. The same retention rate existed following synthetic mid-urethral sling in women who underwent concomitant prolapse repair and those who did not [10]. Rates of retention were comparable to those reported in the TOMUS trial that randomized 597 women to retropubic midurethral sling (RMS) or transobturator midurethral sling (TMS). With 12 months of follow-up, this trial found rates of voiding dysfunction requiring surgery, use of catheter, or both to be 2.7 % in the RMS arm and 0 % in the TMS group [9].

Autologous Pubovaginal Sling

Based on the AUA Guideline Update review, the rate of de novo UI was 9 % for women who underwent autologous fascial sling without bone anchors [10]. The estimated retention rate following autologous fascial sling was 8 % for patients who did not receive concurrent prolapse repair and 5 % for women who did [10]. With 24 months of follow-up, the SISTEr Trial found that 20/326 (6 %) women who underwent autologous pubovaginal sling developed voiding dysfunction that necessitated surgical intervention [11]. Extended 5-year follow-up was reported on patients from the SISTEr Trial. Of the 183 women with 5-year follow-up after sling, 3 (1.6 %) developed de novo UI, 6 (3.3 %) required persistent catheterization, and 4 (2.2 %) required another surgical procedure for voiding dysfunction [6].

Burch Colposuspension

In the AUA Update review, the rate of de novo UI based on 695 women undergoing a Burch colposuspension (BC) was 8 % with 12–23 months of follow-up [10]. The rate of retention following BC was 3 % in women with no simultaneous prolapse treatment and 1 % in women with prolapse repair [10]. The SISTEr Trial found that with a follow-up of 24 months, 0/329 women who underwent a BC experienced voiding dysfunction leading to surgical revision [11]. The extended SISTEr Trial reported that of the 174 women with 5-year follow-up after BC, there were 7 (4 %) with de novo UI and 1 (0.6 %) patient who required persistent catheterization [6].

Marshall–Marchetti–Krantz

Retrospective reviews approximate rates of prolonged urinary retention following the Marshall–Marchetti–Krantz (MMK) procedure to range from 2 % to 4 % [12, 13]. In a retrospective analysis of 151 women treated for SUI with an MMK procedure, 8.7 % of patients had difficulty voiding/retention in the immediate postoperative period, but only 4 (3.8 %) had prolonged difficulty voiding that required urethral dilation. Mean follow-up in this study was 51.5 months [13].

Evaluation and Diagnosis

Several diagnostic tools, such as pressure-flow studies (PFS), video urodynamics, and nomograms combining several factors, have been proposed. Chassagne et al. used pressure-flow studies (PFS) of 35 women who were clinically

obstructed compared to 124 controls with stress urinary incontinence (SUI) to generate a definition of female BOO defined as having a maximum flow rate (Q_{max}) <15 ml/s and detrusor pressure at maximum flow rate ($P_{det}Q_{max}$) >20 cm H_2O [14]. A second study by Defreitas et al., compared 169 women with clinical anatomic BOO to 20 normal female controls without urologic complaints. The recommended cutoffs for BOO was $Q_{max} < 12$ ml/s and $P_{det}Q_{max} > 25$ cm H_2O [15]. Differences in normal values between series may also be related to the normal population used.

Nitti et al. described a combined radiographic and urodynamic approach to the diagnosis of female BOO [16]. In this study, obstruction was defined as radiographic evidence of obstruction between the bladder neck and distal urethra in the presence of a sustained detrusor contraction of any magnitude. Using this definition, patients who were identified as obstructed had a lower mean Q_{max}, higher mean $P_{det}Q_{max}$, and higher mean post-void residual (PVR). In 11.8 % of obstructed patients, Q_{max} was greater than 15 ml/s and in 10.5 % of obstructed patients $P_{det}Q_{max}$ was less than 20 cm H_2O [16]. As a result, the authors felt that video-urodynamics identified cases of obstruction that would have been missed using PFS alone [16].

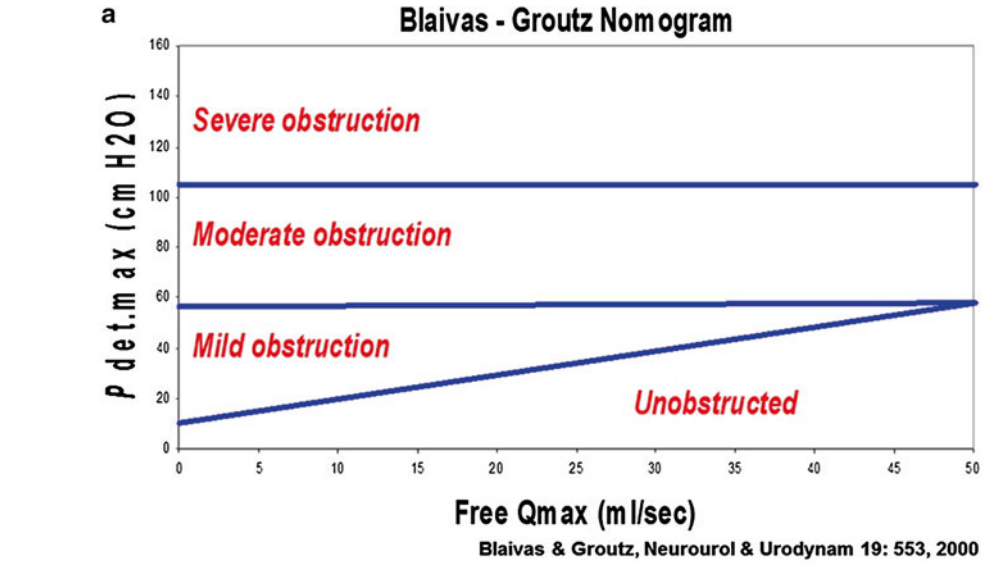
Finally, Blaivas and Groutz published a BOO nomogram for women with lower urinary tract symptoms (LUTS) that combined free-flow values, PFS, and voiding cystourethrography [2]. This nomogram stratified patients into four zones: no obstruction, mild obstruction, moderate obstruction, and severe obstruction, and had a positive correlation with subjective symptoms on AUA Symptom Index score (Fig. 9.1a–c) [2].

Techniques of Repair

There are two basic approaches to correcting urethral obstruction after incontinence surgery—sling incision/excision and urethrolysis. Urethral stricture is a very rare complication after incontinence surgery and will be discussed in Chap. 13. No matter what technique is used, there is always a fine balance between relieving obstruction and the development of recurrent sphincteric incontinence.

Sling Incision/Excision

Simple sling incision is the least invasive and most successful procedure for women who are obstructed after sling surgery. It is indicated as a primary procedure and can be done at any time after the original surgery when a synthetic sling has been done, but is usually reserved for those who are at least a month or two after the original surgery to lessen the likelihood of recurrent sphincteric incontinence. For patients who have undergone biologic slings, we prefer to wait at least 3 months. The technique is straightforward. The patient should be placed in the dorsal lithotomy or Trendelenburg position to achieve optimal visualization of the anterior vaginal overlying the urethra [17]. A Foley catheter should be placed and by placing it on traction, it is usually possible to palpate the sling which appears as a subtle transverse ridge. If that is not successful, urethroscopy with a cystoscope or placement of a urethral sound can aide in the identification of the constricting band by torqueing upward [18]. We prefer to make a transverse incision just distal to the sling; others describe an inverted-U or midline. Some reports advocate using the prior incision or making a lateral incision to release one side of the sling, but by the time we see the patient, the site of the old incision is not apparent [19]. The incision continues down to the surface of the normal urethra distal to the sling and the dissection proceeds proximally until the sling is identified. Sometimes it is obvious, sometimes subtle, and apparent only as a thickened scar. Once identified, we place an Allis clamp on the sling in the midline and exert traction. This takes some tension off the sling and facilitates the dissection between the urethra and sling. We prefer to do the entire dissection sharply with a Metzenbaum scissor (Fig. 9.2a). If the surgical plane is obvious, it can be continued with a fine blunt instrument like a right angle clamp, but great care should be exercised so that the urethra



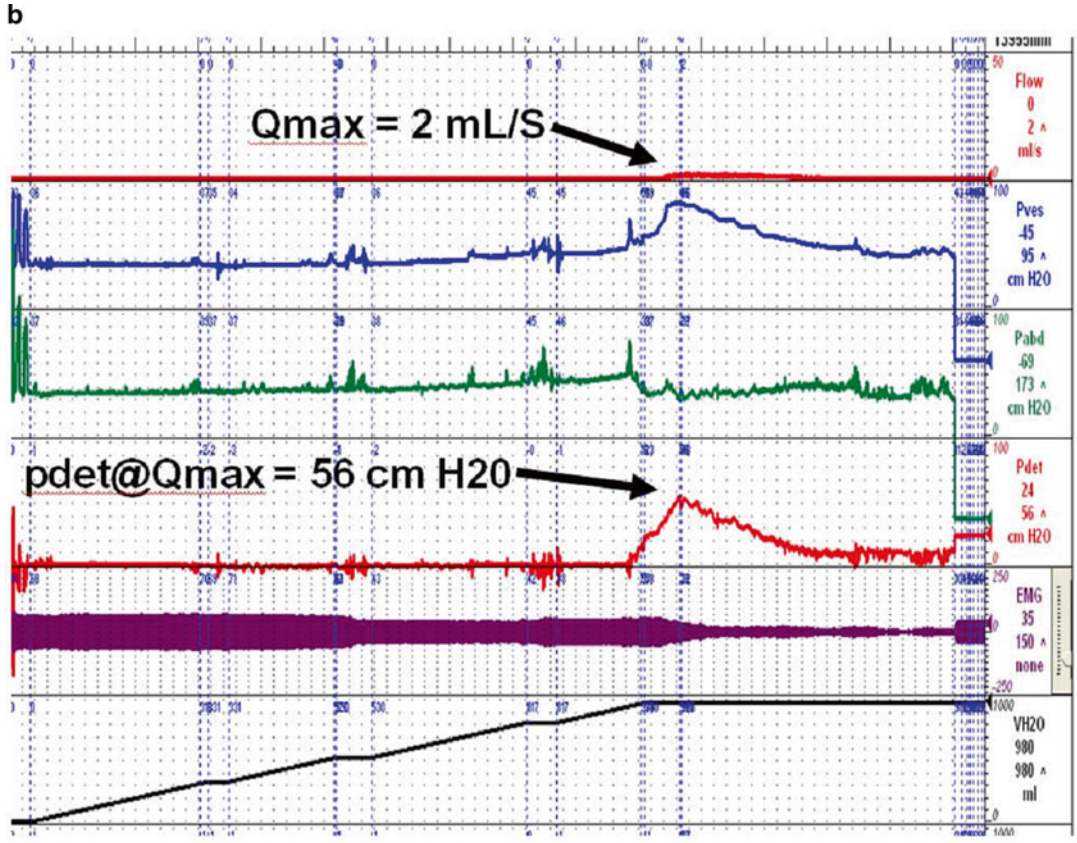


Fig. 9.1 (**a**) Blaivas–Groutz nomogram for bladder outlet obstruction. (**b**) Urodynamic tracing shows severe urethral obstruction (Type 2 in Blaivas-Groutz nomogram). Q_{max}: maximum flow rate; $P_{det}@Q_{max}$: detrusor pressure at maximum flow rate; P_{ves}: vesicle pressure; P_{abd}: abdominal pressure; P_{det}: detrusor pressure; EMG: electromyogram; V_{H2O}: volume of water. (**c**) X-ray exposed at Q_{max} shows obstruction in the distal third of the urethra. Patient underwent excision of the suburethral portion of the sling and subsequently voided normally (Q_{max}: 19 ml/s, voided volume: 150 ml, post-void residual: 49 ml), but developed sphincteric incontinence and underwent successful autologous fascial sling 4 months later. (**a**: Adapted with permission from Blaivas JG, Groutz A. Bladder outlet obstruction nomogram for women with lower urinary tract symptomatology. Neurourol and Urodynam 2000;19:553; **b**, **c**: Used with permission from Blaivas JG, Purohit RS, Weinberger JM, Tsui JF, Chouhan J, Sidhu R, Saleem K. Salvage Surgery after Failed Treatment of Synthetic Mesh Sling Complications. J Urol 2013; 190:1281–1286)

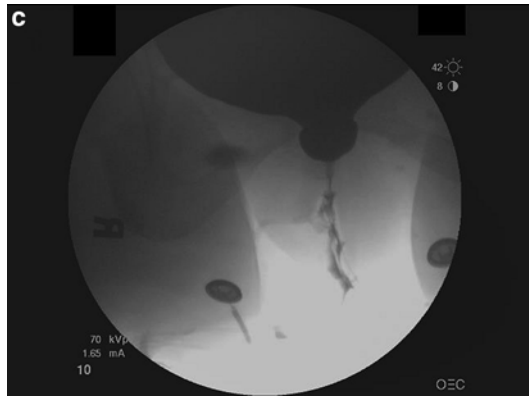


Fig. 9.1 (continued)

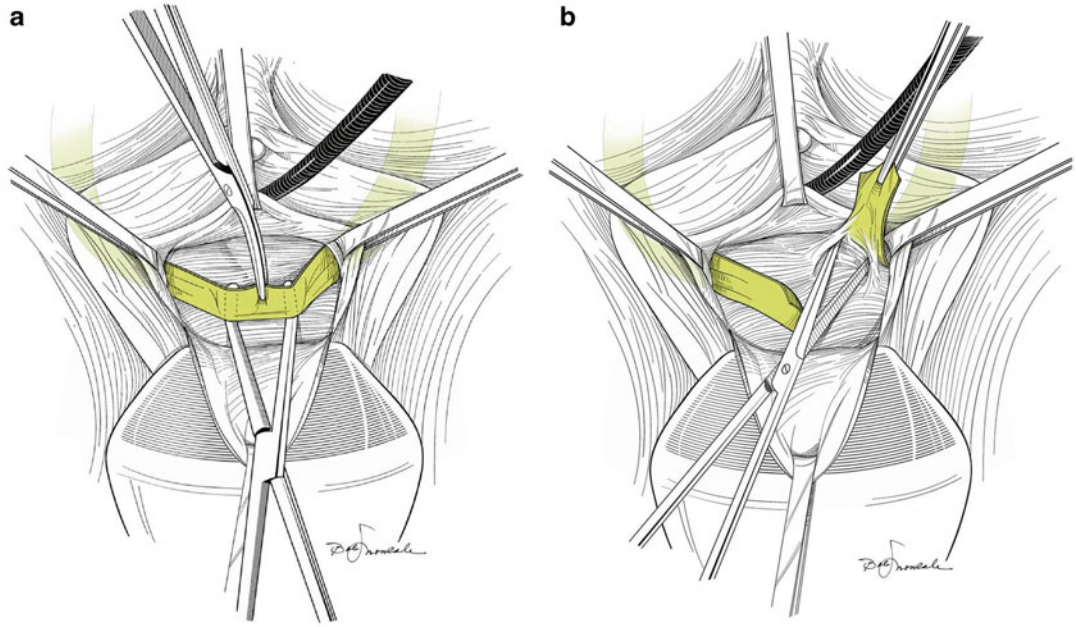


Fig. 9.2 (**a**) Once the sling has been dissected free of the urethra, a right angle clamp is placed between it and the urethra; the sling is transected with a scissors or knife. (**b**) Once the sling has been transected, it usually springs apart. If not, it should be sharply dissected off of the urethra. (Both used with permission from Nitti VW, Carlson KV, Blaivas JG, Dmochowski RR. Early results of pubovaginal sling lysis by midline sling incision. Urology 2002;59:47)

is not damaged. In some cases the sling has been incorporated in the wall of the urethra or has eroded into the lumen. In either instance, it is necessary to excise part of the urethral wall, and we believe the entire vaginal portion of the sling should be excised as well. The defect in the urethra repaired as necessary with absorbable monofilament or chromic sutures.

Once the sling is freed from the periurethral tissue, it is incised in the midline and the cut ends should spring apart (Fig. 9.2b). The ends of the sling material can be removed or left in place [18].

Gomelsky et al. [3] recommend that the suburethral portion of synthetic slings be excised at this point but that biologic slings be simply incised. We do not make such a distinction. If the cut edges of any type sling do not retract, we dissect them out lateral to the urethral wall on either side and then make a decision about whether or not to do more or less urethrolysis as described below.

If the sling cannot be identified by the technique described above, the dissection can be done lateral to the urethra from meatus to bladder neck if necessary and, using this method, we have always been able to identify the sling. At the conclusion of the procedure, a Foley catheter is left indwelling and in most cases, the patient is given an active voiding trial once she has fully recovered from anesthesia. The bladder is filled with saline until the patient is comfortably full, and then the catheter is removed. If she fails the trial, we would prefer to start intermittent self-catheterization, but so far, that has not been necessary in our experience. The patient is discharged home the same day [17].

Urethrolysis

Urethrolysis is indicated in women with obstruction due to periurethral scarring, usually after Burch colposuspension, Marshall–Marchetti–Krantz procedures, and in those who have undergone multiple prior urethral surgeries including sling incision and prior urethrolysis. It is rarely necessary after synthetic sling surgery unless there have been prior failed attempts at sling incision.

Urethrolysis may be performed by a variety of techniques—retropubic, transvaginal, and suprameatal. No matter what method is used, it is important to recognize that, unlike sling incision or excision, there is no clear-cut end point with urethrolysis; deciding when the dissection is complete is a matter of judgment and experience and must be individualized depending on operative findings. We begin the procedure by inserting first a Q-tip to assess urethral mobility and angle; then a Foley catheter is inserted to assess bladder neck mobility by pulling down on the catheter. As the periurethral dissection proceeds, we periodically pull down on the catheter and reinsert the Q-tip to assess the progress being made in freeing up the urethra. The goal of the surgery is to restore some, but not too much mobility. We have not found any measurements to be useful in this except that the Q-tip angle should be restored to 0 or a positive angle if it was negative to begin with.

Retropubic Approach

The retropubic approach was described in 1990 by Webster and Kreder, primarily for urethral obstruction after retropubic colposuspension or urethropexy [20]. Access to the retropubic space is gained through a low midline or Pfannensteil incision. The urethra, bladder neck, and anterior vaginal wall are freed from any adhesions with sharp dissection, including freeing of the urethra anteriorly away from the pubic bone. If there is severe scarring, the dissection can proceed laterally to the ischial tuberosities; however, this will leave a defect in the paravaginal region. An omental flap may be utilized in the space between the urethra and pubic bone to prevent future adhesions, especially in cases of recurrent obstruction after prior urethrolysis [20, 21].

Transvaginal Approach

Transvaginal urethrolysis can be performed through an inverted-U or midline incision that is made in the anterior vaginal wall over the mid urethra. A transverse incision is made over the midurethra or bladder neck as dictated by the local anatomy (Fig. 9.3a–d). The dissection is then carried proximally to the level of the bladder neck or distally to the meatus as necessary to free up the urethra. Unlike the simple sling incision, the dissection proceeds laterally, perforating the endopelvic fascia with a Metzenbaum scissors and the urethra is freed from its attachments to the vaginal sidewall and pubic symphysis as needed using blunt dissection with an index finger, with a Metzenbaum scissors, or right angle clamp. If necessary, it can be completely freed up circumferentially [3]. After circumferential urethrolysis, particularly if it is done as a tertiary procedure, a Martius graft can be mobilized and

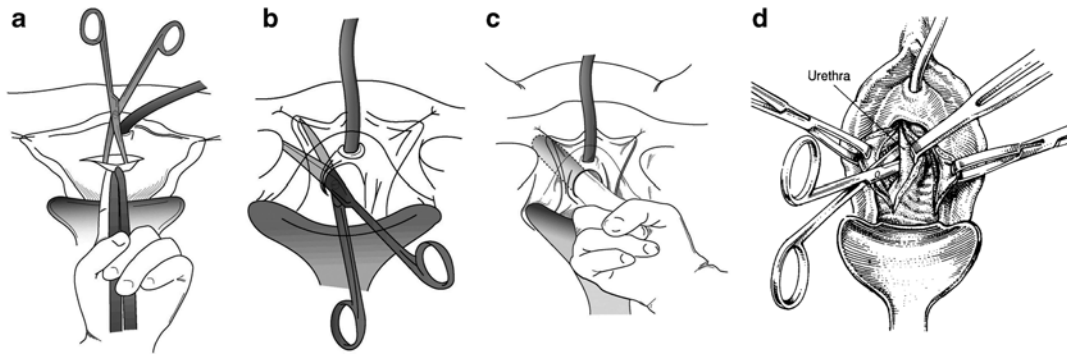


Fig. 9.3 (**a**) A transverse incision is made over the urethra and an Allis clamp placed on the proximal edge of the vaginal incision in the midline. With the non-dominant hand, traction is placed on the Allis clamp and the index finger holding the clamp pushes upward putting the vaginal wall on tension. A plane is dissected with a Metzenbaum scissors between the pubocervical fascia and the vaginal wall. (**b**) Keeping in the plane between the pubocervical fascia, with the curve of the scissors pointed laterally, the dissection proceeds in the direction of the patient's ipsilateral shoulder, hugging the undersurface of the pubis and ileum. The technique is not spread and cut, but rather by opening and closing the tips of the scissors, and you push against the bones. The retropubic space is entered sharply with the scissors or bluntly with an index finger. (**c**) Once the retropubic space is entered, the urethrolysis is completed, by dissecting with an index finger medially and laterally on the undersurface of the bone. (**d**) If a circumferential urethrolysis is necessary, the scissor is directed medially between the pubis and urethra on either side. Once a window is created, a penrose drain is passed around the urethra and, using it as traction, the dissection can be extended proximally or distally. (**a**, **b**, **c**: Used with permission from Blaivas JG, Chaikin. Pubovaginal fascial sling for the treatment of all types of stress urinary incontinence: surgical technique and long-term outcome. Urol Clin North Am 2011;387–15; **d**: Used with permission of Shlomo Raz, MD. With permission of and from Nitti VW, Raz S: Obstruction Following Anti-Incontinence Procedures Diagnosis and Treatment with Transvaginal Urethrolysis. J Urol 1994; 152:93–98)

tunneled between the labia and vaginal dissection. The graft is passed anteriorly to the urethra and wrapped circumferentially around to minimize scarring and recurrent obstruction [22]. In rare instances we have completely circumscribed the entire urethra from meatus to bladder neck. If that is done, we recommend a Martius flap and autologous sling; otherwise the patient will almost assuredly have severe sphincteric incontinence [23]. A urethroscopy is performed to ensure that no urethral injury occurred.

Suprameatal Approach

A variation on the transvaginal approach is the suprameatal technique, which begins with an inverted-U incision between the clitoris and urethral meatus (Fig. 9.4a–d). The apex of the U is anterior to the urethral meatus at the 12 o'clock position [3]. The dissection is done with a Metzenbaum scissors and it is generally easy to find a bloodless plane…for a while. At the proximal most portion of the dissection, there is sometimes a gush of bleeding which is controlled by finger pressure or by temporarily packing with a sponge. We have not found it necessary to look for individual bleeders (nor is it usually possible). It is usually possible to palpate retropubic slings and Burch sutures and to visualize them, but the space is too small to cut them under direct vision. We use a curved Mayo scissors with the curve pointed upward and cut the sling or sutures by feel. The remainder of the dissection and freeing up of the urethra is as described in the transvaginal approach. A Martius flap can also be performed in conjunction with this technique.

Outcomes in the Literature

Success rates for a formal urethrolysis (retropubic, transvaginal, or suprameatal) range from 43 % to 94 % [22, 24–31]. More recently, success rates ranging from 80 % to 100 % have been described for sling incision [8, 17–19]. A review

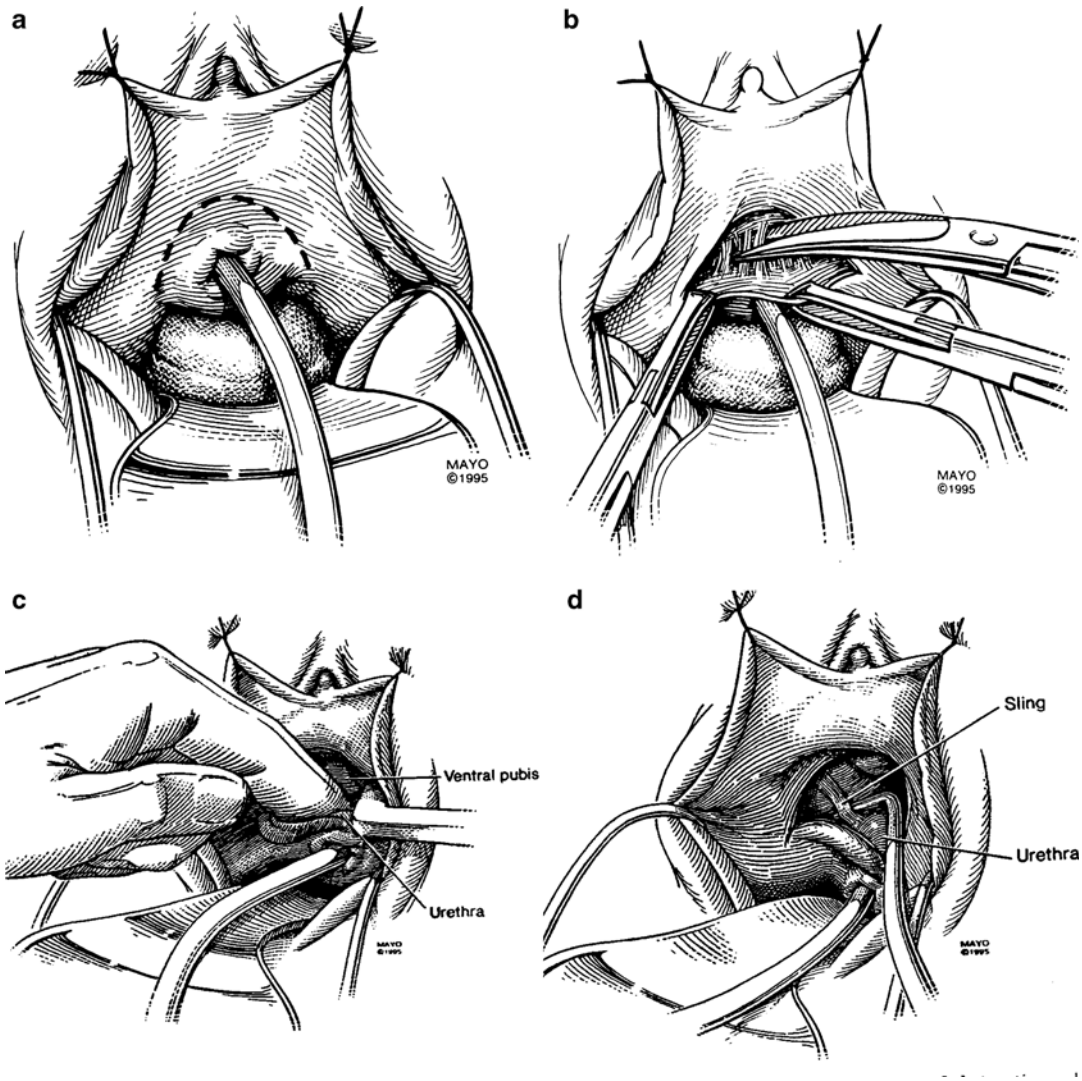


Fig. 9.4 (**a**) An inverted "U" incision is made 0.5–1 cm above the urethral meatus. (**b**).The pubo-urethral ligament is sharply taken down in the midline. (**c**) Finger dissection in the midline is utilized to further free up the urethra. Once the proximal surface of the pubis is reached, the retropubic space can be entered either bluntly or sharply, being careful to apply pressure in an upward direction against the bone so as not to injure the bladder neck. (**d**) Once the retropubic space has been entered, the sling or sutures from retropubic suspension are usually easily palpated, but may be difficult to visualize directly because of the narrow wound. It is usually possible to cut the sling or sutures blindly by palpation and cutting with the tips of the scissors pointing outward against the bone while the index finger of the non-dominant hand is pressing downward on the urethra (not pictured in this figure). (**a**–**d**: From Petrou S P, Brown J. A., Blaivas, J. G.: Suprameatal transvaginal urethrolysis. J Urol 1999; 161: 1268, 1999. Used by permission of Mayo Foundation for Medical Education and Research. All Rights Reserved)

of the literature yields many retrospective studies looking at various types of surgical intervention to relieve iatrogenic obstruction following anti-incontinence procedures in women, but the number of patients is small and so many different types of primary and secondary procedures have been done that it is not possible to come to any firm conclusions except that retropubic urethropexies are much more difficult to treat successfully. Of note, the definition of success varies by study. Many categorize patients by symptom improvement, either complete or partial.

Sling Incision

Goldman et al. described a simple sling incision for iatrogenic urethral obstruction in 14 women. In this retrospective analysis, 11 women had a biologic pubovaginal sling and 3 women had a synthetic midurethral sling. With 12 months of follow-up, 11/14 had complete resolution of symptoms and 2 had significant improvement. Overall, 93 % of women had a complete or significant improvement in symptoms. One patient went on to formal urethrolysis and one underwent surgery for recurrent SUI [17]. Croak et al. reported a retrospective analysis of five women who underwent sling incision after tension-free vaginal tape (TVT) placement for SUI. All women were able to void postoperatively; however, one woman had recurrent SUI and one had persistent dysuria following urethrolysis [8]. Nitti et al. presented a retrospective analysis of 19 women with both storage and voiding symptoms who underwent lysis of a pubovaginal sling. With 12 months follow-up, 84 % had resolution or improvement of their presenting symptoms. SUI recurred in 17 % of women and three women went on to require a formal urethrolysis [18]. Kusada also described five patients who underwent incision of a pubovaginal sling for obstructive symptoms that resolved in all patients [19]. Amundsen et al. reported outcomes on a retrospective series combining sling incision and transvaginal urethrolysis. Success rates for complaints related to voiding were 94 % and 67 % for complaints related to storage [25]. These rates were not subdivided by type of urethrolysis.

Urethrolysis: Retropubic

Anger et al. performed a retrospective review of 16 women who underwent either transvaginal (7 patients) or retropubic urethrolysis (9 patients) after obstructed BC. With a mean follow-up of 7 months, 43 % of women who underwent transvaginal urethrolysis had resolution of obstructive or irritative symptoms compared to 78 % who underwent a retropubic approach [24]. One woman in the transvaginal and one in the retropubic had recurrent SUI [24]. A second retrospective review reported by Petrou and Young included 12 women who underwent retropubic urethrolysis. Ten women felt that their obstruction had been successfully relieved, and two women had improvement but did not characterize it as a success [26]. Three women (25 %) had new or persistent SUI after urethrolysis and two (17 %) had urge incontinence [26]. A second study comparing retropubic versus transvaginal urethrolysis was conducted by Carr and Webster. In this retrospective review of 51 consecutive women who underwent urethrolysis, 35 women underwent the retropubic approach, 15 women the vaginal approach, and 4 women had an infrapubic approach. Success, defined as cure or improvement in symptoms, occurred in 86 % of women who underwent the retropubic lysis and 73 % for vaginal (no statistical difference by technique) [27].

Urethrolysis: Transvaginal

In addition to the studies comparing the transvaginal approach to a retropubic approach that are described above, several analyses were performed on series including only transvaginal urethrolysis. A retrospective review performed by Carey et al. examined 23 women who underwent transvaginal urethrolysis with Martius labial fat pad grafting. With this technique, 20/23 (87 %) of women had complete resolution of symptoms and 3/23 continued to require CIC with a mean follow-up of 15 months [22]. SUI occurred de novo following transvaginal urethrolysis in three women (16 %) and persisted in three women. De novo urge incontinence occurred in 20 % of patients and persisted in 37 % [22]. Goldman et al. reported on 31 patients treated with transvaginal urethrolysis without resuspension. Of these women, 26 (84 %) voided well or had significant improvement in symptoms. SUI persisted in 19 % of women [28]. A third analysis performed by Cross et al. analyzed outcomes following 39 transvaginal urethrolysis procedures for urethral obstruction. With 16 months of follow-up, obstructive symptoms resolved in

84 % of women and UI resolved in 79 %. One patient ultimately required resuspension for SUI and two patients required a secondary urethrolysis procedure [29]. Finally, Austin et al. reported a series of 16 women who underwent transvaginal urethrolysis for irritative and obstructive symptoms. Of these women, 69 % had improvement in irritative symptoms [30]. Three women underwent urethrolysis for obstructive symptoms and only one was able to successfully urinate following the urethrolysis. Two women were scheduled for repeat urethrolysis [30].

Urethrolysis: Suprameatal

With regard to the suprameatal approach, Petrou et al. reported outcomes of 32 consecutive women treated with transvaginal suprameatal urethrolysis who presented with retention or irritative voiding symptoms. Obstructive symptoms resolved in 65 % of women and urgency resolved in 67 %. One patient developed SUI [31].

Repeat Urethrolysis

A review of the literature yields many series that reported success rates of urethrolysis by approach. Another factor to consider when discussing success rates is whether the patient had a prior failed urethrolysis. Scarpero et al. presented a retrospective review of 24 women who underwent repeat urethrolysis for iatrogenic BOO [32]. The first attempted urethrolysis was transvaginal in 23 women and retropubic in 1 woman. The repeat urethrolysis was retropubic in 12 women, transvaginal in 10, and a combined approach in 2 cases. Repeat urethrolysis restored normal emptying with relief of symptoms and normalization of PVR in 22 women (92 %) with a mean follow-up of 14 months. Urge incontinence was completely resolved in 12 %, improved in 69 %, and remained unchanged in 19 %. Stress incontinence recurred in 4/22 (18 %) and persisted in 2/22 (9 %) [32]. Based on these results the authors concluded that a repeat urethrolysis can successfully treat obstructive symptoms; however, rates of improvement of storage symptoms were less promising [32].

Timing of Urethrolysis

In addition to controversy regarding the diagnosis of BOO in women, debate also exists about when to intervene once the diagnosis has been made. Many authors would advocate repair if voiding dysfunction persists for 3 months postoperatively; however, this is an arbitrary waiting period [3]. There is some concern that persistent obstruction could lead to permanent damage to the bladder and continued voiding dysfunction even after urethrolysis. This hypothesis was tested by Leng et al. [33] who performed a retrospective analysis of 15 patients who underwent transvaginal urethrolysis for post-sling voiding dysfunction. Seven patients underwent urethrolysis and had no persistent bladder storage or voiding complaints. An additional eight patients underwent urethrolysis and subsequently required anticholinergics to control significant urgency. Mean time to urethrolysis in those patients who had resolution of symptoms was 9.0 ± 10.1 months, compared to 31.3 ± 21.9 months in patients who continued to complain of urgency ($p = 0.02$). The mean follow-up was 17 months. The authors concluded that prolonged time delay to treatment of obstruction might lead to persistent storage symptoms, such as frequency and urgency. Of note, this is a small, retrospective cohort that overall had a higher than average time delay to treatment of obstruction in both groups [33].

Outcome Measures

Diagnosis of urethral obstruction prompting urethrolysis is typically a clinical diagnosis made by urodynamic findings such as a combination of high detrusor pressure and low flow (Q), or findings on exam such as urethral angulation or tethering. It is also often based on clinical history by correlating the development of de novo voiding

symptoms after anti-incontinence surgery [31, 34]. Studies describing resolution of post-urethrolysis symptoms typically employ a global instrument such as a patient reported outcome measure or an un-validated assessment of pre- and postoperative symptoms. Reports with objective quantification of outcomes with respect to urinary symptoms through symptom questionnaires are rare. Change in PVR has been used as an outcome measure in conjunction with a subjective assessment of relief of storage and voiding symptoms [17, 24]. Other studies define success simply by the ability to void with little to no PVR and patient reported symptom resolution [18]. Success has also been defined in the literature as patient reported resolution of the presenting symptom even in the absence of any additional clinical information (e.g., flow rate or residual). In this context, standardization of comparative postoperative outcomes is difficult.

A standardized outcome measure would aid in comparing outcomes with different techniques for urethrolysis. An ideal standardized outcome measure would assess Q & PVR pre- and postoperatively and incorporate urodynamic criteria, a validated measure of symptoms as well as patient reported outcome measures. Further, some symptoms, such as the ability to void only in a certain position, may not be amenable to being progressively graded. To account for a symptom that cannot be measured quantitatively, a unified outcome measure may include the Patient Global Impression of Improvement (PGI-I) in order to encompass patients' subjective assessment of their health. In this way, a physician may be able to attribute success or failure in a manner that is specific and therefore meaningful to other physicians.

Complications in the Literature

Recurrent Stress Incontinence

Recurrent SUI is a known complication of urethrolysis. Rates of recurrent SUI vary by study and depend on several factors. Some studies include patients who are already experiencing SUI prior to urethrolysis and others perform concomitant resuspension to prevent SUI. Nonetheless, recurrence of SUI after urethrolysis occurred in 3–25 % of patients [22, 26, 31]. Specific rates of recurrent SUI by urethrolysis approach are outlined in the outcomes section.

Overactive Bladder

Another complication that is somewhat difficult to quantify in terms of prevalence is overactive bladder or storage symptoms following urethrolysis. Many patients experience storage symptoms, such as urgency, frequency, urge incontinence, or dysuria prior to urethrolysis. In fact, these symptoms may be the reason the women seeks evaluation after the original anti-incontinence procedure. Rates of persistent storage symptoms range from 10 % to over 50 % [18, 35]. One study by Carey et al. reported the rate of de novo urge incontinence after transvaginal urethrolysis to be 20 % [22]. A retrospective, case-control study by Starkman et al. looked specifically at the prevalence of overactive bladder symptoms following urethrolysis in 40 women. OAB persisted in 56 % of women in this cohort [35]. The study cohort was stratified into two groups: persistent OAB and resolved OAB. Patients who had persistent OAB symptoms were more likely to demonstrate detrusor overactivity during urodynamics; however, there was no difference in symptoms on presentation, rates of elevated PVRs, or rates of CIC between the groups. Not surprisingly, patients with persistent OAB symptoms reported greater symptom bother and decreased improvement in quality of life [35]. Time to urethrolysis did not alter rates of persistent OAB symptoms, unlike the findings in the Leng et al. study [33].

Conclusions

Bladder outlet obstruction (BOO) in women is rare, with an incidence ranging from 2.7 % to 8.3 % [1, 2]. The presentation ranges widely from obstructive symptoms with voiding to irritative

storage symptoms. Given this complex presentation, the combination of several diagnostic tools, such as pressure-flow studies (PFS), video urodynamics, and nomograms, has been recommended. Potential treatment options range from excision of the sling to a complete urethrolysis through a retropubic, transvaginal, or suprameatal approach. Complications following urethrolysis include recurrent stress urinary incontinence and overactive bladder symptoms. Given the complexity of these cases, patients should be closely followed for resolution of symptoms following urethrolysis.

References

1. Carr LK, Webster GD. Bladder outlet obstruction in women. Urol Clin North Am. 1996;23:385.
2. Blaivas JG, Groutz A. Bladder outlet obstruction nomogram for women with lower urinary tract symptomatology. Neurourol Urodyn. 2000;19:553.
3. Gomelsky A, Nitti VW, Dmochowski RR. Management of obstructive voiding dysfunction after incontinence surgery: lessons learned. Urology. 2003;62:391.
4. Groutz A, Blaivas JG. Non-neurogenic female voiding dysfunction. Curr Opin Urol. 2002;12:311.
5. Groutz A, Blaivas JG, Chaikin DC. Bladder outlet obstruction in women: definition and characteristics. Neurourol Urodyn. 2000;19:213.
6. Brubaker L, Richter HE, Norton PA, et al. 5-year continence rates, satisfaction and adverse events of burch urethropexy and fascial sling surgery for urinary incontinence. J Urol. 2012;187:1324.
7. Klutke C, Siegel S, Carlin B, et al. Urinary retention after tension-free vaginal tape procedure: incidence and treatment. Urology. 2001;58:697.
8. Croak AJ, Schulte V, Peron S, et al. Transvaginal tape lysis for urinary obstruction after tension-free vaginal tape placement. J Urol. 2003;169:2238.
9. Richter HE, Albo ME, Zyczynski HM, et al. Retropubic versus transobturator midurethral slings for stress incontinence. N Engl J Med. 2010;362:2066.
10. Dmochowski RR, Blaivas JM, Gormley EA, et al. Update of AUA guideline on the surgical management of female stress urinary incontinence. J Urol. 2010;183:1906.
11. Albo ME, Richter HE, Brubaker L, et al. Burch colposuspension versus fascial sling to reduce urinary stress incontinence. N Engl J Med. 2007;356:2143.
12. McDuffie Jr RW, Litin RB, Blundon KE. Urethrovesical suspension (Marshall-Marchetti-Krantz). Experience with 204 cases. Am J Surg. 1981;141:297.
13. Zorzos I, Paterson PJ. Quality of life after a Marshall-Marchetti-Krantz procedure for stress urinary incontinence. J Urol. 1996;155:259.
14. Chassagne S, Bernier PA, Haab F, et al. Proposed cut-off values to define bladder outlet obstruction in women. Urology. 1998;51:408.
15. Defreitas GA, Zimmern PE, Lemack GE, et al. Refining diagnosis of anatomic female bladder outlet obstruction: comparison of pressure-flow study parameters in clinically obstructed women with those of normal controls. Urology. 2004;64:675.
16. Nitti VW, Tu LM, Gitlin J. Diagnosing bladder outlet obstruction in women. J Urol. 1999;161:1535.
17. Goldman HB. Simple sling incision for the treatment of iatrogenic urethral obstruction. Urology. 2003; 62:714.
18. Nitti VW, Carlson KV, Blaivas JG, et al. Early results of pubovaginal sling lysis by midline sling incision. Urology. 2002;59:47.
19. Kusuda L. Simple release of pubovaginal sling. Urology. 2001;57:358.
20. Webster GD, Kreder KJ. Voiding dysfunction following cystourethropexy: its evaluation and management. J Urol. 1990;144:670.
21. Scarpero HM, Nitti VW. Management of urinary retention and obstruction following surgery for stress urinary incontinence. Curr Urol Rep. 2002;3:354.
22. Carey JM, Chon JK, Leach GE. Urethrolysis with Martius labial fat pad graft for iatrogenic bladder outlet obstruction. Urology. 2003;61:21.
23. Blaivas JG, Purohit RS. Post-traumatic female urethral reconstruction. Curr Urol Rep. 2008;9:397.
24. Anger JT, Amundsen CL, Webster GD. Obstruction after Burch colposuspension: a return to retropubic urethrolysis. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:455.
25. Amundsen CL, Guralnick ML, Webster GD. Variations in strategy for the treatment of urethral obstruction after a pubovaginal sling procedure. J Urol. 2000;164:434.
26. Petrou SP, Young PR. Rate of recurrent stress urinary incontinence after retropubic urethrolysis. J Urol. 2002;167:613.
27. Carr LK, Webster GD. Voiding dysfunction following incontinence surgery: diagnosis and treatment with retropubic or vaginal urethrolysis. J Urol. 1997;157:821.
28. Goldman HB, Rackley RR, Appell RA. The efficacy of urethrolysis without re-suspension for iatrogenic urethral obstruction. J Urol. 1999;161:196.
29. Cross CA, Cespedes RD, English SF, et al. Transvaginal urethrolysis for urethral obstruction after anti-incontinence surgery. J Urol. 1998;159:1199.
30. Austin P, Spyropoulos E, Lotenfoe R, et al. Urethral obstruction after anti-incontinence surgery in women: evaluation, methodology, and surgical results. Urology. 1996;47:890.
31. Petrou SP, Brown JA, Blaivas JG. Suprameatal transvaginal urethrolysis. J Urol. 1999;161:1268.

32. Scarpero HM, Dmochowski RR, Nitti VW. Repeat urethrolysis after failed urethrolysis for iatrogenic obstruction. J Urol. 2003;169:1013.
33. Leng WW, Davies BJ, Tarin T, et al. Delayed treatment of bladder outlet obstruction after sling surgery: association with irreversible bladder dysfunction. J Urol. 2004;172:1379.
34. Goldman HB. Urethrolysis. Urol Clin North Am. 2011;38:31.
35. Starkman JS, Duffy III JW, Wolter CE, et al. The evolution of obstruction induced overactive bladder symptoms following urethrolysis for female bladder outlet obstruction. J Urol. 2008;179:1018.

Vaginal Fistula Repairs

10

Forrest C. Jellison and Shlomo Raz

Introduction

A vaginal fistula is defined by extra-anatomic communication between the vagina and an epithelial lined organ. This communication in the vagina may be straight forward with an abnormal connection to and involve the ureters, bladder, urethra, and rectum. A complex vaginal fistula might involve several different abdominal and pelvic organs which emphasize the importance of a complete workup before attempting to repair.

A vaginal fistula negatively affects an individual's quality of life and prompt repair is suitable in most patients that are properly selected. There are many surgical approaches to repair a fistula, the best repair centers on the first procedure being the most effective. However, there is a lack of high quality comparative studies to guide the surgeon. When selecting the initial surgical approach the surgeon should consider their expertise and experience after a thorough evaluation to determine the etiology and location of the fistula. In this chapter we will focus on the etiology, diagnosis, evaluation, and surgical repair of vesicovaginal (VVF), urethrovaginal (UVF), and rectovaginal fistula (RVF).

F.C. Jellison, M.D.
Female Pelvic Medicine and Reconstructive Surgery, Department of Urology, San Antonio Military Medical Center (SAMMC), Uniformed Services University of the Health Sciences, Fort Sam Houston, TX, USA
e-mail: fjellison@gmail.com

S. Raz, M.D. (✉)
Department of Urology, Division of Pelvic Medicine and Reconstructive Surgery, UCLA School of Medicine, Los Angeles, CA, USA
e-mail: sraz@mednet.ucla.edu

General Principles of Fistula Repair

This chapter provides an overview of the principles for successful repair which involve the timing, condition of the patient, vaginal access, surgical approach, use of adjuvant procedures, and evaluation. The majority of fistulas encountered in developed countries are iatrogenic and will be the focus of this chapter.

Timing

Timing of repair begins with an assessment of risk factors for poor healing (malnutrition, radiation, immunosuppression, or vaginal atrophy) that should be corrected when possible before proceeding with repair. The timing of repair depends mostly on the etiology of the fistula and the experience and comfort level of the surgeon. The surgical timing and approach is individualized for every patient. The first repair should be the most successful in the surgeon's

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_10,

experience, since it is the most important in establishing successful results. Most fistulas are found after the acute period and traditionally it was thought that a period of about 6 months was required for tissue swelling and infection to resolve. However, by allowing edema and inflammation to resolve, this would promote tissue healing so that an optimal repair could be performed. There are similar outcomes with early or delayed repairs with several reports where fistula repair can be performed successfully after 2 weeks [1–3]. The prerequisite for early repair is no obvious infection and that patients do not have an ischemic fistula from radiation or obstructed labor which can impede healing from viability of tissue margins that vary with time.

In the cases of radiation-induced fistula, it is advisable to allow for tissue stabilization, although many fistulas present late when there is no active progression. Assuming there is no infection and the tissue has stabilized, the patient can proceed to surgical reconstruction. Radiation results in extensive tissue ischemia and the reported failure rates can be up to nearly 50 % [4]. Due to high failure rates, adjuvant procedures involving tissue interposition should be performed. Consideration of temporary fecal diversion or in severe cases permanent urinary or fecal diversion may be warranted. Additional findings of diminished bladder capacity are common with the fistula typically located in immobile region of the bladder trigone and may involve the ureteric orifices requiring an abdominal approach to perform bladder augmentation and/or ureteric reimplant.

Special consideration should be given to RVF from Crohn's disease by first contemplating medical therapy before proceeding to surgical repair. Medical therapy with antitumor necrosis factor therapy has a reported success rate of 60 % at 1 year, but this declines to 36 % at long-term follow-up with similar unsatisfactory results from other studies [5, 6]. Other medical treatments include 6-mercaptopurine, and cyclosporine with limited success [7, 8]. Even with advances in medical therapy, surgical repair is the primary basis for long-term cure.

Abdominal or Vaginal Approach, Combined

The goal of surgical repair is to have a durable repair with the least morbidity while preserving continence in the case of urethrovaginal fistula. In deciding the surgical procedure, consideration is made to the location of the fistula, number, size, etiology, quality of the surrounding tissue, and vaginal access all which could limit or change aspects of the surgery. Principles of repair regardless of approach include nonoverlapping sutures, tension-free approximation of tissue, avoid devitalizing of the tissue, removal of foreign bodies, good hemostasis of the surgical field, water tight, multilayer closure with or without interposition of tissue, and postoperative bladder and urethra drainage or fecal diversion. An infratrigonal VVF is typically approached vaginally. While a supratrigonal fistula or difficult vaginal access may be considered a limitation to be approached vaginally by some surgeons, although in our experience this can be repaired vaginally when the fistula is not complex and there is no history of radiation [3, 9]. Other options include the abdominal approach (open or robotic). An indication for an abdominal or robotic approach is based on surgeon preference or when there is a need for concomitant bladder augmentation, ureteric reimplant, intraperitoneal pathology, or bowel diversion.

Both abdominal and vaginal repair of VVF are well established and have excellent success rates with each approach having its advantages [10]. Laparoscopic and robotic repairs have been reported in small case series at centers of excellence with encouraging results, but further study may be warranted before wide scale adoption [11–13]. The advantages of a vaginal approach are decreased morbidity associated with shorter hospitalization; the majority of repairs are performed at an ambulatory surgical center with less than 24 h stay, and decreased complications due to the minimally invasive approach which avoids intraperitoneal injury and large bladder incision. In deciding the approach, a surgeon has to consider their experience, comfort, and familiarity with each approach.

Urethrovaginal fistula repair is tailored by the location, size, and symptoms. A fistula located in the distal urethra may only need an incision of the distal urethra or observation if asymptomatic. If the fistula is large in size (>1–2 cm), radiation induced, or tissue is necrotic, an inflamed use of an interpositional tissue flap is recommended. Interposition with a martius flap is the preferred method due to the location to the urethra and ease in dissection with minimal complications. In cases of extensive urethral destruction, complex reconstruction may require rotational vaginal or labial flaps, neo-urethral reconstruction, autologous fascia sling or bladder neck reconstruction.

As in other urinary fistula, there are no formal guidelines for RVF repair. Most urologists or gynecologists repair RVF vaginally, while colorectal surgeons are more familiar with transanal or abdominal repair. Most of the rectovaginal fistulae are easily accessed by the vaginal route with the abdominal approach reserved for sigmoid colon and proximal rectal fistula. There is limited experience with minimally invasive treatment with fibrin glue or endoscopic management [14]. We will focus on vaginal repair which avoids the morbidity of an abdominal surgery.

Before embarking on vaginal repair, there should be consideration to the fistula location, sphincteric function, quality of the tissues due to radiation or prior surgeries, and if concomitant abdominal pathology or the need of a diverting colostomy needs to be addressed. A high fistula is not an absolute indication for an abdominal repair. A vaginal approach allows for simultaneous anal sphincter reconstruction. The surgeon's expertise and familiarity should be considered for each case.

Concomitant Procedures

Vaginal fistula repair may involve concomitant procedures that depend on the surgical setting. These procedures include autologous fascia sling, ureteric reimplantation, bladder augmentation, fecal diversion, and adjuvant tissue interposition. A simple tissue interposition repair may involve vaginal, peritoneal, or martius flaps or in cases of complex repair require rotational labial flaps, inner thigh rotational or island flaps, omental, gracilis, or myocutaneous flaps.

Stress incontinence after successful VVF repair may cause distress and lead the patient to believe their surgery was a failure. Preoperative evaluation and education is important, so patients understand their surgical options. Concomitant anti-incontinence procedures can be performed with fistula repair and do not increase the risk of recurrence, although it may be better to stage the anti-incontinence procedures [15]. In select instances, we would consider placement of a fascial sling at the time of VVF repair, but in the majority of cases the procedure is staged because even temporary outlet obstruction can lead to fistula recurrence. A synthetic sling is not recommended as it is a foreign body and may lead to fistula recurrence. Our preference is to stage an anti-incontinence procedure to prevent the risk of high pressure voiding that may result in fistula recurrence.

The approach is similar with UVF where in the majority of cases the sling procedure is staged to prevent development of urethral obstruction and the potential increase risk of fistula recurrence. The condition where there is less controversy about placing a fascial sling is when the fistula involves the mid-urethra sphincteric complex or distal third of urethra and there is suspicion that the patient will be incontinent postoperatively. The sling would be placed proximal to the repair at the bladder neck. Some have successfully reported concomitant autologous fascial sling at the time of a fistula repair, while we have not placed a sling distal to our urethral repair due to risk of the sling creating obstruction and high pressure voiding that may result in fistula recurrence.

Ureteric Reimplant and Bladder Augmentation

Diagnostic evaluation can determine the need for additional bladder or ureteral surgery which would require an abdominal approach. An abdominal approach is indicated when there is ureteric obstruction or fistula which would require

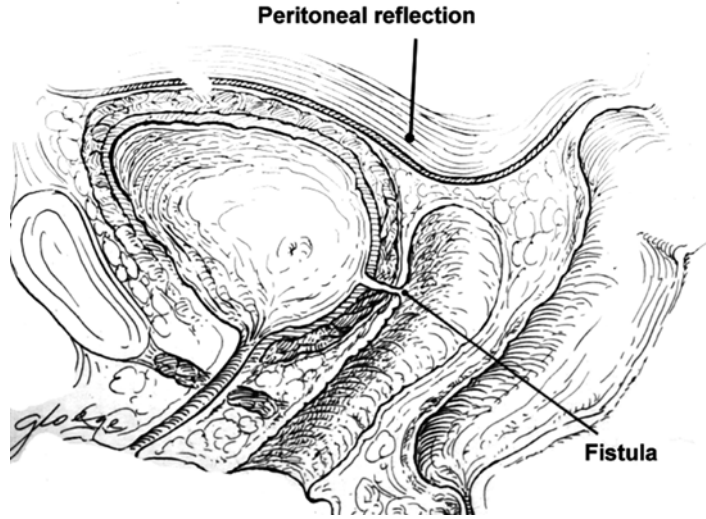


Fig. 10.1 The peritoneum is located near a post hysterectomy vesicovaginal fistula (Copyright © Shlomo Raz, MD)

ureteric reimplantation. Placement of preoperative ureteral stents when a VVF is located near the ureteric orifice may avoid ureteric reimplant. In cases of a small and contracted bladder capacity, an augmentation should be performed.

Fecal Diversion

The decision to perform a temporary diverting colostomy or ileostomy is made on an individual basis as there are no absolute indications when considering a RVF repair. The surgeon may elect to divert stool with complex fistula that are radiation induced, recurrent, large, or a result of malignancy. The diversion is taken down 3–6 months postoperatively after successful repair.

Tissue Interposition-Peritoneal, Martius, Labial, Gluteal Flaps (Inner Thigh Rotational Flaps Based on the Internal Pudendal Artery)

Vaginal repair and reconstruction is complex and requires many techniques to be in the surgeon's armamentarium. Successful repair consists of several layers in the closure of the fistula. The use of interpositional tissue is advised when the fistula is complex, large, a history of radiation, tissue is inflamed, or closure is suboptimal. There are differing opinions when tissue interposition is necessary as there are no definitive indications. Evan et al. in a retrospective study found improved success rates of VVF repair with interpositional flap [16]. There are several described flaps that can be used for interposition. After a hysterectomy the location of the VVF is often at the vaginal cuff and we routinely use a peritoneal flap due to its ideal location, ease of dissection, and it maintains a reliable vascular supply (Fig. 10.1). The results have been excellent with a peritoneal flap with 96 % success rate [9]. A peritoneal flap is an appropriate choice for supratrigonal fistula and in the case of a distal fistula it should be repaired with a martius flap due to its location. Successful repair has been reported at 97 % with a martius flap [9]. The martius flap is well vascularized with the blood supply located superiorly by the external pudendal artery, laterally by the obturator artery, and inferiorly by the posterior labial branches of the internal pudendal artery by which it is usually based (Fig. 10.2). The martius flap is mobilized by transection of the superior and lateral pedicles and its blood supply is based on the inferior pedicle in the majority of cases. Successful repair is subject to adequate mobilization so that the flap is off tension without

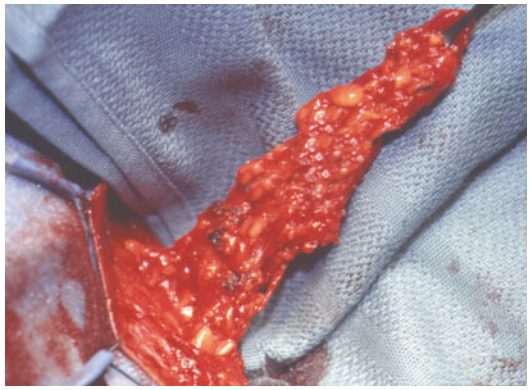


Fig. 10.2 Martius flap based on its inferior pedicle, posterior labial branches of the internal pudendal artery (Copyright © Shlomo Raz, MD)

compromising its blood supply. Disadvantages of a martius flap are that it may not reach a proximal fistula without compromising its viability or resulting in vaginal shortening [9]. Although the surgical harvest of a martius flap is feasible, complications such as hematoma, pain/numbness at surgical site, cosmetic deformity, and sexual dysfunction can rarely occur.

Rotational labial and inner thigh rotational flaps are selected for specific conditions: large vaginal defects, difficult vaginal access requiring a relaxing incision, and subsequent need for vaginal coverage, large, recurrent, or radiation-induced fistula. When there is a large vaginal defect these flaps can provide fibroadipose tissue and skin coverage with a well vascularized blood supply. Full thickness rotational labial flaps, for anterior vaginal wall, or gluteal flaps, for posterior or proximal vaginal wall, are chosen depending on the location of where the flap is needed. A full thickness rotational labial flap is the same fatty tissue of a martius flap with its overlying skin that is rotated to cover an anterior vaginal defect. The fistula is first repaired and then a U-shaped incision is made lateral to the labia majora with the apex located at the posterior fourchette. The flaps' blood supply is from the superior pedicle which is based on the external pudendal artery. This flap is dissected free from the fascia of the pubic bone so that it can be rotated medially to achieve repair. In a small series there has been a successful report of this technique [17].

A full thickness gluteal inner thigh rotational flap is reserved for complex refractory fistula. With the patient in the lithotomy position, a mediolateral episiotomy is made at 5 O' clock extending from the introitus to the vaginal apex. Dissection is continued into the para-rectal space. A 4×12 cm inner thigh flap is prepared by making an inverted U-incision lateral to the labia major extending from the ischial tuberosity inferiorly, and to the pubic rami superiorly. This incision preserves the blood supply from the internal pudendal artery and innervation from the labial branches of the internal pudendal nerve and perineal branches of the posterior cutaneous nerve of the thigh. Dissection is carried to the level of the fascia. The episiotomy is extended to the infero-medial aspect of the flap to allow complete mobility. This creates a lateral gluteal rotational inner thigh flap and a medial labial flap. The labial and gluteal rotational inner thigh flaps are crossed; the inner thigh flap medially and the labia flap laterally. The inner thigh flap is transferred and sutured to the vaginal defect. This is a functional full thickness flap that provides good sensation and adequate vaginal width and depth. A variation of the full thickness inner thigh flap is the Singapore island flap [18]. The dissection of the flap is similar except that the episiotomy is avoided and the flap is tunneled to the defect. The epithelium of the flap is removed except for the area that is covering the fistula. This flap is used in complex fistula repair and may be preferred to the full thickness rotational inner thigh flap when there is already adequate vaginal access.

There are several reports of gracilis myocutaneous flap for radiation-induced fistula in which it is used for vaginal reconstruction [19, 20]. We seldom find it necessary to perform this technique because the rotational gluteal flap can duplicate many of the same functions of this gracilis graft without the associated morbidity and cosmetic defects.

The most described interposition is the omental flap which has increased success rates for abdominal repair in retrospective studies [16, 21]. The omentum is based on the right or left branch of gastroepiploic artery, although typically it is based on the right which is usually

larger and more caudal. In cases of bowel resection the mesentery can be preserved and serve as a useful interposition which has similar properties as the omentum with a well vascularized blood supply and lymphatic drainage to decrease inflammation and promote healing. Other tissue interposition flaps that have been reported are bladder flaps [22–26].

Selection of closure and reconstruction of the urethra after UVF requires expertise and experience due to its complexity. Urethral reconstruction centers on different techniques, primarily urethral closure, vaginal and bladder flap advancement which includes pedicle flap (labia minora and anterior or posterior bladder), and use of grafts [27]. Surgical planning of the urethra reconstruction technique may influence vaginal incision location. In a complex fistula resulting in damage of nearly the entire urethra that can extend potentially to the bladder neck, a urethral reconstruction using vaginal or bladder flap construction with interposition of tissue would be preferred to a primary closure. It would be advised to place ureteral stents as the fistula may distort the anatomy and ureteral injury may be avoided during repair.

Evaluation

History and Physical Examination

Women with VVF and UVF most often present with constant urinary incontinence shortly after a pelvic surgery. The presenting symptoms may be recurrent urinary tract infections with chronic perineal changes exhibited by poor healing and irritated skin. Questioning the degree of difference voided and the amount leaked may give clues to the size and location of the VVF. Timing of the onset of leakage and whether there is stress incontinence or urge incontinence with overactive bladder symptoms before the VVF is important to consider in selecting treatment options and patient counseling. A patient with a UVF in the distal third of the urethra may remain continent and asymptomatic or they will commonly describe a splayed urinary stream. They may additionally complain of urinary leakage after vaginal voiding. When the fistula is in the mid-urethra and part of the external sphincter, the patient may have positional intermittent leakage of urine. Patients may have constant, large amounts of urine leaking indicating there is a large fistula that is located proximal to the mid-urethra in the proximal urethra or bladder neck. Gathering information to determine the etiology and prior surgical attempts to repair the fistula can affect the treatment plan.

On physical examination, there should be careful inspection of the fistula size, number, location, and quality of the surrounding tissue. The location of a VVF after hysterectomy is usually a single fistula at the vaginal cuff, although it may present as a complex VVF with multiple fistulas. Evidence of the fistula site is found with surrounding inflammation with granulation and tissue defect. Adequate vaginal access and the degree of mobility of the tissue surrounding the fistula is revealed by the amount of vaginal prolapse. Nulliparous patients or with a history of radiation may be challenging due to a lack of vaginal access and mobility because of narrow vaginal width. On exam, the integrity of the vaginal mucosa, urethral mobility, and assessment of stress incontinence with provocative maneuvers should be performed.

It is important to differentiate the origin of the vaginal drainage and not to make any assumptions as the fluid may be from the fallopian tube, vaginal secretions, peritoneum, lymph, or urine. The differential diagnosis of VVF is UVF, ureterovaginal, uterovaginal, ectopic ureter, or vaginal infection. It is important to differentiate the origin of vaginal drainage and not to make any assumptions as the fluid may be from other pelvic sources [3].

Patients with RVF have presenting clinical symptoms that include gas, stool, and purulent vaginal discharge. The physician should be aware that colonic or enteric fistula may present with similar symptoms as a RVF. The history should focus on causes of the fistula which is most commonly obstetric trauma, but also includes pelvic surgery, malignancy, history of radiation, pelvic abscess, and inflammatory bowel diseases.

Occasionally, small or intersphincteric rectal vaginal fistula may be asymptomatic. Vaginal and bimanual exam should be performed taking note of the location, number, tissue quality, and size of the fistula. On exam, the fistula is normally clearly visualized and instilling dye into the rectum may be of assistance. The location of the fistula is important in deciding the operative approach and is classified into high and low in relation to the anal sphincter. High fistula may need to be approached abdominally and low fistula transvaginally. Occasionally examination under anesthesia is indicated for a more thorough evaluation. During physical examination anal sphincter tone should be evaluated as this may need concomitant repair.

Diagnosis

The diagnosis of a urinary or rectovaginal fistula can most often be made upon a vaginal examination. A urethral catheter with retrograde filling of the bladder or rectum with dye may demonstrate the fistula on exam. A urinary fistula can be confirmed after administering phenazopyridine once it is excreted in the urine. By placing gauze or a tampon in the vagina, the gauze should turn orange in color in the presence of a fistula. A double dye tampon test can further delineate the origin and location of the fistula by giving the patient phenazopyridine followed by retrograde instillation of dye, methylene blue or indigo carmine, into the bladder through a catheter. A ureterovaginal fistula should be orange in the proximal part of the packing while a VVF or UVF should be blue in the mid or distal packing. A negative tampon test does not rule out a fistula and clinical suspicion is many times required to make the diagnosis.

There are varying opinions and no consensus on the imaging required in the evaluation of a VVF or UVF. Many patients have a complex history with postoperative complications and there are medico-legal implications that should be considered [28]. Our practice is to completely evaluate the patient to attempt to address all problems at the initial repair. A voiding cystogram during filling may demonstrate the fistula; however, the intradetrusor pressure may need to be increased during voiding to visualize small fistulas with the patient positioned in the lateral and oblique position. The lateral views may best demonstrate the fistula when it has a direct connection between the bladder and vaginal or when the connection is indirect and enters a collection/sinus tract before draining into the vagina. The VCUG can identify additional findings of a UVF which can be found concomitantly with VVF, the degree of vaginal prolapse, and stress incontinence [29]. Demonstration of preoperative stress incontinence may change the treatment plan by the addition of an anti-incontinence procedure or it may alter patient expectations, of postoperative leakage.

Upper tract evaluation to assess for abnormal findings of hydronephrosis or urinary extravasation from obstruction or fistula with CT urogram should be performed, although there are no formal recommendations to guide the surgeon. There is a 12 % upper tract injury with VVF [30]. Should there be further questions regarding ureteric involvement, a retrograde pyelogram would be justified as it is the most sensitive in detection of upper tract injury, although a CT urogram with reconstructions may be adequate in our experience [31]. Urine cytology is recommended for those with a history of malignancy or pelvic radiation.

It is our routine practice to perform a cystoscopy to evaluate for a urethral fistula and consider it mandatory when there is a history of hematuria or radiation. Urethroscopy should be performed with a short beaked rigid cystoscope (urethroscope or hysteroscope) or flexible cystoscope to allow full visualization of the urethra; the light and the irrigant are at the same level allowing direct vision and expansion of the urethral wall. A 30 and 70° optic lens allows identification of bladder abnormalities while a 0 or 15° lens is better for visualization of urethral foreign bodies or lesions. The fistula size and location in relation to the bladder neck, trigone, and ureteric orifice are determined on cystoscopy. If the fistula involves the bladder neck, it should be discussed with the patient as it may affect continence

after repair. Findings on a cystoscopy can determine if ureteral stents are necessary and if a combined vaginal and abdominal approach are appropriate when there is ureteric involvement.

It is important to document preoperative sexual function and discuss potential postoperative complications. Vaginal stenosis is a potential complication that can be corrected with a subsequent vaginoplasty in most cases. Vaginal shortening may result when a martius flap has insufficient length for a proximal fistula or as a result of Latzko partial colpocleisis. The peritoneal flap is better situated for proximal fistula repair to prevent vaginal shortening.

Further radiologic evaluation with a CT of the abdomen and pelvis should be performed in cases of prior malignancy or in patients without other risk factors for RVF. Gastrografin enema may identify the location of the rectovaginal fistula. Proctosigmoidoscopy and colonoscopy may establish the diagnosis and evaluate for malignancy, especially in the case of radiation-induced fistula where about a third are malignant [32]. If there is any concern for malignancy, the fistula should be biopsied.

Anal sphincter tone should be evaluated preoperatively with physical examination. Nearly 50 % of patients have fecal incontinence which should be discussed and potentially treated simultaneously with fistula repair [33]. Our practice is to routinely obtain an endoanal ultrasound when the cause of the fistula is from trauma after vaginal delivery. Endoanal ultrasound and anal manometry testing can provide valuable information regarding sphincteric function and defects preoperatively.

Vesicovaginal Fistula

Background

Vesicovaginal fistula is an abnormal extra-anatomic connection between the bladder and vagina. Women with VVF suffer enormous amounts of physical, social, and psychological limitations. It is uncommon in western countries, although it remains a widespread problem in undeveloped countries due to obstructed labor [34]. In developed countries, VVF is most often a complication of pelvic surgery (hysterectomy) which we will direct the majority of our attention. VVF may associate with UVF and/or RVF [9, 35]. VVF usually presents with constant urinary incontinence that is distressing and may be intensified as a result of a surgical complication.

Etiology

VVF in the USA and developed countries are the result of gynecologic pelvic surgery in over 80 % of cases, with the remaining causes being comprised from radiation, malignancy, trauma, and obstetric instrumentation during childbirth [29]. Hysterectomy accounts for 91 % of the gynecologic pelvic surgeries that resulted in VVF [9]. A total of 600,000 hysterectomies are performed annually in the USA and nearly a third of women have hysterectomies for benign disease [36–38]. The reported incidence of fistula after hysterectomy for benign disease is reported to be 0.1 to 0.4 %. The risk of fistula increases about tenfold to 1–4 % after radical hysterectomy [39]. The majority of hysterectomies in the USA are performed abdominally with a Cochrane review reporting the risk of fistula formation is similar regardless of the approach, although there is increased risk of injury of urinary tract with laparoscopic hysterectomy [40, 41]. A national database registry study in Sweden found that abdominal and laparoscopic surgery had the highest fistula rate [39]. Fistula formation after hysterectomy is thought to be the result of unrecognized injury to the urinary tract at the time of surgery. The injury may be directly to the bladder or from inadvertently placed sutures that result in tissue necrosis. These injuries result in a urinoma that accumulates and drains through the vaginal cuff [42]. Preoperative risk factors for fistula formation after hysterectomy for benign and malignant disease are diabetes, smoking, history of c-section, endometriosis, pelvic inflammatory disease, and radiation [43–45]. Intraoperative findings of pelvic adhesions, bladder injury, extensive surgery, and higher stage cancer have higher risk of fistula [43–46]. Performing a subtotal hysterectomy with preservation of the cervix

decreased the fistula rate which may be the result of a less extensive surgery [42]. Attention to avoiding injury to the urinary tract and performing a cystoscopy during difficult dissections where bladder injury is suspected may prevent a fistula [28]. It may be helpful to retrograde fill the bladder with dye or saline in these select cases to detect injury. Observation of the urine draining from the foley during hysterectomy should be clear and if there is question further investigation is indicated.

Pelvic surgery with mesh-augmented repair is another cause of fistula. There are reports from transvaginal mesh causing VVF at low rates 0.29 % [47]. A mid-urethral sling may inadvertently injure the bladder and cause a VVF [48]. This reinforces the importance of a cystoscopy at the time of sling placement to prevent urinary fistula. As the number of transvaginal mesh surgeries has been increasing, there may have been a rise in the number of urinary fistula from mesh complications [49]. Whether this trend may reverse as a result of decreased transvaginal mesh-augmented repairs due to the FDA safety communication in July 2011 regarding complications related to transvaginal mesh for POP remains to be seen.

Radiation-induced fistula represents a minor portion of VVF. The mechanism of injury is from obliterative arteritis resulting in ischemia which also produces inflammation of encompassing tissue [50]. Presentation of radiation fistula can occur acutely or be delayed for several years [3, 29]. Suspicion of recurrent cancer or secondary malignancy must be considered with a history of radiation fistula.

Treatment

Conservative Treatment

The goal of surgical repair is to have resolution of the fistula with the least morbidity. In select circumstances it is reasonable to attempt a trial of catheter for about 4 weeks [51]. There are reports of spontaneous resolution of fistulas that are simple and small with the overriding principle that there should be no delay in definitive repair [52–54]. Consideration of endoscopic treatment with fulguration and fibrin glue has been successfully reported in small case series when fistulas are less than 3.5 mm in size [55, 56]. This is a reasonable approach when patients meet these defined criteria; however, few patients are candidates for these conservative or minimally invasive procedures and require surgical repair. Patients with a history of fistulas that are complex, large, or radiation induced should proceed with definitive repair as minimally invasive treatment is futile.

Transvaginal Repair

Prior to surgery a detailed physical examination, urine analysis with culture if indicated, cystoscopy, and VCUG are performed with CT urogram with 3D reconstruction of the ureters in selected cases. We describe our basic technique and adjuvant procedures performed in complex cases. With the patient in lithotomy position, surgical repair begins with vaginal exposure with a ring retractor and vaginal speculum. The key to performing this repair is identification of the fistula tract. A cystoscopy is performed to identify the fistula and a wire is placed through it. Bilateral stent placement is done when the fistula is near the ureteric orifices. A 16–18 Fr catheter is inserted into the bladder. The vaginal cuff is grasped with Allis clamps or with stay sutures to expose the fistulous tract which is dilated with sounds over the guide wire to allow the passage of a 8–10 Fr catheter. The catheter is important in the exposure and retraction of the bladder during the repair. A circumferential incision is made less than 1 cm from the fistula track. An inverted U-incision is made on the anterior vaginal wall and it is mobilized 3–4 cm to create the anterior vaginal flap (Fig. 10.3). A posterior vaginal wall flap is created from the cuff to expose the prerectal fascia, the vesico-rectal space, and the posterior cul de sac where the peritoneal flap can be retrieved.

The fistula tract is isolated and closed with 2-0 or 3-0 delay absorbable interrupted sutures. Care is taken to incorporate the entire fistulous tract and the bladder wall. At the end of the closure, diluted indigo-carmine tests the integrity of the repair. We omit excision of tract unless there is concern of malignancy or extensive necrotic tissue. The fistula tract is not routinely excised

because it provides excellent anchoring tissue for closure, avoids creating a larger defect to repair that may prevent the need for ureteric reimplant, and prevents bleeding from the fistula tract edges that can become devitalized from electrocautery during control of bleeding [3, 57]. The bladder is filled with indigo carmine once the fistula tract is closed to ensure there is no extravasation. A second layer of sutures 1 cm from the fistula and imbricated over the tract with 2-0 or 3-0 delay absorbable interrupted suture for the second layer of closure. A double layer of peritoneal flap is dissected from the vesico-rectal space in the cul de sac, mobilized and advanced 2–3 cm distal to the fistula closure (Fig. 10.4). This flap is approximated with 3-0 absorbable interrupted sutures. A small segment of the distal flap is excised and the posterior flaps are advanced and closed beyond the fistula side with absorbable 2-0 interrupted suture to result in a four-layer closure (Fig. 10.5).

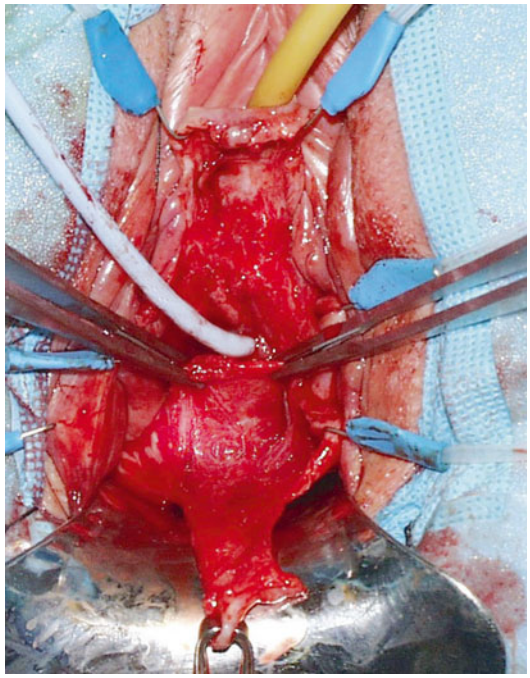


Fig. 10.3 Anterior vaginal flap that has been mobilized (Copyright © Shlomo Raz, MD)

Latzko Partial Colpocleisis

The Latzko partial colpocleisis is an alternative technique to our transvaginal vesicovaginal fistula repair. Our approach avoids vaginal shortening and overlapping suture line that may result in recurrence. Other authors report low recurrence rates and vaginal shortening only when there is already a shortened vagina [58]. Potential advantages of this approach are decreased morbidity from less blood loss and shorter operating time. The Latzko technique is attempted for proximal post hysterectomy fis-

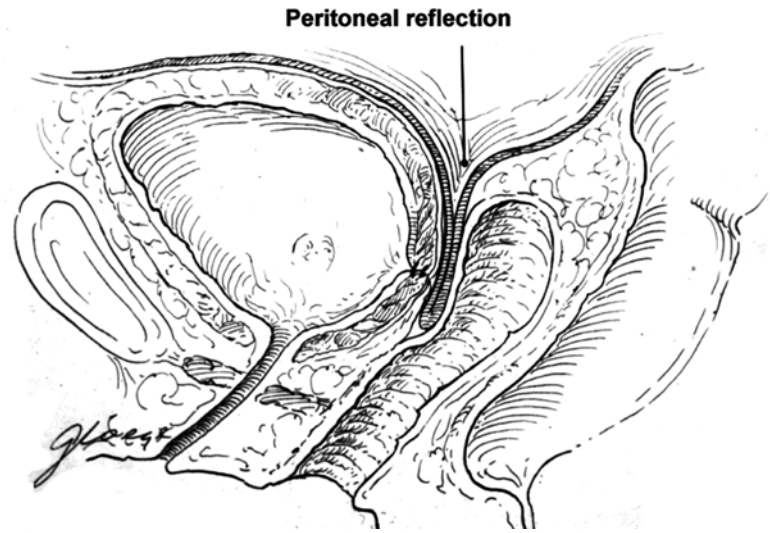


Fig. 10.4 The peritoneum is advanced distal to the fistula closure (Copyright © Shlomo Raz, MD)

tula. It involves a circumferential elliptoid incision around the fistula with wide mobilization of the vaginal epithelium in all directions.

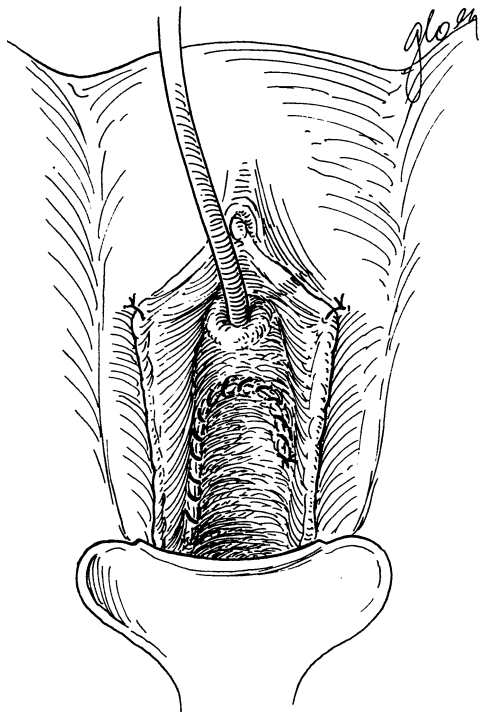


Fig. 10.5 Repair and closure of the fistula with four layers (Copyright © Shlomo Raz, MD)

The fistula tract is closed and the repair is reinforced by an inverted layer of the perivesical tissue. The suture lines are overlapping in this repair (Figs. 10.6a–c and 10.7a–c).

Abdominal Repair (Fig. 10.8a–e)

An abdominal approach may include open, laparoscopic or robotic repair. Indications for an abdominal approach include intraperitoneal pathology, concomitant ureteric reimplant, or bladder augmentation, or need for fecal diversion. Abdominal repair is typically approached with the O'Conor technique. Our standard approach begins with a midline incision that extends from the umbilicus to the pubic bone. Once entering the peritoneum the bladder is identified by retrograde filling of the catheter. A vaginal probe is inserted and retracted superiorly. The bladder is dissected free from the vagina until the fistulous tract is encountered. A decision to bivalve the bladder is made if the fistula tract is not visibly patent and open. The bladder is bivalved extending the incision to include the fistula which can be biopsied if there is concern for malignancy. The vaginal wall is dissected and separated from the bladder 3–4 cm surrounding the fistula. The bladder and the vagina are closed in two separate layers and indigo carmine is injected to

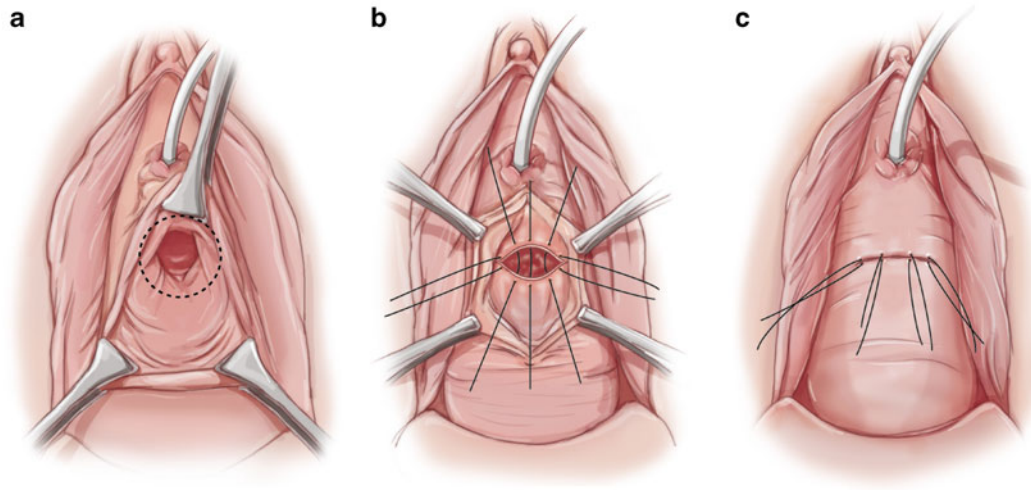


Fig. 10.6 (**a**, **b**) Circumferential incision and mobilization of the vaginal epithelium around the fistula. (**c**) Closure of vaginal epithelium results in overlapping suture lines

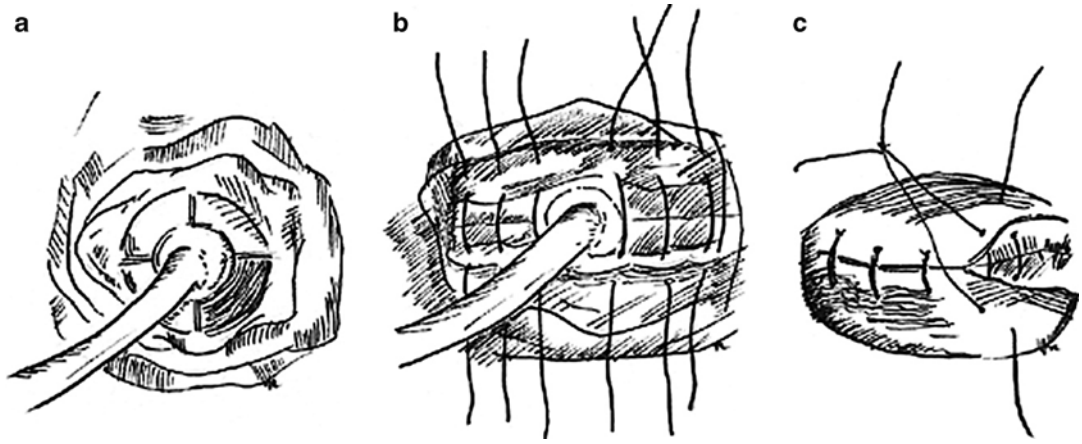


Fig. 10.7 (**a**, **b**) Circumferential incision and dissection of the epithelium around the fistula with inverted closure of the fistula tract and perivesical tissue in two layers. (**c**) Closure of vaginal epithelium results in nonoverlapping suture lines (Copyright © Shlomo Raz, MD)

assure complete closure. Interposition of tissue flap between the layers is added for security. The most described interposition is the omental flap which has increased success rates in retrospective studies or free flap of peritoneum can be used [16, 21].

Laparoscopic/Robotic

The first description of a laparoscopic VVF repair was by Nezhat in 1994 [59]. The robotic repair was first described in 2005, which is a platform that allows more surgeons to perform the technical aspects of this surgery of suturing and knot tying that are technically demanding with laparoscopic surgery [60]. The success rates are about 90 % or greater in the few case series reporting on these techniques [11–13, 61, 62]. There is one study comparing open to robotic repair of VVF with similar outcomes [11]. As in open repair, the robotic approach allows the surgeon to perform ureteric reimplant when indicated. These emerging technologies appear to be promising for the surgeon skilled in robotic or laparoscopic surgery. Outcome data on robotic repairs remains limited as this is an emerging technology. Vaginal surgery has decreased operating time, costs, and can be performed in an ambulatory surgical center.

Complications

Complications of vaginal bleeding, bladder spasm, and infection increase the risk of recurrence. These should be treated to prevent fistula recurrence. Fistula recurrence is the most significant complication and every attempt should be made to prevent it. Postoperative antibiotics are routinely given for about 2 weeks postoperatively. Anticholinergics or B & O suppositories may be needed to prevent bladder spasms. Patients should be advised to have pelvic rest for 3 months postoperatively. Recurrent fistula should be treated with a tissue interposition and at least a 3 month delay in repair. Rare complications of injury to the ureters, bowel, and rectum occur and should be discussed.

Urethrovaginal Fistula

Background

A UVF is an abnormal connection between the urethra and the vagina that may be the result of obstetric, iatrogenic, neoplasm, trauma, or infection. This should not be confused or grouped together with VVF because the etiology, surgical repair, and potential complications of urethrovaginal

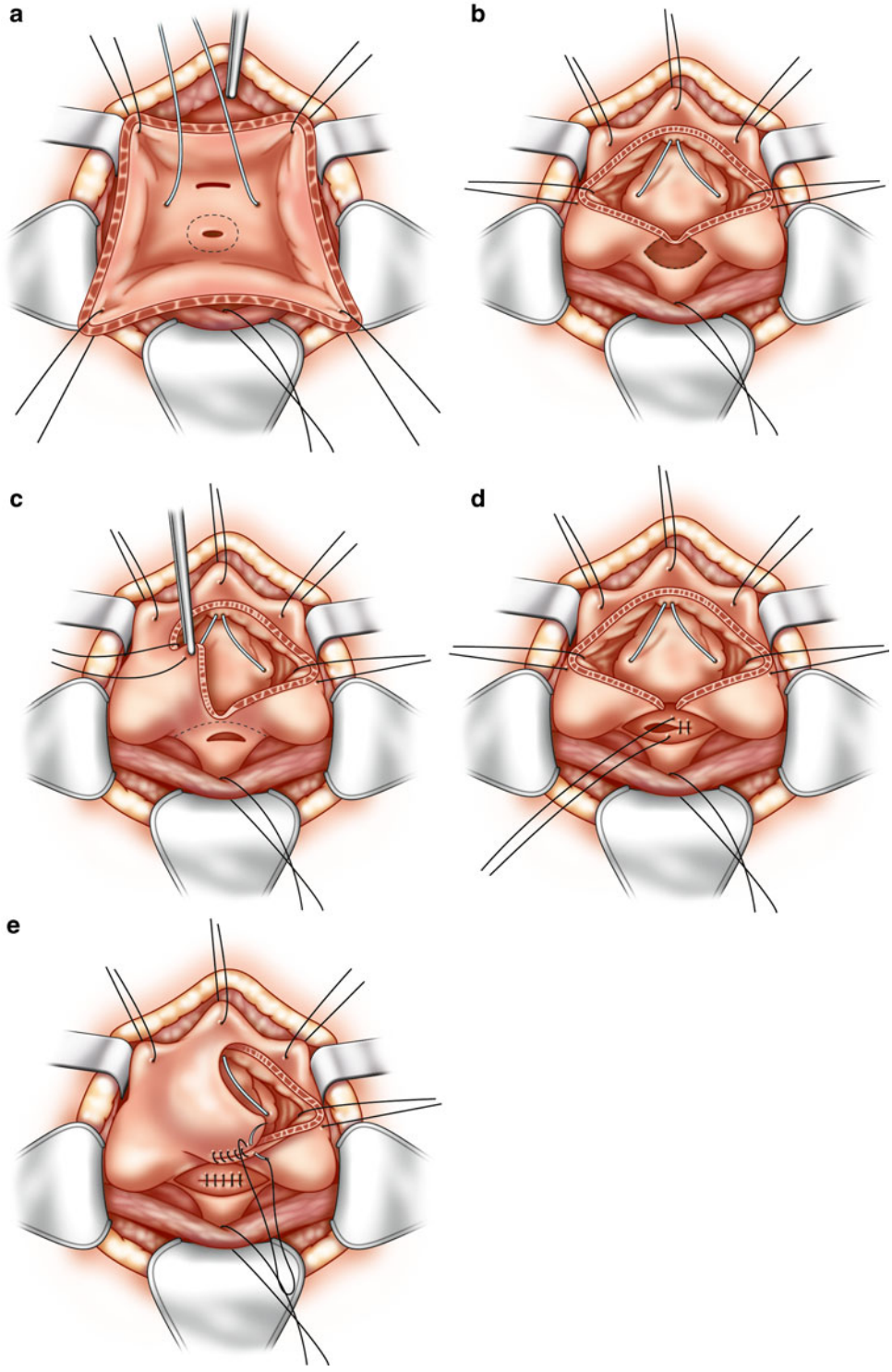


Fig. 10.8 (**a**–**e**) Open approach for vesicovaginal fistula. (**a**) An incision is made in the anterior bladder wall and retraction sutures are placed to expose the fistula. Ureteral stents identify the ureters during repair. (**b**) The incision in the bladder is extended to include and excise the fistula tract. The vaginal tissue in the fistula tract is excised. (**c**) The bladder and vagina are separated using sharp dissection along the *dotted line*. This is a difficult plane to develop, but it is important to mobilize and separate the bladder and vagina for a successful repair. (**d**) The vagina is closed with tension-free interrupted sutures. (**e**) The bladder and vagina are closed in separate layers. Tissue can be interposed between the repairs at this time

fistula differ. UVF is a rare condition due to the female urethra being seldom involved in injury because of its short length and protection from the pubic bone [63, 64]. The majority of UVFs in developed countries are iatrogenic and arise from pelvic surgery or radiation and less often from obstetric procedures during childbirth [29, 34, 65].

Etiology

UVFs in developed countries are the main focus and can be divided into two main categories; causes from vaginal/pelvic procedures which make up the majority and less often from radiation. Currently with the increased use of mesh, mesh exposure or erosion into the urethra needs to be considered as a source of the fistula. There are case reports of synthetic mid-urethral slings causing urinary fistulas [48, 66]. This mechanism of injury is likely unrecognized iatrogenic injury of the urethra from urethral perforation which increases the risk of fistula formation [67–69]. Urethral diverticulectomy surgery is the most common surgical cause of UVF and contributes to 25 % of patients who undergo pelvic surgery [29]. This may be the result of incomplete excision of the diverticulum or inadequate urethral closure without sufficient tissue interposition.

Radiation fistula formation can present immediately or can occur years after exposure and may contribute to 15 % of UVFs in one series [3, 29]. There should be consideration of malignancy when patients have a history of pelvic cancer or radiation treatment.

Rare cases of UVF in the USA may be the result of trauma, injury during childbirth, malignancy, or infection. As childbirth techniques have improved, there are less injuries and trauma contributing to UVF [34]. The use of forceps or instruments may result in laceration of the urethra that if not identified and repaired can lead to UVF. Blunt trauma with pelvic fracture rarely can cause an avulsion of the urethra or develop into an UVF with an incidence range of 0–6 % [4]. Instrumentation of the urethra is another unusual cause of fistula [70, 71]. Chronic indwelling foley can cause pressure necrosis of the bladder neck and distal urethral which can form a hypospadic urethra and UVF [72, 73].

The majority of UVFs in undeveloped countries originate from prolonged obstructed labor and are not iatrogenic as in western countries. These UVFs are due to ischemia and commonly involve the bladder and urethra with extensive tissue loss. The mid-urethral sphincteric complex may be irreversibly damaged making for a tenuous repair with unwanted outcomes [4, 74].

Treatment

Transvaginal Repair

This is a description of a UVF that is closed primarily (Fig. 10.9). Surgical repair begins with vaginal exposure with a ring retractor and vaginal speculum. A foley catheter is inserted into the urethra. A small foley catheter is inserted in the fistula with dilation of the tract if necessary. Injection of retrograde dye in the urethral meatus

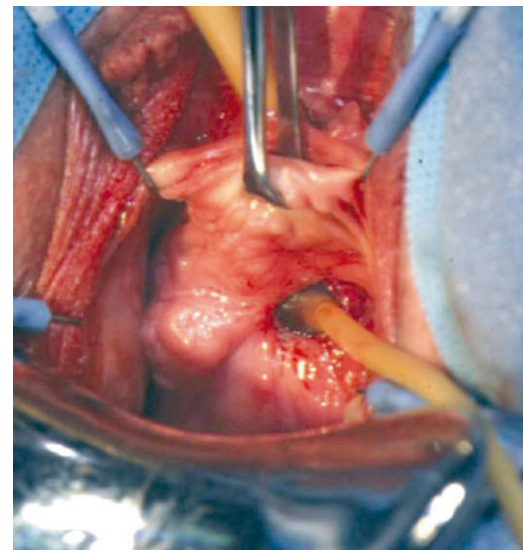


Fig. 10.9 Urethrovaginal fistula with a foley used as a retractor (Copyright © Shlomo Raz, MD)

may help to identify a small fistula. An inverted U incision is made on the anterior vaginal wall. The anterior vaginal wall flap is dissected and freed so that it has mobility to advance 2 cm distal to the fistula. The anterior vaginal wall flap is dissected lateral and proximal to the fistula which facilitates creation of the flap by avoiding scarring and friable tissue. Lateral and distal vaginal flaps are dissected which expose the fistula tract and the periurethral fascia. Once the vaginal wall is separated and is adequately mobilized, a transverse incision of the periurethral tissue is made at the level of the fistula as in a urethral diverticulectomy repair. Superior and inferior flaps of the periurethral fascia are created, isolating the urethral wall with the catheter in place. The fistula is closed in two layers with the urethra as the first layer closed transversely like a Heineke–Mikulicz technique. The periurethral fascia is closed in a transverse fashion to cover the area of reconstruction (Fig. 10.10). The fistula tract is not routinely excised as mentioned previously. Optional creation of a martius flap to cover the periurethral fascia (radiation, multiple surgeries, large defects, poor tissue quality) is performed. The vaginal wall flap is advanced to cover the area of reconstruction. The foley catheter is left for 2–3 weeks and removed with a negative voiding trial or VCUG.

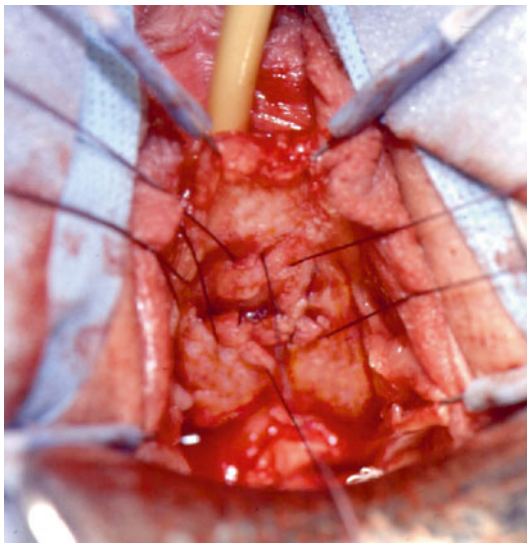


Fig. 10.10 Transverse closure of the periurethral fascia (Copyright © Shlomo Raz, MD)

Complications

Complications should be discussed preoperatively so the patient has realistic expectations after repair. Patients may develop obstructive voiding due to urethral stenosis in 5–20 % of cases [64, 65]. There is a 50 % chance they will develop stress incontinence symptoms requiring an anti-incontinence procedure [64]. Patients requiring extensive urethral reconstruction or a history of radiation with an immobile poorly vascularized urethra may fail fistula repair necessitating a bladder neck closure and urinary diversion.

Rectovaginal Fistula

Background

Rectovaginal fistula is an extra-anatomic epithelial connection between the rectum and vagina. It is a disabling disease that severely devastates and impacts an individual's social life and self-esteem.

Etiology

RVF is most often a complication after a traumatic vaginal delivery that occurs in about 0.1 % of vaginal deliveries in modern developed countries [75, 76]. Fistula formation is the result of high grade rectal lacerations, grade 3 and 4, involving the perineal body and rectum that is unrecognized or becomes infected after repair. Risk factors for high grade rectal lacerations at time of vaginal delivery are midline episiotomies, use of forceps, first vaginal delivery, and increased birth weight of the fetus [77]. Investigation should be given to additional causes of RVF from pelvic surgery including low anterior resection, synthetic mesh for POP, hysterectomy, pessary (Fig. 10.11), colorectal or gynecologic malignancy, history of radiation, pelvic abscess, and inflammatory bowel diseases which include Crohn's disease, ulcerative colitis, and diverticulitis [78–80].

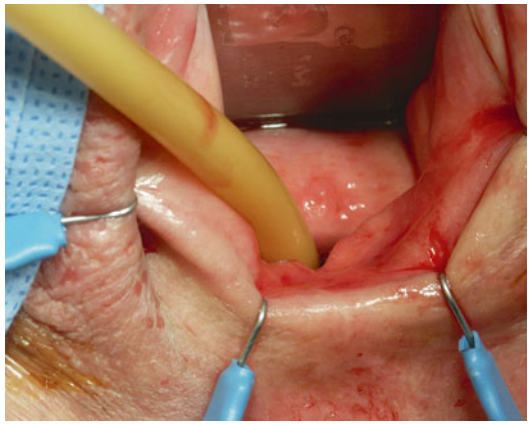


Fig. 10.11 A rectovaginal fistula that resulted from prolonged tissue necrosis and ischemia from a pessary (Copyright © Shlomo Raz, MD)

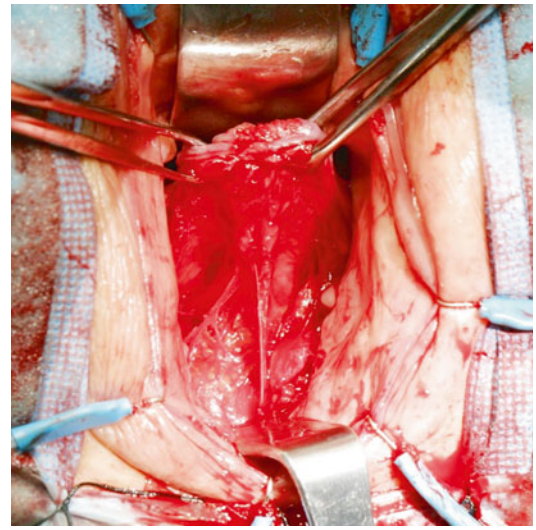


Fig. 10.12 Perirectal fascia is advanced 2–3 cm beyond the rectovaginal fistula repair (Copyright © Shlomo Raz, MD)

Treatment

Surgical Procedure

Tissue Interposition

The majority of RVF repairs involve interposition of tissue to prevent recurrence with little added morbidity. Interposition of a martius flap after a transvaginal repair of low fistula is our preference and typically reserves gluteal rotational inner thigh flaps for high, difficult vaginal access, large defects, or fibrotic vaginal tissue that is suboptimal for fistula closure.

Vaginal Repair

Routine administration of broad spectrum antibiotics and mechanical bowel preparation are given preoperatively. The transvaginal repair is performed with a multilayer closure with routine use of tissue interposition. Fecal diversion is performed selectively in cases of complex fistula. Complex fistulas are defined as radiation induced, recurrent, large, or a result of malignancy.

The patient is positioned in high lithotomy position and the fistula is exposed with a ring retractor. A foley is inserted into the fistula tract and can be used a retractor. A U-incision is made on the posterior vaginal wall and it is mobilized 3–4 cm to create a vaginal flap. The vaginal wall is dissected free on the lateral wall and the pre-rectal fascia is dissected to create a flap that will be cover the fistula at the end of the procedure. The fistula tract is closed in two layers with interrupted delayed 3-0 absorbable suture that results in a water-tight closure. The first layer includes the rectal and vaginal wall. The second layer includes the perirectal fascia that is advanced 2–3 cm over the fistula repair (Fig. 10.12). A martius flap that had been previously prepared is placed for additional coverage. A vaginal flap is advanced for a four-layer closure (Fig. 10.13). There is also description of use of biological material to reinforce the fistula repair [81].

Transanal Repair

The transanal approach is most commonly used for low fistula. It begins with the patient in prone jackknife position. A rectal advancement flap is created that includes the mucosa, sub mucosa, and the circular muscular fibers (internal sphincter). The flap is dissected 5 cm proximal to the fistula with its proximal base being twice the width of the apex. The fistula tract is excised and the rectal side of the fistula is closed leaving the vaginal side open.

Perineal Repair

The perineal approach involves a two-step procedure which is more morbid than transvaginal

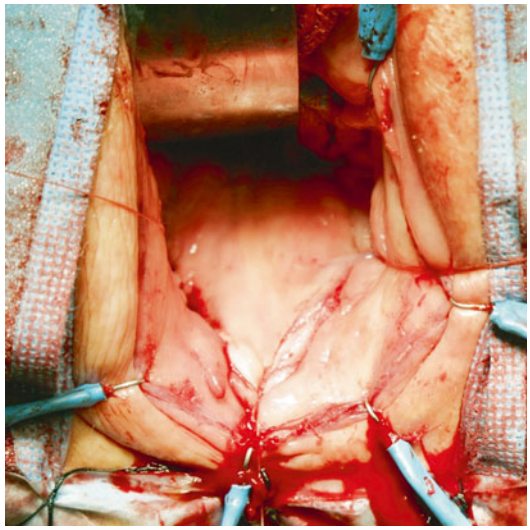


Fig. 10.13 The repair is completed with advancement of a vaginal flap for a four-layer closure (Copyright © Shlomo Raz, MD)

repair. Perineal repair is used primarily for perineal fistula that many times involves the anal sphincter integrity. First a catheter is inserted into the fistula and the overlying tissue is incised creating a perineoproctotomy. Optimally, the fistula tract is excised and then the layers that were divided are approximated without tension. The vaginal and rectal mucosa are separated and closed in two layers. The second step is a sphincteroplasty and rebuilding of the perineal body. The internal and external sphincters are approximated and the perineal body is reconstructed. In our experience, we perform adjuvant procedures to improve the repair by making the incision asymmetric and excising only the epithelium, so the subcutaneous fat can be crossed over and interposed to provide an additional layer.

Complications

Complications of hematoma and infection increase the risk of recurrence which should be prevented. Preoperative broad coverage antibiotics are given. In cases of recurrent fistula the patient should have a fecal diversion before exploration and repair. The repair should include interposition of tissue.

Summary

Vesicovaginal, urethrovaginal, and rectovaginal fistulas are significant complications that require complex repair. There is controversy surrounding the ideal timing, approach, technique, and use of adjuvant procedures in repair. Although there is debate in management, the principles in the diagnosis, treatment, and surgical repair of fistulas decrease the risk of complications and improve patient outcomes. Surgeon experience and principles of repair dictate the chosen repair. In our experience, the majority of fistulas can be successfully repaired transvaginally thereby limiting the postoperative morbidity, complications, and hospital stay.

References

1. Shelbaia AM, Hashish NM. Limited experience in early management of genitourinary tract fistulas. Urology. 2007;69:572.
2. Blaivas JG, Heritz DM, Romanzi LJ. Early versus late repair of vesicovaginal fistulas: vaginal and abdominal approaches. J Urol. 1995;153:1110.
3. Rutman MP, Raz S, Rodriguez LV. Female urology. 3rd ed. Philadelphia: Elsevier; 2008. p. 794–801.
4. Pushkar DY, Sumerova NM, Kasyan GR. Management of urethrovaginal fistulae. Curr Opin Urol. 2008;18:389.
5. Ricart E, Panaccione R, Loftus EV, et al. Infliximab for Crohn's disease in clinical practice at the Mayo Clinic: the first 100 patients. Am J Gastroenterol. 2001;96:722.
6. Sands BE, Blank MA, Patel K, et al. Long-term treatment of rectovaginal fistulas in Crohn's disease: response to infliximab in the ACCENT II Study. Clin Gastroenterol Hepatol. 2004;2:912.
7. Present DH, Korelitz BI, Wisch N, et al. Treatment of Crohn's disease with 6-mercaptopurine. A long-term, randomized, double-blind study. N Engl J Med. 1980;302:981.
8. Present DH, Lichtiger S. Efficacy of cyclosporine in treatment of fistula of Crohn's disease. Dig Dis Sci. 1994;39:374.
9. Eilber KS, Kavaler E, Rodriguez LV, et al. Ten-year experience with transvaginal vesicovaginal fistula repair using tissue interposition. J Urol. 2003;169:1033.
10. Richman MB, Goldman HB. Vesicovaginal fistula: abdominal approach. In: Vasavada SP, Appell RA, Sand PK, Raz S, editors. Female urology, urogynecology, and voiding dysfunction. New York: CRC; 2005. p. 783–95.

11. Gupta NP, Mishra S, Hemal AK, et al. Comparative analysis of outcome between open and robotic surgical repair of recurrent supra-trigonal vesico-vaginal fistula. J Endourol. 2010;24:1779.
12. Abdel-Karim AM, Mousa A, Hasouna M, et al. Laparoscopic transperitoneal extravesical repair of vesicovaginal fistula. Int Urogynecol J. 2011;22:693.
13. Gozen AS, Teber D, Canda AE, et al. Transperitoneal laparoscopic repair of iatrogenic vesicovaginal fistulas: Heilbronn experience and review of the literature. J Endourol. 2009;23:475.
14. D'Ambrosio G, Paganini AM, Guerrieri M, et al. Minimally invasive treatment of rectovaginal fistula. Surg Endosc. 2012;26:546.
15. Arrowsmith SD. Genitourinary reconstruction in obstetric fistulas. J Urol. 1994;152:403.
16. Evans DH, Madjar S, Politano VA, et al. Interposition flaps in transabdominal vesicovaginal fistula repairs: are they really necessary? Urology. 2001;57:670.
17. Carr LK, Webster GD. Full-thickness cutaneous martius flaps: a useful technique in female reconstructive urology. Urology. 1996;48:461.
18. Wee JT, Joseph VT. A new technique of vaginal reconstruction using neurovascular pudendal-thigh flaps: a preliminary report. Plast Reconstr Surg. 1989; 83:701.
19. Wang Y, Hadley HR. The use of rotated vascularized pedicle flaps for complex transvaginal procedures. J Urol. 1993;149:590.
20. Gerges DG, Mesfen W. [Repair of a complex vesicovaginal fistula using a musculocutaneous flap of the gracilis muscle]. J Urol (Paris). 1984;90:491.
21. Ayed M, El Atat R, Hassine LB, et al. Prognostic factors of recurrence after vesicovaginal fistula repair. Int J Urol. 2006;13:345.
22. Gil-Vernet JM, Gil-Vernet A, Campos JA. New surgical approach for treatment of complex vesicovaginal fistula. J Urol. 1989;141:513.
23. Mohseni MG, Hosseini SR, Alizadeh F, et al. Bladder mucosal autograft: an effective method for repair of vesicovaginal fistula. Adv Biomed Res. 2019;1:77.
24. Eisen M, Jurkovic K, Altwein JE, et al. Management of vesicovaginal fistulas with peritoneal flap interposition. J Urol. 1974;112:195.
25. James MH, Tisdale BE, Davies TO, et al. The urachal flap: a previously unreported tissue flap in vesicovaginal fistula repair. Female Pelvic Med Reconstr Surg. 2013;19:148.
26. Salup RR, Julian TB, Liang MD, et al. Closure of large postradiation vesicovaginal fistula with rectus abdominis myofascial flap. Urology. 1994;44:130.
27. Ackerman AL, Blaivas J, Anger JT. Female urethral reconstruction. Curr Bladder Dysfunct Rep. 2010;5:225.
28. Hilton P. Vesico-vaginal fistula: new perspectives. Curr Opin Obstet Gynecol. 2001;13:513.
29. Lee RA, Symmonds RE, Williams TJ. Current status of genitourinary fistula. Obstet Gynecol. 1988;72:313.
30. Goodwin WE, Scardino PT. Vesicovaginal and ureterovaginal fistulas: a summary of 25 years of experience. J Urol. 1980;123:370.
31. Brandes S, Coburn M, Armenakas N, et al. Diagnosis and management of ureteric injury: an evidence-based analysis. BJU Int. 2004;94:277.
32. Allen-Mersh TG, Wilson EJ, Hope-Stone HF, et al. The management of late radiation-induced rectal injury after treatment of carcinoma of the uterus. Surg Gynecol Obstet. 1987;164:521.
33. Tsang CB, Madoff RD, Wong WD, et al. Anal sphincter integrity and function influences outcome in rectovaginal fistula repair. Dis Colon Rectum. 1998;41:1141.
34. De Ridder D. An update on surgery for vesicovaginal and urethrovaginal fistulae. Curr Opin Urol. 2011;21:297.
35. Eilber KS, Rosenblum N, Rodriguez LV. Vesicovaginal fistula: complex fistulae. In: Vasavada SP, Appell RA, Sand PK, Raz S, editors. Female urology, urogynecology, and voiding dysfunction. New York: CRC; 2005. p. 761–82.
36. Farquhar CM, Steiner CA. Hysterectomy rates in the United States 1990-1997. Obstet Gynecol. 2002;99:229.
37. Carlson KJ, Nichols DH, Schiff I. Indications for hysterectomy. N Engl J Med. 1993;328:856.
38. Whiteman MK, Hillis SD, Jamieson DJ, et al. Inpatient hysterectomy surveillance in the United States, 2000-2004. Am J Obstet Gynecol. 2008;198:34 e1.
39. Forsgren C, Altman D. Risk of pelvic organ fistula in patients undergoing hysterectomy. Curr Opin Obstet Gynecol. 2010;22:404.
40. Falcone T, Walters MD. Hysterectomy for benign disease. Obstet Gynecol. 2008;111:753.
41. Nieboer TE, Johnson N, Lethaby A et al. Surgical approach to hysterectomy for benign gynaecological disease. Cochrane Database Syst Rev 2009; CD003677.
42. Forsgren C, Lundholm C, Johansson AL, et al. Hysterectomy for benign indications and risk of pelvic organ fistula disease. Obstet Gynecol. 2009;114:594.
43. Duong TH, Gellasch TL, Adam RA. Risk factors for the development of vesicovaginal fistula after incidental cystotomy at the time of a benign hysterectomy. Am J Obstet Gynecol. 2009;201:512 e1.
44. Likic IS, Kadija S, Ladjevic NG, et al. Analysis of urologic complications after radical hysterectomy. Am J Obstet Gynecol. 2008;199:644 e1.
45. Bai SW, Huh EH, Jung da J, et al. Urinary tract injuries during pelvic surgery: incidence rates and predisposing factors. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:360.
46. Duong TH, Taylor DP, Meeks GR. A multicenter study of vesicovaginal fistula following incidental cystotomy during benign hysterectomies. Int Urogynecol J. 2011;22:975.
47. Caquant F, Collinet P, Debodinance P, et al. Safety of trans vaginal mesh procedure: retrospective study of 684 patients. J Obstet Gynaecol Res. 2008;34:449.
48. Starkman JS, Meints L, Scarpero HM, et al. Vesicovaginal fistula following a transobturator midurethral sling procedure. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:113.

49. Rogo-Gupta L, Rodriguez LV, Litwin MS, et al. Trends in surgical mesh use for pelvic organ prolapse from 2000 to 2010. Obstet Gynecol. 2012;120:1105.
50. Perez CA, Grigsby PW, Lockett MA, et al. Radiation therapy morbidity in carcinoma of the uterine cervix: dosimetric and clinical correlation. Int J Radiat Oncol Biol Phys. 1999;44:855.
51. Miller EA, Webster GD. Current management of vesicovaginal fistulae. Curr Opin Urol. 2001;11:417.
52. Hilton P. Urogenital fistula in the UK: a personal case series managed over 25 years. BJU Int. 2012;110:102.
53. Waaldijk K. The immediate management of fresh obstetric fistulas. Am J Obstet Gynecol. 2004;191:795.
54. Bazi T. Spontaneous closure of vesicovaginal fistulas after bladder drainage alone: review of the evidence. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:329.
55. Stovsky MD, Ignatoff JM, Blum MD, et al. Use of electrocoagulation in the treatment of vesicovaginal fistulas. J Urol. 1994;152:1443.
56. Shekarriz B, Stoller ML. The use of fibrin sealant in urology. J Urol. 2002;167:1218.
57. Shaker H, Saafan A, Yassin M, et al. Obstetric vesicovaginal fistula repair: should we trim the fistula edges? A randomized prospective study. Neurourol Urodyn. 2011;30:302.
58. Karram MM. In: Walters MD, Karram MM, editors. Urogynecology and reconstructive pelvic surgery. 3rd ed. Philadelphia, PA: Mosby Elsevier; 2007. p. 445–71.
59. Nezhat CH, Nezhat F, Nezhat C, et al. Laparoscopic repair of a vesicovaginal fistula: a case report. Obstet Gynecol. 1994;83:899.
60. Melamud O, Turbow B, Shanberg AM. Robot-assisted laparoscopic vesicovaginal fistula repair. J Urol. 2005;173:134.
61. Sotelo R, Moros V, Clavijo R, et al. Robotic repair of vesicovaginal fistula (VVF). BJU Int. 2012;109:1416.
62. Shah SJ. Laparoscopic transabdominal transvesical vesicovaginal fistula repair. J Endourol. 2009;23:1135.
63. Leach GE. Urethrovaginal fistula repair with Martius labial fat pad graft. Urol Clin North Am. 1991;18:409.
64. Gebhart JB, Lee RA. Urethrovaginal fistula. In: Valsavada SP, Appell RA, Sand PK, Raz S, editors. Female urology, urogynecology, and voiding dysfunction. New York: Marcel Dekker; 2005. p. 797–810.
65. Pushkar DY, Dyakov VV, Kosko JW, et al. Management of urethrovaginal fistulas. Eur Urol. 2006;50:1000.
66. Siegel AL. Urethral necrosis and proximal urethrovaginal fistula resulting from tension-free vaginal tape. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:661.
67. Reisenauer C, Wallwiener D, Stenzl A, et al. Urethrovaginal fistula–a rare complication after the placement of a suburethral sling (IVS). Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:343.
68. Flisser AJ, Blaivas JG. Outcome of urethral reconstructive surgery in a series of 74 women. J Urol. 2003;169:2246.
69. Glavind K, Larsen EH. Results and complications of tension-free vaginal tape (TVT) for surgical treatment of female stress urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2001;12:370.
70. Carlin BI, Klutke CG. Development of urethrovaginal fistula following periurethral collagen injection. J Urol. 2000;164:124.
71. Hilton P. Urethrovaginal fistula associated with 'sterile abscess' formation following periurethral injection of dextranomer/hyaluronic acid co-polymer (Zuidex) for the treatment of stress urinary incontinence–a case report. BJOG. 2009;116:1527.
72. Zimmern PE, Hadley HR, Leach GE, et al. Transvaginal closure of the bladder neck and placement of a suprapubic catheter for destroyed urethra after long-term indwelling catheterization. J Urol. 1985;134:554.
73. McGuire EJ, Savastano J. Comparative urological outcome in women with spinal cord injury. J Urol. 1986;135:730.
74. Ahmad S, Nishtar A, Hafeez GA, et al. Management of vesico-vaginal fistulas in women. Int J Gynaecol Obstet. 2005;88:71.
75. Senatore Jr PJ. Anovaginal fistulae. Surg Clin North Am. 1994;74:1361.
76. Venkatesh KS, Ramanujam PS, Larson DM, et al. Anorectal complications of vaginal delivery. Dis Colon Rectum. 1989;32:1039.
77. Angioli R, Gomez-Marin O, Cantuaria G, et al. Severe perineal lacerations during vaginal delivery: the University of Miami experience. Am J Obstet Gynecol. 2000;182:1083.
78. Choi JM, Nguyen V, Khavari R, et al. Complex rectovaginal fistulas after pelvic organ prolapse repair with synthetic mesh: a multidisciplinary approach to evaluation and management. Female Pelvic Med Reconstr Surg. 2012;18:366.
79. Yong PJ, Garrey MM, Geoffrion R. Transvaginal repair and graft interposition for rectovaginal fistula due to a neglected pessary: case report and review of the literature. Female Pelvic Med Reconstr Surg. 2011;17:195.
80. Champagne BJ, McGee MF. Rectovaginal fistula. Surg Clin North Am. 2010;90:69.
81. Ellis CN. Outcomes after repair of rectovaginal fistulas using bioprosthetics. Dis Colon Rectum. 2008;51:1084.

Repair of Urethral Diverticula

11

Kelly C. Johnson and Eric S. Rovner

Introduction

The diagnosis and surgical approach to female urethral diverticula (UD) can be an extremely challenging aspect of female urology. UD are known for a complex variety of presentations, ranging from asymptomatic, incidentally found lesions on examination or on imaging, to painful vaginal masses associated with incontinence, stones, or tumors. Each case poses a unique challenge due to variations in anatomy, as well as the location, size, and complexity of these lesions.

Development of imaging modalities, such as ultrasound and MRI, in the past 30 years has greatly contributed to advanced understanding of UD. With the expanding use of such imaging techniques, the diagnosis and evaluation of UD continues to evolve. Once confirmation of the diagnosis is achieved, definitive therapy typically consists of surgical excision and reconstruction. Successful operative excision and reconstruction requires advanced knowledge of the relevant surgical anatomy, as well as creativity and occasionally improvisation in the operating room.

K.C. Johnson, M.D.
Chesapeake Urology, Owings Mills, MD, USA
e-mail: kjohnson@cua.md

E.S. Rovner, M.D. (✉)
Department of Urology, Medical University of South Carolina, Charleston, SC, USA
e-mail: rovnere@musc.edu

Pathophysiology and Etiology

UD represent an epithelialized cavity dissecting within the fascia of the urethropelvic ligament [1] (Figs. 11.1 and 11.2). This defect is often an isolated cyst-like appendage with a discreet connection to the lumen of the urethra, called the neck or ostia. Complicated anatomical patterns are possible, and in certain cases the UD may partially extend ("saddlebag" UD) around the urethra, anterior [2], or circumferentially about the urethra [3].

The periurethral glands are the probable site of origin of acquired UD [1]. Huffman characterized the periurethral glands as being located mostly dorsolateral to the urethra, arborizing proximally along the urethra, but also draining into ducts located in the distal one-third of the urethra [4]. Importantly, he noted that periductal and interductal inflammation was also commonly found. In support of his observations and an infectious or acquired etiology of UD, in over 90 % of cases, the ostia is located posterolaterally in the mid to distal urethra which corresponds anatomically to the location of the periurethral glands [5, 6].

Peters and Vaughn found that there was a strong association with concurrent or previous infection with *Neisseria gonorrhea* and UD [7]. However, the initial infection and subsequent reinfections might also originate from a variety of sources including *E. coli*, and other coliform bacteria, as well as flora within the

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_11,

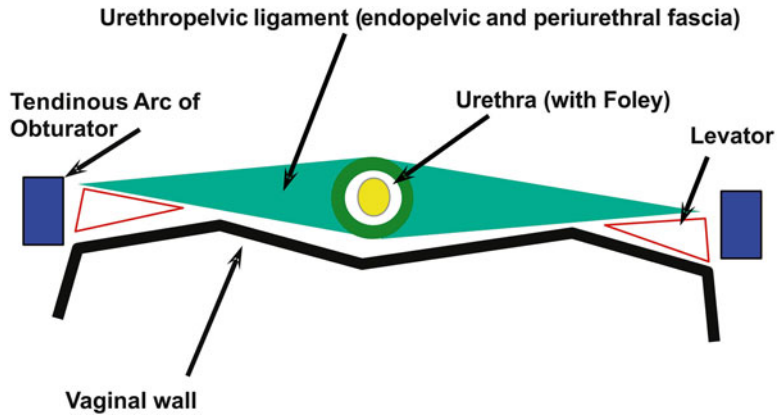


Fig. 11.1 Representative anatomy of the mid urethra in a coronal plane (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

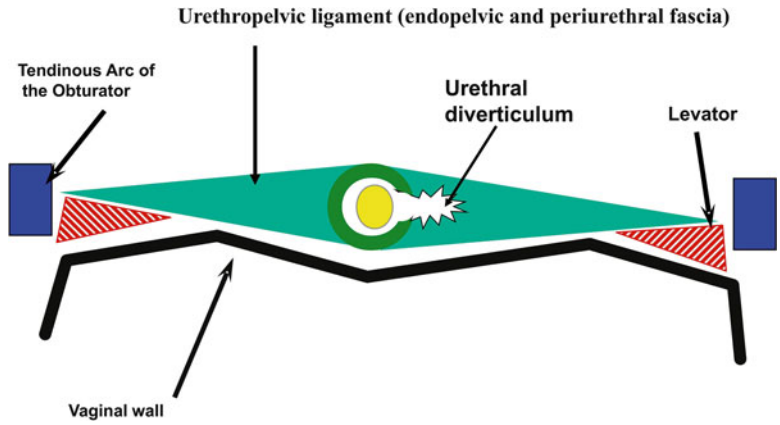


Fig. 11.2 Diagram of urethral diverticulum. The urethral diverticulum forms within and between the layers of the urethropelvic ligament (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

vagina. Nevertheless, UD have been classically attributed to recurrent infection of the periurethral glands with obstruction, suburethral abscess formation and consequent rupture of these infected glands into the urethral lumen. Continual filling and collection of urine in the resultant cavity may result in stasis, recurrent infection, and possible eventual epithelialization of the cavity forming a permanent diverticulum [8]. Reinfection, inflammation, and recurrent obstruction of the neck of the cavity are hypothesized to result in patient symptoms and enlargement of the diverticulum. However, it should be noted that Daneshgari and colleagues have also reported noncommunicating urethral diverticula diagnosed by MRI [9].

Diverticular Anatomy

Typically, UD represent an epithelialized cavity with a single connection to the lumen of the urethra. The size of the lesion may vary from just a few millimeters to several centimeters. In addition, the size may vary over time due to inflammation, intermittent obstruction of the ostia, and subsequent drainage into the urethral lumen.

The epithelium of UD may consist of columnar, cuboidal, stratified squamous, or transitional cells. In some cases, the epithelium is absent and the wall of the UD consists only of fibrous tissue. These lesions are found within the periurethral fascia, bordered by the anterior wall

of the vagina ventrally. In the sagittal plane, UD are most often centered in the middle third of the urethra with the luminal connection or ostia located posterolaterally. The sac may extend distally along the urethra and vaginal wall, almost to the meatus or proximally to the level of the bladder neck, underneath the trigone of the bladder. An array of configurations can be noted on imaging and at surgical exploration. In the axial plane, the UD cavity may extend laterally along the urethral wall and in some cases around the dorsal side of the urethra or wrap circumferentially around the entire urethra. UD may be bilobed (dumbbell shaped) extending across the midline. Multiple loculations can be common, and at least 10 % of patients have multiple UD at presentation. Varying degrees of sphincteric compromise may exist due to the location of diverticulum relative to the proximal and distal urethral sphincter mechanisms. This is especially important to note when considering surgical repair.

Evaluation/Work-Up

The diagnosis and complete evaluation of UD can be made with a combination of a thorough history, physical exam, urine culture and analysis, cystourethroscopy, and selected imaging studies. A urodynamic evaluation may also be utilized in select cases.

Presentation

Most patients with UD present between the 3rd and 7th decades of life [10–14]. The classic presentation has been historically described as the "three Ds": dysuria, dyspareunia, and dribbling (post-void). However, none of these symptoms are sensitive or specific for UD. Although presentation is quite variable, the most common symptoms are irritative (frequency, urgency, etc.) lower urinary tract symptoms (LUTS), pain, and infection [7, 15–17]. Dyspareunia will be noted by 12–24 % of patients [15, 16]. Approximately 5–32 % of patients complain of post-void dribbling [11, 15]. Recurrent cystitis or urinary tract infection is also a frequent presentation in one-third of subjects [11, 15] likely due to stasis of urine in the UD. Multiple bouts of recurrent cystitis should alert the physician to the possibility of a UD. Other complaints include vaginal pain or mass, hematuria, vaginal discharge, obstructive symptoms, urinary retention, and incontinence (stress or urge). Up to 20 % of patients diagnosed with UD might be completely asymptomatic. Some patients may present with a tender or non-tender anterior vaginal wall mass, which upon gentle compression may reveal retained urine or purulent discharge per the urethral meatus. Although spontaneous rupture of UD is extremely rare, urethrovaginal fistula may result under these circumstances [18].

The size of the diverticulum does not correlate with symptoms. Finally, symptoms may wax and wane, and even resolve for long periods of time. This may be related to periodic and repeated episodes of infection and inflammation.

As many symptoms associated with UD are not specific, patients often can be misdiagnosed and treated for years before the diagnosis of UD is made. In one series of 46 consecutive women eventually diagnosed with UD, the mean interval from onset of symptoms to diagnosis was 5.2 years [19]. This underscores the importance of a baseline level of suspicion and a thorough pelvic exam in patients complaining of LUTS or other symptoms potentially associated with UD.

Physical Examination

During physical examination, the anterior vaginal wall should be palpated for masses and tenderness. The location, size, and consistency of a suspected UD should be noted. Most UD are located ventrally over the middle and proximal portions of the urethra, corresponding to the area of the anterior vaginal wall 1–3 cm inside the introitus (Fig. 11.3). Configuration of the UD may have significant implications when undertaking surgical excision and reconstruction. UD may also extend proximally towards the bladder neck. Such UD may produce distortion of the bladder outlet and trigone on cystoscopy or on radiographic imaging and special care should be taken during

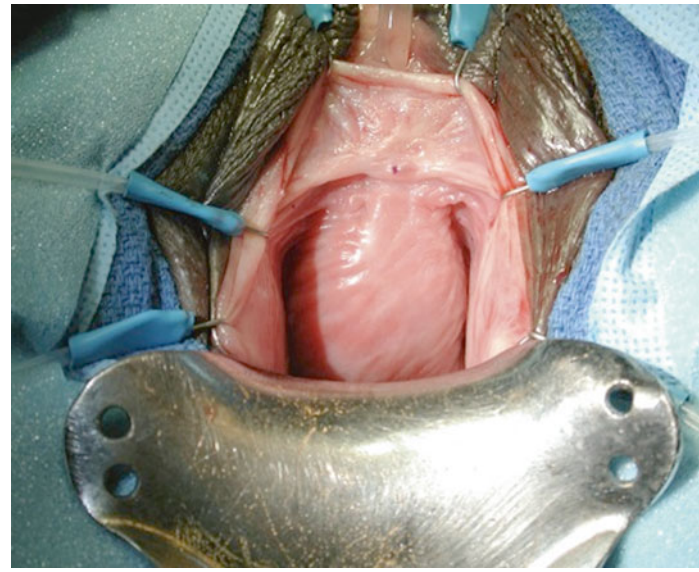


Fig. 11.3 Intraoperative image of a large urethral diverticulum

surgical excision and reconstruction due to concerns for bladder and ureteral injury as well as the potential development of postoperative voiding dysfunction and incontinence. Distal vaginal masses or perimeatal masses may represent other lesions, including Skene's glands abnormalities. The differentiation between these lesions often cannot be made by physical examination alone, and may require additional radiological imaging. A particularly hard anterior vaginal wall mass may indicate a calculus, vaginal wall fibroid, or cancer within the UD and mandates further investigation. During physical exam, the urethra may be gently "stripped" or "milked" distally in an attempt to express purulent material or urine from within the UD cavity. Although often described for the evaluation of UD, this maneuver does not produce the diagnostic discharge per urethral meatus in the majority of patients [20].

Vaginal walls should be assessed for atrophy, rugation, and elasticity. Poorly estrogenized, atrophic tissues are important to note if surgery is being considered. These tissues are often surgically mobilized and may be used for flaps during excision and reconstruction. The distal vagina and vaginal introitus are also assessed for capacity. These factors may impact surgical planning, as a narrow introitus can make surgical exposure difficult and may mandate an episiotomy. Finally, during physical examination, a provocative maneuver to elicit stress incontinence should be performed as well as an assessment of any vaginal prolapse.

Urine Studies

Urinalysis and urine culture should be performed. The most common organism isolated in patients with UD is *E. coli*. However, other gram negative enteric flora as well as *N. gonorrhea*, *Chlamydia*, *Streptococcus*, and *Staphylococcus* are often present [16, 21]. A sterile urine culture does not exclude infection, as these patients are often on antibiotic therapy at presentation. In patients with irritative symptoms or where there is suspicion of malignancy, a urine cytology can be checked.

Cystourethroscopy

Cystourethroscopy is performed, both in an attempt to visualize the UD ostia, as well as to evaluate for other potential causes of the patient's LUTS. A flexible cystoscope or a specially designed rigid female cystoscope is most helpful in evaluating the female urethra. The short beak maintains the flow

of the irrigation solution immediately adjacent to the lens and thus aids in distention of the relatively short (as compared to the male) urethra, permitting improved visualization. It may also be advantageous to compress the bladder neck while simultaneously applying pressure to the diverticular sac with an assistant's finger. Luminal discharge of purulent material can often be seen with this maneuver or with digital compression of the UD during urethroscopy. Again, the UD ostia is most often located posterolaterally at the level of the midurethra, but can be very difficult to identify in some patients. The success in identifying a diverticular ostia on cystourethroscopy is quite variable, and reported to be between 15 % and 89 % [11, 15, 20]. Failure to visualize an ostia on cystourethroscopy should not influence the decision to proceed with further investigations or surgical repair.

Urodynamics

For patients with UD and urinary incontinence or significant voiding dysfunction, a urodynamic study may be helpful [22–24]. Urodynamics can document the presence or absence of stress urinary incontinence prior to repair. Approximately 50 % of women with UD will demonstrate SUI on urodynamic evaluation [11, 25]. A videourodynamic study combines both a voiding cystourethrogram and a urodynamic study, thus consolidating the diagnostic evaluation and decreasing the number of required urethral catheterizations during the patient's work-up. For patients undergoing surgery for UD with coexistent bothersome stress urinary incontinence demonstrated on physical examination, or urodynamically demonstrable SUI, or those found to have an open bladder neck on preoperative evaluation, a concomitant anti-incontinence surgery can be offered. Multiple authors have described successful concomitant repair of urethral diverticula and stress incontinence in the same operative setting [11, 25–27]. Alternatively, on urodynamic evaluation, a small number of patients may have evidence of bladder outlet obstruction due to the obstructive or mass effects of the UD on the urethra. It should be noted that SUI may coexist with obstruction [28, 29] but nevertheless, both conditions can be treated successfully with a carefully planned operation.

Imaging

A number of imaging techniques have been applied to the study of female UD. Currently available techniques for the evaluation of UD include double-balloon positive-pressure urethrography (PPU), voiding cystourethrography (VCUG) (Fig. 11.4), intravenous urography

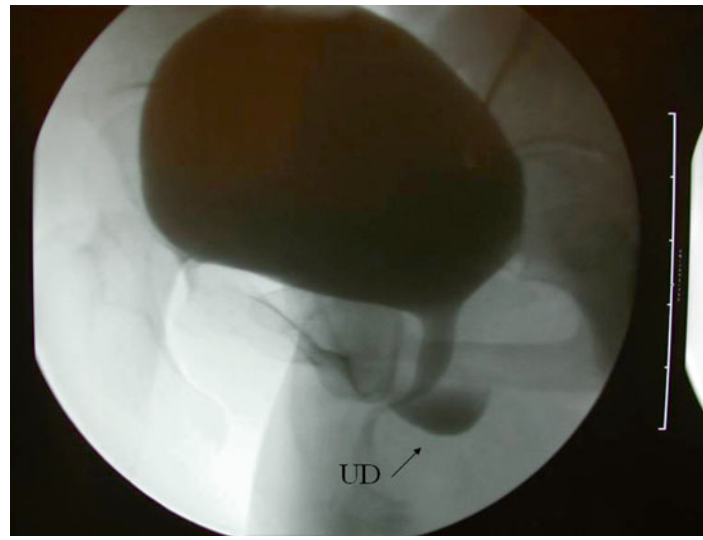


Fig. 11.4 A voiding cystourethrogram demonstrates a urethral diverticulum (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

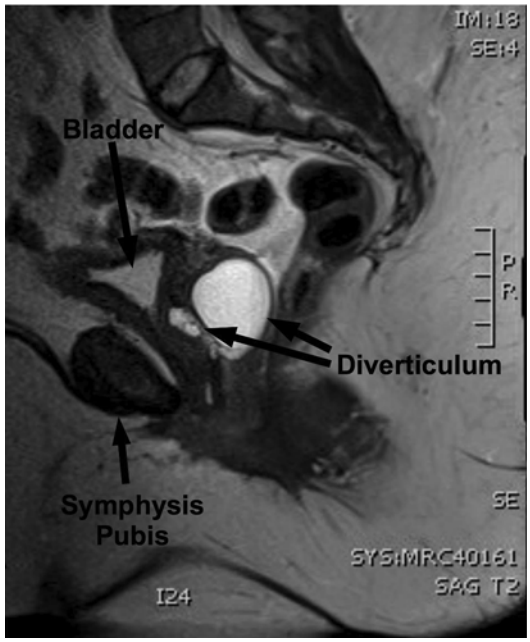


Fig. 11.5 Sagittal MRI demonstrating a urethral diverticulum

(IVU), ultrasound (US), and magnetic resonance imaging (MRI) (Fig. 11.5), with or without an endoluminal coil (eMRI). MRI has become the imaging modality of choice in many centers due to its relatively noninvasive nature, and ability to visualize the anatomy in multiple planes [30–32].

Surgical Repair

Indications for Repair

Although often highly symptomatic, not all urethral diverticula mandate surgery. Some patients may be asymptomatic at presentation, with the lesion diagnosed incidentally. Whether these lesions will progress in size, symptoms or complexity over time is not known. For these reasons, and due to the lack of symptoms in selected cases, some patients may not desire surgical therapy. However, it should be noted that there are multiple reports in the literature of carcinomas arising in UD [29, 33–40] which may be asymptomatic and may not be prospectively identified on radiologic imaging [41].

Symptomatic patients, including those with dysuria, refractory bothersome post-void dribbling, recurrent UTIs, dyspareunia and pelvic pain, may be offered surgical excision. Those with UD and symptomatic bothersome stress urinary incontinence can be considered for a concomitant anti-incontinence procedure at the time of UD excision.

Techniques for Repair

Alternative Techniques

A variety of surgical interventions for urethral diverticula have been reported since 1805 when Hey described transvaginal incision of the UD and packing of the resulting cavity with lint [1]. Approaches have included transurethral and open [42, 43] marsupialization, endoscopic unroofing [44, 45], fulguration [46], incision and obliteration with oxidized cellulose [47] or polytetrafluoroethylene [48], coagulation, and excision with reconstruction. Most commonly, a complete excision and reconstruction is performed. However, for distal lesions, a transvaginal marsupialization as described by Spence and Duckett may reduce operative time, blood loss and recurrence rates [42, 43, 49]. During this procedure, care must be taken to avoid aggressively extending the incision proximally which could result in vaginal voiding or potentially damage the proximal and distal sphincteric mechanism, resulting in postoperative stress incontinence. Therefore, this approach is probably only applicable to UD in very select cases involving the distal one-third of the urethra, and as such, it is not commonly performed.

Excision and Reconstruction

Excision with reconstruction is the most common surgical approach to UD in the modern era. The principles of the urethral diverticulectomy operation have been well described. There are only a few minor issues about which some surgeons may disagree including the type of vaginal incision (inverted "U" vs. inverted "T"), whether it is necessary to remove the entire mucosalized portion of the lesion, and finally, the optimal type of

postoperative catheter drainage (urethra only versus urethra and suprapubic).

Complex urethral reconstructive techniques for the repair of UD have been described. Fall described the use of a bipedicled vaginal wall flap for urethral reconstruction in patients with UD and urethrovaginal fistula [50]. Laterally based vaginal flaps have also been utilized as an initial approach to UD [51, 52]. Complex anatomical configurations may exist and many novel approaches have been described for complicated anterior or circumferential lesions [2, 3, 53]. The technique described herein is similar to that described by Leach and Raz [17] based on earlier work by Benjamin et al. [54] and Busch and Carter [55].

Preoperative Preparation

Prophylactic antibiotics can be utilized preoperatively to ensure sterile urine at the time of surgery. Patients can also be encouraged to strip the anterior vaginal wall following voiding, thereby consistently emptying the UD and preventing urinary stasis and recurrent UTIs. This may not be possible in those with noncommunicating UD or in those who have significant pain related to the UD. Application of topical estrogen creams for several weeks prior to surgery may be beneficial in some patients with postmenopausal atrophic vaginitis in improving the quality of the tissues with respect to dissection and mobilization. Preoperative parenteral antibiotics are often administered, especially for those with recurrent or persistent UTIs.

Patients with symptomatic stress urinary incontinence can be offered simultaneous anti-incontinence surgery. Preoperative videourodynamics may be helpful in evaluating the anatomy of the UD, assessing the competence of the bladder neck, and confirming the diagnosis of stress incontinence. In patients with SUI and UD, Ganabathi and others have described excellent results with concomitant needle bladder neck suspension in these complex patients [11, 56]. More recently, pubovaginal fascial slings have been utilized in patients with UD and stress urinary incontinence with satisfactory outcomes [19, 26, 27]. According to the AUA Stress Urinary Incontinence Guidelines, synthetic slings should not be used synchronously at the time of the surgical repair of UD [57]. There may be an increased risk of sling erosion in such circumstances.

Further complicating these cases may be associated pain, dyspareunia, voiding dysfunction, urinary tract infections, and urinary incontinence. These associated symptoms are often, but not always improved or eliminated with surgery. Therefore, the importance of appropriate preoperative patient counseling regarding surgical repair and postoperative expectations of cure cannot be overemphasized.

Procedure

The patient is placed in the lithotomy position with all pressure points well padded. The use of padded adjustable stirrups for the lower extremities greatly enhances operative access to the perineum. A standard vaginal antiseptic preparation is applied. A weighted vaginal speculum and Scott retractor with hooks aid in exposure. A posterolateral episiotomy may be beneficial in some patients for additional exposure although the midurethral (and therefore somewhat distal in the vaginal canal) location of most UD usually preclude the need for this. A Foley catheter is placed per urethra and a suprapubic tube may be utilized for additional postoperative urinary drainage. Often, a small caliber urethral catheter is utilized during the case and the placement of a suprapubic tube during the procedure ensures maximal postoperative urinary drainage. If desired, a suprapubic tube is placed at the start of the procedure either using the Lowsley retractor or percutaneously under direct transurethral cystoscopic visual guidance. Placement of a suprapubic tube at the end of the case is not advisable, as this will require traversing the fresh urethral suture line and risk disruption of the repair.

An inverted "U" is marked out along the anterior vaginal wall with the base of the "U" at the level of the distal urethra and the limbs extending to the bladder neck or beyond (Fig. 11.6). Care is taken to ensure that the limbs of the "U" are wider proximally (towards the bladder neck) to ensure adequate vascularity at the distal lateral

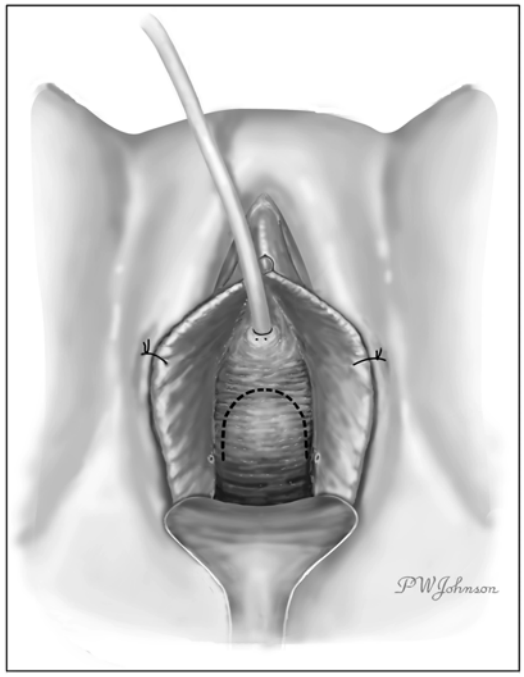


Fig. 11.6 An inverted U-shaped incision (*dashed line*) on the anterior vaginal wall (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

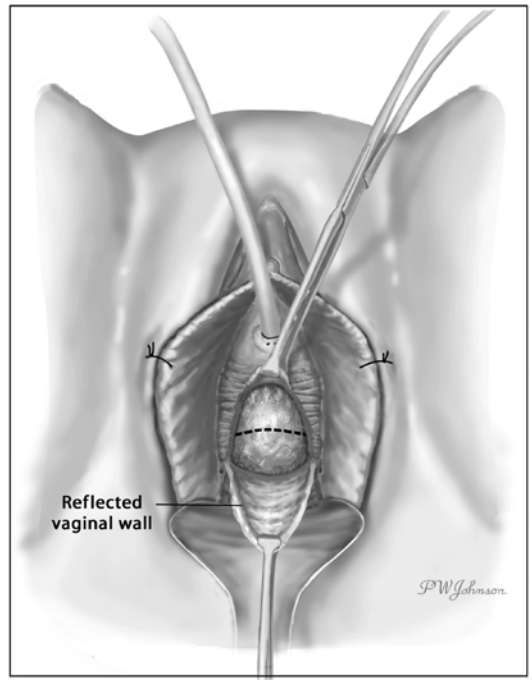


Fig. 11.7 After reflection of the anterior vaginal wall, a transverse incision is made in the periurethral fascia. The *dotted line* represents the intended incision line (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

margins of the anterior vaginal wall flap. As opposed to the inverted "T" incision, the inverted "U" incision provides excellent exposure laterally at the level of the midvagina and can be extended proximally as needed for lesions that extend beyond the bladder neck. Injectable saline can be infused along the lines of the incision to facilitate dissection. An anterior vaginal wall flap is created by careful dissection with Metzenbaum scissors in the potential space between the vaginal wall and the periurethral fascia. The use of sufficient counter-traction during this portion of the procedure is important in maintaining the proper plane of dissection. Care is taken to preserve the periurethral fascia and avoid inadvertent entry into the UD.

A distinct layer of periurethral fascia is usually interposed between the vaginal wall and the UD. Preservation and later reconstruction of this layer is of paramount importance to prevent recurrence, close dead space, and avoid urethro-vaginal fistula formation postoperatively. Pseudodiverticula have been described where this layer of tissue is considerably attenuated or even absent [58]. In these patients, an interpositional flap or graft such as a pubovaginal sling may be utilized for reconstruction.

The periurethral fascia is incised transversely (Fig. 11.7). Proximal and distal layers of periurethral fascia are carefully developed using Metzenbaum scissors, avoiding entrance into the UD. The UD is then grasped and dissected back to its origin on the urethra within the leaves of the periurethral fascia (Fig. 11.8). In many cases it is necessary to open the UD to facilitate dissection from the surrounding tissues. The ostia or connection to the urethra is identified and the walls of UD are completely removed. Every effort should be made to remove the entire mucosalized surface of the UD in order to prevent recurrence [11, 59]. This may involve removing small adherent or inflamed portions of the urethral wall, especially

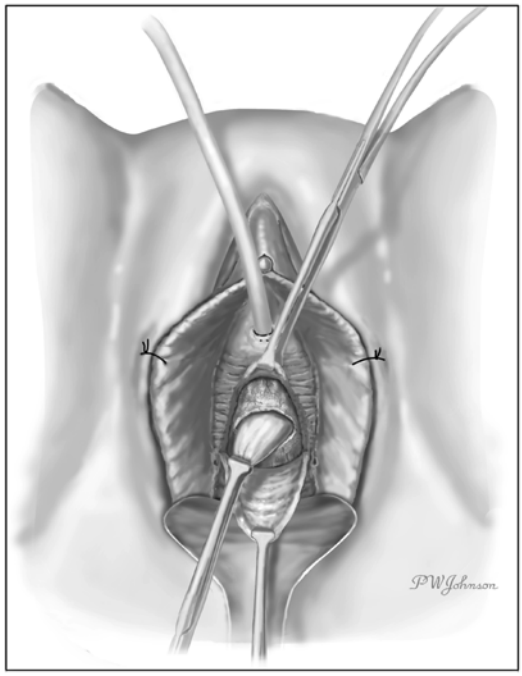


Fig. 11.8 The UD sac is freed from the periurethral fascia (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

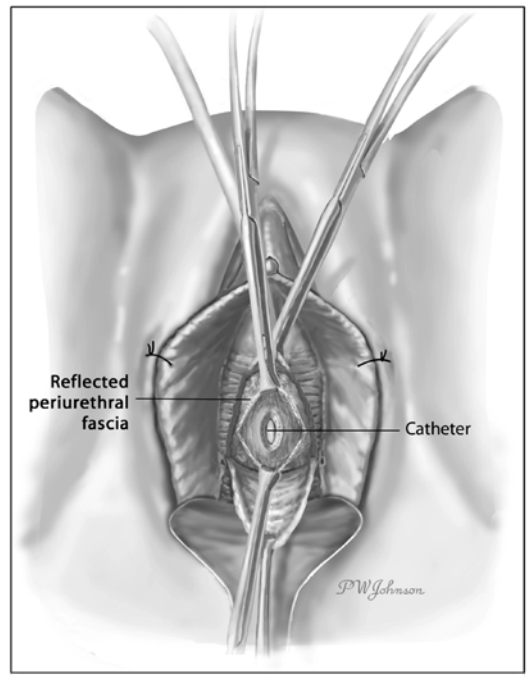


Fig. 11.9 The urethral catheter is seen after complete excision of the UD sac (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

in the area of the ostia. All abnormal tissue in the area of the ostia should be removed if possible to ensure that no mucosal elements of the UD wall remain which could result in postoperative urine leakage and recurrence. Elaborate methods of identifying the full extent of the UD cavity have been described including catheterization of the UD with urinary [12, 60] and Fogarty [61] catheters, packing the UD with gauze [62], infusing and staining the UD with methylene blue, and the use of silicone [63] or cryoprecipitate [64] to create a solid mass and ease dissection.

The Foley catheter is usually seen following complete excision of UD (Fig. 11.9). The urethra can be reconstructed over as small as a 12F Foley catheter without long term risk of urethral stricture [1] and should be closed in a watertight fashion with running or interrupted 4.0 synthetic absorbable suture. The closure should be tension-free and water tight (Fig. 11.10). In rare circumstances, a UD may extend circumferentially around the urethra and require segmental resection of the involved portion of the urethra and complex reconstruction [3, 65].

The periurethral fascial flaps are reapproximated with interrupted 3.0 synthetic absorbable suture in a perpendicular orientation to the urethral closure line to minimize overlap and the risk of postoperative urethrovaginal fistula formation (Fig. 11.11). Care is taken to secure the periurethral fascial flaps in order to close all the dead space.

If desired, a fibro-fatty labial (Martius) flap can be harvested at this point and placed over the periurethral fascia as an additional layer of closure [66]. In patients with poor quality tissues, attenuated periurethral fascia, or in whom significant inflammation is encountered intraoperatively, a well vascularized adjuvant flap such as a Martius flap may reduce the risk of wound breakdown and subsequent complications such as urethrovaginal fistula.

The anterior vaginal wall flap is then repositioned and reapproximated with running 2.0 synthetic absorbable suture. This completes a three

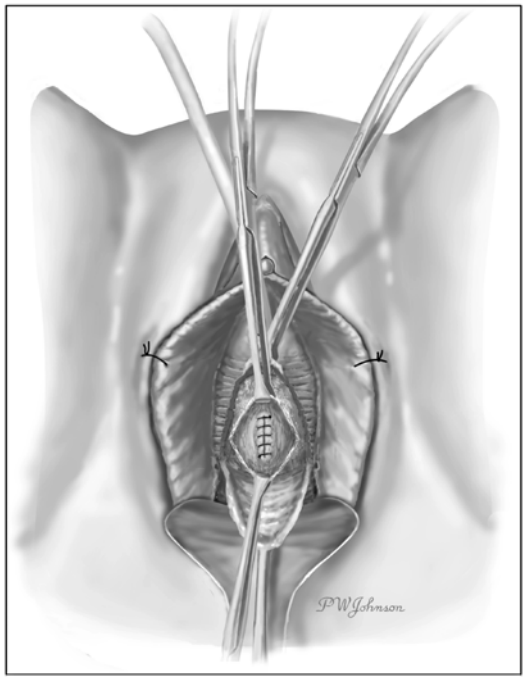


Fig. 11.10 The urethra is closed with absorbable suture (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

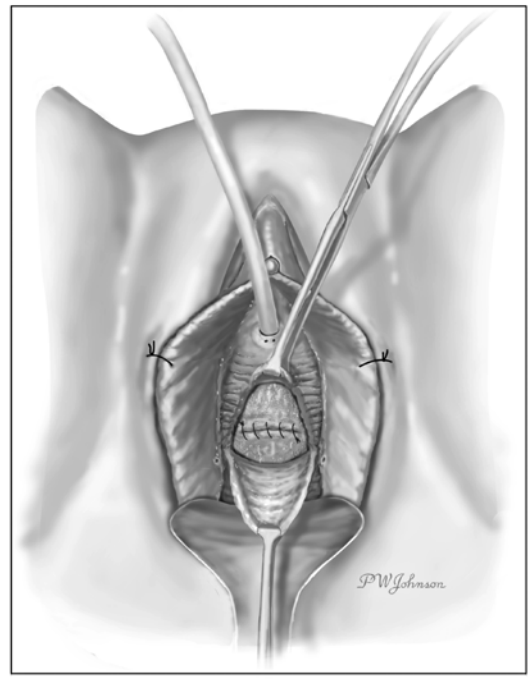


Fig. 11.11 The periurethral fascia is closed with care to obliterate any dead space (Used with permission from Rovner ES. Urethral diverticula. In: Female Urology, 3rd ed. Edited by Raz S, Rodriguez LV. Philadelphia: Saunders Elsevier; 2008)

layer closure (four layers if a Martius flap is utilized). An antibiotic impregnated vaginal pack is placed.

Postoperative Care

Antibiotics are continued for 24 h postoperatively. The vaginal packing is removed and the patient discharged home with closed urinary drainage. Antispasmodics are used to reduce bladder spasms. A pericatheter VCUG is obtained at 10–14 days postoperatively. If there is no extravasation, the catheters are removed. If extravasation is seen, then repeat pericatheter VCUGs are performed weekly until resolution is noted. In the vast majority of cases, extravasation will resolve in several weeks with this type of conservative management [67].

Complications

Careful adherence to the principles of transvaginal urethral diverticulectomy should minimize postoperative complications. Nevertheless, complications may arise. Large diverticula (>4 cm) or those associated with a lateral or horseshoe configuration may be associated with a greater likelihood of postoperative complications [68]. Common complications include recurrent UTI's (up to 31.3 %), urinary incontinence (1.7–16.1 %), or recurrent UD (1–25 %). Urethrovaginal fistula (0.9–8.3 %) is a devastating complication of urethral diverticulectomy [69]. A fistula located beyond the sphincteric mechanism should not be associated with symptoms other than perhaps a split urinary stream and or vaginal voiding. As such, an asymptomatic distal urethrovaginal fistula may not require repair, although some patients may request repair. Conversely, a proximal fistula located at the bladder neck or at the midurethra in patients with an incompetent bladder neck will likely result in considerable symptomatic urinary leakage. These patients should undergo repair with the use of an

adjuvant tissue flap such as a Martius flap to provide a well vascularized additional tissue layer. The actual timing of the repair relative to the initial procedure is controversial. Rare complications include urethral stricture (up to 5.2 %), hypospadias, distal urethral necrosis, bladder injury, urethral injury, and vaginal scarring or narrowing with consequent dyspareunia [69]. Meticulous attention to surgical technique, good hemostasis, avoidance of infection, preservation of the periurethral fascia and a well vascularized anterior vaginal wall flap, and multilayered closure with nonoverlapping suture lines should minimize the potential for postoperative urethrovaginal fistula formation.

Persistence of Symptoms Following Urethral Diverticulectomy

Some patients will have persistence or recurrence of their initial symptoms postoperatively. The finding of a UD following a presumably successful urethral diverticulectomy may occur as a result of a new medical problem (e.g., UTI, etc.), a new UD, or alternatively, as a result of recurrence of the original lesion. Recurrence of UD may be due to incomplete removal of the UD, inadequate closure of the urethra or residual dead space, or other technical factors. Lee noted recurrent urethral diverticulum in 8/85 patients at follow-up of between 2 and 15 years from the initial UD resection [70]. Repeat urethral diverticulectomy surgery can be challenging due to altered anatomy, scarring, and the difficulty in identifying the proper anatomic planes.

Summary

Urethral diverticulectomy surgeries are often challenging but ultimately very satisfying for the patient and surgeon. Many of these patients are highly symptomatic and experience relief of their symptoms with successful surgery. Adherence to principles of reconstructive surgery including careful dissection and preservation of the vascular supply of flaps, avoidance of overlapping suture lines, and watertight closure are important to ensure a satisfactory result.

References

1. Rovner ES. Urethral diverticula. In: Raz S, Rodriguez LV, editors. Female urology. 3rd ed. Philadelphia: Saunders, Elsevier; 2008. p. 825.
2. Vakil B, Wai C, Nihira M. Anterior urethral diverticulum in the female: diagnosis and surgical approach. Obstet Gynecol. 2003;102:1179.
3. Rovner ES, Wein AJ. Diagnosis and reconstruction of the dorsal or circumferential urethral diverticulum. J Urol. 2003;170:82.
4. Huffman JW. The detailed anatomy of the paraurethral ducts in the adult human female. Am J Obstet Gynecol. 1948;55:86.
5. Lang ED, Davis HJ. Positive pressure urethrography: a roentgenographic diagnostic method for urethral diverticula in the female. Radiology. 1959;72:410.
6. MacKinnon M, Pratt JH, Pool T. Diverticulum of the female urethra. Surg Clin North Am. 1959;39:953.
7. Peters III W, Vaughan Jr ED. Urethral diverticulum in the female. Etiologic factors and postoperative results. Obstet Gynecol. 1976;47:549.
8. Routh A. Urethral diverticula. BMJ. 1890;1:361.
9. Daneshgari F, Zimmern PE, Jacomides L. Magnetic resonance imaging detection of symptomatic noncommunicating intraurethral wall diverticula in women. J Urol. 1999;161:1259.
10. Johnson CM. Diverticula and cyst of the female urethra. J Urol. 1938;39:506.
11. Ganabathi K, Leach GE, Zimmern PE, et al. Experience with the management of urethral diverticulum in 63 women. J Urol. 1994;152:1445.
12. Moore TD. Diverticulum of the female urethra. An improved technique of surgical excision. J Urol. 1952; 68:611.
13. Ginsburg D, Genadry R. Suburethral diverticulum: classification and therapeutic considerations. Obstet Gynecol. 1983;61:685.
14. Pathak UN, House MJ. Diverticulum of the female urethra. Obstet Gynecol. 1970;36:789.
15. Davis BL, Robinson DG. Diverticula of the female urethra: assay of 120 cases. J Urol. 1970;104:850.
16. Davis HJ, TeLinde RW. Urethral diverticula: an assay of 121 cases. J Urol. 1958;80:34.
17. Leach GE, Schmidbauer H, Hadley HR, et al. Surgical treatment of female urethral diverticulum. Semin Urol. 1986;4:33.
18. Nielsen VM, Nielsen KK, Vedel P. Spontaneous rupture of a diverticulum of the female urethra presenting with a fistula to the vagina. Acta Obstet Gynecol Scand. 1987;66:87.
19. Romanzi LJ, Groutz A, Blaivas JG. Urethral diverticulum in women: diverse presentations resulting in diagnostic delay and mismanagement. J Urol. 2000;164:428.
20. Leach GE, Bavendam TG. Female urethral diverticula. Urology. 1987;30:407.
21. Hoffman MJ, Adams WE. Recognition and repair of urethral diverticula: a report of 60 cases. Am J Obstet Gynecol. 1965;92:106.

22. Reid RE, Gill B, Laor E, et al. Role of urodynamics in management of urethral diverticulum in females. Urology. 1986;28:342.
23. Summitt Jr RL, Stovall TG. Urethral diverticula: evaluation by urethral pressure profilometry, cystourethroscopy, and the voiding cystourethrogram. Obstet Gynecol. 1992;80:695.
24. Bhatia NN, McCarthy TA, Ostergard DR. Urethral pressure profiles of women with urethral diverticula. Obstet Gynecol. 1981;58:375.
25. Bass JS, Leach GE. Surgical treatment of concomitant urethral diverticulum and stress urinary incontinence. Urol Clin North Am. 1991;18:365.
26. Faerber GJ. Urethral diverticulectomy and pubovaginal sling for simultaneous treatment of urethral diverticulum and intrinsic sphincter deficiency. Tech Urol. 1998;4:192.
27. Swierzewski III SJ, McGuire EJ. Pubovaginal sling for treatment of female stress urinary incontinence complicated by urethral diverticulum. J Urol. 1993; 149:1012.
28. Bradley CS, Rovner ES. Urodynamically defined stress urinary incontinence and bladder outlet obstruction can coexist in women. J Urol. 2004;171:757.
29. Gonzalez MO, Harrison ML, Boileau MA. Carcinoma in diverticulum of female urethra. Urology. 1985; 26:328.
30. Pathi SD, Rahn DD, Sailors JL, Graziano VA, Sims RD, Stone RJ, McIntire DD, Wai CY. Utility of clinical parameters, cystourethroscopy, and magnetic resonance imaging in the preoperative diagnosis of urethral diverticula. Int Urogynecol J. 2013;24(2):319–23.
31. Dwarkasing RS, Dinkelaar W, Hop WC, Steensma AB, Dohle GR, Krestin GP. MRI evaluation of urethral diverticula and differential diagnosis in symptomatic women. AJR Am J Roentgenol. 2011;197(3): 676–82.
32. Blander DS, Rovner ES, Schnall MD, Ramchandani P, Banner MP, Broderick GA, Wein AJ. Endoluminal magnetic resonance imaging in the evaluation of urethral diverticula in women. Urology. 2001;57(4): 660–5.
33. Marshall S, Hirsch K. Carcinoma within urethral diverticula. Urology. 1977;10:161.
34. Patanaphan V, Prempree T, Sewchand W, et al. Adenocarcinoma arising in female urethral diverticulum. Urology. 1983;22:259.
35. Prudente d T, Dias Montellato NI, Arap S, et al. Carcinoma in diverticulum of female urethra. Urol Int. 1978;33:393.
36. Rajan N, Tucci P, Mallouh C, et al. Carcinoma in female urethral diverticulum: case reports and review of management. J Urol. 1993;150:1911.
37. Seballos RM, Rich RR. Clear cell adenocarcinoma arising from a urethral diverticulum. J Urol. 1995; 153:1914.
38. Tesluk H. Primary adenocarcinoma of female urethra associated with diverticula. Urology. 1981;17:197.
39. Thomas RB, Maguire B. Adenocarcinoma in a female urethral diverticulum. ANZ J Surg. 1991;61:869.
40. Tines SC, Bigongiari LR, Weigel JW. Carcinoma in diverticulum of the female urethra. AJR Am J Roentgenol. 1982;138:582.
41. Chung DE, Purohit RS, Girshman J, Blaivas JG. Urethral diverticula in women: discrepancies between magnetic resonance imaging and surgical findings. J Urol. 2010;183(6):2265–9.
42. Spence HM, Duckett Jr JW. Diverticulum of the female urethra: clinical aspects and presentation of a simple operative technique for cure. J Urol. 1970;104:432.
43. Roehrborn CG. Longterm follow-up study of the marsupialization technique for urethral diverticula in women. Surg Gynecol Obstet 1988;167:191.
44. Lapides J. Transurethral treatment of urethral diverticula in women. Trans Am Assoc Genitourin Surg. 1978;70:135.
45. Spencer WF, Streem SB. Diverticulum of the female urethral roof managed endoscopically. J Urol. 1987; 138:147.
46. Saito S. Usefulness of diagnosis by the urethroscopy under anesthesia and effect of transurethral electrocoagulation in symptomatic female urethral diverticula. J Endourol. 2000;14:455.
47. Ellick M. Diverticulum of the female urethra: a new method of ablation. J Urol. 1957;77:243.
48. Mizrahi S, Bitterman W. Transvaginal, periurethral injection of polytetrafluoroethylene (polytef) in the treatment of urethral diverticula. Br J Urol. 1988; 62:280.
49. Spence HM, Duckett Jr JW. Motion picture: simple operation for cure of diverticula of female urethra. Trans Am Assoc Genitourin Surg. 1969;61:78.
50. Fall M. Vaginal wall bipedicled flap and other techniques in complicated urethral diverticulum and urethrovaginal fistula. J Am Coll Surg. 1995;180:150.
51. Appell RA, Suarez BC. Experience with a laterally based vaginal flap approach for urethral diverticulum. J Urol. 1982;127:677.
52. Woodhouse CRJ, Flynn JT, Molland EA, et al. Urethral diverticulum in females. BJU. 1980;52:305.
53. Clyne OJ, Flood HD. Giant urethral diverticulum: a novel approach to repair. J Urol. 2002;167:1796.
54. Benjamin J, Elliott L, Cooper JF, et al. Urethral diverticulum in adult female. Clinical aspects, operative procedure, and pathology. Urology. 1974;3:1.
55. Busch FM, Carter FH. Vaginal flap incision for urethral diverticula. In: Western Section Meeting, American Urological Association, Honolulu; 1973.
56. Lockhart JL, Ellis GF, Helal M, et al. Combined cystourethropexy for the treatment of type 3 and complicated female urinary incontinence. J Urol. 1990;143:722.
57. Dmochowski RR, Blaivas JM, Gormley EA, Juma S, Karram MM, Lightner DJ, Luber KM, Rovner ES, Staskin DR, Winters JC, Appell RA. Update of AUA guideline on the surgical management of female stress urinary incontinence. J Urol. 2010;183(5):1906–14.
58. Leng WW, McGuire EJ. Management of female urethral diverticula: a new classification. J Urol. 1998; 160:1297.

59. Fortunato P, Schettini M, Gallucci M. Diagnosis and therapy of the female urethral diverticula. Int Urogynecol J. 2001;12:51.
60. Kohorn EI, Glickman MG. Technical aids in investigation and management of urethral diverticula in the female. Urology. 1992;40:322.
61. Wear JB. Urethral diverticulectomy in females. Urol Times. 1976;4:2.
62. Hyams JA, Hyams MN. A new operative procedure for the treatment of diverticulum of the female urethra. J Urol. 1939;43:573.
63. Hirschhorn RC. A new surgical technique for removal of urethral diverticula in the female patient. J Urol. 1964;92:206.
64. Feldstein MS. Cryoprecipitate coagulum as an adjunct to surgery for diverticula of the female urethra. J Urol. 1981;126:698.
65. Tamada S, Iwai Y, Tanimoto Y, et al. Urethral diverticula surrounding the urethra in women: report of 2 cases. Hinyokika Kiyo—Acta Urologica Japonica. 2000;46:639.
66. Dmochowski R. Urethral diverticula: evolving diagnostics and improved surgical management. Curr Urol Rep. 2001;2:373.
67. Schwab CW, Rovner ES. Utility of radiologic imaging and prolonged catheterization following complex lower urinary tract reconstruction. Presented at the Mid-Atlantic AUA Sectional meeting, Boca Raton, FL; 2003.
68. Porpiglia F, Destefanis P, Fiori C, et al. Preoperative risk factors for surgery female urethral diverticula. Our experience. Urol Int. 2002;69:7.
69. Rovner E. Bladder and female urethral diverticula. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. Campbell-Walsh urology. 10th ed. Philadelphia: Elsevier, Saunders; 2012.
70. Lee RA. Diverticulum of the female urethra: postoperative complications and results. Obstet Gynecol. 1983;61:52.

Augmentation Cystoplasty

12

Geneviève Nadeau and Sender Herschorn

Introduction

Augmentation cystoplasty (AC) is a surgical procedure used to increase bladder storage capacity or improve detrusor compliance. Performed by urologists for more than a century, the technique has evolved over the years, using different segments of the gastrointestinal (GI) tract. It is frequently combined with other procedures depending on the need for correction of associated problems (bladder outlet correction, ureteral obstruction, etc.), and more recently has been performed laparoscopically or robotically. Even though its role has declined over the years, decreasing from 192 operations in 2000 in the United Kingdom to 120 in 2010 [1], it remains of paramount importance for the reconstructive urologist to be proficient with this procedure. This chapter will outline the indications and technique of bladder augmentation as well as focus on the short- and long-term complications.

G. Nadeau, M.D., M.Sc., F.R.C.S.C.
Division of Urology, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, ON, Canada
e-mail: Genevieve.nadeau.6@ulaval.ca

S. Herschorn, B.Sc., M.D.C.M., F.R.C.S.C. (✉)
Division of Urology, Department of Surgery, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, ON, Canada
e-mail: s.herschorn@utoronto.ca

History

First described in dogs in 1888 by Tizzoni and Foggi [2] and in 1899 in humans by von Mikulicz [3], augmentation cystoplasty was reported by Couvelaire in 1950 to treat tuberculosis cystitis [4]. The use of different segments of the GI tract has been described: colon in 1912 by Charghi [5], sigmoid by Bisgard in 1943 [6], cecum by Couvelaire [4], stomach in 1956 by Sinaiko [7], and finally the classic detubularized ileal patch by Goodwin in 1959 [8]. A wide variety of other organic tissues (peritoneum, omentum, gallbladder, skin, etc.) have also been unsuccessfully attempted and abandoned [9]. Finally, the introduction of clean intermittent catheterization (CIC) by Lapides in 1972 allowed the procedure to gain widespread acceptance [10].

Indications

Considering that AC is an invasive procedure, it should be offered only after more conservative interventions such as behavioral modification, anticholinergics, botulinum toxin (BTX-A), or sacral neuromodulation have been considered and failed, as recommended by the 4th International Consultation on Incontinence [11]. Many conditions may necessitate AC (Table 12.1), whether in the setting of upper tract deterioration due to high bladder storage pressures (detrusor leak point pressure over 40 cm H_2O) or unacceptable incontinence

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_12,

Table 12.1 Indications for augmentation cystoplasty

	Indications
Congenital	Myelodysplasia
	Tethered spinal cord
	Exstrophy (classic, cloacal, epispadias)
	Sacral agenesis
	Caudal regression
	Posterior urethral valves
Acquired neurogenic	Spinal cord injury
	Spinal tumors
	Myelitis
	Multiple sclerosis
	Idiopathic
Acquired non-neurogenic	Detrusor overactivity
	Defunctionalized bladder in patients on dialysis
Infectious	Tuberculosis
	Schistosomiasis
Inflammatory	Interstitial cystitis
	Radiation cystitis
	Chemotherapy-induced cystitis
Iatrogenic	Intraoperative loss of bladder wall (after extirpative surgery for malignancy)
	Urinary undiversion

secondary to contracted bladder volume and decreased compliance or overactivity [12].

Neurogenic conditions caused by congenital, acquired, or traumatic etiologies, especially myelodysplasia in children and spinal cord injuries in adults, are probably the most frequent indications. Although there is considerable variation in institutional rates, it has been estimated that 5–30 % of patients with spina bifida will undergo AC [13, 14]. Multiple sclerosis, with its inherent progressive neuromuscular deterioration that may make intermittent self-catheterization difficult [15], is preferably managed with medical therapy such as anticholinergics and botulinum toxin but occasional cases may be amenable to AC.

Patients with severe refractory idiopathic overactive bladder (OAB) or rarely interstitial cystitis can also be offered such a procedure, but this should be a last resort option for carefully selected cases, as pointed out by the American Urological Association guidelines [16, 17].

Benign diseases with decrease in compliance from collagen deposition in the bladder wall are other indications. Tuberculosis cystitis, which used to be the number one indication in the past, is now a rarity [15]. Schistosomiasis, an endemic parasitic infection found primarily in the Middle East and Africa, may cause bladder wall fibrosis in 2 % of cases [1]. Reduced bladder capacity may be improved by AC.

Pelvic radiation therapy or surgical resection for colorectal, gynecological, or urological malignancies may also compromise the urinary tract and necessitate AC.

Finally, a defunctionalized bladder in a patient on dialysis awaiting renal transplantation might warrant AC to restore proper lower urinary tract function and protect the allograft, although its timing (before, after, or even concomitantly with the transplantation) is controversial [18], the concern being the risk of severe sepsis in the immunosuppressed patient [1].

Contraindications

Poor baseline renal function may potentially expose patients to severe electrolyte abnormalities and worsening renal function and is a relative contraindication. However, in patients with renal dysfunction that is a direct result of bladder dysfunction with elevated storage pressure, AC may be appropriate and help stabilize renal function [19]. Other relative contraindications include inflammatory bowel disease (Crohn's disease), irradiated bowel, short gut syndrome, bladder tumors, and liver failure [20].

Preoperative Surgical Considerations

Extensive history and physical exam, serum chemistries (complete blood count, electrolyte and creatinine levels, coagulation factors), urine cytology, urinalysis, and cultures are required. All patients should also undergo anatomic and functional assessment of the urinary tract with cystoscopy (to exclude intravesical abnormalities

such as tumors), urodynamics (to characterize bladder capacity, compliance, or uninhibited detrusor contractions, to assess detrusor and Valsalva leak point pressures, and to rule out infravesical obstruction), cystography (to rule out vesicoureteral reflux), and upper tract imaging (ultrasound or computerized tomography; to document the presence of hydronephrosis or stone disease). A history of bowel disease may require preoperative bowel imaging studies or colonoscopy. A voiding diary can help in planning for the final reservoir size by assessing 24-h urine volume [21], although the augmentation usually enlarges with time. The need for latex precautions for at-risk patients with spinal dysraphism should also be kept in mind. Planning for other concomitant procedures such as a continent catheterizable stoma (using the Mitrofanoff or Monti principles), ureteral reimplantation, or bladder neck management is an essential prerequisite before considering the intervention [22]. Finally, since CIC is frequently a necessary adjunct, adequate manual dexterity and cognitive abilities are prerequisites to be considered an adequate surgical candidate and it is crucial, in the preoperative visits, to reinforce the importance of compliance with CIC postoperatively [23].

Bowel Preparation and Antibiotic Coverage

Preoperative mechanical bowel preparation continues to be an issue of controversy. In the colorectal surgery literature, it does not lower the rate of postoperative complications (wound infection, intraperitoneal abscess, or anastomotic leak) [24, 25] and some patients have reported adverse events from it [26–28]. There are two pediatric studies that have reported no increase in complications after AC following no preoperative mechanical bowel preparation [29, 30]. However, there are no prospective randomized trials so caution should be exercised especially in patients with ventriculoperitoneal shunts that may be exposed to fecal contamination intraoperatively [31]. Even though there is no consensus among the studies with the duration, the dosage, and the type of bowel preparation to use, we agree with the most recent CDC guidelines (published in 1999) [32] and still routinely prescribe mechanical bowel preparation and clear fluids diet the day prior to the intervention.

The periprocedural systemic administration of an antimicrobial agent to reduce infectious risks in contaminated urology surgery [33] is an evidence-based supported concept, but the literature is not clear about the optimal therapeutic regimen (type of medication, dosage, and route of administration). Available practice guidelines recommend a combination of either second- or third-generation cephalosporin with an aminoglycoside and metronidazole, with special caution for patients with prosthetic devices such as ventriculoperitoneal shunts or orthopedic hardware [32, 33].

Choice of Intestinal Segment

Different parts of the GI tract from stomach to sigmoid have been used for bladder augmentation, each having its pros and cons (Table 12.2), but none being the ideal segment.

Ileum is unquestionably the most widely used bowel segment in reconstructive urology. The versatility it provides allows the surgeon to refashion it in a multitude of different techniques that have been elaborated over the years [34]. With overall fewer complications than other bowel segments, it is mobile, easy to handle, with a constant blood supply, and available in large quantity [35]. However, ileum might not be ideally suited in situations where the mesentery is short, or for those with significant adhesions or prior small bowel resections [23]. Caution should be also exercised when considering ileocystoplasty for patients who underwent prior pelvic radiation therapy [36].

If functional or anatomic factors preclude the use of ileum, colon is often the second choice. Advantages of colocystoplasty include a thick muscular wall, large lumen, and abundant mesentery guaranteeing adequate bladder capacity and maneuverability [37], while disadvantages include more mucus production with increased

Table 12.2 Advantages and disadvantages of different tissues used for augmentation cystoplasty

Tissue	Advantages	Disadvantages
Stomach	Produces less mucus Lower incidence of bacteriuria Rich blood supply More appropriate for patients with renal insufficiency	Risk of bladder ulcers and perforation Hematuria–dysuria syndrome Vit B12 malabsorption
Jejunum		Severe electrolyte abnormalities Risk of profound dehydration Iron and calcium deficiencies
Ileum	Mobile, small diameter, easy to handle Well-defined, reliable blood supply Less severe metabolic complications	Lipid malabsorption (Vit A, D, E, K) Vit B12 deficiency (anemia) Incidence of bowel obstruction more common than colon Lack of bile salt reabsorption (diarrhea) Metabolic acidosis Sometimes short mesentery
Colon	Transverse colon safer if prior pelvic radiation Fewer nutritional problems Redundant in spina bifida patients Antireflux tunnels easily made	Metabolic acidosis Produces more mucus than ileum (increased risk of UTIs and stones)
Ureter	Urothelial lined Requires no intestinal resection	Limited availability (need for hydroureter)
Autoaugmentation	Urothelial lined Requires no intestinal resection	Technically demanding Limited gain in capacity and compliance Risk of perforation
Tissue engineering	Theoretically unlimited donor tissue	Still experimental

Data from Abou-Elela A. Augmentation cystoplasty: in pretransplant recipients. In: Ortiz J, Andre J, eds. Understanding the Complexities of Kidney Transplantation. Rijeka, Croatia: InTech; 2011:279–330; and Duel BP, Gonzalez R, Barthold JS. Alternative techniques for augmentation cystoplasty. J Urol. 1998;159(3):998–1005

risk of urinary tract infections (UTI) and stone formation [38]. More specifically, the sigmoid is sometimes redundant in patients with neurogenic bowel dysfunction which makes it an attractive alternative for this population [22]. Finally, the cecum, in conjunction with the terminal ileum, can be used as a continent catheterizable channel [39]. The ileocecal valve provides the continence mechanism, but its resection renders the patient vulnerable to diarrhea [40], which occasionally may be severe and difficult to manage. This may render the patient at risk for malabsorption and fecal incontinence [35].

Gastrocystoplasty as an alternative has been mostly reported in the pediatric literature [41, 42]. Despite clear advantages to the use of the stomach such as less mucus production, a lower incidence of bacteriuria, and a rich blood supply, the technique has declined in popularity due to significant limitations, mostly from debilitating hematuria–dysuria syndrome [43].

The use of jejunum results in severe metabolic disorders in 25–40 % of cases and therefore should not ordinarily be utilized [44, 45]. Although not formerly contraindicated, its use should be limited to those extremely rare cases where any other segment of the GI tract is not available for the augmentation, a situation we have not yet encountered.

Alternative tissues have also been described. Several series have reported satisfactory outcomes with ureterocystoplasty [38, 46, 47]. This necessitates a hydroureter from a nonfunctioning renal unit and is practically rarely applicable [47, 48]. In autoaugmentation (or detrusor myectomy) of the bladder, the creation of a urothelial pseudodiverticulum is technically demanding and usually only provides a slight gain in capacity

and compliance [49]. With such disappointing efficacy, the technique is rarely reported.

Finally, none of the bowel segments has been shown to be clearly superior to another in all circumstances so the reconstructive urologist needs to be proficient in several techniques making use of various segments in order to individualize the decision and optimize results.

Technique

With the ultimate goals of lowering urinary storage pressures, preserving renal function, maximizing continence, and hopefully maintaining volitional voiding, the clam ileocystoplasty is the most commonly reported technique.

With the patient supine or in the low lithotomy position, a midline infraumbilical incision is usually adequate to gain intraperitoneal access, but a short Pfannenstiel incision has also been described [50]. With a Foley catheter in the surgical field, the native bladder is distended. The loose areolar tissue is bluntly dissected to expose the anterior and perivesical spaces [51]. With stay sutures on either side, the bladder is bivalved in the coronal or sagittal plane [11]. A "smile" incision has also been described [52]. The bladder incision should be a broad opening to create a large anastomosis and prevent an hourglass deformity [35]. Occasionally, supratrigonal cystectomy can be done for conditions such as severe interstitial cystitis [53].

A 15–40 cm segment of ileum is then harvested approximately 20 cm proximal to the ileocecal valve on a broad well-vascularized pedicle that may be identified by transillumination. A side-to-side stapled or hand-sewn ileoileostomy reestablishes bowel continuity cephalad to the isolated segment and the mesenteric defect may be closed to prevent internal hernias, or alternatively left open if judged to be wide enough. The isolated segment is irrigated thoroughly until return is clear to prevent intraperitoneal soiling as much as possible. It is then detubularized on its antimesenteric side to prevent intraluminal pressure rises from peristaltic contractions. The segment is reconfigured into a U, S, or W shape [36, 54] (Figs. 12.1 and 12.2). The surgeon should make sure that the segment reach down into the pelvis without tension or twist on the mesentery. The ileal patch can then be reapproximated to the edges of the cystotomy, starting posteriorly, with one to two layers of 2-0 continuous absorbable sutures. The vesico-intestinal anastomosis is tested for watertightness and drains are inserted prior to closing the abdominal wall in a standard fashion. The goal is to create a new reservoir in as spherical a configuration as possible to maximize the surface area as per Laplace's law.

More recently, minimally invasive (laparoscopic and robot-assisted) approaches have become more frequently reported from a number of institutions. Depending on the surgeon's experience and expertise, most of the suturing can be performed intra- or extracorporeally [55]. More technically demanding, these minimally invasive techniques have the advantages of reduced perioperative pain and morbidity, shorter hospital stay, and cosmetic superiority, with outcomes similar to the open surgery [20, 56–58].

Postoperative Care

As for any major abdominal surgery, intravenous fluids and bowel rest are maintained for several days with close monitoring of inputs and outputs. Routine nasogastric tube drainage is not required as it has not been shown to lead to faster bowel function recovery [59]. Antibiotic prophylaxis for 24 h [60], thromboprophylaxis, and early ambulation are also recommended. A closed-suction pelvic drain is kept until output tapers off or once peritoneal fluid is confirmed with fluid chemistries [21].

Proper drainage and irrigation of the augment, initially several times a day with 50–100 mL of sterile saline and then gradually reduced to an as-needed basis, are necessary to prevent mucus from accumulating, plugging the catheters and disrupting the fresh anastomosis. We usually favor a Foley urethral catheter (16–18 Fr) and a suprapubic (SP) Malecot catheter (20–22 Fr). Both tubes are kept for approximately 4 weeks, after which a cystogram is performed. The Foley is then removed if there is no extravasation and patients resume spontaneous voiding. Post-void

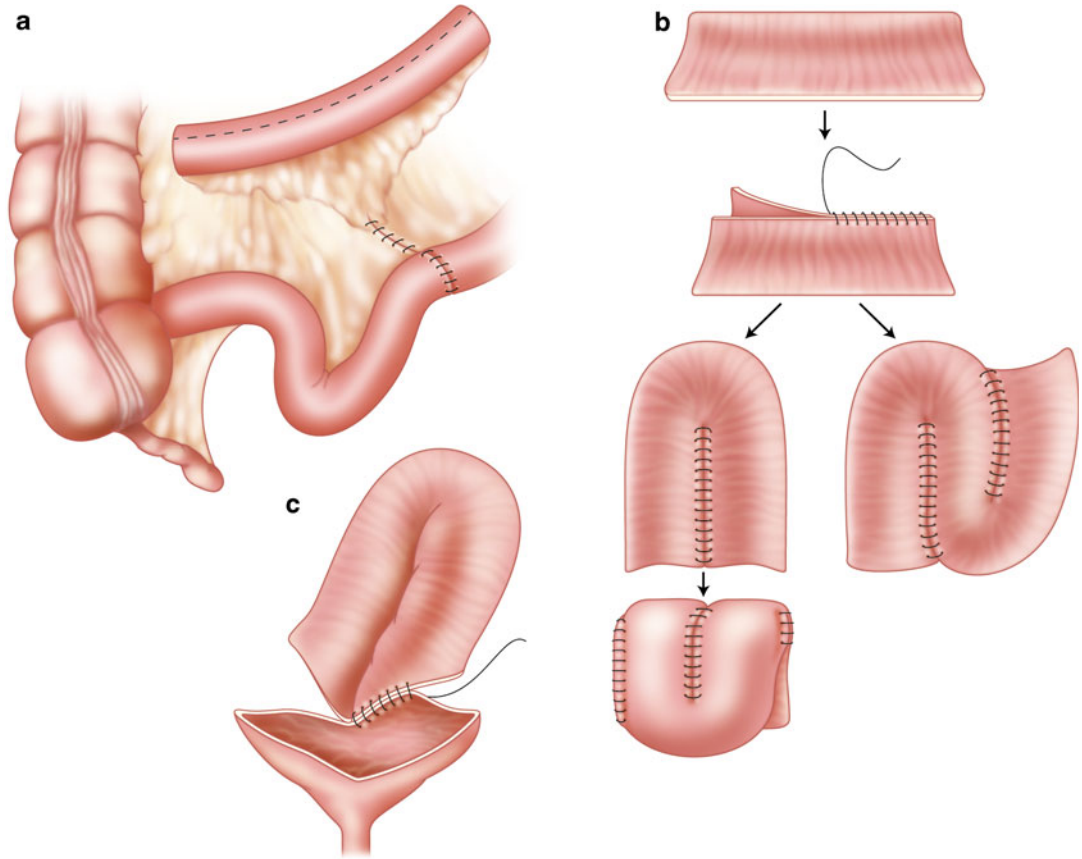


Fig. 12.1 (**a**–**c**) (**a**) Ileocystoplasty. A 20- to 40-cm segment of ileum at least 15 cm from the ileocecal valve is removed and opened on its antimesenteric border. Ileoileostomy reconstitutes the bowel. (**b**) The opened ileal segment should be reconfigured. This can be done in a U, S, or W configuration. It can be further folded as a cup patch. (**c**) The reconfigured ileal segment is anastomosed widely to the native bladder

residuals are closely monitored initially as well as on a long-term basis, and CIC is initiated when needed. The SP tube may be kept for 1–2 more weeks longer and used for daily bladder irrigation. Although there is no evidence to support it, antimicrobial prophylaxis can be administered at the time of catheter removal since bacterial colonization has likely occurred [33].

Complications

Flood and colleagues reported an overall 28 % early and 44 % late complication rate in this difficult group of patients, with a chance of requiring a reintervention ranging from 15 % to 40 % [61–63]. Table 12.3 lists the possible consequences of such procedure.

Early Complications

Cardiovascular, respiratory, thromboembolic, and gastrointestinal complications can occur in the early postoperative course, as for any major abdominal surgery. The most common ones include wound infection or dehiscence (5–6 %), prolonged ileus (5 %), anastomotic leakage (2–4 %), catheter obstruction from mucus, ventriculoperitoneal shunt sepsis (0–20 %) [64], and peroneal nerve palsy [19]. Contemporary publications in the literature report a mortality rate of 0–3.2 % [19].

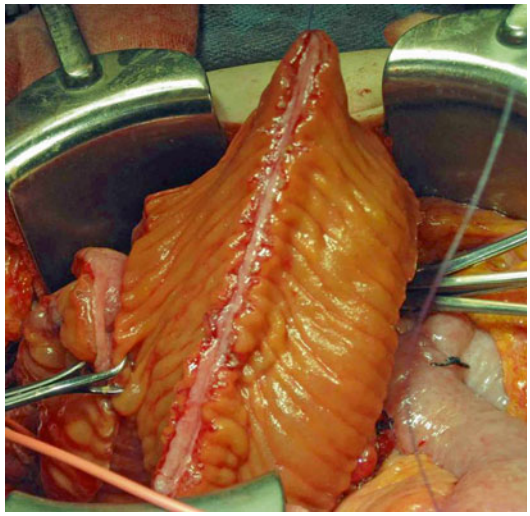


Fig. 12.2 A 40 cm length of ileum is shown. The segment has been isolated from the GI tract and reconfigured. The antimesenteric border was incised and the bowel segment was detubularized into an inverted U shape. It will be anastomosed to the bladder

Late Complications

Bacteriuria

Asymptomatic bacteriuria in patients on CIC is nearly universal regardless of the segment considered and should not be treated except for infection with urease-splitting organisms [65]. Recurrent episodes of symptomatic cystitis do require treatment, but symptoms may be nonspecific. Risk factors predisposing to UTI include mucus accumulation, stasis, and CIC [66]. Symptomatic urinary tract infection which occurs in 5–40 % of patients [19, 67, 68] requires antibiotic treatment. With regard to prophylaxis, as recommended by the European Association of Urology, low-dose, long-term, antibacterial prophylaxis may be an option for patients with recurrent UTI, but there is a risk of emergence of multiresistant organisms [69].

Table 12.3 Complications of augmentation cystoplasty

Early	Late
Infection	Metabolic disturbances
Wound infection	Metabolic acidosis
Intraperitoneal abscess	Hypokalemia
UTI	Hypocalcemia
Ventriculoperitoneal shunt sepsis	Hypomagnesemia
	Hyperammonemia (encephalopathy)
Wound dehiscence	Mucus accumulation
Prolonged ileus	Urolithiasis
Peroneal nerve palsy	Bacteriuria/Pyelonephritis
Anastomotic leak	Bowel dysfunction
Urinary	Diarrhea
Intestinal	Fecal incontinence
	Bowel obstruction
Fistula	Neoplasia
Hemorrhage	Bladder perforation
Death	Upper tract deterioration/Renal dysfunction
	Malabsorption
	Vitamins A, D, E, K deficiencies
	Bile salts (gallbladder stones)
	Vitamin B12 deficiency (megaloblastic anemia)
	Hematuria–dysuria syndrome (gastric)
	Drug absorption toxicities
	Bone demineralization/Impaired growth
	Urinary retention/Diverticularization
	Incontinence

Data from Herschorn S WB. Bladder augmentation. In: HB G, ed. Complications of female incontinence and pelvic reconstructive surgery. Cleveland, OH: Humana Press; 2013:171–187; Flood HD, Malhotra SJ, O'Connell HE, Ritchey MJ, Bloom DA, McGuire EJ. Long-term results and complications using augmentation cystoplasty in reconstructive urology. Neurourol Urodyn. 1995;14(4):297–309; and de Petriconi R. Metabolic aspects of bowel use in urologic surgery. Ann Urol (Paris). 2007;41(5):216–236

Urolithiasis

The incidence of calculi in augmented bladders ranges from 10 % to 50 % [70, 71]. Stone composition is more commonly struvite or calcium oxalate [72]. Stasis, incomplete emptying, excessive mucus production, and chronic bacteriuria, especially if urease-splitting organisms, predispose to stone formation [73, 74]. Patients with a continent catheterizable channel (which may not drain the bladder completely), those using urethral CIC (compared to those voiding spontaneously), and patients with urease-splitting bacteriuria are at increased risk [19, 75]. Permanent sutures can serve as a nidus and should be avoided as much as possible [35]. Prevention strategies include increased fluid intake, routine bladder irrigation, prompt treatment of UTI, and staple exclusion at the time of surgery [74].

Perforation

Bladder perforation is a potentially life-threatening complication and is due to either overdistension or trauma from catheterization. It has been reported up to 13 % [76]. Patients with neurogenic bladders, those with competent bladder necks, those without a catheterizable channel, and those who abuse alcohol appear to be at an increased risk [19, 77–79]. Patients can present with an acute abdomen, but symptoms can be more subtle in neurologically impaired patients. The diagnosis can be made with a CT cystogram but requires a high index of suspicion [76] and low threshold for exploratory laparotomy [79]. The area of perforation is usually at the vesico-intestinal anastomosis or within the weaker bowel wall [80]. Emphasizing the importance of compliance with regular CIC can help obviate the risk of early perforation [76].

Mucus Production

The average daily mucus production is about 40 g and does not taper off with time despite villous atrophy [1, 81]. With time, accumulated proteinaceous material can become a nidus for infection and stone or impair adequate bladder emptying [19]. Colonic segments produce more mucus than ileum [82]. Daily irrigation can help reduce mucus retention [83]. These can be augmented with acetylcysteine or urea irrigations which help dissolve mucus [82] or oral ranitidine which may help to reduce mucus production [84].

Renal Function

Renal function deterioration can occur in about 20 % of patients after AC [23]. The etiology of renal dysfunction may be urinary stone disease, bacteriuria, high detrusor pressures, vesicoureteral reflux, unrecognized obstruction, and lack of compliance with CIC [85]. The patient's initial kidney function predicts long-term outcome, but in all cases close postoperative monitoring is warranted with periodic ultrasound imaging and laboratory studies.

Neoplasia

Bladder cancer following AC is a recognized risk, but remains a rare complication, with an incidence of approximately 1 % [86]. The risk may be similar in patients with CIC and bladder dysfunction due to neurologic abnormalities, exstrophy, and posterior urethral valves [87]. Tumors are usually adenocarcinomas that occur at the vesico-intestinal junction [86]. The exact etiologic mechanism of the development of neoplasia is not well understood, but proposed hypotheses include chronic inflammation from prolonged exposure of the intestinal epithelium bathed in urine, the production of carcinogenic nitrosamines by chronic bacteriuria, and/or mixed cell-to-cell interactions from juxtaposition of tissues from two different origins: intestinal and urothelial [88–90]. We do advocate routine surveillance with endoscopy and urine cytology on a long-term basis despite inconsistent evidence to support it, considering the long latency period of about 15 years [91, 92]. Although there is currently no consensus, we begin surveillance about 5–10 years after initial surgery and follow patients every year or every other year thereafter.

Metabolic Disturbances

Translocation of functional enteric epithelium into the urinary tract can result in electrolyte and acid–base abnormalities which are directly

related to the segment of intestine used, the amount of time urine spends in contact with the bowel mucosa, and renal function [38, 93]. The length of bowel segment used in AC is usually relatively short which partly reduces the incidence of metabolic disorders compared to other types of diversion [94]. The classic electrolyte pattern for ileum and colon is hyperchloremic metabolic acidosis [39], from net absorption of NH_4 and to a lesser extent bicarbonate losses [95]. Low serum potassium as well as hypocalcemia and hypomagnesemia can also occur, but they are less common.

Nutritional disorders are rare if the surgeon spares the terminal ileum, which is the sole site of absorption for vitamin B12. Resection of the distal 20 cm should be avoided to prevent such deficiency which may cause megaloblastic anemia and neurologic changes [95]. If more than 100 cm of ileum is resected, lipid malabsorption is virtually inevitable and there is also fat-soluble vitamin (A, D, E, and K) malabsorption [96].

When stomach is used, a hypokalemic hypochloremic metabolic alkalosis can occur [97]. The exclusion of a jejunal segment is rarely used in urology due to the severe hyponatremic hypochloremic hyperkalemic metabolic acidosis that may result [98].

Other consequences from metabolic disturbances include hyperammonemia secondary to the inability of the liver to clear it, which could lead to encephalopathy [95], and bone demineralization in adults or impaired bone growth in children [99] from chronic acidosis. Finally, certain drugs (digoxin, methotrexate, phenylalanine, antibiotics, theophylline) can be reabsorbed by the intestinal segment and cause toxicity [94].

Bowel Dysfunction

Patients can present a prolonged paralytic ileus, especially in those with neurological impairment as they often already suffer from slow transit [100]. Intestinal obstruction from adhesions can occur in the early or late postoperative setting and usually resolves with conservative management [83]. With time, removal of the ileocecal valve may yield a decreased stool transit time and result in intractable diarrhea, steatorrhea (from fat malabsorption), or distressing fecal incontinence [21].

Pregnancy

Successful pregnancies and deliveries after AC have been reported and are becoming more common. There is a substantial rate of obstetrical complications, mostly preterm labor and preeclampsia, the latter being more challenging to diagnose considering the inaccuracy of urinalysis for proteinuria due to mucus from the enteric segment [101]. Antenatal close monitoring is recommended and should include regular midstream urine cultures [102] in order to treat any UTI and minimize the risk of renal scarring and impairment. In spite of this increased risk of ascending bacteria, pyelonephritis, and upper tract obstruction over the general population [103], lower urinary tract function is usually not adversely affected by pregnancy or delivery, as reported by Greenwell et al. [104]. Unless the patient has had bladder neck reconstruction or insertion of an artificial urinary sphincter [105], vaginal delivery seems preferable. If an elective cesarean section is necessary, a classical upper-segment rather than a lower-segment section should be favored [19], with urologic assistance to avoid the potential risk of damaging the vascular pedicle of the bowel segment, in which case eventual ischemic enteric contraction could occur [39].

Outcomes

From a clinical standpoint, we aim for patients to be free of symptomatic UTIs, continent, and with a stable renal function without hydronephrosis or stones. Only a few small retrospective case series (Level 4 evidence) have reported outcomes, but comparisons are difficult due to lack of standard terminology and the use of non-validated questionnaires [83]. The success rate, defined as achieving continence with or without CIC, has ranged from 67 % to 100 % [61, 63, 106]. The average rate for CIC after AC is 54 % (range

14–100 %) [104]. The improvement in quality of life for individuals undergoing enterocystoplasty is substantial. However, discrepancy exists between neuropathic and non-neuropathic patients regarding their satisfaction as assessed by means of a questionnaire. Indeed, the latter group is overall only 50–60 % satisfied on a long-term basis [11] compared to over 90 % satisfaction for the neuropathic patients [107], results that are probably largely influenced by the degree of unacceptability of the preoperative condition.

From a urodynamic standpoint, successful cases will have cystometrograms indistinguishable from normal, and on fluoroscopic studies, it has been demonstrated that the initial contraction occurs in the detrusor residue and subsequently in the intestinal segment [108]. Outflow resistance is also a critical factor to assess if patients have leakage postoperatively [108]. In any case, a delicate balance exists between continence and retention. Patients' expectations and motivation highly influence their long-term satisfaction and sense of well-being after surgery.

The cost-effectiveness of AC has been compared to intradetrusor injections of botulinum toxin A (BTX-A) for refractory neurogenic OAB and favors BTX-A over a 5-year period. However, AC was deemed to be cheaper if the complication rate was below 14 % or if the efficacy of BTX-A was less than 5 months [109].

Overall, excellent outcomes can be expected with a combination of reliable surgical techniques in addition to careful patient selection as well as appropriate preoperative counseling and postoperative follow-up [110].

Follow-Up

Despite the fact that this surgery has been practiced for decades, no evidence-based guidelines are available to recommend what kind of follow-up should be performed. Nevertheless, if we transpose the recommendations for urinary diversions post-cystectomy, and consider the potential complications and underlying disease of the patients, annual monitoring appears reasonable. Thus, in addition to history (focused on continence, frequency of intermittent catheterization, UTI, mucus production, etc.) and physical exam, upper urinary tract surveillance (comprising serum electrolytes, renal function tests, and imaging) starting at 6 weeks, then at longer intervals, and then annually may be done. Adequate oral fluid intake should be emphasized and those voiding spontaneously should check their post-void residuals regularly. Urinary cytology may be misleading because of the presence of a variety of cells. As previously mentioned, we advocate an annual or biennial cystoscopy to screen bowel and bladder mucosa for any suspicious change (redness, growth) or foreign body (stone, mucus). Long-term follow-up is necessary because of the persisting potential for problems over the years.

Experimental Options

Augmentation cystoplasty using either enteric or urothelial tissues is not a panacea and experimentation is ongoing in the quest for alternative tissues to use.

Since its beginning in the early 1990s, the field of tissue engineering has made tremendous progress [111]. Currently, there are two types of technologies that have been investigated. These are unseeded and seeded technologies [112]. Unseeded technology involves implantation of bioactive or resorbable synthetic matrices into the host organ and allowing the body's natural capability to use the matrix as a scaffold for cell growth or tissue regeneration [111]. In comparison, seeded technology involves cells derived from biopsy that are expanded in vitro, seeded onto a natural (porcine small intestinal submucosa) or synthetic (polyglycolic acid) scaffold, and the composite is then placed back into the host for continued regeneration. Among the most exciting results, Kurokawa et al. have reported utilizing cultured oral mucosal epithelial cell sheets grafted on demucosalized seromuscular colonic flaps [113], while Atala et al. described a model created with autologous urothelial and muscle cells obtained from bladder biopsy seeded on collagen-polyglycolic acid scaffolds, and wrapped in omentum after implantation [114]. Unfortunately, despite

significant advances in the field of bioengineering, the search for a perfect material remains experimental and ongoing, with few published trials on humans available yet [114].

Conclusion

Although intradetrusor botulinum toxin and sacral neuromodulation have emerged as competing minimally invasive therapeutic options, AC remains an essential component to the reconstructive urologist's armamentarium, with the most popular technique using a detubularized patch of ileum. It provides satisfactory clinical outcomes and acceptable morbidity, but the potential for long-term complications warrants prolonged follow-up and monitoring.

References

1. Biers SM, Venn SN, Greenwell TJ. The past, present and future of augmentation cystoplasty. BJU Int. 2012;109(9):1280–93.
2. Tizzoni G, Foggi A. Die weiderherstellung der harnblase. Zentralbl Chir. 1888;15:921–4.
3. von Mikulicz J. Zur Operation der angeborenen Blasenspalte. Centralbl Chir. 1899;26:641–3.
4. Couvelaire R. La petite vessie des tuberculeux génito-urinaires: essai de classification, places et variantes des cysto-intestinoplasties. J Urol Medicale Chir. 1950;56:381–434.
5. Charghi A, Charbonneau J, Gauthier GE. Colocystoplasty for bladder enlargement and bladder substitution: a study of late results in 31 cases. J Urol. 1967;97(5):849–56.
6. Bisgard JD. Substitution of the urinary bladder with a segment of sigmoid: an experimental study. Ann Surg. 1943;117(1):106–9.
7. Sinaiko E. Artificial bladder from segment of stomach and study of effect of urine on gastric secretion. Surg Gynecol Obstet. 1956;102(4):433–8.
8. Goodwin WE, Winter CC, Barker WF. Cup-patch technique of ileocystoplasty for bladder enlargement or partial substitution. Surg Gynecol Obstet. 1959;108(2):240–4.
9. Elbahnasy AM, Shalhav A, Hoenig DM, Figenshau R, Clayman RV. Bladder wall substitution with synthetic and non-intestinal organic materials. J Urol. 1998;159(3):628–37.
10. Lapides J, Diokno AC, Gould FR, Lowe BS. Further observations on self-catheterization. J Urol. 1976;116(2):169–71.
11. Smith A, Dmochowski R, Hilton P, et al. Surgery for urinary incontinence in women. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence—4th international consultation. 2009th ed. Paris: Health Publications; 2009. p. 1239–40.
12. Scales CD, Wiener JS. Evaluating outcomes of enterocystoplasty in patients with spina bifida: a review of the literature. J Urol. 2008;180(6):2323–9.
13. Kaefer M, Pabby A, Kelly M, Darbey M, Bauer SB. Improved bladder function after prophylactic treatment of the high risk neurogenic bladder in newborns with myelomentingocele. J Urol. 1999;162(3 Pt 2):1068–71.
14. Lendvay TS, Cowan CA, Mitchell MM, Joyner BD, Grady RW. Augmentation cystoplasty rates at children's hospitals in the United States: a pediatric health information system database study. J Urol. 2006;176(4 Pt 2):1716–20.
15. Reyblat P, Ginsberg D. Augmentation cystoplasty: what are the indications? [Review]. Curr Urol Rep. 2008;9(6):452–8.
16. Gormley EA, Lightner DJ, Burgio KL, et al. Diagnosis and treatment of overactive bladder (non-neurogenic) in adults: AUA/SUFU guideline. J Urol. 2012;188(6 Suppl):2455–63.
17. Hanno PM, Burks DA, Clemens JQ, et al. AUA guideline for the diagnosis and treatment of interstitial cystitis/bladder pain syndrome. J Urol. 2011;185(6):2162–70.
18. Dinckan A, Turkyilmaz S, Tekin A, et al. Simultaneous augmentation ileo-cystoplasty in renal transplantation. Urology. 2007;70(6):1211–4.
19. Greenwell TJ, Venn SN, Mundy AR. Augmentation cystoplasty. BJU Int. 2001;88(6):511–25.
20. Elliott SP, Meng MV, Anwar HP, Stoller ML. Complete laparoscopic ileal cystoplasty. Urology. 2002;59(6):939–43.
21. Rao PKIA, Sabanegh ES. Augmentation cystoplasty. http://emedicine.medscape.com/article/443916-overview (2013). Accessed 31 Mar 2013.
22. Sajadi KP, Goldman HB. Bladder augmentation and urinary diversion for neurogenic LUTS: current indications. Curr Urol Rep. 2012;13(5):389–93.
23. Stone AR, Nanigian D. Augmentation cystoplasty for overactive bladder. In: Kreder K, Dmochowski R, editors. The overactive bladder: evaluation and management. London: Informa Healthcare; 2007. p. 359–69.
24. Bucher P, Mermillod B, Gervaz P, Morel P. Mechanical bowel preparation for elective colorectal surgery: a meta-analysis. Arch Surg. 2004;139(12):1359–64. discussion 1365.
25. Slim K, Vicaut E, Launay-Savary MV, Contant C, Chipponi J. Updated systematic review and meta-analysis of randomized clinical trials on the role of mechanical bowel preparation before colorectal surgery. Ann Surg. 2009;249(2):203–9.
26. Belsey J, Epstein O, Heresbach D. Systematic review: adverse event reports for oral sodium phosphate and polyethylene glycol. Aliment Pharmacol Ther. 2009;29(1):15–28.

27. Franga DL, Harris JA. Polyethylene glycol-induced pancreatitis. Gastrointest Endosc. 2000;52(6):789–91.
28. Committee ADRA. Electrolyte disturbances with oral phosphate bowel preparations. Aust Adv Drug React Bull 1997;16(2) p. 5.
29. Gundeti MS, Godbole PP, Wilcox DT. Is bowel preparation required before cystoplasty in children? J Urol. 2006;176(4 Pt 1):1574–6. discussion 1576–1577.
30. Victor D, Burek C, Corbetta JP, et al. Augmentation cystoplasty in children without preoperative mechanical bowel preparation. J Pediatr Urol. 2012;8(2):201–4.
31. Canning DA. Re: Augmentation cystoplasty in children without preoperative mechanical bowel preparation. J Urol. 2012;188(6):2368–9.
32. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection. Centers for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. Am J Infect Control. 1999; 27(2):97–132.
33. Wolf JS, Bennett CJ, Dmochowski RR, et al. Best practice policy statement on urologic surgery antimicrobial prophylaxis. J Urol. 2008;179(4): 1379–90.
34. Bailly GG, Herschorn S. Urinary Diversion. In: Corcos J, Schick E, editors. Textbook of the neurogenic bladder. 2nd ed. London: Informa Healthcare; 2008. p. 670–85.
35. Abou-Elela A. Augmentation cystoplasty: in pretransplant recipients. In: Ortiz J, Andre J, editors. Understanding the complexities of kidney transplantation. Rijeka: InTech; 2011. p. 279–330.
36. Adams MC, Joseph DB. Augmentation cystoplasty. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. Campbell-Walsh urology, vol. IV. 10th ed. Philadelphia, PA: Saunders, Elsevier; 2012. p. 3471–91.
37. Hendren WH, Hendren RB. Bladder augmentation: experience with 129 children and young adults. J Urol. 1990;144(2 Pt 2):445–53. discussion 460.
38. Duel BP, Gonzalez R, Barthold JS. Alternative techniques for augmentation cystoplasty. J Urol. 1998;159(3):998–1005.
39. Herschorn S, Welk BK. Bladder augmentation. In: Goldman HB, editor. Complications of female incontinence and pelvic reconstructive surgery. Cleveland, OH: Humana; 2013. p. 171–87.
40. Fromm D. Ileal resection, or disease, and the blind loop syndrome: current concepts of pathophysiology. Surgery. 1973;73(5):639–48.
41. Gosalbez R, Woodard JR, Broecker BH, Parrott TS, Massad C. The use of stomach in pediatric urinary reconstruction. J Urol. 1993;150(2 Pt 1):438–40.
42. Sumfest JM, Mitchell ME. Gastrocystoplasty in children. Eur Urol. 1994;25(2):89–93.
43. Nguyen DH, Bain MA, Salmonson KL, Ganesan GS, Burns MW, Mitchell ME. The syndrome of dysuria and hematuria in pediatric urinary reconstruction with stomach. J Urol. 1993;150(2 Pt 2):707–9.
44. Clark SS. Electrolyte disturbance associated with jejunal conduit. J Urol. 1974;112(1):42–7.
45. Klein EA, Montie JE, Montague DK, Kay R, Straffon RA. Jejunal conduit urinary diversion. J Urol. 1986;135(2):244–6.
46. Churchill BM, Aliabadi H, Landau EH, et al. Ureteral bladder augmentation. J Urol. 1993;150(2 Pt 2):716–20.
47. Hitchcock RJ, Duffy PG, Malone PS. Ureterocystoplasty: the 'bladder' augmentation of choice. Br J Urol. 1994;73(5):575–9.
48. Johal NS, Hamid R, Aslam Z, Carr B, Cuckow PM, Duffy PG. Ureterocystoplasty: long-term functional results. J Urol. 2008;179(6):2373–5. discussion 2376.
49. MacNeily AE, Afshar K, Coleman GU, Johnson HW. Autoaugmentation by detrusor myotomy: its lack of effectiveness in the management of congenital neuropathic bladder. J Urol. 2003;170(4 Pt 2):1643–6. discussion 1646.
50. Redman JF, Barthold JS. Experience with ileal augmentation cystoplasty using a short pfannenstiel incision. J Urol. 1996;155(5):1726–7.
51. Aleman MA, Abdelmalak JB, Rackley RR. Augmentation cystoplasty. In: Goldman HB, Vasavada SP, editors. Current clinical urology: female urology: a practical clinical guide. Totowa, NJ: Humana; 2007. p. 251–9.
52. Blaivas JG, Weiss J, Desai P, Flisser AJ, Stember D, Stahl P. Long-term followup of augmentation enterocystoplasty and continent diversion in patients with benign disease. J Urol. 2005;173(5):1631–4.
53. Gil Vernet SG. Neurogenic bladder, neuromuscular bladder and intestinal bladder. Acta Urol Belg. 1962;30:405–20.
54. Adams MC, Joseph DB. Urinary tract reconstruction in children. In: Wein A, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. Campbell-Walsh urology, vol. 4. Philadelphia: Saunders, Elsevier; 2007. p. 3656–702.
55. Rackley RR, Abdelmalak JB. Laparoscopic augmentation cystoplasty. Surgical technique. Urol Clin North Am. 2001;28(3):663–70.
56. Gill IS, Rackley RR, Meraney AM, Marcello PW, Sung GT. Laparoscopic enterocystoplasty. Urology. 2000;55(2):178–81.
57. Kang IS, Lee JW, Seo IY. Robot-assisted laparoscopic augmentation ileocystoplasty: a case report. Int Neurourol J. 2010;14(1):61–4.
58. Meng MV, Anwar HP, Elliott SP, Stoller ML. Pure laparoscopic enterocystoplasty. J Urol. 2002;167(3): 1386.
59. Inman BA, Harel F, Tiguert R, Lacombe L, Fradet Y. Routine nasogastric tubes are not required following cystectomy with urinary diversion: a comparative analysis of 430 patients. J Urol. 2003;170(5):1888–91.
60. Botto H, Naber KG, Bishop MC, Jarlier V, Lim V, Norby R. Antimicrobial policy in prophylaxis and treatment of nosocomial urinary tract infection. In: Naber KG, Pechere JC, Kumazawa J, Khoury S,

Gerberding JL, Schaeffer AJ, editors. Nosocomial and health care associated infections in urology. Plymouth: Health Publications; 2001. p. 177–92.
61. Flood HD, Malhotra SJ, O'Connell HE, Ritchey MJ, Bloom DA, McGuire EJ. Long-term results and complications using augmentation cystoplasty in reconstructive urology. Neurourol Urodyn. 1995; 14(4):297–309.
62. Welk B, Herschorn S, Law C, Nam R. Population based assessment of enterocystoplasty complications in adults. J Urol. 2012;188(2):464–9.
63. Herschorn S, Hewitt R. Patient perspective of long-term outcome of augmentation cystoplasty for neurogenic bladder. Urology. 1998;52(4):672–8.
64. Yerkes EB, Rink RC, Cain MP, Luerssen TG, Casale AJ. Shunt infection and malfunction after augmentation cystoplasty. J Urol. 2001;165(6 Pt 2):2262–4.
65. Akerlund S, Campanello M, Kaijser B, Jonsson O. Bacteriuria in patients with a continent ileal reservoir for urinary diversion does not regularly require antibiotic treatment. Br J Urol. 1994;74(2):177–81.
66. Worth PH. The treatment of interstitial cystitis by cystolysis with observations on cystoplasty. A review after 7 years. Br J Urol. 1980;52(3):232.
67. Khoury J, Timmons S, Corbel L, Webster G. Complications of enterocystoplasty. Urology. 1992; 40(1):9–14.
68. Mitchell M, Kulb T, Backes D. Intestinocystoplasty in combination with clean intermittent catheterization in the management of vesical dysfunction. J Urol. 1986;136(1 Pt 2):288–91.
69. Stöhrer M, Blok B, Castro-Diaz D, et al. EAU guidelines on neurogenic lower urinary tract dysfunction. Uroweb. 2011. http://www.uroweb.org/guidelines/online-guidelines/. Accessed 11 Jan 2014.
70. Palmer LS, Franco I, Kogan SJ, Reda E, Gill B, Levitt SB. Urolithiasis in children following augmentation cystoplasty. J Urol. 1993;150(2 Pt 2):726–9.
71. Sakakibara R, Hattori T, Uchiyama T, Kamura K, Yamanishi T. Uroneurological assessment of spina bifida cystica and occulta. Neurourol Urodyn. 2003;22(4):328–34.
72. Benway BM, Bhayani SM. Lower urinary tract calculi. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. Campbell-Walsh urology, vol. III. 10th ed. Philadelphia: Saunders, Elsevier; 2012. p. 2521–30.
73. Khoury AE, Salomon M, Doche R, et al. Stone formation after augmentation cystoplasty: the role of intestinal mucus. J Urol. 1997;158(3 Pt 2):1133–7.
74. DeFoor W, Minevich E, Reddy P, et al. Bladder calculi after augmentation cystoplasty: risk factors and prevention strategies. J Urol. 2004;172(5 Pt 1): 1964–6.
75. Nurse D, McInerney P, Thomas P, Mundy A. Stones in enterocystoplasties. Br J Urol. 1996;77(5):684–7.
76. DeFoor W, Tackett L, Minevich E, Wacksman J, Sheldon C. Risk factors for spontaneous bladder perforation after augmentation cystoplasty. Urology. 2003;62(4):737–41.
77. Novak T, Salmasi A, Mathews R, Lakshmanan Y, Gearhart J. Complications of complex lower urinary tract reconstruction in patients with neurogenic versus nonneurogenic bladder—is there a difference? J Urol. 2008;180(6):2629–34.
78. Fox J, Husmann D. Continent urinary diversion in childhood: complications of alcohol abuse developing in adulthood. J Urol. 2010;183(6):2342–6.
79. Metcalfe PD, Casale AJ, Kaefer MA, et al. Spontaneous bladder perforations: a report of 500 augmentations in children and analysis of risk. J Urol. 2006;175(4):1466–70. discussion 1470–1461.
80. Braverman RM, Lebowitz RL. Perforation of the augmented urinary bladder in nine children and adolescents: importance of cystography. AJR Am J Roentgenol. 1991;157(5):1059–63.
81. Murray K, Nurse DE, Mundy AR. Secreto-motor function of intestinal segments used in lower urinary tract reconstruction. Br J Urol. 1987;60(6):532–5.
82. Gillon G, Mundy AR. The dissolution of urinary mucus after cystoplasty. Br J Urol. 1989;63(4): 372–4.
83. Farnham SB, Cookson MS. Surgical complications of urinary diversion. World J Urol. 2004;22(3): 157–67.
84. George V, Gee J, Wortley M, Stott M, Gaches C, Ashken M. The effect of ranitidine on urine mucus concentration in patients with enterocystoplasty. Br J Urol. 1992;70(1):30–2.
85. Fontaine E, Leaver R, Woodhouse CR. The effect of intestinal urinary reservoirs on renal function: a 10-year follow-up. BJU Int. 2000;86(3):195–8.
86. Soergel TM, Cain MP, Misseri R, Gardner TA, Koch MO, Rink RC. Transitional cell carcinoma of the bladder following augmentation cystoplasty for the neuropathic bladder. J Urol. 2004;172(4 Pt 2): 1649–51. discussion 1651–1642.
87. Higuchi T, Granberg C, Fox J, Husmann D. Augmentation cystoplasty and risk of neoplasia: fact, fiction and controversy. J Urol. 2010;184(6): 2492–6.
88. Husmann DA, Rathbun SR. Long-term follow up of enteric bladder augmentations: the risk for malignancy. J Pediatr Urol. 2008;4(5):381–5. discussion 386.
89. Filmer RB, Spencer JR. Malignancies in bladder augmentations and intestinal conduits. J Urol. 1990;143(4):671–8.
90. Balachandra B, Swanson PE, Upton MP, Yeh MM. Adenocarcinoma arising in a gastrocystoplasty. J Clin Pathol. 2007;60(1):85–7.
91. Golomb J, Klutke CG, Lewin KJ, Goodwin WE, deKernion JB, Raz S. Bladder neoplasms associated with augmentation cystoplasty: report of 2 cases and literature review. J Urol. 1989;142(2 Pt 1): 377–80.
92. Hamid R, Greenwell TJ, Nethercliffe JM, Freeman A, Venn SN, Woodhouse CR. Routine surveillance cystoscopy for patients with augmentation and substitution cystoplasty for benign urological conditions: is it necessary? BJU Int. 2009;104(3):392–5.

93. Koch MO, McDougal WS, Reddy PK, Lange PH. Metabolic alterations following continent urinary diversion through colonic segments. J Urol. 1991;145(2):270–3.
94. de Petriconi R. Metabolic aspects of bowel use in urologic surgery. Ann Urol (Paris). 2007;41(5):216–36.
95. Gilbert SM, Hensle TW. Metabolic consequences and long-term complications of enterocystoplasty in children: a review. J Urol. 2005;173(4):1080–6.
96. Burkhard FC, Kessler TM, Mills R, Studer UE. Continent urinary diversion. Crit Rev Oncol Hematol. 2006;57(3):255–64.
97. Gosalbez R, Woodard JR, Broecker BH, Warshaw B. Metabolic complications of the use of stomach for urinary reconstruction. J Urol. 1993;150(2 Pt 2):710–2.
98. Tanrikut C, McDougal WS. Acid-base and electrolyte disorders after urinary diversion. World J Urol. 2004;22(3):168–71.
99. Hochstetler JA, Flanigan MJ, Kreder KJ. Impaired bone growth after ileal augmentation cystoplasty. J Urol. 1997;157(5):1873–9.
100. Singh G, Thomas DG. Bowel problems after enterocystoplasty. Br J Urol. 1997;79(3):328–32.
101. Niknejad KG, Atala A. Bladder augmentation techniques in women. Int Urogynecol J Pelvic Floor Dysfunct. 2000;11(3):156–69.
102. Fenn N, Barrington JW, Stephenson TP. Clam enterocystoplasty and pregnancy. Br J Urol. 1995; 75(1):85–6.
103. Hautmann RE, Volkmer BG. Pregnancy and urinary diversion. Urol Clin North Am. 2007;34(1):71–88.
104. Greenwell TJ, Venn SN, Creighton S, Leaver RB, Woodhouse CR. Pregnancy after lower urinary tract reconstruction for congenital abnormalities. BJU Int. 2003;92(7):773–7.
105. Hill DE, Kramer SA. Management of pregnancy after augmentation cystoplasty. J Urol. 1990;144(2 Pt 2):457–9. discussion 460.
106. Kreder K, Das AK, Webster GD. The hemi-Kock ileocystoplasty: a versatile procedure in reconstructive urology. J Urol. 1992;147(5):1248–51.
107. Hasan ST, Marshall C, Robson WA, Neal DE. Clinical outcome and quality of life following enterocystoplasty for idiopathic detrusor instability and neurogenic bladder dysfunction. Br J Urol. 1995;76(5):551–7.
108. Futter NG, Collins WE. Intestinal cystoplasty. Long-term functional result. Urology. 1974;3(4):434–6.
109. Padmanabhan P, Scarpero HM, Milam DF, Dmochowski RR, Penson DF. Five-year cost analysis of intra-detrusor injection of botulinum toxin type A and augmentation cystoplasty for refractory neurogenic detrusor overactivity. World J Urol. 2011;29(1):51–7.
110. Khavari R, Fletcher SG, Liu J, Boone TB. A modification to augmentation cystoplasty with catheterizable stoma for neurogenic patients: technique and long-term results. Urology. 2012;80(2):460–4.
111. Stanasel I, Mirzazadeh M, Smith JJ. Bladder tissue engineering. Urol Clin North Am. 2010;37(4):593–9.
112. Colvert JR, Kropp BP, Cheng EY. Bladder augmentation: current and future techniques. AUA Update Ser. 2003;22(32):250.
113. Kurokawa S, Morita T, Shiroyanagi Y, Yamato M, Okano T, Kobayashi E. Novel augmentation cystoplasty utilizing cultured oral mucosal epithelial cell sheets grafted on demucosalized seromuscular colonic flaps. Eur Urol Suppl. 2007;6(2):96. abstract 294.
114. Atala A, Bauer SB, Soker S, Yoo JJ, Retik AB. Tissue-engineered autologous bladders for patients needing cystoplasty. Lancet. 2006;367(9518):1241–6.

Urethroplasty for Female Urethral Stricture

13

Chad Baxter

Introduction

Female urethral stricture disease has been historically treated with urethral dilation, which has demonstrated high recurrence rates. There is growing evidence that formal urethroplasty should be pursued early in the care of female urethral stricture disease as surgery can provide durable results [1]. This chapter presents the various techniques for female urethroplasty that are presented in the recent literature.

Background

Urethral stricture disease, while sometimes idiopathic, is commonly iatrogenic. Urethral dilations, catheterization, urinary tract endoscopy, urethral surgery, and radiation are all implicated. Urethral strictures may occur as a consequence of urethral infection, urethral diverticulum, atrophy with subsequent fibrosis, primary urethral carcinomas, leiomyomas, teratomas, and trauma [2–8].

Female urethral stricture disease is rarely diagnosed and rarely reported in the contemporary urological literature. All causes of bladder outlet obstruction have an estimated incidence of 3–8 % of women presenting with obstructive symptoms [9].

Of these women with obstruction, even fewer have proven urethral stricture. Estimates range between 4 and 13 %. Thus the true incidence of urethral stricture appears to lie between 0.1 and 1 % [10–12].

Despite the low incidence of urethral stricture disease, urethral dilation and urethrotomy have historically been employed to manage a wide variety of lower urinary tract symptoms in women [11, 13–15]. McLean and Emmett reported that the first urethral dilation took place in 1923, and, by the 1960s, series with as many as 800 patients were being reported [15]. Dilation of the female urethra has been advocated to treat recurrent urinary tract infections, bladder pain syndromes, urinary urgency, urinary frequency, overactive bladder, and interstitial cystitis symptoms [13, 14]. Contemporary literature has demonstrated that urethral dilation has no benefit and may be harmful for patients in the absence of demonstrable urethral stricture disease [16].

Santucci and the Urologic Diseases in America Project reported in 2008 that urethral dilation for various lower urinary tract symptoms appears ineffective, common, costly, and mostly unnecessary [16]. They note that while there were less than 40 reports of true female urethral strictures in the contemporary literature at that time, more than 1.2 million office visits for female urethral stricture occurred in the United States between 1992 and 2000 at a cost of $61 million per year.

C. Baxter, M.D. (✉)
Department of Urology, David Geffen School of Medicine at UCLA, Santa Monica, CA, USA
e-mail: cbaxter@mednet.ucla.edu

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_13,

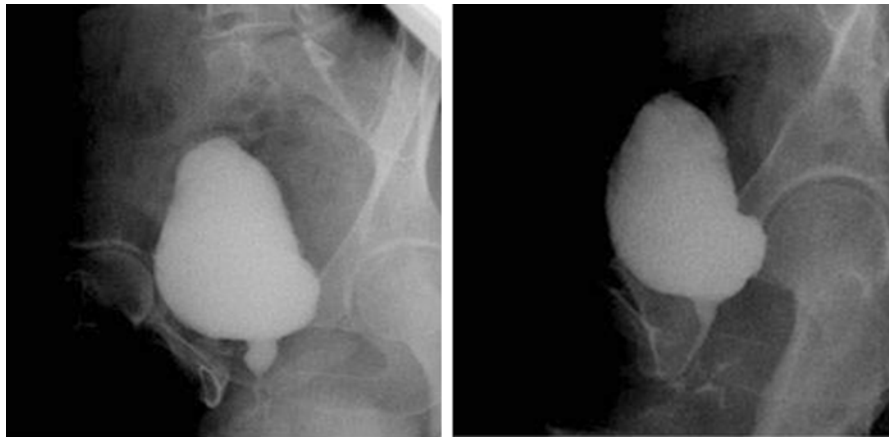


Fig. 13.1 Voiding cystourethrogram showing proximal urethral dilatation with distal urethra narrowing and distortion of bladder morphology from spherical to oblong (Both: Used with permission from Groutz A, Blaivas JG, Chaikin DC. Bladder outlet obstruction in women: Definition and Characteristics. Neurourol Urodyn 2000, 19:213–220)

Since the first case report in 1828 [17], no more than 200 cases have been reported in the English language literature. The majority of reports describe small, single-surgeon series and lack objective preoperative or postoperative measures of success.

Given the low number of reported urethroplasties, there exists no consensus for surgical treatment and several different surgical techniques have been reported and will be reviewed here.

Evaluation

As Blaivas et al. note, the diagnosis of urethral stricture in women requires a high index of suspicion. Similar to other forms of bladder outlet obstruction such as prolapse, bladder neck dysfunction, and detrusor external sphincter dyssynergia, symptoms of urethral stricture may include urinary frequency, dysuria, weak or dribbling stream, and recurrent urinary tract infections. Cystoscopy provides the most reliable assessment, though some experts advocate urethral calibration as well. Urethral calibration may offer information of scar density through haptic feedback [1]. Periurethral fibrosis may also be measured by translabial or transvaginal ultrasound, though the efficacy of ultrasound for this purpose in women is not well defined. Detrusor pressure-flow studies allow determination of bladder outlet obstruction and may be graded according to the Blaivas–Groutz nomogram [11] and voiding cystourethrogram or cystoscopy allows one to determine the location of obstruction (Fig. 13.1).

Preoperative Considerations

Female urethroplasty can be categorized by the surgical approach, nature of any needed tissue graft, and whether to perform concomitant bladder outlet procedure. The urethra is approached either dorsally or ventrally. A dorsal approach is perhaps less familiar to many urologists and care must be taken to avoid injury to the crura and body of the clitoris. A dorsal approach may also facilitate later anti-incontinence procedures, prevent sacculation of the reconstructed urethra, and minimize risk of urethrovaginal fistula or hypospadias [18]. A ventral approach, conversely, is familiar to urologists experienced in many of the transvaginal anti-incontinence procedures and allows for easier visualization of the urethra to the level of the bladder neck.

Graft tissue may be either a local rotational flap, pedicle flap, or free flap harvested from vaginal, labial, or oral mucosa. Free or pedicle flaps may minimize distortion of local tissue compared to rotational flaps, but are also associated with harvest site morbidity including paresthesia, anesthesia, and altered cosmesis.

The decision to perform a concomitant anti-incontinence procedure should be based on preoperative evidence of stress incontinence or, in the opinion of some experts, in the presence of contrast entering the urethra to the level of the stricture during fluoroscopic imaging while performing a Valsalva maneuver and cough [1]. Many experts advocate dorsal, as opposed to ventral, urethroplasty techniques to facilitate concomitant or delayed placement of ventral urethral sling [18].

Given the infrequent occurrence of urethral strictures and more infrequent reports of their repair in the literature, there is a paucity of data precluding a recommendation of one approach or graft over another.

Postoperative Care

Duration of catheterization, use of imaging, and antibiotic therapy differs among reported series of urethral reconstructions. Many surgeons advocate leaving a catheter indwelling for 2–4 weeks, obtaining a voiding cystourethrogram at time of catheter removal, and maintaining antibiotics, usually low dose Ciprofloxacin or trimethoprim-sulfamethoxazole, for the duration of catheterization.

Surgical Techniques

Ventral Incision and Anastomosis

Short strictures located primarily on the ventral aspect of the urethral lumen may be incised longitudinally with transverse closure consistent with the Heineke–Mikulicz principle. Long or circumferential dense scar may be recalcitrant to this technique:

1. In dorsal lithotomy position, prepare the vagina with betadine, drape, and facilitate exposure with a Lone Star or similar retractor.
2. Place a foley catheter or, under cystoscopic guidance, intubate urethra and stricture with a guide wire.
3. Develop an anterior vaginal wall flap exposing the underlying periurethral fascia.
4. Transversely incise the periurethral fascia to expose the underlying urethra.
5. Incise the urethra longitudinally through the stricture.
6. Calibrate urethra preferably to 28 Fr, place 20 Fr catheter.
7. Repair urethra with transverse closure using absorbable suture, such as 4-0 poliglecaprone, in an interrupted fashion.
8. Close periurethral fascia with interrupted absorbable suture, such as 3-0 polyglactin.
9. Advance the anterior vaginal wall flap beyond the underlying suture line of the periurethral fascia.
10. Close vaginal wall with running, locking 3-0 polyglactin.

Excision and end-to-end anastomosis is not often used due to the risk of urethral ischemia distal to the excision. Rovner reported that this approach was useful in treating complex urethral diverticulae [19].

Vaginal Inlay Flap

Vaginal inlay flaps have been separately reported by several investigators [20, 21]:

1. After intubating the urethra with a catheter, wire, or guide, an anterior vaginal wall flap is created with an inverted-U incision, the apex of which is at the urethral meatus (Fig. 13.2a–d).
2. The ventral aspect of the urethra is incised in the midline from the meatus, through the stricture, to a point of normal caliber urethra proximal to the stricture.
3. The dorsal aspect of the urethra may need to be incised in the midline to facilitate approximation of the flap.
4. The apex of the vaginal wall flap is then approximated with absorbable suture, such as

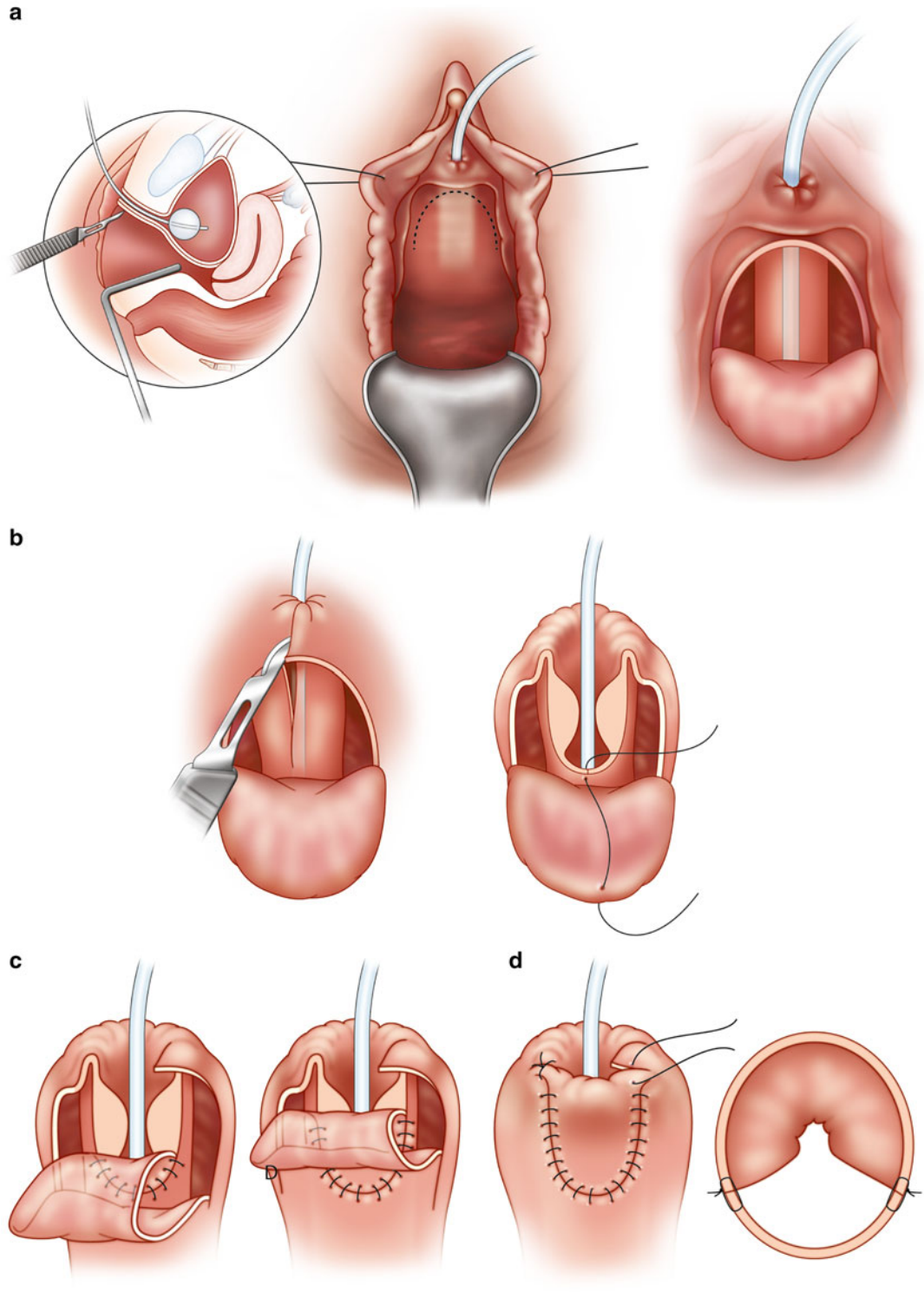


Fig. 13.2 (**a**–**d**) Vaginal inlay flap. (**a**) Anterior vaginal wall is exposed with labial retraction and weighted speculum. Foley catheter is placed per urethra to guide dissection. An anterior vaginal wall, inverted-U vaginal wall flap, is dissected superficial to the periurethral fascia. (**b**) Periurethral fascia is opened sharply and the urethra incised longitudinally from distal to proximal traversing the stricture. (**c**) The anterior vaginal wall flap is folded upon itself and the apex of the flap affixed to the proximal extent of the urethrotomy. Repair is continued with running, locking absorbable suture (e.g., 4-0 poliglecaprone). (**d**) The final repair thus consists of vaginal wall as the inferior portion of the urethra

4-0 poliglecaprone, to the proximal extent of the urethral incision.
5. A large caliber urethral catheter is then placed.
6. The vaginal wall flap is approximated with running, locking absorbable suture to the urethra along either side of the flap until the urethra has been reconstructed distally to the urethral meatus.

Free Labia Minora Skin Flap

The free labia minora skin flap is an alternative to the vaginal inlay flap and is well described by Rehder et al. [22]:

1. The vagina is prepped and draped in the usual surgical fashion.
2. An anterior vaginal wall flap or midline anterior vaginal wall incision beneath the urethra may be performed to expose the urethra (Fig. 13.3a–e).
3. The urethra, intubated with urethral sound, catheter, or wire, is incised in the midline to level proximal to the stricture.
4. Donor tissue site is selected from medial aspect of the labia minora.
5. An elliptical graft is sharply harvested, its size corresponding to the length and width of the urethral defect.
6. The graft donor site is repaired with interrupted absorbable suture.
7. The graft is anastamosed to the urethra with running, locking absorbable 4-0 polyglyconate or poliglecaprone over a catheter.
8. Vaginal wall is then closed with running, locking absorbable suture.

Consideration should be given to placing a Martius flap harvested through either a separate vulvar incision or from the lateral aspect of the anterior vaginal wall flap incision.

Pedicle Flap from the Labia Minora

This procedure is similar to the free labial graft, except that the donor tissue is isolated on a pedicle and tunneled under the vulvovaginal wall to the site of urethral dissection (Fig. 13.4a–d). The remainder of the urethroplasty is similarly performed [23].

Ventral Buccal Graft

Buccal grafts, widely used in male anterior urethral reconstruction, are well described for female urethral reconstruction by Bergland et al. [24]:

1. Prior to the buccal graft harvest, the urethra is exposed via an anterior vaginal wall flap. The intubated urethra is incised from the meatus through the stricture to healthy tissue proximally.
2. A suprapubic catheter can be placed at this time so that postoperative manipulation of the urethra may be minimized.
3. The buccal graft is harvested as described by Morey and McAninch [25] with a width of 2–2.5 cm in width, and tailored to fill the length of the urethral defect.
4. The graft is then approximated to the urethrotomy defect with running, locking absorbable suture. Berglund et al., reported using 5-0 polyglyconate.
5. A 28 Fr sound is used to calibrate the urethra. An 18 Fr foley catheter is then placed, as well as a suprapubic catheter.
6. The graft is covered with periurethral soft tissue or Martius flap if periurethral soft tissue is inadequate.
7. The vaginal wall is closed with running, locking absorbable suture.

Dorsal Buccal Graft [18]

1. Suprameatal incision is made from 9 O'clock to 3 O'clock position between urethra and clitoral cavernous tissue (Fig. 13.5a–d).
2. The urethra is dissected to the level of the bladder neck, with the anterior component of the striated urethral sphincter mobilized from the urethral wall.
3. The bladder neck is marked with an absorbable suture, such as 4-0 polyglyconate.
4. An incision is made in the midline over the intubated urethra, full thickness through spongiosal and mucosal tissues, from bladder neck to meatus.
5. The buccal graft is harvested, 6 cm × 2.5 cm.

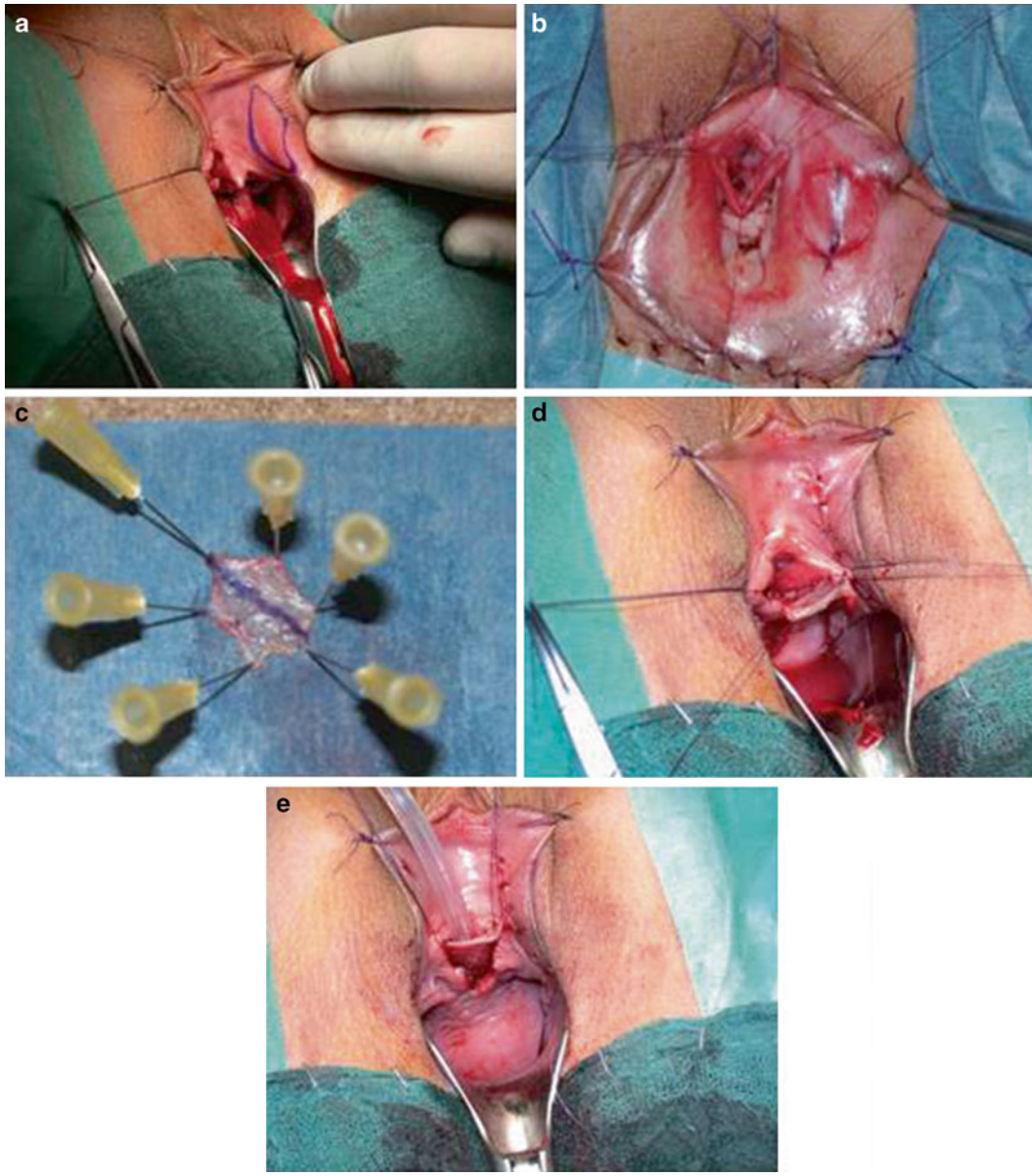


Fig. 13.3 (**a**–**e**) (**a**) After exposure of urethra and incision through the region of stenosis, an elliptical graft is marked. (**b**) The graft is excised and the donor site repaired. (**c**) The graft. (**d**) The graft is sewn to the urethra with interrupted absorbable suture. (**e**) The anterior vaginal wall defect after graft placement (**a**–**e**: Used with permission from Rehder P, Glodny B, Pichler R et al. Dorsal urethroplasty with labia minora skin graft for female urethral strictures. BJU Int 2010 Oct;106(8):1211-4)

6. The graft is sewn to the mucosa of the urethrotomy with absorbable 4-0 polyglyconate suture.
7. The urethra is quilted to the clitoral body to cover the grafted urethra.
8. A Martius flap may be deployed to facilitate blood supply and prevent fixation of urethra to the posterior aspect of the pubis.
9. The vulvar incision is then closed with running, locking absorbable suture.

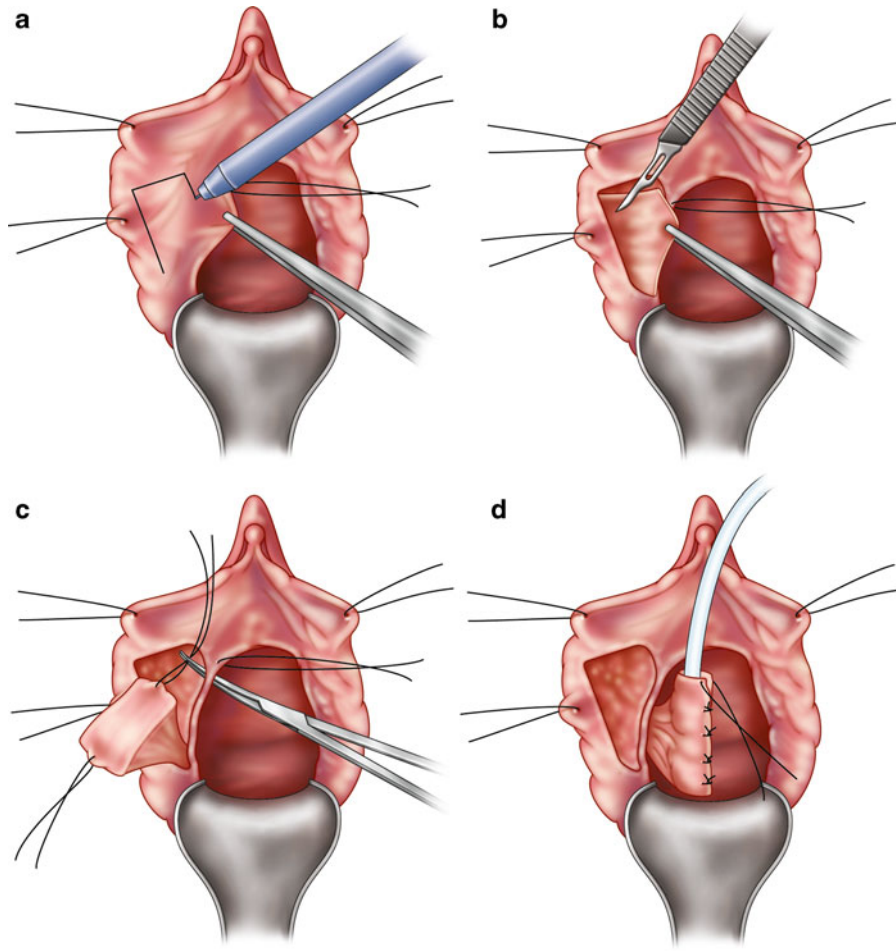


Fig. 13.4 (**a**–**d**) (**a**) Graft site is marked. (**b**) The graft is harvested taking care to preserve the pedical. (**c**, **d**) The graft is tunneled and affixed to the urethra

Dorsal Vestibular Flap Urethroplasty [26]

1. Midline incision anterior to the urethra between the meatus and clitoris is performed sharply (Fig. 13.6a–c).
2. The dorsum of the urethra is exposed and anterior component of sphincter reflected from urethra.
3. The urethra is incised in the midline.
4. If a Martius flap is needed, it is now harvested and rotated into position between urethra and pubis.
5. The vestibular flap is dissected free and rotated into the urethrotomy.
6. Urethral reconstruction performed with running, locking 4-0 polyglyconate suture, calibrated to 28 Fr.
7. Vestibular incisions closed with absorbable suture.

Free Vaginal Wall In-Lay Graft [27]

1. A transverse segment of anterior vaginal wall measuring as large as 6 cm × 2.5 cm is harvested (Fig. 13.7a–c). The harvest site is then closed with running, locking absorbable 4-0 polyglyconate suture.

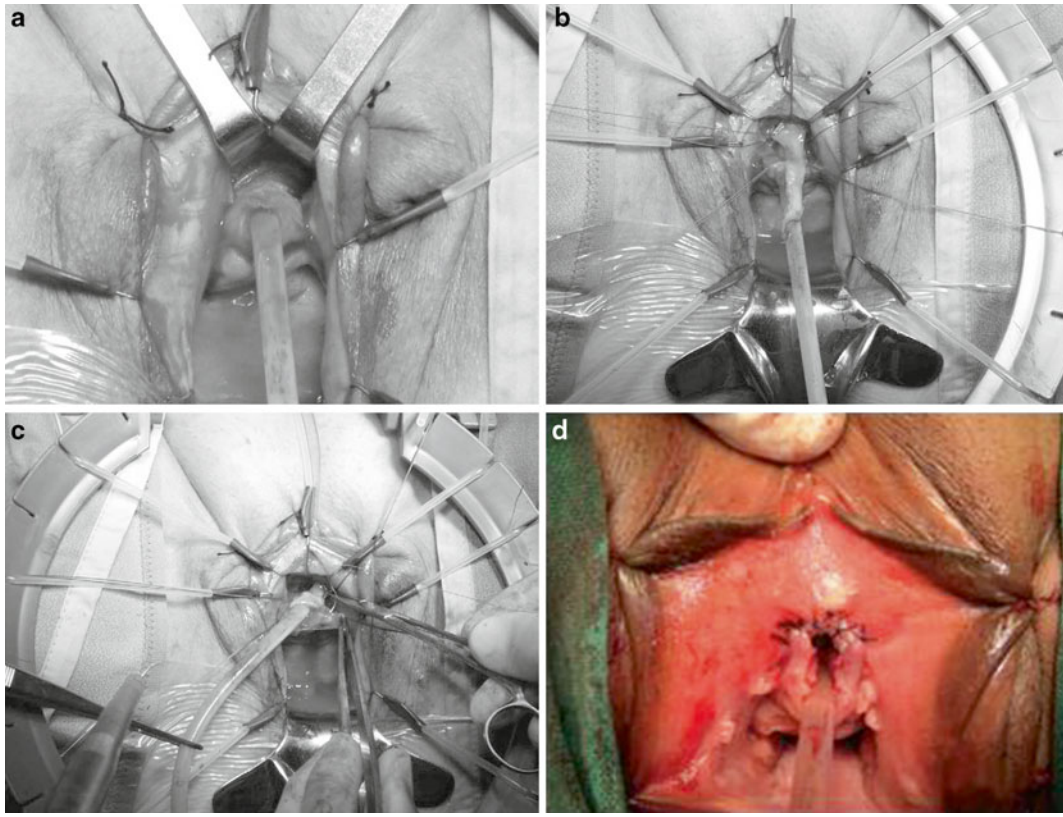


Fig. 13.5 (**a**–**d**) (**a**) with a urethral catheter in place, a suprameatal incision is performed. (**b**) The urethra is mobilized to the bladder neck and incised through the stricture. (**c**) The graft is then placed. (**d**) Finally, the meatus is reformed with interrupted suture (**a**–**c**: Used with permission from Migliari R, Leone P, Berdondini E et al. Dorsal buccal mucosa graft urethroplasty for female urethral strictures. J Urol 2006; 176:1473-6; **d**: Used with permission from Sharma GK, Pandey A, Bansal H et al. Dorsal onlay lingual mucosal graft urethroplasty for urethral strictures in women. BJU Int 2009; Oct 29: 1309-1312)

2. Urethral dissection is performed, either dorsal or ventral, and urethrotomy is performed.
3. The free graft is then anastamosed within the urethrotomy to a calibrated lumen of 28–30 Fr.
4. Vaginal wall is then closed.

Summary

Female urethral stricture disease is an infrequently reported entity despite more than 100,000 physician office visits each year in the United States for symptoms mimicking the condition. The vast majority of female patients with obstructive voiding symptoms have a dynamic dysfunction as opposed to a fixed urethral stricture. Presumed fixed urethral obstruction has been historically managed by urethral dilation and rarely endoscopic urethrotomy. In patients with proven urethral stricture disease, minimally invasive treatments including dilation and endoscopic incision generally fail with early recurrence and the need for additional procedures or intermittent catheterization. Urethroplasty appears to be a viable option for definitive therapy. Given the low number of reported urethral strictures, small series in even the most specialized urology practices, and variety of surgical techniques, the optimal treatment is not yet known. The dorsal

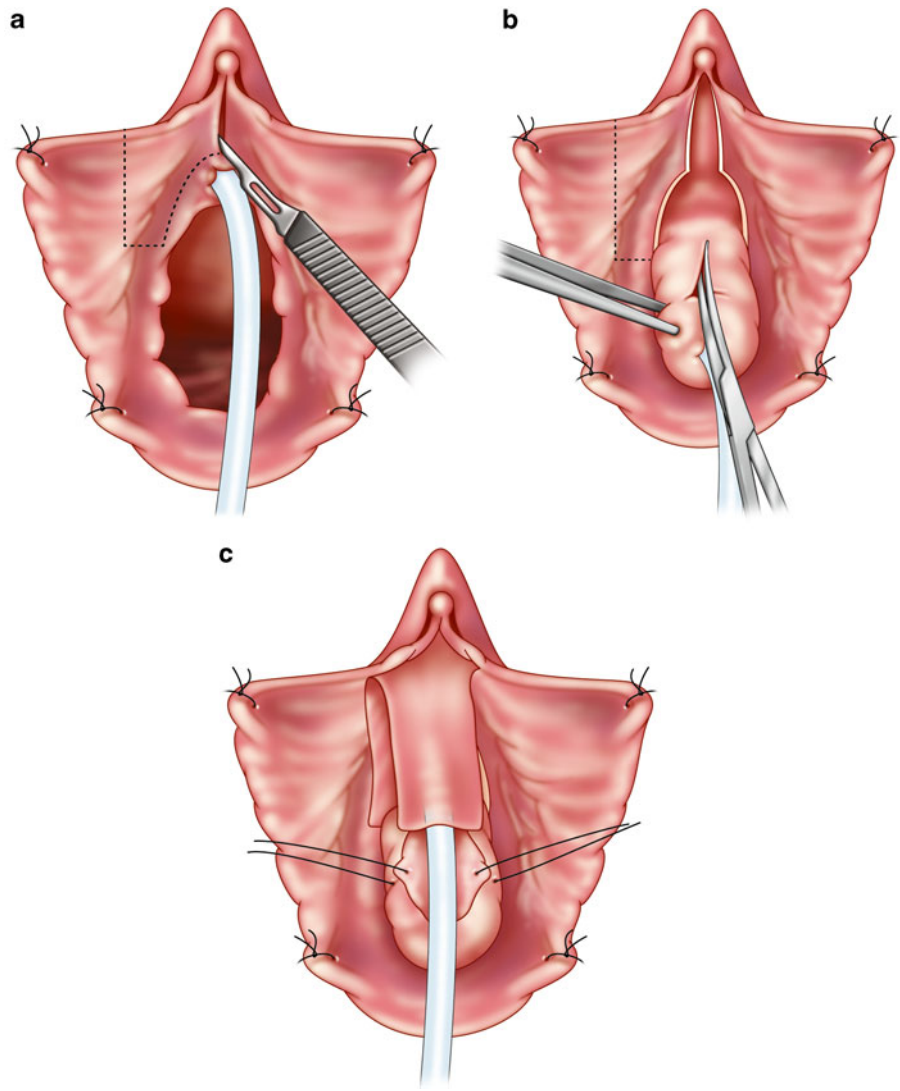


Fig. 13.6 (**a**–**c**) (**a**) With labia retracted, a suprameatal incision is performed. (**b**) The dorsal urethra is incised through the stricture. (**c**) The vestibular flap is harvested

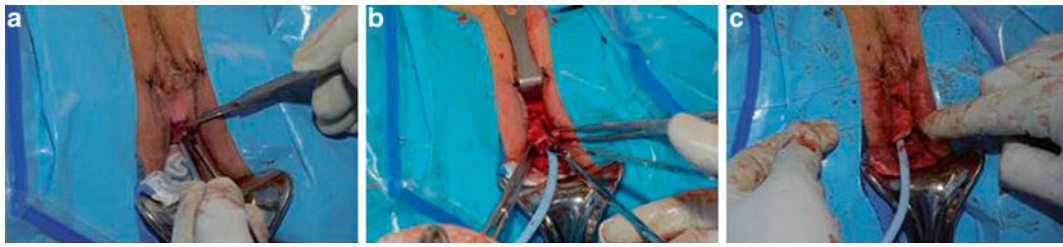


Fig. 13.7 (**a**–**c**) (**a**) A strip of vaginal wall is harvested. (**b**) Dorsal incision of the urethra through the stricture. (**c**) Anastomosis of the graft to the urethra (**a**–**c**: Used with permission from Petrou SP, Rogers AE, Parker AS et al. Dorsal vaginal graft urethroplasty for female urethral stricture disease. BJU Int 2012; May 17: E1090-E1095)

approach to the proximal female urethra may be associated with a greater postoperative incidence of stress urinary incontinence when compared to ventral approaches, but this remains to be proven. Anti-incontinence procedures such as urethral sling placement or suspensions are not routinely performed concomitant to urethroplasty, but again evidence for this recommendation is lacking. Local tissue flaps, when available, appear to be very effective. When local tissue is inadequate, distal grafts, e.g., buccal mucosa, appear efficacious.

References

1. Blaivas JG, Santos JA, Tsui JF, et al. Management of urethral stricture in women. J Urol. 2012;188:1778–82.
2. Indudhara R, Vaidyanathan S, Radotra BD. Urethral tuberculosis. Urol Int. 1992;48:436–8.
3. Romero Perez P, Schiefenbusch Munne E, Lobato Encinas JJ, Mira Llinares A. [Female urethral stenosis caused by vulvar dystrophy]. Arch Esp Urol. 1990;43:341–6.
4. Pugliese JM, Morey AF, Peterson AC. Lichen sclerosus: review of the literature and current recommendations for management. J Urol. 2007;178:2268–76.
5. Desai S, Libertino JA, Zinman L. Primary carcinoma of the female urethra. J Urol. 1973;110:693–5.
6. Yamashita T, Masuda H, Yano M, et al. Female urethral fibroepithelial polyp with stricture. J Urol. 2004;171:357.
7. Ozel B, Ballard C. Urethral and paraurethral leiomyomas in the female patient. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17:93–5.
8. Nieuwenhuijs JL, De Jong TP. Two cases of unusual urethral complications after resection of sacrococcygeal teratoma. J Pediatr Surg. 2003;38:E14–5.
9. Carr LK, Webster GD. Bladder outlet obstruction in women. Urol Clin North Am. 1996;23:385–91.
10. Nitti VW, Tu LM, Gitlin J. Diagnosing bladder outlet obstruction in women. J Urol. 1999;161:1535–40.
11. Groutz A, Blaivas JG, Chaikin DC. Bladder outlet obstruction in women: definition and characteristics. Neurourol Urodyn. 2000;19:213–20.
12. Kuo HC. Videourodynamic characteristics and lower urinary tract symptoms of female bladder outlet obstruction. Urology. 2005;66:1005–9.
13. Carson CC, Segura JW, Osborne DM. Evaluation and treatment of the female urethral syndrome. J Urol. 1980;124:609.
14. Rutherford AJ, Hinshaw K, Essenhigh DM, Neal DE. Urethral dilatation compared with cystoscopy alone in the treatment of women with recurrent frequency and dysuria. Br J Urol. 1988;61:500.
15. McLean P, Emmett JL. Internal urethrotomy in women for recurrent infection and chronic urethritis. J Urol. 1969;101:724.
16. Santucci RA, Payne CK, Saigal CS. Urologic Disease in America Project: Office dilation of the female urethra: a quality of care problem in the field of urology. J Urol. 2008;180:2068–75.
17. Earle H. Contraction of the female urethra. Lond M Gaz. 1828–9;3:470.
18. Migliari R, Leone P, Berdondini E, et al. Dorsal buccal mucosa graft urethroplasty for female urethral strictures. J Urol. 2006;176:1473–6.
19. Rovner ES, Wein AJ. Diagnosis and reconstruction of the dorsal or circumferential urethral diverticulum. J Urol. 2003;170:82–6.
20. Schwender CE, Ng L, McGuire E, Gormley EA. Technique and results of urethroplasty for female stricture disease. J Urol. 2006;175:976–80.
21. Gormley EA. Vaginal flap urethroplasty for female urethral stricture disease. Neurourol Urodyn. 2010;29 Suppl 1:S42–5.
22. Rehder P, Glodny B, Pichler R, et al. Dorsal urethroplasty with labia minora skin graft for female urethral strictures. BJU Int. 2010;106(8):1211–4.
23. Tanello M, Frego E, Simeone C, Cosciani CS. Use of pedicle flap from the labia minora for the repair of female urethral strictures. Urol Int. 2002;69:95–8.
24. Berglund RK, Vasavada S, Angermeier K, Rackley R. Buccal mucosa graft urethroplasty for recurrent stricture of female urethra. Urology. 2006;67:1069–71.
25. Morey AF, McAninch JW. Technique of harvesting buccal mucosa for urethral reconstruction. J Urol. 1996;155:1696–7.
26. Montorsi F, Salonia A, Centemero A, et al. Vestibular flap urethroplasty for strictures of the female urethra: impact on symptoms and flow patterns. Urol Int. 2002;69:12–6.
27. Tsivian A, Sidi AA. Dorsal graft urethroplasty for female urethral stricture. J Urol. 2006;176:611–3.

Excision of Vaginal Cysts

14

Nirmala Pillalamarri and Harvey Winkler

Introduction

Benign lesions of the vagina or vulva include cystic tumors, solid tumors, and dermatoses. Of these benign lesions, cysts are the most common. A cyst is an enclosed pocket or pouch of tissue that can be filled with materials such as air, fluid, or pus. Several types of vaginal and vulvar cysts exist and can range in size from millimeters to many centimeters. The evaluation, management, and treatment of vaginal and vulvar cysts has remained rather constant over the years and will be explored in this chapter.

Embryology and Anatomy

Embryology

A rudimentary understanding of embryology of the genital tract is useful when evaluating vaginal and vulvar anomalies such as cysts [1]. The urogenital system is comprised of the urinary tract (kidneys, ureters, bladder, and urethra) while the genital tract contains the gonads, ductal system, and external genitalia. The formation of the urogenital system in an embryo begins when a fold of intermediate mesoderm forms along each side of the abdominal aorta in early development. These folds are termed the urogenital ridges, each of which then separates into a nephrogenic ridge and a genital ridge.

The Urinary Tract

Creation of the urinary tract begins when the nephrogenic ridges give rise to the mesonephric, or Wolffian, ducts. These ducts connect the mesonephric kidneys to the cloaca, a distal pouch where the urinary, genital, and alimentary tracts culminate. By the 5th week of fetal life, each mesonephric duct gives rise to a ureteric bud. Subsequently the ureteric bud becomes a metanephric duct, also called the ureter. The ureter causes formation of the metanephros, which becomes the adult kidney. Concomitantly, the mesonephric kidney degenerates by the 10th week of life.

The Genital Tract

At 5 weeks of gestation, the gonads are undifferentiated and have the capability of becoming either ovaries or testes. After differentiation into ovaries, the formation of the genital tract begins. The paramesonephric ducts, also known as the mullerian ducts, develop bilaterally from an invagination of the coelomic epithelium between the gonad and mesonephric duct at 6 weeks of

N. Pillalamarri, M.D.
H. Winkler, M.D., F.A.C.O.G. (✉)
Division of Female Pelvic Medicine and Reconstructive Surgery,
North Shore—Long Island Jewish Health System, Great Neck, NY, USA
e-mail: npillalama@nshs.edu; hwinkler@nshs.edu

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_14,

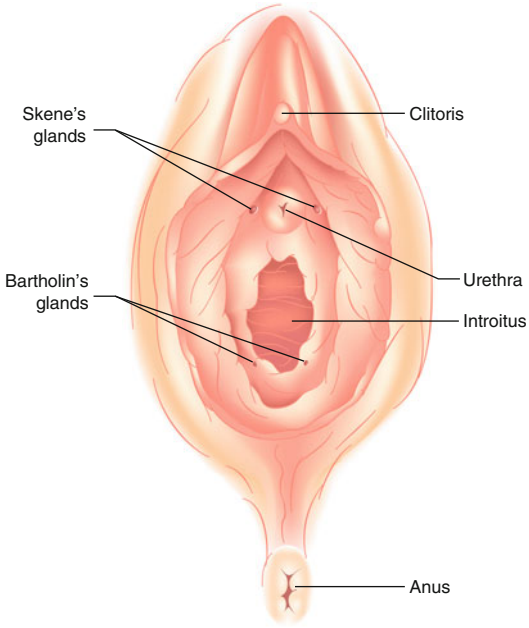


Fig. 14.1 Bartholin gland and Skene/paraurethral gland duct openings within the vagina

gestation. Both the mullerian and mesonephric ducts end at the posterior aspect of the cloaca. The cloaca divides to form the urogenital sinus anteriorly, and the rectum posteriorly. The urogenital sinus is composed of three parts, including the vesicle portion that later becomes the bladder, the middle portion that becomes the urethra, and the caudal portion that becomes the distal vagina including the Bartholin (greater vestibular) glands, urethral glands, and Skene (paraurethral) glands (Fig. 14.1). The most inferior aspects of the mesonephric and metanephric ducts enter the trigone of the bladder. At 12 weeks of gestation, the inferior portion of the mullerian ducts fuses to create the uterovaginal canal. By 20 weeks, the uterovaginal canal develops into the uterus and upper vagina. The superior aspects of the mullerian ducts do not fuse, instead becoming the fallopian tubes.

The Vagina

The formation of the vagina starts with the development of two solid sinovaginal bulbs in the superior aspect of the vaginal portion of the urogenital sinus. These bulbs grow distally and form a solid vaginal plate. As the central aspect of the vaginal plate disintegrates, the lumen of the lower vagina is created and completed by 20 weeks. This lower vaginal lumen is separated from the more superior urogenital sinus and uterovaginal canal by the hymenal membrane. This membrane disintegrates centrally before birth, leaving behind a circumferential tissue band within the distal vagina and proximal to the vaginal introitus [2].

Anatomy

Vaginal Structure and Histology

Knowledge of pertinent normal lower genital tract anatomy is also useful when identifying vaginal and vulvar cysts [3]. The vagina, an empty tubular structure with a lumen that is held in place by its surrounding muscular and connective tissue support structures, sits between the bladder and rectum. The most proximal aspect of the vagina, the vaginal apex, heads posteriorly toward the ischial spines. Normal vaginal length is generally between 7 and 10 cm. In the absence of prior total hysterectomy, the cervix is present at the vaginal apex. The outer portion of the cervix (the ectocervix) is made of nonkeratinized stratified squamous cells and the inner portion and canal of the cervix (the endocervix) is comprised of simple columnar cells. Where these two different cell types meet is termed the transformation zone. The vaginal walls consist of three layers. The first layer in direct communication with the vaginal lumen is the vaginal epithelium (also referred to as "mucosa" despite an absence of glands) that is made of nonkeratinized stratified squamous epithelium. Deep to the mucosa is the muscularis that contains smooth muscle, collagen, and elastin. The deepest layer is the adventitia that is made of collagen and elastin connective tissue. The muscularis and adventitia together are referred to as the fibromuscular layer, and is commonly referred to as the pubovesicocervical fascia anterior to the vagina or the rectovaginal fascia posterior to the vagina. On the lateral aspects of the vagina, the adventitia extends out to the pelvic walls and contains fat, lymphatics, and neurovasculature.

Vaginal Neurovasculature

Blood is supplied to the vagina from the descending cervical branch of the uterine artery and branches of the internal iliac artery including the vaginal, middle rectal, and internal pudendal arteries. Lymphatic drainage of the upper two-thirds of the vagina is to the obturator nodes as well as the external and internal iliac nodes, while the lower third of the vagina drains to the inguinal nodes [4]. Portions of the inferior hypogastric plexus are responsible for sympathetic innervation of the vagina [5].

The Urethra

The urethra, which averages 3 cm in length, rests on and is supported by an endopelvic fascial layer and the anterior vaginal wall [6, 7]. It is comprised of four layers: mucosa, submucosa, internal urethral sphincter made of smooth muscle, and striated external urethral sphincter made of skeletal muscle. The inner surface of the proximal urethra is lined by transitional epithelium while the distal urethra is lined by stratified squamous epithelium [8]. This epithelium is continuous with bladder epithelium proximally and with that of the vaginal vestibule distally. Paraurethral glands open into the urethral lumen within this layer in the dorsal or vaginal side of the distal urethra [9]. The most pronounced of these paraurethral glands are the *Skene glands* that can be visible on the inner surface of the external urethral meatus. Blood supply to the urethra comes from the internal pudendal, vaginal, and inferior vesical arteries, while nerve supply comes from the pudendal nerve for somatic control and portions of the inferior hypogastric plexus for autonomic control [10]. The lymphatics of the distal third of the urethra drain into the superficial or deep inguinal nodes, whereas the proximal two-thirds drain into the external iliac, internal iliac, and obturator nodes.

The Vulva

The vulva is the term for the female external genitalia and includes the mons pubis, labia majora, labia minora, clitoris, vestibule, vestibular bulbs, vestibular glands, Bartholin (greater vestibular) glands, Skene (paraurethral) glands, external urethral meatus, and vaginal orifice. The skin over the mons pubis and labia majora has hair and a fatty subcutaneous layer. The obliterated processus vaginalis, or *canal of Nuck*, and the round ligament exit the inguinal canal and terminate within the fat or skin of the labia majora. The skin over the labia minora does not have hair and its subcutaneous tissue is devoid of fat.

The vaginal vestibule is an area in between the labia minora. The specific boundaries include the clitoris anteriorly, the fourchette posteriorly, the Hart line laterally, and the hymen medially. Distal to the Hart line the exposed tissue is a keratinized stratified squamous epithelium, while proximally it is nonkeratinized stratified squamous epithelium. The vestibule also contains the urethral, vestibular, Bartholin, and Skene gland openings.

The Bartholin glands are the female equivalent of the male bulbourethral, or Cowper, glands. One gland is situated at 5 O'clock while the other rests at the 7 O'clock position and each gland possesses a duct that opens into the vaginal vestibule. The inner surfaces of these glands are lined by columnar cells that secrete mucus to provide lubrication [11].

Evaluation and Management

History and Physical

A history and physical exam is the starting point of any vaginal or vulvar cyst workup. Specific symptoms to address in the patient's history of present illness include the presence or absence of vaginal or vulvar pain, dyspareunia, urethral versus vaginal discharge, spotting, post-void dribbling, leakage of urine, dysuria, incomplete voiding, or palpable mass. The patient should be questioned regarding recent vaginal trauma or placement of foreign body into the vagina. Pertinent aspects in the history include age, known congenital urogenital tract anomalies, obstetrical history including vaginal trauma and repair, sexual activity, recurrent urinary tract infections, prior vaginal surgery, and history of pelvic radiation therapy. Gynecological history including menstrual

cycles, prior vaginal infections, endometriosis, fibroids, and exposure to diethylstilbestrol (DES) in utero may be specifically applicable to the patient's presenting condition.

A targeted physical examination should include bimanual and speculum exams. A pelvic organ prolapse assessment to rule out prolapse as a cause for vaginal bulge symptomatology should be completed. Special attention should be taken to palpation of the anterior vaginal wall; a compressible bulge and discharge per urethra should raise suspicion for urethral diverticulum. Vaginal discharge should be evaluated. Masses such as cystic-appearing bulges should be examined with special attention taken to note the size, location, and tenderness of the cyst.

Differential Diagnosis

Vaginal Cysts

The differential diagnosis of vaginal cysts (Table 14.1) includes epidermal inclusion cysts, mullerian cysts, Gartner duct cysts (mesonephric remnants), and mucous inclusions (adenosis). Less common types of vaginal cysts include endometriotic cysts and vaginitis emphysematosum. Vaginal endometriotic cysts are infrequently encountered as the vagina is a rare location for endometriotic implants to manifest [12]. Although the etiology remains unclear, the benign and self-limited condition of multiple gas-filled cysts lining the vaginal wall (vaginitis emphysematosum) has also been described [13].

Table 14.1 Differential diagnosis of vaginal and vulvar cysts

Vaginal cyst	Vulvar cyst
Epidermal inclusion cyst	Epidermal inclusion (sebaceous) cyst
Mullerian cyst	Bartholin (greater vestibular) gland cyst
Gartner duct cyst (mesonephric remnant)	Skene (paraurethral) gland cyst
Adenosis (mucous inclusions)	Cyst of the canal of Nuck (hydrocele)
Endometriotic cyst	Leiomyoma
Vaginitis emphysematosum	Urethral diverticulum

Vulvar Cysts

The differential diagnosis of vulvar cysts (Table 14.1) includes vulvar epidermal inclusion (sebaceous) cysts, Bartholin (greater vestibular) gland cysts, Skene (paraurethral) gland cysts, cysts of the canal of Nuck (hydroceles), leiomyoma, and urethral diverticulum (Fig. 14.2; see Chap. 11 on Repair of Urethral Diverticula).

Overall, the frequency of presentation with a vaginal cyst is uncommon. Patients are more likely to present with symptomatic vulvar cysts. Kondi-Pafiti et al. [14] reviewed 40 cases of benign vulvar and vaginal cysts, the majority of which were asymptomatic. The most common symptomatic cyst was a Bartholin gland cyst. The most frequently encountered cyst was a mullerian cyst (12), followed by a Bartholin gland cyst (11), epidermal inclusion cyst (10), Gartner's duct cyst (5), endometrioid cyst (1), and one unspecified cyst. Deppisch [15] studied the classification of 64 surgically excised vaginal cysts and found that Gartner's duct cysts were uncommon, mullerian cysts made up 1/3 of cases, and epithelial inclusion cysts were the most common

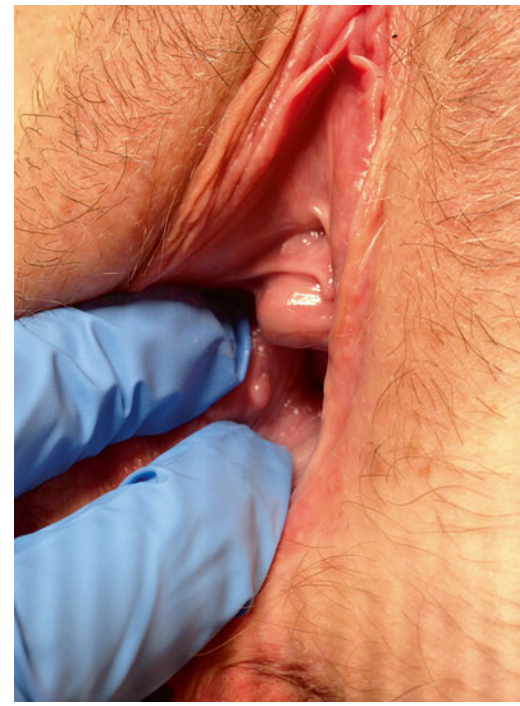


Fig. 14.2 Urethral diverticulum

type of vaginal cyst. These studies are consistent with the general acceptance that epithelial inclusion cysts, mullerian cysts, and Bartholin gland cysts more often cause symptoms and are therefore more likely identified than other types of vulvar or vaginal cysts.

Evaluation of Vaginal Cysts

Epidermal Inclusion Cysts

Epidermal inclusion cysts are often asymptomatic and discovered during routine gynecological exam. However, symptoms may include vaginal pain or dyspareunia and presence of a palpable mass. The appearance is round, pinkish-white in color, and variable in size (Figs. 14.3, 14.4, 14.5, and 14.6). The fluid content can be viscous. Histopathology confirms the diagnosis and reveals epithelial cells lined by a cyst wall made of nonkeratinized stratified squamous epithelium. Asymptomatic cysts can be observed, while drainage or excision should be performed for symptomatic cysts or if there is concern for infection. Additionally, the cyst should be biopsied and drained or excised for patients with a history of cervical, vaginal, or vulvar intraepithelial neoplasia. Hoffman et al. [16] reported on 26 women who were treated for vaginal intraepithelial neoplasia, 22 of whom had undergone prior hysterectomy for benign causes, cervical intraepithelial neoplasia, or malignancy. Five patients were

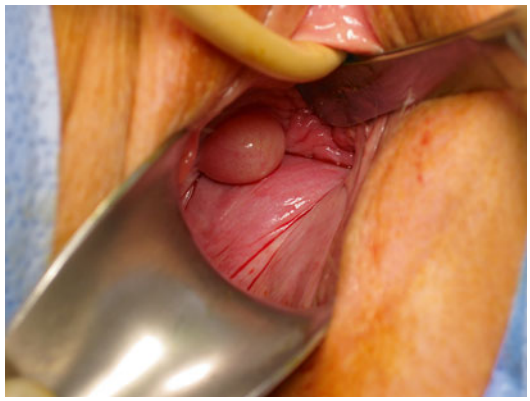


Fig. 14.3 Epidermal inclusion cyst arising from right lateral vaginal wall

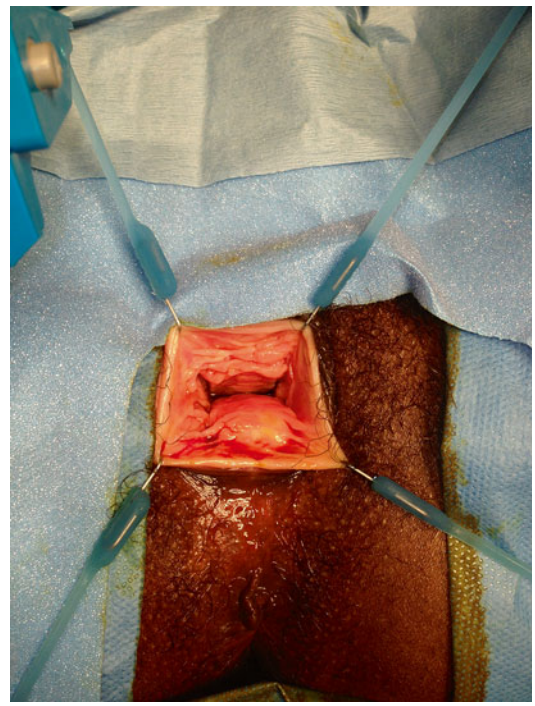


Fig. 14.4 Epidermal inclusion cyst arising from posterior vaginal wall

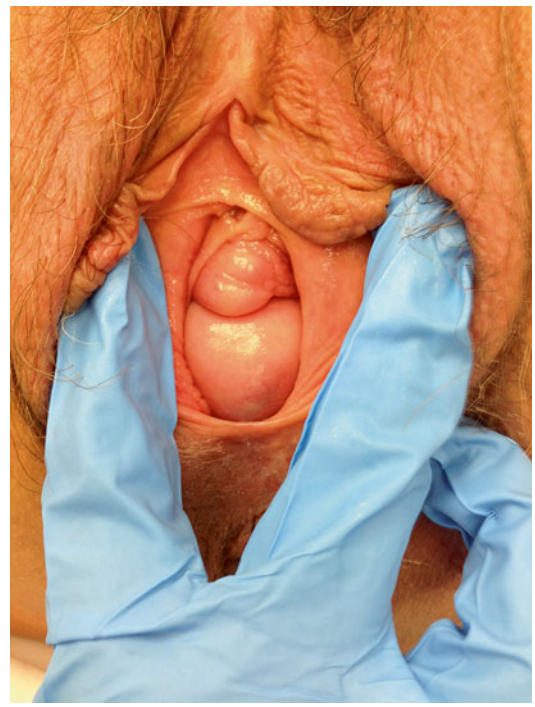


Fig. 14.5 Epidermal inclusion cyst (visually difficult to distinguish from pelvic organ prolapse)

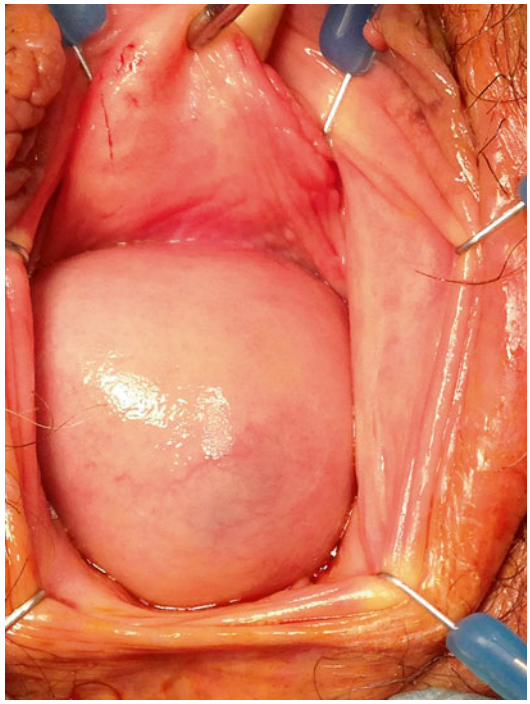


Fig. 14.6 Showing base of epidermal inclusion cyst shown in Fig. 14.4 arising from mid-portion of anterior vaginal wall

noted to have vaginal cuff inclusion cysts that were found to be positive for a neoplastic process. This finding suggests that when a vaginal epithelial inclusion cyst is identified in a patient with a prior intraepithelial neoplastic process or cancer, biopsy of the cyst should be performed.

Gartner Duct Cysts

On physical exam these cysts are typically identified within a lateral wall of the vagina. Gartner duct cysts are mesonephric (Wolffian) duct remnants. During embryonic development the mesonephric ducts are involved in the formation of the urogenital system. In females, after the fundamental urinary system is created, these ducts regress and sometimes leave a remnant behind causing a Gartner duct cyst. They are usually asymptomatic and encountered during routine gynecological exam. When present, symptoms include vaginal pain, dyspareunia, or the awareness of the presence of a mass. Histologically, they are lined with cuboidal or columnar epithelium. Asymptomatic cysts can be observed while symptomatic ones

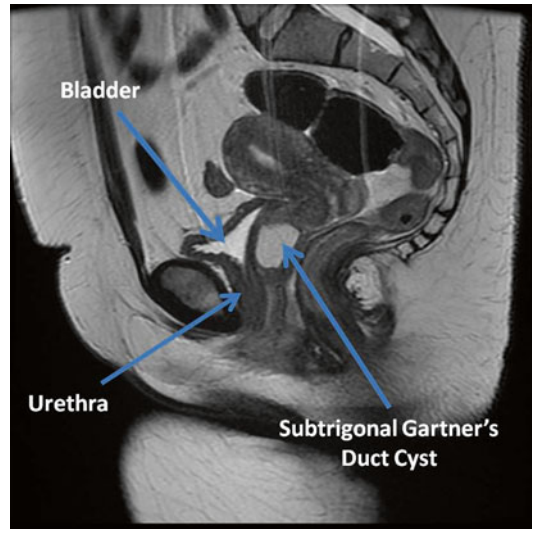


Fig. 14.7 MRI sagittal view of Gartner duct cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

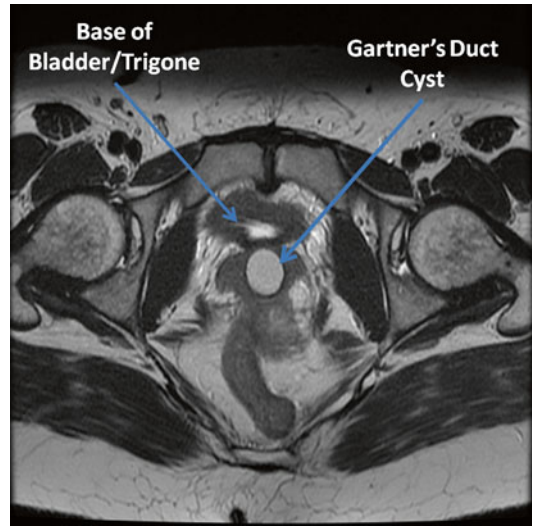


Fig. 14.8 MRI transverse view of Gartner duct cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

should be marsupialized or excised. If surgical management is pursued, it is important to image the lower reproductive tract because Gartner duct cysts can track cephalad as far as into the broad ligament and an awareness of the cyst dimensions may prove helpful during dissection (Figs. 14.7 and 14.8). Furthermore, imaging is performed to

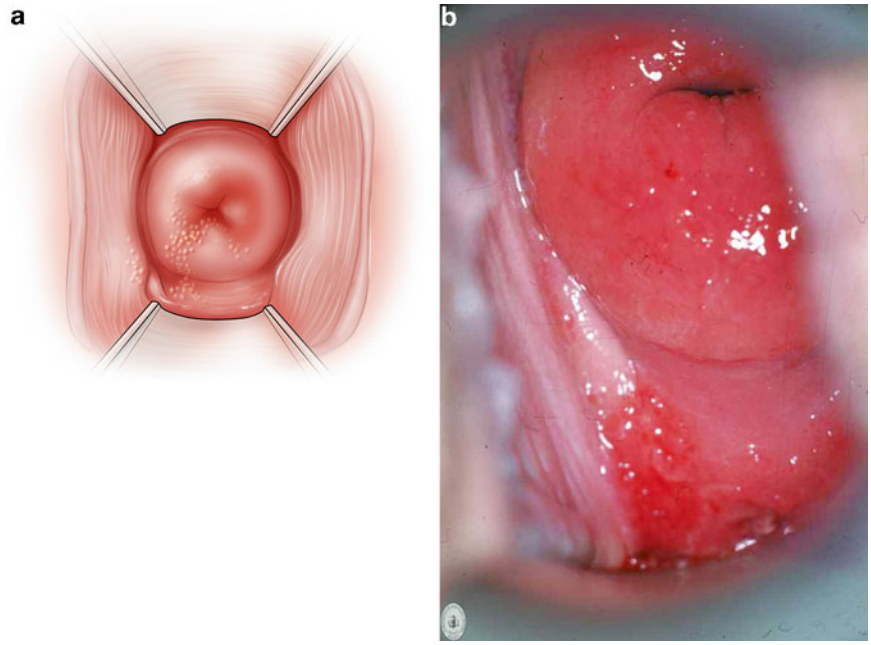


Fig. 14.9 (**a**, **b**) Vaginal adenosis

detect renal anomalies with which Gartner duct cysts may be associated [17]. Bats et al. [18] reported a case of malignant transformation of a Gartner cyst in a 67-year-old woman who presented with vaginal bleeding. Pathology from excision revealed clear cell carcinoma. Although the risk of developing carcinoma from a Gartner duct cyst is extremely rare, we suggest performing at least a biopsy if not an excision in patients with vaginal bleeding.

Adenosis

Vaginal adenosis is a condition where mucous inclusions (glandular tissue) are present within the vaginal epithelium. The etiology can be spontaneous, but many affected patients were exposed to diethylstilbestrol (DES) in utero. Symptoms include vaginal discharge, irritation, pruritis, dyspareunia, and postcoital spotting. The area of affected vaginal wall appears erythematous and grainy or possibly with multiple small (millimeter) cysts. Histology displays normal vaginal stratified squamous mucosa containing regions of columnar epithelium. Those patients with a history of DES exposure are at increased risk for developing vaginal clear cell adenocarcinoma. Because of the risk of underlying carcinoma, a colposcopic exam should be performed with biopsy. The condition is usually self-limited; however, if persistent or symptomatic, treatment with carbon dioxide laser coagulation or excision can be considered. See Fig. 14.9a, b.

Vaginitis Emphysematosum

Vaginitis emphysematosum is an uncommon abnormality that produces multiple subcentimeter gas-filled cysts within small connective tissue spaces in the vaginal wall giving it a corrugated appearance. As reported by Leder et al. [19] the etiology of this benign bacterial vaginitis is uncertain, but most reported cases have noted the presence of vaginitis caused by *Trichomonas vaginalis* or *Gardnerella vaginalis*. The condition is often discovered in gravid or immunocompromised patients and usually does not cause symptoms. When present, symptoms may include vaginal discharge and pruritis. The condition can be observed on imaging such as X-ray

or Computed Tomography (CT) scan. Histology reveals gas spaces beneath the vaginal epithelial pegs that are lined by inflammatory multinucleated giant cells. Vaginitis emphysematosum is benign and self-limiting.

Mullerian Cysts

As most vaginal cysts, mullerian cysts are most often asymptomatic and found on incidental exam. However, symptoms may include dyspareunia and presence of a palpable mass detected either manually or while attempting to insert a tampon. They appear round, smooth, pink, variable in size, and fluid content feels gelatinous when palpated. On exam, it is not possible to distinguish mullerian cysts from inclusion or Gartner's duct cysts. Histology reveals a columnar epithelial lining. Once again, asymptomatic cysts can be observed whereas incision and drainage or excision should be performed for symptomatic or infected cysts. There have been several case reports documenting vaginal mullerian cysts. In one report, a woman presented with an 8 cm posterior vaginal wall cyst that contained mucoid material [20], while another case reported a large anterior vaginal wall cyst that was evaluated with radiologic studies and subsequently excised [21]. There has been one unusual report of adenocarcinoma arising from a vaginal mullerian cyst in a 48-year-old presenting with a 3 cm ruptured vaginal cyst [22]. This finding highlights the need to have a low threshold for biopsy when larger or persistent anatomical abnormalities are encountered.

Imaging

The role of radiography in evaluation of a vaginal cyst depends on the location and type of cyst suspected after performing a history and physical exam. Based on the initial evaluation, it may be prudent to obtain preoperative information regarding cyst characteristics and its proximity to neighboring structures. For example, Gartner duct cysts (mesonephric remnants) may extend as far cranially as into the broad ligament and preoperative imaging may help determine the extent of intraoperative dissection that will be necessary. Additionally, since Gartner duct cysts can be associated with renal anomalies, preoperative imaging may expose findings such as renal agenesis or ectopic ureter. Sheih et al. [23] found 13 cases of cysts in the pelvis that were associated with ipsilateral renal agenesis or dysplasia after 280,000 children with renal abnormalities were screened with ultrasounds. Seven of the 13 cases found to have pelvic cysts were girls with Gartner duct cysts.

Preoperative imaging may be useful in identifying additional cysts that were not visible on physical exam. Wai et al. [24] reported a case of a 20-year-old woman who presented with two vaginal cysts on exam. Magnetic Resonance Imaging (MRI) was obtained preoperatively and not only demarcated the arrangement of her vaginal cysts, but also identified additional larger cysts that had not been appreciated on physical examination.

Evaluation of Vulvar Cysts

Vulvar Epidermal Inclusion Cysts

Vulvar epidermal inclusion cysts, also known as sebaceous cysts, are the most common vulvar cysts. They form on areas where hair follicles or sebaceous glands are present. Additionally, clitoral epidermal inclusion cysts have been described in patients with a history of female genital mutilation. Rouzi et al. [25] reported on 21 females presenting anywhere between 2 and 20 years after having had a type I female genital mutilation procedure, whereas Ofodile et al. [26] described 19 cases. Most vulvar inclusion cysts are asymptomatic. When present, symptoms include pain to touch and bothersome appearance. These cysts appear firm, are often mobile, and contain caseous material. They run the risk of becoming infected and developing into cellulitis or an abscess. Obstruction of the duct leads to buildup of secretions and consequently cyst formation. Treatment includes warm compresses to drain the obstructed duct. Recurrent, persistent, or enlarging cysts can be treated with incision and drainage, punch biopsy with subsequent drainage, or excision. The type of excision may depend on the cyst size (to be described later in

the chapter). In a randomized prospective study of 60 patients with nonspecifically located noninfected vulvar epidermal inclusion cysts, Lee et al. [27] compared results after treatment with the punch biopsy technique versus excision. The study group found that those treated with the punch biopsy method produced superior cosmetic results, while there was no difference in recurrence rates over the 16-month time period they were studied.

Bartholin Gland Cysts

Like other vulvar cysts, Bartholin gland duct cysts form when the duct gets obstructed and accumulation of gland secretions leads to cyst formation. If the cyst becomes infected an abscess forms. A single organism may be causative or the infection may be polymicrobial. Anaerobic and aerobic bacteria are often the organisms involved, and *Neisseria gonorrhoeae* or *Chlamydia trachomatis* only rarely. In fact, Bhide et al. [28] found that 74 % of 78 cases reviewed revealed a positive microbial culture, and aerobes were the most common organisms involved. None of the cases contained *Neisseria gonorrhoeae* or *Chlamydia trachomatis*. Mechanical trauma can be a risk factor for cyst formation and stimulation from sexual activity can lead to cyst enlargement. Premenopausal women are most commonly affected and are usually benign. According to the New York State Department of Health census data reported in a study done in 1996 by Visco et al. [29], the incidence of Bartholin gland carcinoma was 0.023 per 100,000 woman-years in premenopausal women and 0.114 per 100,000 woman-years in postmenopausal women. Malignancies encountered include squamous carcinoma and adenocarcinoma. Although cancer is rare, Bartholin gland duct cysts that present after age 40 should be evaluated for malignancy. If excision is not to be performed, then biopsy of the cyst wall during drainage should occur. Ben-Harosh et al. [30] reported a case of a young woman who presented with a firm and irregular Bartholin gland. Pathology after excision revealed gland hyperplasia. This finding suggests that a tissue diagnosis should be made for any gland cyst with a nodular texture. Biopsy plus drainage or cyst excision should be performed, not drainage alone. Symptoms of Bartholin gland cysts and abscesses include pain to touch or during activity. Glands are usually unilaterally affected and it is rare to have simultaneous bilateral gland involvement. Upon inspection, cysts appear round and feel tense upon palpation (Figs. 14.10 and 14.11). If an abscess is present, there may be associated epithelial erythema and tenderness. Management options for Bartholin gland cysts include biopsy in women over the age of forty, observation if asymptomatic, incision and drainage, marsupialization, or gland excision. Abscesses cannot be observed and contents must be drained.

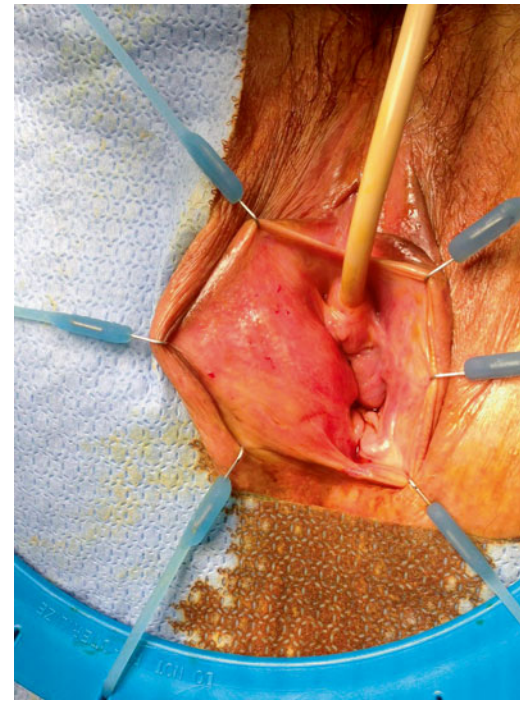


Fig. 14.10 Bartholin gland cyst

Skene Gland Cysts

Skene gland cysts occur after gland ductal occlusion (Fig. 14.12). The Skene glands are located at the distal urethra and are the largest paraurethral glands. Similar to other vulvar gland pathology, a cyst can become infected and form an abscess. Risk factors may include local infection or trauma. Symptoms include dyspareunia, localized

pain, voiding dysfunction, and urinary obstruction. Treatment of Skene gland cysts or abscesses is with excision. Abscesses should be treated with antibiotics prior to excision to decrease the risk of spreading infection. There may be a role for cystourethroscopy as well. Lucioni et al. [31] found that Skene gland cysts may benefit from assessment to determine proximity to urethra and bladder. Cystourethroscopy was used to rule out other pathology such as urethral diverticulum or fistula and can be performed during the same operative setting prior to initiating surgical excision.

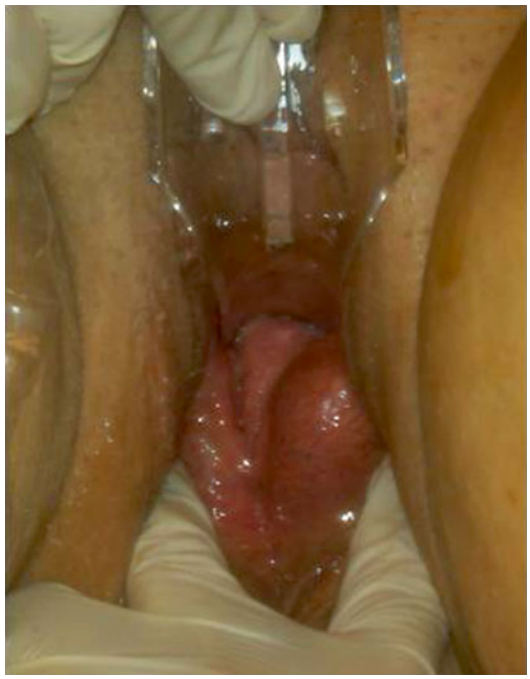


Fig. 14.11 Bartholin gland cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

Cysts of the Canal of Nuck

A cyst of the canal of Nuck (hydrocele of the canal of Nuck) forms due to a persistent processus vaginalis. This type of cyst is a fluid-filled hernia of the peritoneum that accompanies the round ligament and extends from the inguinal canal into the labia majora. It presents with inguinal swelling with or without unilateral labia majora pain and swelling. The mass is irreducible. It can be confused with an inguinal hernia (may contain intestines); however intestinal contents should not be present if the mass is truly a cyst of the canal of Nuck. Transillumination can be used to evaluate for a cystic versus a solid mass. The finding should be evaluated for herniation preoperatively. Ultrasound or MRI are the imaging modalities of choice to aid in diagnosis

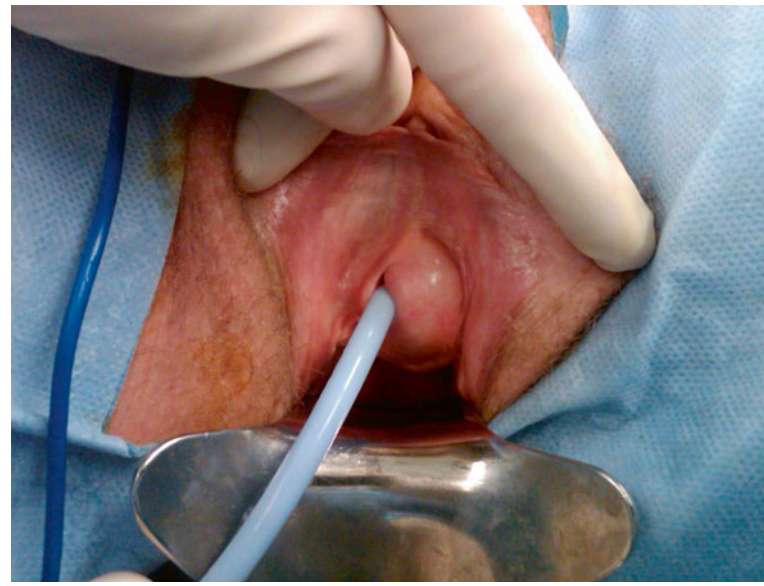


Fig. 14.12 Skene gland cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

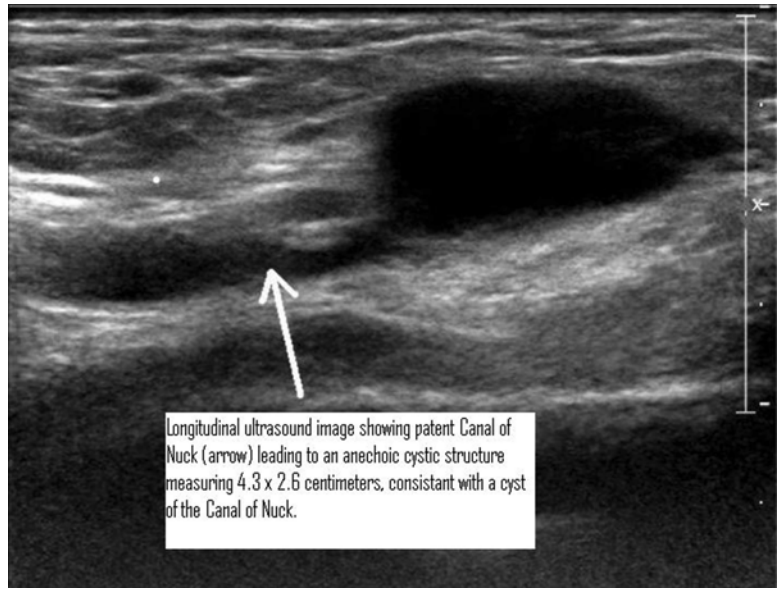


Fig. 14.13 Longitudinal ultrasound image of cyst of the Canal of Nuck (Used with permission from Jagdale R, Agrawal S, Chhabra S, Jewan SY. Hydrocele of the Canal of Nuck: Value of Radiological Diagnosis. Journal of Radiology Case Reports. 2012 June; 6(6): 18-22. www.jrcr.org/916)

and confirm preoperative suspicion. Ultrasound can be performed while the patient performs the Valsava maneuver to evaluate for this type of cyst compared to inguinal hernia (Fig. 14.13). Both ultrasound and MRI would reveal an ovoid tubular structure within the inguinal canal or labia majora [32]. Treatment is excision of the cyst and correction of any herniation that may be present. Diagnosis is confirmed with histopathology after excision.

Surgical Intervention

Operative Technique

Surgical management options for vaginal cysts include incision and drainage, marsupialization, and definitive management with excision.

Incision and Drainage

Incision and drainage can provide immediate but temporary relief of symptoms. Once the incision edges reseal, secretions may recollect within the cyst. Therefore, the goal with an incision and drainage is to drain cyst contents and create a new epithelialized tract for continued drainage. The procedure can be performed as an outpatient. The patient is placed in dorsal lithotomy position and the area overlying the cyst is cleaned with an antiseptic agent. A local anesthetic such as 1 or 2 % lidocaine is infused into the cyst and the overlying vaginal epithelium where the incision is to be made. A 1 cm incision is made with a scalpel (number 15 blade) through the vaginal epithelium and cyst wall. The tips of a hemostatic or tonsil clamp may be placed within the cyst and used to lyse adhesions, thereby facilitating drainage. The fluid does not need to be cultured unless abscess is suspected. Ideally, the incision should be kept patent with a device such as a Word catheter. The tip of a Word catheter (Fig. 14.14), which is the diameter of a number 10 French Foley catheter, is placed within the cyst cavity and the balloon tip is then inflated at the opposite end of the catheter with sterile saline until the balloon is large enough to prevent the catheter from falling out (about 3 ml [33]). As there are openings on both ends of the catheter, this device allows drainage of cyst contents into the vagina. The catheter should remain in situ for 4 weeks during which time nothing should be placed in the vagina [33–35]. Broad-spectrum antibiotics to cover aerobes and anaerobes are only warranted in the case of abscess.

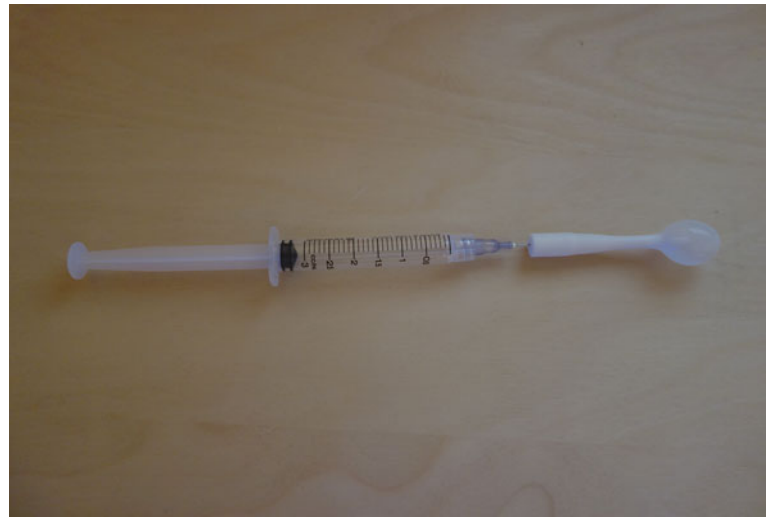


Fig. 14.14 Word catheter

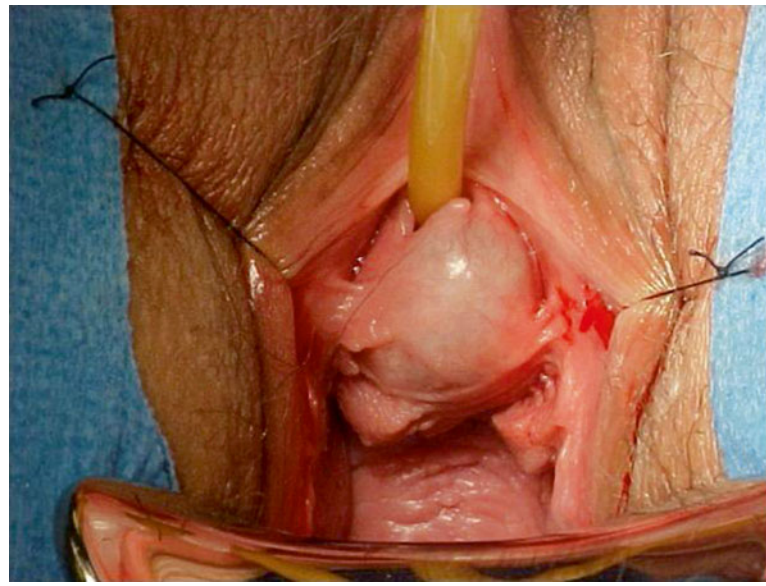


Fig. 14.15 Skene gland cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

Marsupialization

Marsupialization is a technique that was devised due to the high recurrence rates following incision and drainage without epithelialization of the tract. It is a way to create a new duct drainage tract without the use of an external device such as a Word catheter. The procedure is most commonly performed in an ambulatory operating room setting but can also be done in an office or emergency room. The patient is placed in dorsal lithotomy position. Analgesia can be achieved with local anesthesia, sedation plus local anesthesia, regional anesthesia, or most commonly general anesthesia. The vagina and vulva are prepped and draped in the usual sterile manner. A 3 cm incision with a scalpel is made in the vaginal epithelium overlying the cyst while taking care not to rupture the cyst wall (Fig. 14.15). The cyst wall is then incised (the contents will drain) with a scalpel and the cyst incision is extended to the same

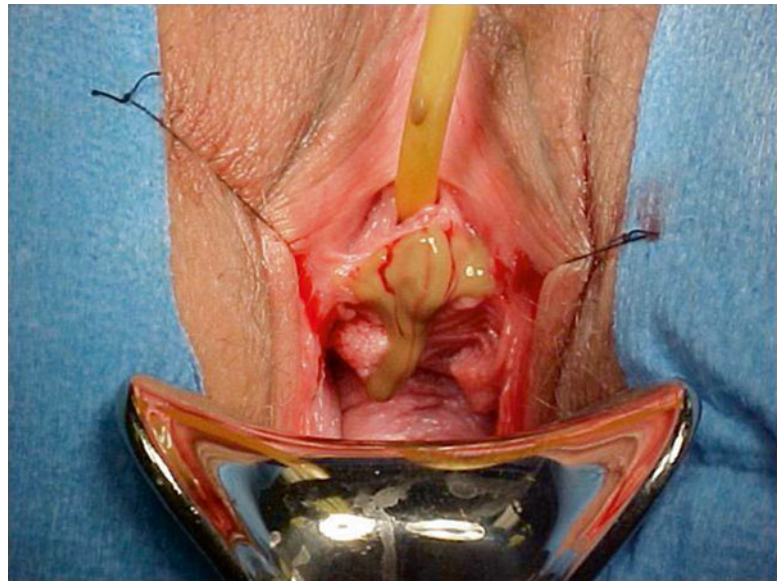


Fig. 14.16 Cyst wall incision (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

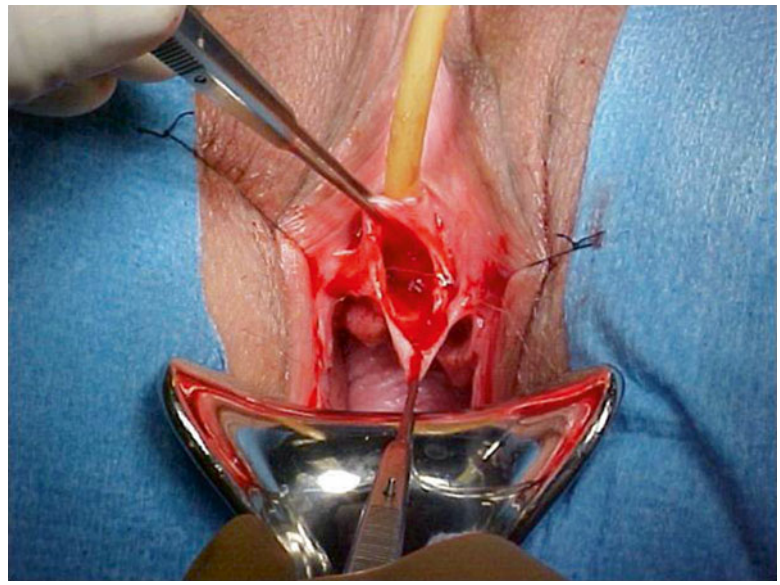


Fig. 14.17 Lysis of adhesions within cyst wall (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

size as the vaginal epithelial incision (Fig. 14.16). The cyst wall is then everted with the use of Allis clamps that are placed circumferentially around the incision to grasp the cyst wall plus overlying vaginal epithelium in each bite. A hemostat is used to lyse adhesions within the cyst and the cyst is then irrigated (Fig. 14.17). The cyst wall edge is then circumferentially sutured to its overlying vaginal epithelium with either 2-0 or 3-0 delayed-absorbable—Vicryl® (Ethicon, Inc, Blue Ash, OH) is commonly used—interrupted sutures (Fig. 14.18). Generally by 4–6 weeks after the procedure, the opening of the duct decreases in size to less than 1 cm. Risks of marsupialization include cyst recurrence and rarely abscess formation. Marsupialization requires better surgical site

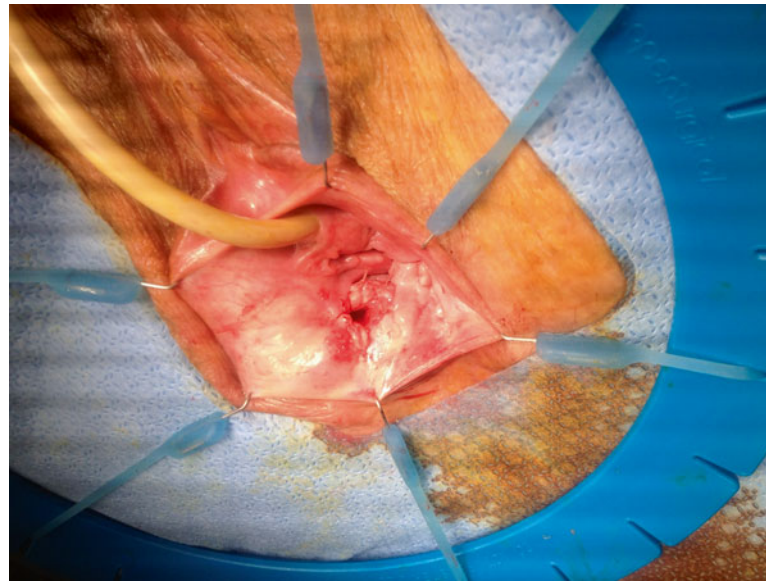


Fig. 14.18 End result after marsupialization of cyst wall to vaginal epithelium

exposure, a larger incision, more suturing, greater levels of analgesia, and more time to perform than incision and drainage. For these reasons, the use of marsupialization has decreased since the introduction of Word catheter use at the time of incision and drainage.

Excision

The procedure for excision of a vaginal cyst is similar regardless of the type of cyst. When performing an office physical exam, one should inspect the size of the cyst. Consideration should be taken to perform excision in the operating room for larger cysts. Although there is no cutoff as to what size cyst is unlikely to be successfully excised in the office setting, cysts greater than 2 cm generally require dissection that may be too extensive for an office procedure. In addition, preoperative assessment may be performed with imaging to evaluate the relationship of the cyst to surrounding structures depending on its vaginal location on exam. If preoperative evaluation reveals the cyst to be in close proximity to the bladder or ureters, the procedure should take place under general anesthesia. Prior to cyst excision, consideration can be taken to placing ureteral catheters. Integrity of the urethra and bladder can be evaluated intraoperatively with the use of cystourethroscopy. Surgical intervention in the operating room may be performed under local or general anesthesia. The patient is placed in the dorsal lithotomy position and then prepped and draped in the usual sterile fashion.

The technique for excision of a vaginal cyst is as follows. Initially, adequate exposure of the cyst must be ensured. Vaginal retractors or a Lone Star Retractor System™ (Cooper Surgical, Inc., Stafford, Texas) may be used to assist with visualization (Fig. 14.19). Stay sutures with 0 Vicryl may be placed to keep orientation above or below the cyst, such as at the 12 O'clock position. The vaginal epithelium is infused with a dilute vasopressin solution using a thin 22-gauge or 25-gauge needle. It is our practice to dilute 20 units of vasopressin in 100 ml normal saline. The purpose of this infiltration is for hydrodissection and hemostasis. A scalpel is used to make a longitudinal superficial incision through the vaginal epithelium overlying the cyst wall. The vaginal wall is then dissected circumferentially off the cyst until the base is reached and the cyst is entirely freed (Fig. 14.20). Dissection is carried out sharply with Metzenbaum scissors or a knife as well as bluntly while using electrocautery sparingly as needed.

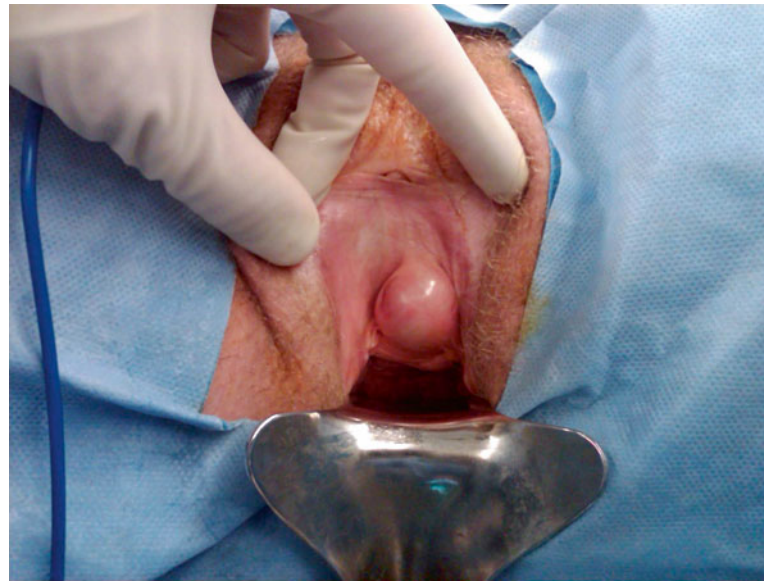


Fig. 14.19 Skene gland (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH.)

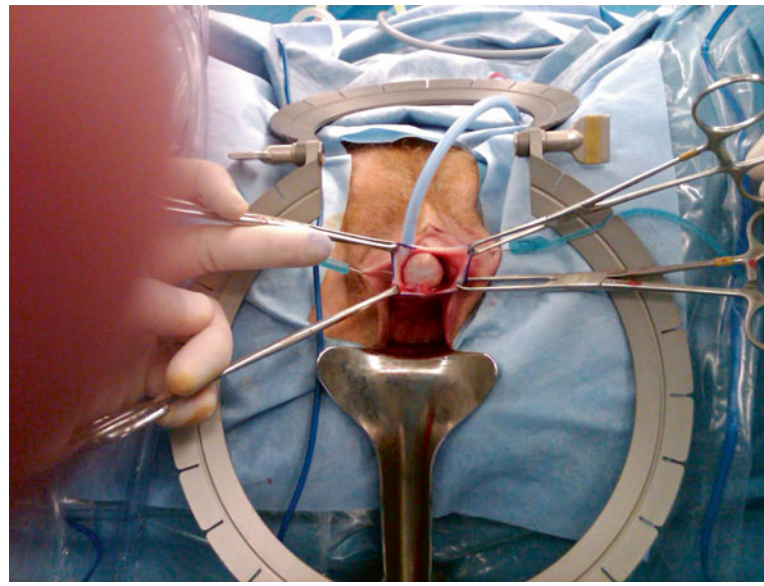


Fig. 14.20 Vaginal wall dissection off the underlying cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

The direction of dissection is inferior to superior as this technique minimizes disruption in visualization of the surgical field with blood. Attention is taken not to rupture the cyst during the process. The base is often the location of blood supply for the cyst, and therefore after the cyst is removed it may bleed. This base should be sutured with 3-0 Vicryl in a figure-of-eight fashion or in a running-locking technique until hemostasis is noted. The vascular supply to the cyst can also be clamped with a hemostat and suture ligated with 2-0 or 3-0 Vicryl. Electrocautery may be used with careful consideration of underlying structures. Complete removal of the cyst (Fig. 14.21) is essential because if residual tissue is left behind, cyst formation may recur. If potential space (Fig. 14.22) is present in

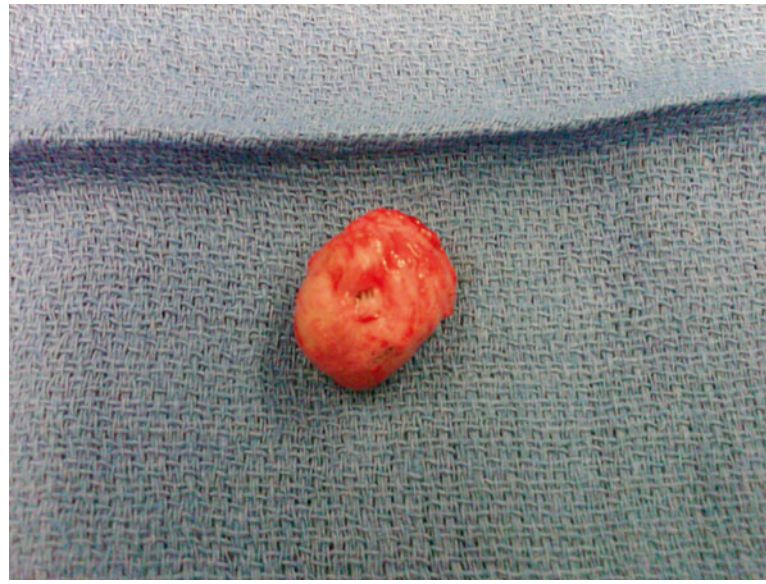


Fig. 14.21 Skene gland cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

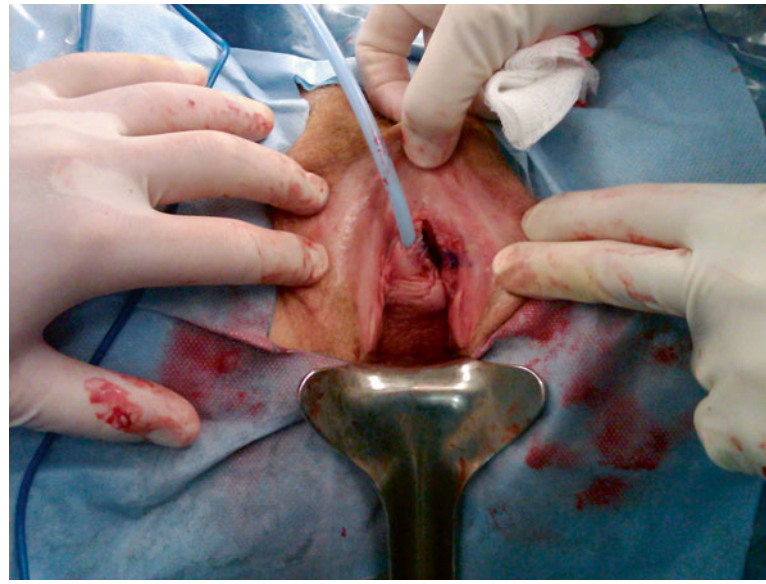


Fig. 14.22 Potential space after excision of cyst (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

the fibromuscular layer deep to the vaginal epithelium, this layer should be closed with 2-0 or 3-0 Vicryl sutures in an interrupted fashion. The overlying vaginal epithelium is then sutured in a running or running-locking fashion with 2-0 Vicryl (Fig. 14.23). The overlying vaginal epithelium rarely needs to be trimmed prior to closure as trimming may cause tension leading to scarring, pain, or dyspareunia.

In the cases of larger cysts that impede anatomical visualization or with inadvertent cyst rupture (Figs. 14.24 and 14.25), the cyst may be intentionally drained and then the cyst wall is dissected (Figs. 14.26, 14.27, and 14.28) or destroyed [24]. Potential space and vaginal epithelium should be closed in the aforementioned manner (Figs. 14.29, 14.30, and 14.31). The excised cyst or a portion of the cyst wall should

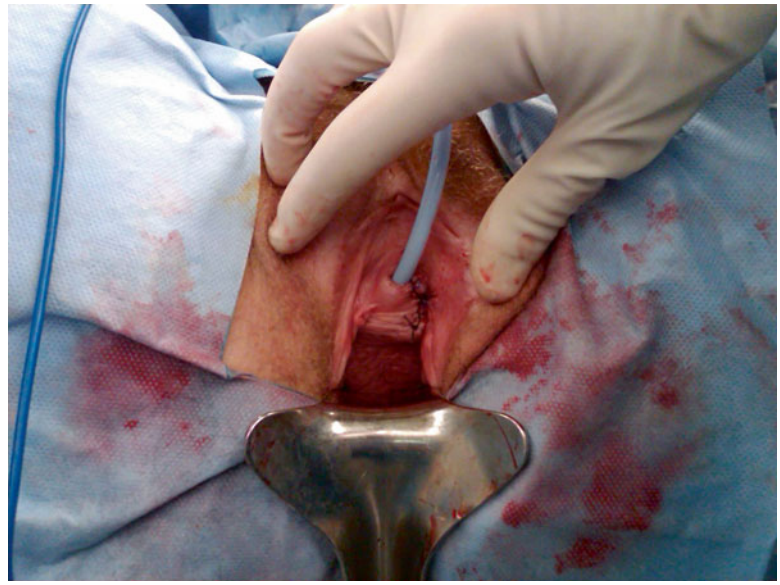


Fig. 14.23 Closure of vaginal epithelium (Courtesy of Howard Goldman, MD, Department of Urology, Cleveland Clinic, Cleveland, OH)

be sent to pathology for examination. We recommend cystourethroscopy to be performed at the end of any vaginal cyst excisional procedure to evaluate for urinary tract injury. Risks of excision include bleeding, hematoma formation, cellulitis, cyst recurrence, pain, dyspareunia, and damage to deep and surrounding anatomical structures.

Vulvar Considerations

The techniques for surgical management of vulvar cysts such as incision and drainage, marsupialization, and excision are similar to that of vaginal cysts, with a few variations based on the type of vulvar cyst that is present.

Vulvar Epidermal Inclusion Cysts

Asymptomatic and noninfected vulvar epidermal inclusion cysts can be observed. Excision can be performed for symptomatic cysts where the type of excision is determined based on cyst size. Cysts that are 1 cm or smaller can be excised utilizing a wedge incision technique. The wedge-shaped specimen removes its encompassing skin, subcutaneous tissue, and the cyst in its entirety. Cysts larger than 1 cm require the usual excision technique starting with a skin incision, sharp dissection of skin off the underlying cyst wall, and complete removal of the cyst. Attention must be paid not to grab the cyst wall during dissection

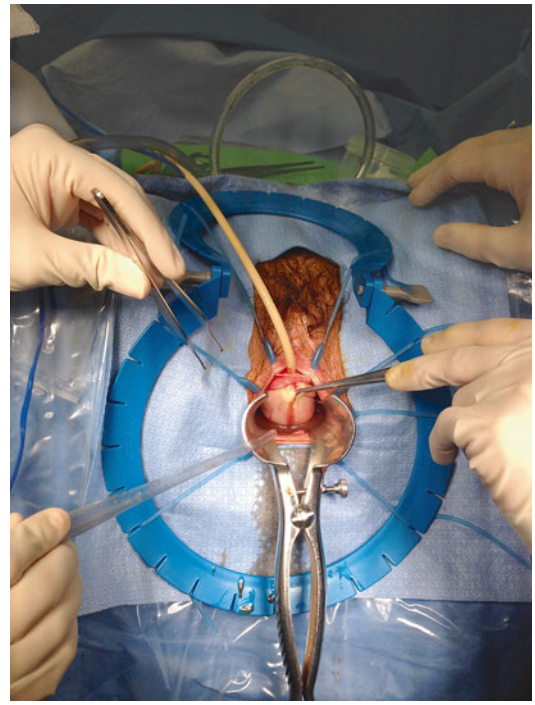


Fig. 14.24 Cyst rupture during dissection of anterior vaginal wall cyst

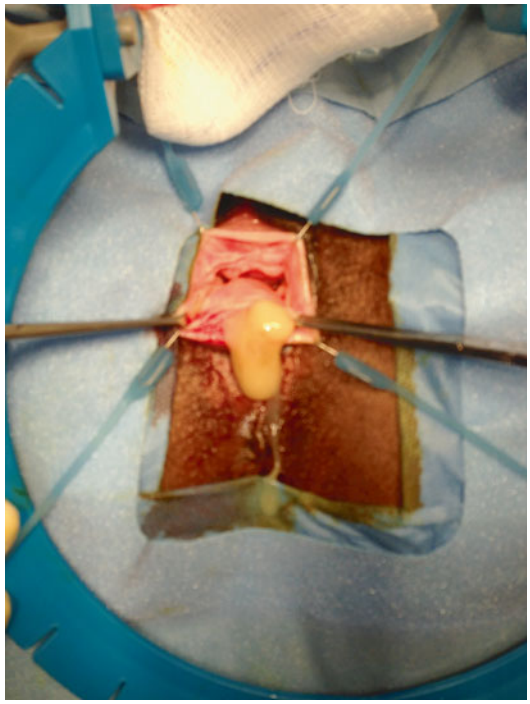


Fig. 14.25 Drainage of a posterior vaginal cyst contents

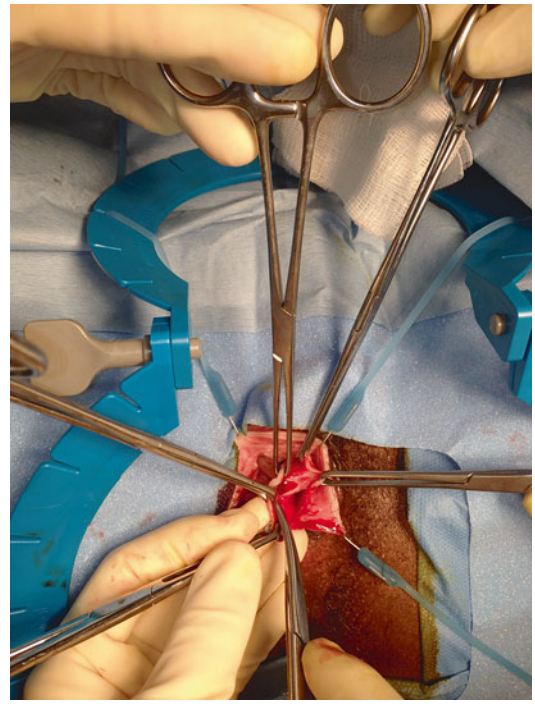


Fig. 14.27 Dissection of posterior vaginal cyst wall off of vaginal epithelium and underlying endopelvic fascia

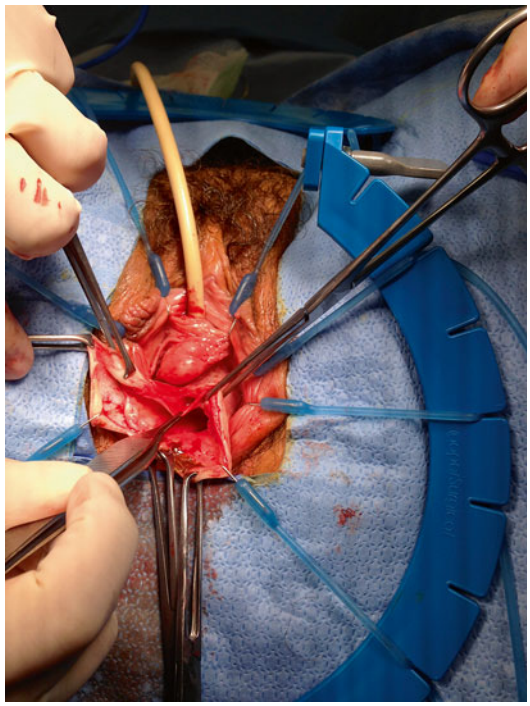


Fig. 14.26 Dissection of posterior vaginal cyst wall off of vaginal epithelium and underlying endopelvic fascia

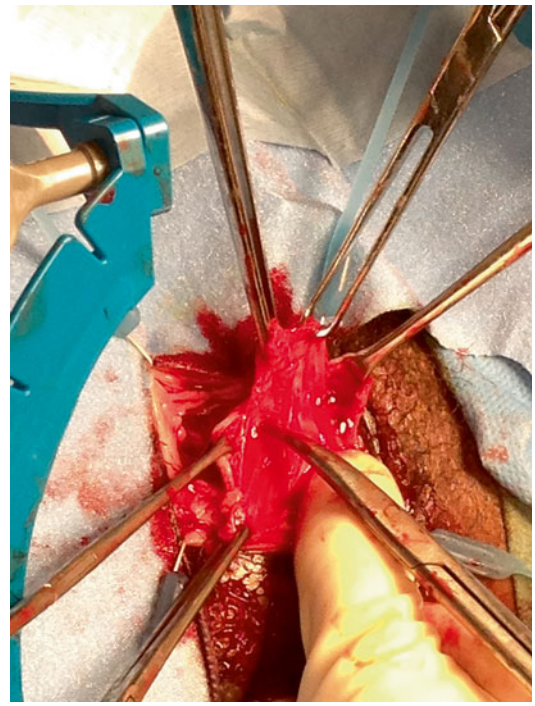


Fig. 14.28 Allis clamps lifting posterior vaginal cyst wall

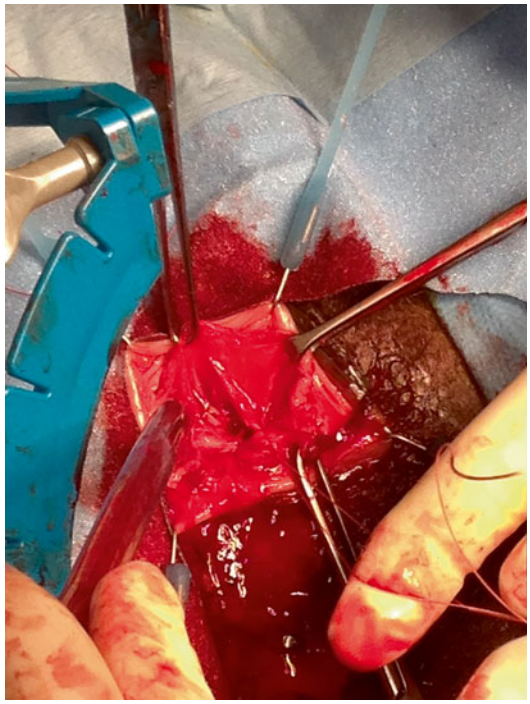


Fig. 14.29 Closure of potential space after removal of posterior vaginal cyst

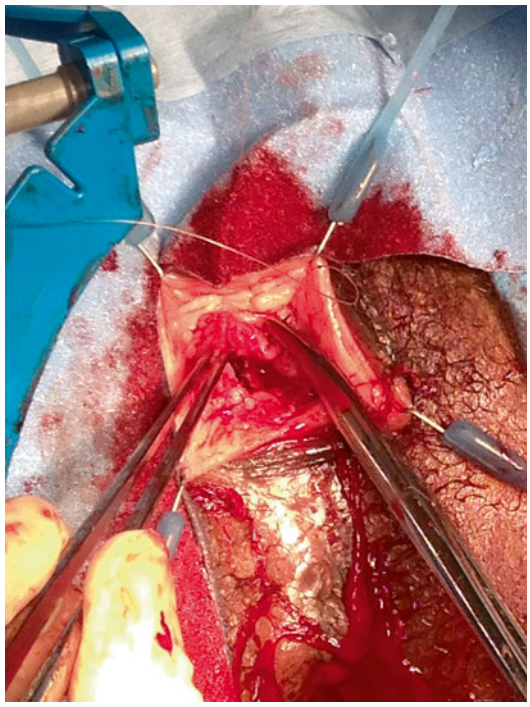


Fig. 14.30 Closure of posterior vaginal epithelium after excision of cyst

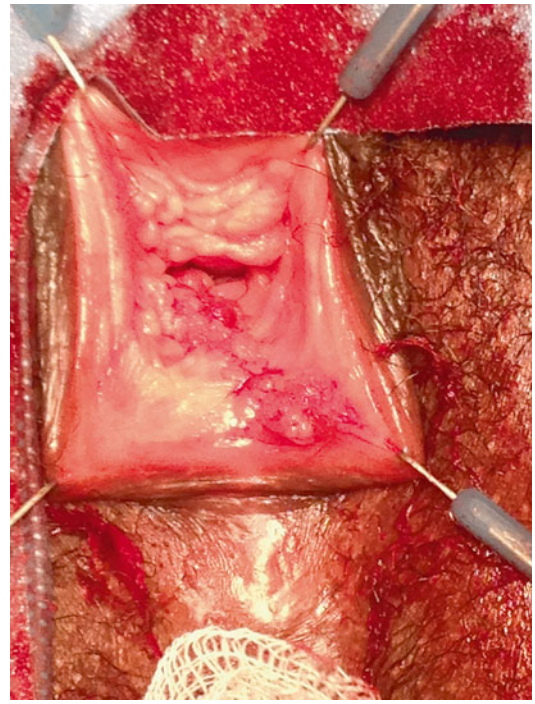


Fig. 14.31 Completed closure of posterior vaginal wall

in order to avoid rupture. The skin can be held with Adson forceps while fine dissection is carried out with Stevens tenotomy scissors. The wound is closed in layers and excess skin is cut as needed for the skin to realign properly. After excision of vulvar cysts, skin is closed in an interrupted or subcuticular running fashion with 4-0 delayed-absorbable sutures. A pressure dressing can be applied to prevent hematoma formation. Alternatively to excision, a punch biopsy of the lesion and drainage of cyst contents through the biopsy site can be performed.

Bartholin Gland Cysts

As Bartholin gland duct cysts and abscesses are common enough and often symptomatic, they require special mention. Abscesses and symptomatic cysts should be treated. Treatment options include incision and drainage with or without Word catheter placement, marsupialization, and excision. Other procedures to obliterate the cyst wall such as incision plus the use of silver nitrate stick insertion into the cyst or abscess [36, 37] or carbon dioxide laser vaporization [38, 39]

have been described but are not commonly employed. A recent review by Wechter et al. [33] looked at recurrence rates after various treatments of Bartholin gland cysts and abscesses including aspiration, incision and drainage, marsupialization, excision, silver nitrate ablation, and carbon dioxide laser excision. Recurrence rates varied from 0 to 38 % and were highest after incision or aspiration alone. There were no recurrences noted after marsupialization; however no best treatment approach was identified. Patients must be counseled regarding the risk of cyst reaccumulation when any portion of the gland is left in situ. Patients also need to be made aware that gland function and ability to form vaginal secretions may be reduced if the gland is removed in its entirety. These facts along with the patient's age and personal history of Bartholin gland cyst or abscess recurrence should be used when selecting a procedure.

The technique for Bartholin gland cyst incision and drainage, marsupialization, and excision may be carried out as mentioned above. A linear incision is made on the medial aspect of the vestibule overlying the cyst roughly 1 cm distal and parallel to the hymen. The incision should be on the vaginal mucosa and not on the vulvar skin. This location mimics the site of the glands' natural orifices at the 5 and 7 O'clock positions. If performing an incision and drainage, the incision is generally less than 1 cm. After drainage, a Word catheter should be placed and remain in situ for at least 4 weeks. Kushnir et al. [40] described the use of a small loop of plastic tubing that was secured in place after incision and drainage of a Bartholin gland abscess when a Word catheter was not available. The technique was found to be a successful replacement to the Word catheter as there was no evidence of the abscess and the patient healed 3 weeks after the procedure.

If marsupialization or excision is to take place, the incision is created in the same location but is extended to 3 cm in length while remaining parallel with the hymen throughout the incision. Cultures should be obtained for *N. gonorrhoeae* and *C. trachomatis* during drainage. Complete excision should not be performed in the presence of suspected infection prior to the administration of antibiotics. If excision is being performed, care must be taken as the rectum is located just posterior to the gland. A finger may be placed in the rectum during dissection to differentiate between rectum and cyst. Generally the blood supply arises from the posterosuperior aspect of the cysts and therefore dissection should be carried out heading toward this final location. The vascularity of the Bartholin gland and its associated cysts requires that dead space remaining after cyst excision should be closed in layers to prevent hematoma formation. Surgical complications include bleeding from venous channels that can cause significant hematoma formation. In addition to suture ligation techniques, direct pressure should be applied and a drain may be placed intraoperatively if needed.

Postoperative care includes ice packs initially to decrease swelling and pain from scarring. Nothing should be placed per vagina for at least 4 weeks, at which time the site should be inspected. Warm sitz baths and proper hygiene should be encouraged during recovery. Broad-spectrum antibiotics need only be given when treating abscesses with accompanying cellulitis or if postoperative cellulitis develops. Although rare, there have been case reports of rectovaginal fistula developing as a complication to Bartholin gland excision. Zoulek et al. [41] describe a case of a 43-year-old woman who was found to have a rectovaginal fistula immediately after having had a Bartholin gland excision procedure. Care should be taken to close dead space in layers in attempt to prevent this rare but serious complication.

Cysts of the Canal of Nuck

Surgical treatment of a cyst of the canal of Nuck begins with a vertical incision into the labia majora to expose the underlying cyst. If blood vessels are encountered while dissecting in the subcutaneous tissue, they can be suture ligated with 4-0 Vicryl. The cyst is then grasped with Allis clamps and freed using sharp dissection on all sides except the superior attachment. Metzenbaum scissors utilizing the flash technique are used to ensure there is no underlying intestine and subsequently the cyst is entered. A finger is placed into the cyst to palpate the external inguinal

ring. Any peritoneal lining is excised from the cyst, and the external inguinal ring is closed. The superior portion of the cyst is then closed with a purse-string suture and the cyst is excised and freed. The vulva including the skin is then closed in layers with 3-0 Vicryl sutures. If an inguinal hernia is present, an inguinal hernia repair should be performed. Although surgical excision is the treatment of choice, sonographically guided aspiration has been performed for symptom relief. This technique does entail the risk of bowel puncture in the rare instance of intestinal herniation, so special care must be taken [42].

Summary

In conclusion, both vulvar and vaginal cysts are often asymptomatic and discovered on routine gynecological examination. The evaluation of these cysts includes a detailed history, thorough physical examination, and possibly imaging. Asymptomatic cysts can be observed while symptomatic or infected cysts should be actively managed. Patient demographics and cyst characteristics should guide the clinician as to what surgical procedure to employ. Techniques include incision and drainage, marsupialization, and excision. Incision and drainage with epithelialization of the drainage tract as well as marsupialization have good outcomes with low complication and recurrence rates. Definitive treatment is achieved by cyst excision. Clinicians should have a low threshold to biopsy of any concerning or recurrent lesion.

References

1. Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG, Calver LE. Anatomic disorders. In: Williams gynecology. Beijing: McGraw-Hill; 2008. p. 402–3.
2. Laufer MR. Diagnosis and management of congenital anomalies of the vagina. UpToDate.com. UpToDate, Inc. February 2013. Web. March 2013.
3. Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG, Calver LE. Anatomic disorders. In: Williams gynecology. Beijing: McGraw-Hill; 2008. p. 787–8.
4. Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG, Calver LE. Anatomic disorders. In: Williams gynecology. Beijing: McGraw-Hill; 2008. p. 786.
5. Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG, Calver LE. Anatomic disorders. In: Williams gynecology. Beijing: McGraw-Hill; 2008. p. 790.
6. Rahn DD, Bleich AT, Wai CY, Roshanravan SM, Wieslander CK, Schaffer JI, Corton MM. Anatomic relationships of the distal third of the pelvic ureter, trigone, and urethra in unembalmed female cadavers. Am J Obstet Gynecol. 2007;197(6):668.e1–4.
7. DeLancey JO. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol. 1994;170(6):1713–20.
8. Bent AE, Cundiff GW, Swift SE. Anatomy of the pelvic viscera. In: Ostegard's urogynecology and pelvic floor dysfunction. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2008. p. 21.
9. Huffman J. Detailed anatomy of the parautethral ducts in the adult human female. Am J Obstet Gynecol. 1948;55:86–101.
10. Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG, Calver LE. Anatomic disorders. In: Williams gynecology. Beijing: McGraw-Hill; 2008. p. 791.
11. Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG, Calver LE. Anatomic disorders. In: Williams gynecology. Beijing: McGraw-Hill; 2008. p. 796–7.
12. Cabral RJ, Perez GD, Martins SC, Ribeiro SA. Suburethral endometrioma. Actas Urologicas Espanolas. 2007;31(2):153–6.
13. Aboud KA, Hawsawi KA, Ramesh V. Vaginitis emphysematosa. Sex Transm Infect. 2002;78:155.
14. Kondi-Pafiti A, Grapsa D, Papakonstantinou K, Kairi-Vassilatou E, Xasiakos D. Vaginal cysts: a common pathologic entity revisited. Clin Exp Obstet Gynecol. 2008;35(1):41–4.
15. Deppisch LM. Cysts of the vagina: classification and clinical correlations. Obstet Gynecol. 1975;45(6):632–7.
16. Hoffman MS, Roberts WS, LaPolla JP, Sterghos Jr S, Cavanagh D. Neoplasia in vaginal cuff epithelial inclusion cysts after hysterectomy. J Reprod Med. 1981;34(6):412–4.
17. Dywer PL, Rosamilia A. Congenital urogenital anomalies that are associated with the persistence of Gartner's duct: a review. Am J Obstet Gynecol. 2006;195(2):354–9.
18. Bats AS, Metzger U, Le Frere-Belda MA, Brisa M, Lecuru F. Malignant transformation of a Gartner cyst. Int J Gynecol Cancer. 2009;19(9):1655–7.
19. Leder RA, Paulson EK. Vaginitis emphysematosa: CT and review of the literature. Am J Radiol. 2001;176:623–5.
20. Jayaprakash S, Lakshmidevi M, Kumar SG. A rare case of posterior vaginal wall cyst. BMJ Case Reports. 2011. pii: bcr0220113804. doi: 10.1136/bcr.02.2011.3804

21. Lucente V, Benson JT. Vaginal mullerian cyst presenting as an anterior enterocele: a case report. Obstet Gynecol. 1990;76(5 Pt 2):906–8.
22. Lee KS, Park KH, Lee S, Kim JY, Seo SS. Adenocarcinoma arising in a vaginal mullerian cyst: a case report. Gynecol Oncol. 2005;99(3):767–9.
23. Sheih C, Hung C, Wei C, Lin C. Cystic dilations within the pelvis in patients with ipsilateral renal agenesis or dysplasia. J Urol. 1990;144:324–7.
24. Wai CY, Corton MM, Miller M, Sailors J, Schaffer JI. Multiple vaginal wall cysts: diagnosis and surgical management. Obstet Gynecol. 2004;103(5 Pt 2):1099–102.
25. Rouzi AA, Sindi O, Radhan B, Ba'aqeel H. Epidermal clitoral inclusion cyst after type I female genital mutilation. Am J Obstet Gynecol. 2001;185(3):569–71.
26. Ofodile FA, Oluwasanmi JO. Post-circumcision epidermoid inclusion cysts of the clitoris. Plast Reconstr Surg. 1979;63(4):485–6.
27. Lee HE, Yang CH, Chen CH, Hong HS, Kuan YZ. Comparison of the surgical outcomes of punch incision and elliptical excision in treating epidermal inclusion cysts: a prospective, randomized study. Dermatol Surg. 2006;32(4):520–5.
28. Bhide A, Nama V, Patel S, Kalu E. Microbiology of cysts/abscesses of Bartholin's gland: review of empirical antibiotic therapy against microbial culture. J Obstet Gynaecol. 2010;30(7):701–3.
29. Visco AG, Del Priore G. Postmenopausal Bartholin gland enlargement: a hospital-based cancer risk assessment. Obstet Gynecol. 1996;87(2):186–90.
30. Ben-Harosh S, Cohen I, Bornstein J. Bartholin's gland hyperplasia in a young woman. Gynecol Obstet Invest. 2008;65(1):18–20.
31. Lucioni A, Rapp DE, Gong EM, Fedunok P, Bales GT. Diagnosis and management of periurethral cysts. Urol Int. 2007;78(2):121–5.
32. Ozel A, Kirdar O, Halefoglu AM, Erturk SM, Karpat Z, Lo Russo G, Maldur V, Cantisani V. Cysts of the canal of Nuck: ultrasound and magnetic resonance imaging findings. J Ultrasound. 2009;12(3):125–7.
33. Wechter ME, Wu JM, Marzano D, Haefner H. Management of Bartholin duct cysts and abscesses: a systematic review. Obstet Gynecol Surv. 2009;64(6):395–404.
34. New instrument for office treatment of cyst and abscess of Bartholin's gland. JAMA. 1964;190:777–8.
35. Haider Z, Condous G, Kirk E, Mukri F, Bourne T. The simple outpatient management of Bartholin's abscess using the Word catheter: a preliminary study. Aust NZ J Obstet Gynaecol. 2007;47(2):137–40.
36. Mungan T, Uğur M, Yalçin H, Alan S, Sayilgan A. Treatment of Bartholin's cyst and abscess: excision versus silver nitrate insertion. Eur J Obstet Gynecol Reprod Biol. 1995;63(1):61–3.
37. Yüce K, Zeyneloglu HB, Bükülmez O, Kisnisci HA. Outpatient management of Bartholin gland abscesses and cysts with silver nitrate. Aust NZ J Obstet Gynaecol. 1994;34(1):93–6.
38. Lashgari M, Keene M. Excision of Bartholin duct cysts using the CO2 laser. Obstet Gynecol. 1986;67(5):735–7.
39. Figueiredo AC, Duarte PE, Gomes TP, Borrego JM, Marques CA. Bartholin's gland cysts: management with carbon-dioxide laser vaporization. Rev Bras Ginecol Obstet. 2012;34(12):550–4.
40. Kushnir VA, Mosquera C. Novel technique for management of Bartholin gland cysts and abscesses. J Emerg Med. 2009;36(4):388–90.
41. Zoulek E, Karp DR, Davila DW. Rectovaginal fistula as a complication to a Bartholin gland excision. Obstet Gynecol. 2011;118(2 Pt 2):489–91.
42. Stickel WH, Manner M. Female hydrocele (cyst of the canal of Nuck): sonographic appearance of a rare and little-known disorder. J Ultrasound Med. 2004;23(3):429–32.

Transvaginal Closure of Bladder Neck

15

Teresa L. Danforth and David A. Ginsberg

Abbreviations

CIC	Clean intermittent catheterization
BNC	Bladder neck closure
SPT	Suprapubic catheter
TPN	Total parenteral nutrition
SBO	Small bowel obstruction
TV	Transvaginal
TP	Transperineal

Background

Transvaginal closure of the bladder neck in the female patient is indicated for those with a devastated outlet, usually caused by chronic indwelling urethral catheter placement. Many of these patients have a neurogenic etiology for either urinary retention or urinary incontinence. Chronic catheterization leads to urethral erosion and destruction, ultimately resulting in a patulous urethra that cannot be maintained in the bladder. Management is limited as many of these patients are debilitated due to their comorbid conditions. There is often an inadequate amount of residual urethra to allow for placement of a pubovaginal sling, and many of these patients are unwilling or unable to undergo urinary tract reconstruction. Therefore, the best option is often bladder neck closure with suprapubic tube (SPT) placement. Transvaginal closure is an outstanding option that does not require an abdominal incision, making it a viable minimally invasive option; however, vaginal techniques can be technically challenging for inexperienced vaginal surgeons. Primary complications include fistula formation, bladder stones, SPT site leakage or stenosis, and wound infection. Transvaginal closure of the bladder neck carries less morbidity but may require more than one procedure to achieve continence.

Indications for Transvaginal Closure of the Bladder Neck

Transvaginal closure of the bladder neck in a female patient is indicated for the devastated bladder outlet, usually caused by chronic indwelling urethral catheter placement. Chronic urethral catheters are placed for a variety of indications, including urinary incontinence refractory to other treatments and urinary retention. Many patients with indwelling urethral catheters have a neurogenic etiology (i.e., multiple sclerosis, spinal cord injury, spinal dysraphism, or stroke) as the cause of their lower urinary tract dysfunction.

T.L. Danforth, M.D.
Department of Urology, State University of New York at Buffalo, Buffalo, NY, USA
e-mail: danforth@buffalo.edu

D.A. Ginsberg, M.D. (✉)
Department of Urology, University of Southern California, Los Angeles, CA, USA
e-mail: ginsberg@med.usc.edu

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_15,

However, as the population ages and treatment of incontinence in the debilitated aging patient continues to be a challenge, chronic indwelling catheter usage may be seen with increasing frequency in this patient population as well.

The typical clinical situation would be one in which a patient undergoes placement of a urethral catheter as she is unwilling or not medically able to undergo any more invasive form of treatment for urinary incontinence or retention. These patients are generally not optimal candidates for clean intermittent catheterization (CIC) for reasons such as physical debilitation, poor hand function, or simply unwillingness to catheterize. In addition, most skilled nursing facilities are unable to maintain patients on a regular CIC program; indwelling catheter placement is a much easier initial option for both the patient and the nursing staff. Male patients with urinary incontinence have the option of using a condom catheter; unfortunately, there is not a similar alternative for female patients. Chronic indwelling catheters are known to have complications including urinary tract infections, urinary tract calculi, catheter plugging, cellular toxicity, and malignancy [1]. Furthermore, pressure from the catheter, balloon, and poor management of catheter by the patient or ancillary staff all contribute to urethral erosion and destruction (Fig. 15.1) [2]. With urethral erosion, further leakage around the catheter can occur which frequently leads to upsizing of the catheter or the balloon, resulting in even further damage to the urethra over time. The urethra become patulous and catheters are frequently pulled out or cannot be maintained in the bladder leading to a decreased bladder capacity, as the bladder can no longer fill and cycle. The damage can lead to traumatic hypospadias and/or anterior urethral erosion to the level of the pubic symphysis.

Management options for these patients are limited. Unfortunately many of these patients are debilitated secondary to their medical comorbidities and poor nutritional status. The use of pads or diapers can be problematic for management of pressure ulcers and wounds, which are commonplace in this subset of patients. Suprapubic catheters have been successfully used in some patients; however, many patients will still have significant leakage per urethra due to the damage caused by the initial indwelling urethral catheter. Transvaginally placed slings, although theoretically are useful as they allow for continued access to the bladder through the native urethra, do not usually give enough support to achieve continence. In addition, there may not be an adequate amount of residual urethral length to allow for sling placement if the urethral damage is severe enough.

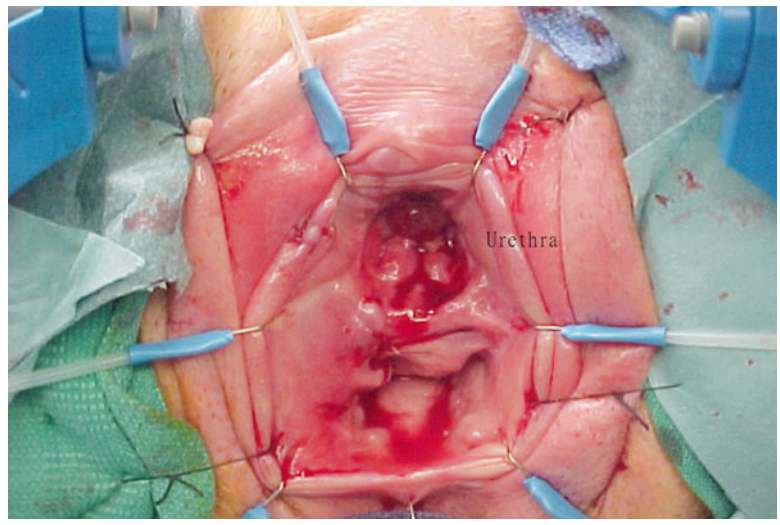


Fig. 15.1 Urethral erosion as demonstrated by a wide, patulous urethra

Reconstruction of the lower urinary with various methods have been described but many patients are not willing or medically appropriate to undergo such procedures. In patients who are willing and able to undergo urinary tract reconstruction, closure of the bladder neck is usually achieved transabdominally at the same time as their reconstruction. This type of closure is more invasive but has been reported to have lower rates of fistula formation postoperatively compared to transvaginal repair.

Transvaginal bladder neck closure (BNC) with SPT placement is reserved for those patients whom are not candidates for more invasive reconstruction. The primary concern with this procedure is fistula formation between the closed bladder neck and the vagina and may be more technically challenging for inexperienced pelvic surgeons.

Preoperative Evaluation

Preoperatively the most important decision is which approach to take in managing the patient's incontinence. Andrews et al. described a series of 57 patients with long-term indwelling catheters of which 39 were managed successfully with SPT alone [1]. Similarly, Eckford et al. reported that 11 of 50 women with multiple sclerosis managed with indwelling catheters were happy with an SPT alone even with a small degree of intermittent leakage from the urethra [3]. It is important to recognize that some patients, depending on the degree of urethral destruction, may have enough improvement in their incontinence with an SPT alone that they may not need further surgical intervention. If this fails due to a poor outlet, then treatment will likely focus on an obstructing sling vs. bladder neck closure. Wantanabe et al. reported that candidates for pubovaginal sling must have an intact bladder neck with 1 cm of proximal urethral tissue in order to obtain effective compression of the urethra, which may or may not be the case in patients with a chronic indwelling catheter [4]. If bladder neck closure is to be done, then the decision between transvaginal versus transabdominal approach needs to be resolved.

When considering abdominal versus vaginal approaches, various factors must be considered: morbidity of the procedure, planned concomitant procedures, prior surgeries, surgeon experience [1, 5]. Certainly avoiding an abdominal incision allows for decreased morbidity, but the transvaginal approach is associated with higher rates of fistula and/or failure [1, 2, 5]. A discussion with the patient regarding the lower morbidity but higher risk of failure and potential need for reoperation must take place to set appropriate expectations. Surgeon experience should also be taken into account, as less experienced vaginal surgeons may not fare as well with this approach. Levy et al. reported on a series of 12 patients, 4 of whom underwent transvaginal closure of the bladder neck alone with a 50 % success rate [5]. The two patients who failed and the subsequent 8 patients underwent combined abdominal and transvaginal approach with 100 % success. Levy suggests that surgeons without significant experience operating vaginally should consider an abdominal approach to achieve higher success. Ginger et al. also reported an 11 % leakage rate in 26 patients undergoing abdominal BNC as opposed to a 100 % leakage rate in 2 patients who underwent transvaginal approach [2].

As already discussed, patients with chronic indwelling catheters are often debilitated and malnourished. Poor preoperative nutrition status is associated with poor wound healing, increased infection rate, higher pulmonary complication rate, prolonged hospitalization, and higher mortality rates [6]. Hebbar et al. reviewed studies looking at the use of total parenteral nutrition (TPN) or enteral feeds preoperatively and the rates of complications. From the VA TPN Cooperative Study which used 7–15 days of preoperative TPN, patients with severe malnourishment were found to have a dramatic drop in complication rate from 42.9 to 5.3 % with the use of TPN; however, pooled data of all patients did not show any significant difference. There was no difference between the use of TPN vs. enteral feeds. Therefore, one could consider using preoperative nutrition in the severely malnourished patient.

Procedure

Various techniques have been described to perform a transvaginal closure of the bladder neck [1–3, 7–10]. The patient is placed in dorsal lithotomy position with adequate exposure to the anterior vaginal wall using labial retraction sutures and/or a self-retaining retractor, with or without a posterior weighted speculum. If an SPT is not already present, one can be placed with various techniques. Eckford et al. describe a two stage technique in which an SPT is placed during the first procedure (percutaneously or open) followed by a second procedure for transvaginal BNC if the patient continues to leak [3]. A Lowsley suprapubic tractor may be used to aid in SPT placement if desired [7].

The anterior vaginal wall is infiltrated with injectable saline to aid in the dissection of the vaginal wall from the urethra and bladder. Two incisions are made along the anterior vaginal wall: one circumferentially around the urethral meatus and the second as an inverted wide-based anterior vaginal wall flap beginning from the urethral meatus extending past the bladder neck (Fig. 15.2a–f) [9]. The urethra is dissected laterally over the periurethral fascia to the retropubic space and off of the urethropelvic ligaments followed by transection of the urethra off of the urethropelvic ligament dorsally to the inferior margin of the pubic symphysis. This allows for complete mobilization of the remaining urethra and bladder neck (Fig. 15.2b). The necrotic urethral tissue is then removed, which may in fact be the entire urethra, thus making bladder neck mobility extremely important. If there is viable urethral tissue, one can utilize a technique described by Rovner et al. in which the anterior urethra is divided toward the bladder neck and the bivalved urethra is rotated in an anterior and cephalad direction and secured to the anterior bladder wall with two layers of absorbable suture (Fig. 15.2d). This rotates the suture line anteriorly, toward the retropubic space and underneath the pubic symphysis, minimizing overlying suture lines during closure of the vaginal wall flap (Fig. 15.2e, f). In addition, if possible, we also try to then secure the sutures used to close the bladder neck to the undersurface of the pubic symphysis, further placing the bladder neck closure anteriorly. Mobilization of the closure upward should minimize the risk of postoperative fistula formation. The vaginal wall flap is closed with absorbable suture and packing is placed.

Nielson et al. describe a technique in which two chromic sutures are passed through the SPT site via the Lowsley tractor and used to tag the edges of the urethral closure [7]. These sutures are then later used to invaginate the urethral mucosa and pull the urethra away from the vaginal closure.

Flaps or graft placements are generally not necessary in primary repair. In cases where the perivesical tissues may be compromised or in patients with history of prior pelvic radiation, one can consider using a Martius flap for interposition [8]. In patients who have failed prior attempts, a combined abdominal and vaginal approach with omental, peritoneal, or Martius flaps have been described [3, 9].

Postoperative Management

Appropriate postoperative management is critical to success of abdominal or transvaginal closure of the bladder neck. Ginger et al. found an association with poor postoperative catheter care and leakage [2]. They found that 13 of 29 patients had documented poor catheter management by their caregivers which included poor securing of the SPT, inadequate catheter irrigation, clogging, kinking, and dislodgement. 7 of 8 patients with persistent leakage postoperatively (6 urethral and 2 around SPT) were associated with poor catheter care. This stresses that not only an adequate catheter size should be used but also that catheters need to be appropriately secured at all times and irrigated if necessary to ensure that it is draining appropriately.

Equally important to adequate catheter drainage is management of detrusor overactivity. Ginger et al. demonstrated detrusor overactivity in 12 of 23 patients preoperatively [2]. Stoffel et al. also showed that 10 of 12 neurogenic patients had poorly compliant bladders

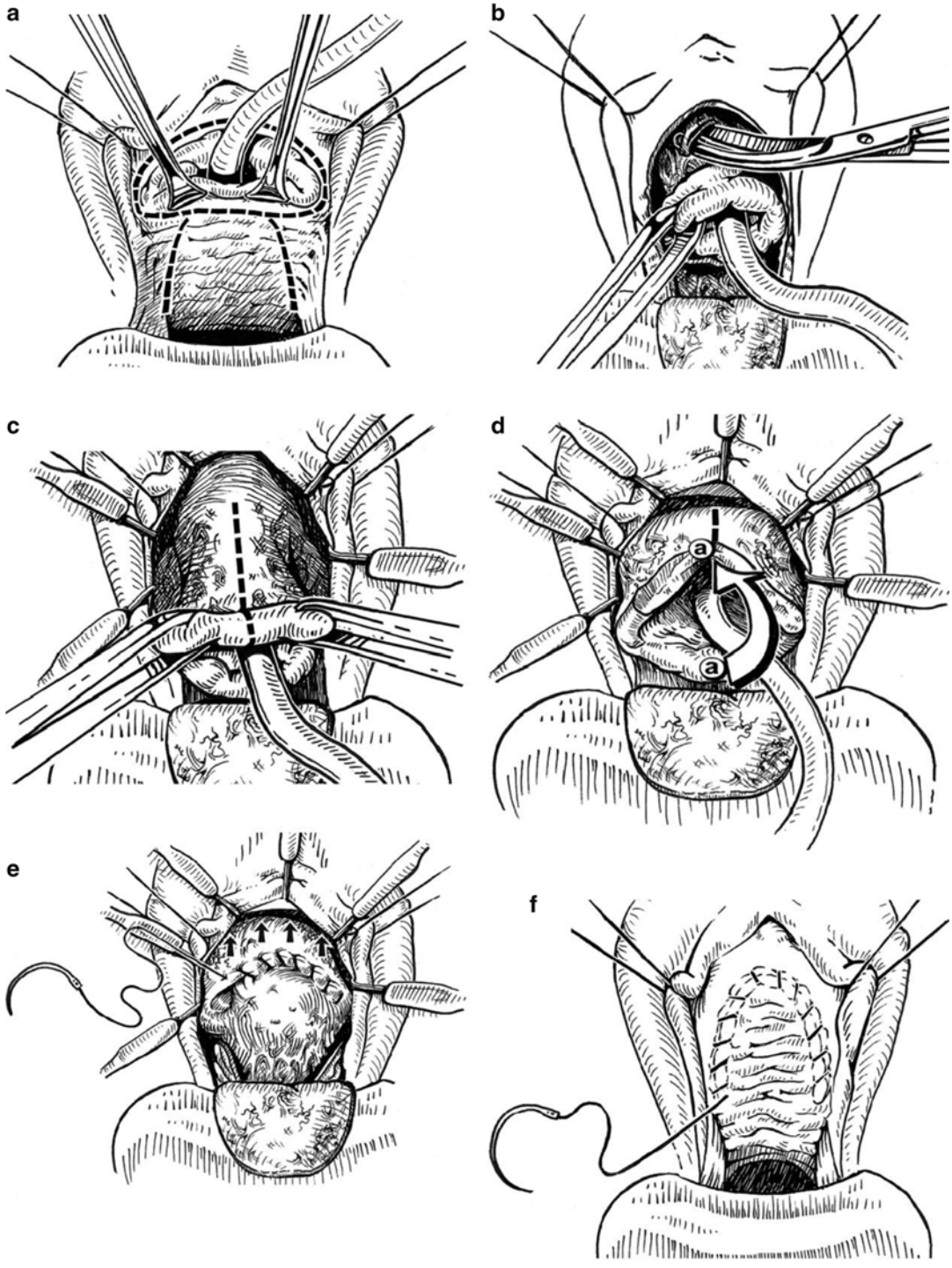


Fig. 15.2 (**a**–**f**) TV BNC with posterior urethral flap. (**a**) Vaginal wall flap developed and dorsal semilunar incision is made above the urethra. (**b**) Dissection continued above urethra into retropubic space. Pubourethral and urethropelvic ligaments taken down, mobilizing urethra and anterior bladder neck. (**c**) Dorsal urethra bivalved and incision carried onto anterior bladder neck for 2–3 cm. (**d**) Dorsally bivalved urethra then rotated cephalad toward anterior bladder wall incision. (**e**) Ventral urethral flap affixed high on anterior bladder wall, such that when bladder rotates into anatomic position, suture line rotates under symphysis pubis. (**f**) Vaginal wall closed as second layer with no overlapping suture lines (All: Used with permission from Rovner ES, Goudelocke CM, Gilchrist A, Lebed B. Transvaginal bladder neck closure with posterior urethral flap for devastated urethra. Urology 2011;78(1):208-212)

on fluorourodynamics prior to BNC with urinary diversion [10]. Although an SPT should allow for continuous bladder drainage, physiologically an open bladder neck prompts a bladder contraction. Higher pressures in the bladder put stress on the suture line, thus increasing the risk of postoperative fistula formation. Administration of anticholinergics will potentially decrease this risk and should be used postoperatively (or continued if patient is already on them) [11]. The use of onabotulinumtoxinA at the time of BNC has not yet been studied, but presumably would allow for lower bladder pressures and higher success rates.

There are various complications associated with transvaginal closure of the bladder neck, the most important of which is continued leakage of urine and development of a vesicovaginal fistula. A postoperative cystogram should be performed 2–3 weeks following the procedure to assess the integrity of the repair. If a leak is suspected but cannot be identified on cystogram, often it can be identified on examination. Methylene blue or indigo carmine irrigation in the bladder can be used to easily identify the site of leakage if it cannot be identified.

If there is a leak, continued catheter drainage can be attempted with the hopes of eventual closure. No study has looked at conservative management of leakage after an attempted transvaginal BNC. In uretero-ileal anastomosis or abdominal BNC with fistula, it has been suggested that conservative management with continued drainage for several weeks with frequent catheter irrigations and placement of bilateral percutaneous nephrostomy tubes may lead to resolution of a fistula [11]. If a patient has a leak on cystogram with bladder filling, but is clinically dry, these patients can usually be managed with regular SPT changes monthly and do not require further intervention unless the fistula progresses and becomes clinically relevant. For a matured fistula tract, repeat surgery may be considered. The choice of transvaginal versus transabdominal versus combined approach is at the discretion of the surgeon. Those surgeons with more vaginal experience may consider a repeat transvaginal approach with or without a Martius flap. Less experienced vaginal surgeons may consider an abdominal approach or combined abdominal and transvaginal approach. If the repair is not salvageable, more invasive measures for urinary diversion may be considered including cystectomy with continent or incontinent diversion.

Complications

Fistula rates for transvaginal BNC are variable, ranging from 0 to 100 % (Table 15.1) [1–3, 5, 7, 9–12]. After revision, fistula rates drop to 0–25 % [1, 3, 9–11]. Abdominal BNC fistula rates are notably lower with both primary repair (0–18 %) and after revision (0–6 %) [1, 2, 10, 11, 13, 14].

Other complications of transvaginal BNC surgery include bladder stones, SPT site leakage, SPT site stenosis, leakage around SPT, and wound infection. If associated bladder augmentation procedures are performed, further complications can arise including intestinal fistulae, stomal stenosis, small bowel obstruction (SBO), and poor bladder compliance.

Summary

Overall, transvaginal closure of the bladder neck is well tolerated and carries less morbidity than an abdominal approach; however, patients may require more than one procedure to achieve continence.

Table 15.1 Comparison of complication rates of transvaginal and transabdominal BNC

Author	Approach (number of patients)	Follow-up	Complications	Fistula rate
Zimmern et al. [12]	Transvaginal (6)			TV 0 %
Nielsen et al. [7]	Transvaginal (5)	35 months (10–78)	SPT site stenosis 20 %	TV 20 %
Eckford et al. [3]	Transvaginal (50)	6.5 years (2–17)[a]	Bladder stones 42 % Catheter blockage 79 % Redo SPT 11 %	TV 22 % 8 % after 2nd revision
Levy et al. [5]	Transvaginal (4) Combined (10)[b]	15.6 months (6–40)		TV 50 % Combined 0 %
Andrews et al. [1]	SPT (39) Urethral Recon (6) Transvaginal (8) Abdominal with augment(4)	4.6 years (0.5–9.5)	Bladder stones 6 % SPT site leakage 6 %	Urethral Recon 33 % TV 50 % 25 % after revision Abdominal 0 %
Shpall et al. [11]	Abdominal with augment (39)	36.9 months (7–173)	Stomal Dysfunction 15 % Wound Infection 3 % Retained sponge 3 % Stones 13 %	Abdominal 15% 3 % after revision[c]
O'Connor et al. [14]	Abdominal with SPT (15) Abdominal with Continent Stoma (12) Abdominal with Ileovesicostomy (5)	78.6 months (12–164)	Vesicocutaneous fistula 6 % Enterocutaneous fistula 3 % SBO 3 % Stomal stenosis 9 % Bladder stone 6 %	Abdominal 17 % 6 % after revision
Stoffel et al. [10]	Transperineal (4)[d] Transvaginal (8)[d]	20 months (9.2–27)	Poor bladder compliance 8 %	75 % (all patients 1 procedure) TV 12.5 % after revision TP 0 % after revision
Spahn et al. [13]	Abdominal with continent diversion (17)	68 months (12–129)	Vesico-intestino-cutaneous fistula 6 % Stomal Stenosis 24 % Stones 12 %	Abdominal 18 % 0 % after revision
Ginger et al. [2]	Transvaginal (2) Abdominal (26) Perineal (1)	38.2 months (0.9–104)	SPT dysfunction 24 % Wound infection 21 % SPT leakage 3 % SBO 3 % Urosepsis 3 % Stones 10 %	TV 100 % Abdominal 12 % Perineal 100 %
Rovner et al. [9]	Transvaginal (11)	9.6 months (1–36)	Cellulitis 9 %	9 % 0 % after revision

[a]Only 19 patients had data for review
[b]2 patients were failed transvaginal BNC
[c]One patient had spontaneous resolution of fistula
[d]Patients underwent concomitant bladder augmentation

References

1. Andrews HO, Shah PJR. Surgical management of urethral damage in neurologically impaired female patients with chronic indwelling catheters. BJU. 1998;82:820–4.
2. Ginger VAT, Miller JL, Young CC. Bladder neck closure and suprapubic tube placement in a debilitated patient population. Neurourol Urodyn. 2010;29:382–6.
3. Eckford SB, Kohler-Ockmore J, Feneley RCL. Long-term follow-up of transvaginal urethral closure and suprapubic cystostomy for urinary incontinence in women with multiple sclerosis. BJU. 1994;74:319–21.
4. Wantanabe T, Rivas DA, Smith R, et al. The effect of urinary tract reconstruction on neurologically impaired women previously treated with an indwelling urethral catheter. J Urol. 1996;156:1926–8.
5. Levy JB, Jacobs JA, Wein AJ. Combined abdominal and vaginal approach for bladder neck closure and permanent surprapubic tube: urinary diversion in the neurologically impaired woman. J Urol. 1994;152:2081–2.
6. Hebbar R, Harte B. Do preoperative nutritional interventions improve outcomes in malnourished patients undergoing elective surgery? Cleve Clin J Med. 2007;74 suppl 1:S8–10.
7. Nielson KT, Bruskewitz RC. Female urinary incontinence treated by transvaginal urethral closure and suprapubic catheter. Int Urol Nephrol. 1989;21:603–8.
8. Rosenblum N, Nitti VW. Female urethral reconstruction. Urol Clin N Am. 2011;38:55–64.
9. Rovner ES, Goudelocke CM, Gilchrist A, Lebed B. Transvaginal bladder neck closure with posterior urethral flap for devastated urethra. Urology. 2011;78(1):208–12.
10. Stoffel JT, McGuire EJ. Outcome of urethral closure in patients with neurologic impairment and complete urethral destruction. Neurourol Urodyn. 2006;25:19–22.
11. Shpall AI, Ginsberg DA. Bladder neck closure with lower urinary tract reconstruction: technique and long-term follow-up. J Urol. 2004;172:2296–9.
12. Zimmern PE, Hadley RH, Leach GE, Raz S. Transvaginal closure of the bladder neck and placement of a suprapubic catheter for destroyed urethra after long-term indwelling catheterization. J Urol. 1985;134(3):554–7.
13. Spahn M, Kocot A, Loeser A, Kneitz B, Riedmiller H. Last resort in devastated bladder outlet: bladder neck closure and continent vesicostomy – long-term results and comparison of different techniques. Urology. 2010;75(5):1185–92.
14. O'Connor RC, Stapp EC, Donnellan SM, Hovey RM, TSE VWM, Stone AR. Long-term results of suprapubic bladder neck closure for treatment of the devastated outlet. Urology. 2005;66(2):311–15.

Surgical Therapy for Fecal Incontinence

16

Kelly A. Garrett

Introduction

Fecal incontinence is defined as the involuntary passage or the inability to control the elimination of stool or fecal material from the anus [1]. Incontinence can be characterized as being passive—the involuntary discharge of stool or gas without awareness, urge—the discharge of stool in spite of active attempts to retain bowel contents, or seepage—the leakage of stool following an otherwise normal evacuation. In addition, symptoms can range from mild leakage to complete loss of control of both liquid and solid stool. Nevertheless, this problem can be socially devastating and can have significant emotional and psychological impact on quality of life. Fecal incontinence is one of the most common causes of institutionalization in the elderly and it accounts for significant expense. There is limited information regarding the economic burden of this disease and the total costs remain difficult to measure. In a study following 63 patients with fecal incontinence, it was estimated that the average lifetime cost associated with treatment and follow-up was $17,166 per patient in 1996 with average facility charges associated with sphincteroplasty to be $8,555 per procedure [2].

The prevalence of fecal incontinence is difficult to estimate, as it is frequently underreported due to embarrassment and reluctance of patients to discuss symptoms with their physicians. In a recent study, more than two-thirds of women with symptoms of incontinence had never discussed their condition with a physician. The lack of care-seeking for this symptom was hindered by embarrassment, perception that symptoms are a normal part of aging, development of personal coping skills, and the perception that there is no treatment available, among other reasons [3]. Nonetheless, quoted prevalence rates vary from 1.4 % to 19 % with higher rates in nursing home residents, parous females, patients with cognitive impairment or neurologic disorders, and the elderly [3–6]. Even though it is primarily a problem in the elderly population, younger groups are affected as well. Obstetric factors can be implicated in this latter group as the incidence of temporary or permanent fecal incontinence after vaginal delivery can reach 3 % or more [7]. This population, however, is complex because although we know that anal sphincter injury is an important factor, it has been shown that mode of delivery does not affect the prevalence of fecal incontinence [8].

Although it is difficult to estimate the exact incidence and prevalence of this condition, we know that the causes are many times multifactorial. Continence depends on many elements such

K.A. Garrett, M.D., F.A.C.S., F.A.S.C.R.S. (✉)
Department of General Surgery, Division of Colon and Rectal Surgery, Weill Cornell Medical College/ New York Presbyterian Hospital, New York, NY, USA
e-mail: Keg9034@med.cornell.edu

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_16,

as colonic transit, stool consistency, rectal reservoir function, anorectal sensation, muscle innervation, and internal and external sphincter muscle function. Interfering with one or more of these factors can lead to issues with incontinence.

Etiology

Obstetric Injury

Obstetric injury is the most commonly cited cause of incontinence in females [9]. At 3–6 months after delivery, as many as 13 to 25 % of women report fecal incontinence [10, 11]. However, the prevalence falls to 1 to 6 percent by 12 months postpartum [12, 13]. Sphincteric injury is clinically recognized in approximately 10 % of all vaginal deliveries but many other women may have unrecognized damage to the sphincter. Risk factors for sphincteric disruption include forceps delivery, occipitoposterior position, a prolonged second stage of labor, mediolateral episiotomy, and primiparity [14–16]. Additionally, as touched upon previously, women who give birth vaginally and do not suffer a sphincter laceration, and even those who undergo cesarean delivery, may also develop fecal incontinence [8, 17]. This may be related to pelvic floor denervation resulting from compression or traction injury to the pudendal nerves.

Congenital

Anorectal malformations represent a spectrum of defects that are characterized by absence of an external anal orifice. They are categorized as being low (perineal fistula, vestibular fistula) or high (rectourethral fistula, rectovesical fistula, anal atresia without fistula, rectal atresia, or persistent cloaca). Anorectal malformations occur in approximately 1 in 5,000 live births. Operative procedures depend on the type of deformity but the goal is to create a perineal opening with adequate sensory and motor control [18]. Even with adequate surgical repair, it is well known that these patients have many issues with pelvic floor dysfunction characterized by constipation and fecal incontinence as well as urinary and sexual dysfunction [19, 20]. Reported rates of incontinence vary, but in a large retrospective study from Germany, complete continence was found in only 27 % of patients and 74 % of patients had some degree of soiling. Only approximately 50 % of this cohort followed a bowel management program consisting of enemas, suppositories, and/or anal plugs and still more than 80 % of these patients had persistent soiling [20].

Iatrogenic

Fecal incontinence is a common sequelae of anorectal surgery. The most common procedures to cause symptoms of incontinence are those for anal fissure and for fistula-in-ano. Although both of these procedures involve cutting some degree of sphincter muscle, the mere use of an anal retractor can cause damage to the internal sphincter muscle with resultant postoperative seepage or leakage of stool.

The theory behind treatment for anal fissure is reduction of elevated sphincter tone. The first line of treatment is usually medical treatment such as topical nitroglycerin, topical calcium channel blocker, or botulinum toxin injection. When conservative treatment fails, surgical treatment is usually indicated. The most common surgical procedure to treat this condition is lateral transection of the internal sphincter muscle or lateral internal sphincterotomy. This procedure is highly effective for treatment of anal fissure but fecal incontinence is a reported complication. In a study from Brazil, it was noted that the rates of incontinence were decreased depending on the amount of internal sphincter muscle that was divided. When less than 25 % (<1 cm) of the sphincter muscle was divided, there were no patients that suffered from postoperative fecal incontinence [21].

Perianal infections or abscesses are one of the most common benign anorectal disorders treated by colon and rectal surgeons. Of all patients who present with an initial perianal abscess, up to one-third will develop a chronic or recurrent anal

fistula [22]. Although the principal goal is to eradicate the fistula and minimize the risk of recurrence, it is also important to preserve continence. There are many different surgical procedures available to treat anal fistulas. The most effective procedure is fistulotomy which entails division of a variable degree of anal sphincter muscle. Although the success rate for this procedure can approximate 90 %, postoperative incontinence has been noted in up to 40 % of patients. Patients who are predisposed to incontinence include those with baseline incontinence, patients with a history of anal operations, women with anterior based fistulas, and patients with high-tracts involving a significant amount of sphincter muscles [23–25].

Procedures other than anorectal surgery can result in incontinence. Although the vast majority of patients with rectal cancer can now be treated with sphincter sparing procedures, there is still frequently postoperative compromise of anorectal function. While sphincter function may be preserved, capacity of the neo-rectum, maximum tolerable volume, and rectal compliance may be reduced resulting in an increased stool frequently and episodes of incontinence. Postoperative continence is even poorer if treatment with radiation and chemotherapy is used [26].

Neurogenic

Denervation of the pelvic floor muscles, specifically the puborectalis and the external anal sphincter, has been described in up to 80 % of patients with idiopathic fecal incontinence. Descending perineal syndrome has been implicated in this denervation. Similar to the mechanism causing postpartum pudendal neuropathy, chronic straining for stool can also cause traction injury to the perineal branches of the pudendal nerve. A vicious cycle then results in further weakness of the pelvic floor and the subsequent need for more straining. This theoretically leads to denervation causing incontinence [7, 27].

Spinal cord injuries and neurologic conditions can also cause incontinence. The pathophysiology leading to incontinence in these patients is complex. Colonic transit time is prolonged leading to constipation and often fecal impaction. The ability to voluntarily contract or relax the external anal sphincter is absent or reduced while the function of the internal sphincter muscle is normal. When the rectum is full the internal sphincter will relax; however, the patient may be unable to completely relax the external anal sphincter. This may contribute to constipation and impaction. This, in combination with an intact rectoanal inhibitory reflex (RAIR) leads to leakage of liquid stool around hard impacted stool in the rectum and incontinence [28].

Rectal Prolapse

Rectal prolapse can be associated with constipation or incontinence. Approximately 50–75 % of patients with rectal prolapse report fecal incontinence [29]. The pathophysiology causing incontinence is multifactorial. The prolapsed rectum causes chronic stretching of the anal sphincter muscles, inhibition of the internal anal sphincter muscle due to constant stimulation of the RAIR, mechanical disruption of the sphincter, impairment of anorectal sensation, and denervation of the pelvic floor muscles [7]. Improvement of continence after surgical correction of prolapse occurs in approximately two-thirds of patients [29, 30].

Assessment

As with any diagnosis, a proper and complete history and physical examination is necessary. In the case of fecal incontinence, concentration is mainly toward the perineal exam. Patients are examined in the left lateral decubitus or the prone jackknife position. First external inspection is performed. Observation should be made if the patient wears a pad, what is on the pad, and if there is stool externally on the skin. Documenting the presence of previous surgical scars or evidence of a previous obstetric injury is necessary. Inspection should be made for fistulous openings or any other significant deformities.

Notation should be made if the anus is patulous or open when the buttocks are separated. With the patient bearing down, the physician should inspect for hemorrhoidal, mucosal, or full thickness rectal prolapse. While straining, if the perineum balloons down, this indicates weakness of the pelvic floor or in more severe cases descending perineum syndrome.

Digital rectal exam should assess resting anal sphincter tone which is a function of the internal sphincter. With the finger in the rectum, the patient should be asked to squeeze simulating their ability to hold in a bowel movement. Assessment can be made if the squeeze is normal, decreased, poor, or absent which will determine external sphincter function. The examiner can feel the amount and consistency of stool in the rectum or if the patient is impacted with hard stool. Digital exam may reveal a rectocele by pushing the anterior wall of the rectum anteriorly and downward into the vagina. By performing a bimanual exam with a finger in the vagina and the rectum, the thickness of rectovaginal septum can be evaluated. By asking the patient to squeeze and then bear down, one can determine for the presence of anismus or paradoxical contraction.

Anoscopy, proctoscopy, or flexible sigmoidoscopy can be performed in the office to look for inflammation or proctitis. This can explain symptoms of diarrhea or significant mucus production. Other pathologies can cause significant mucus production such as a solitary rectal ulcer which can frequently be found in patients with rectal prolapse or internal intussusception or large villous adenomas. Findings during physical examination should be described and recorded properly. Other studies can be ordered or added as adjuncts to physical examination on an as needed basis.

Physiologic Testing

Anorectal Manometry

Anorectal manometry provides an objective assessment of anal sphincter resting and squeeze pressures as well as an evaluation of rectal sensation, rectoanal reflexes, and rectal compliance. There are different types of systems available including a water-perfused probe with multiple closely spaced sensors or a solid-state probe with micro-transducers. The latter tend to be easier to calibrate and possibly more accurate [31–33]. Although manometry gives a reliable, reproducible, and objective assessment of anal sphincter function, the findings do not consistently correlate with severity of fecal incontinence. Anal pressures in normal individuals have a large range and vary with age and gender. Patients with low values may be continent whereas high pressures do not guarantee continence. Nevertheless, the test may influence management decisions, but it may not reliably predict postoperative results.

Pudendal Nerve Terminal Motor Latency

Pudendal Nerve Terminal Motor Latency (PNTML) measurement is an assessment of pudendal nerve function. This test can be performed in conjunction with anal manometry and specifically measures neuromuscular integrity between the terminal portion of the pudendal nerve and the anal sphincter (Fig. 16.1) [1].

This test employs a disposable electrode that is placed around the gloved fingertip and inserted into the rectum (Fig. 16.2). Transrectal stimulation of the pudendal nerve is performed while measuring the time from electrical stimulus of the pudendal nerve to the onset of the electrical response in the muscles of the pelvic floor (Fig. 16.3). Prolonged PNTML indicates pudendal neuropathy. Unfortunately normal latencies do not exclude nerve injury as only the fastest remaining conducting fibers are recorded [34]. In addition, there can be anatomic overlap of the pudendal innervation on both sides of the external anal sphincter [35].

Endorectal Ultrasound

In women with suspected obstetrical injury or patients who have a history of anorectal procedures, endorectal ultrasound is a simple test for

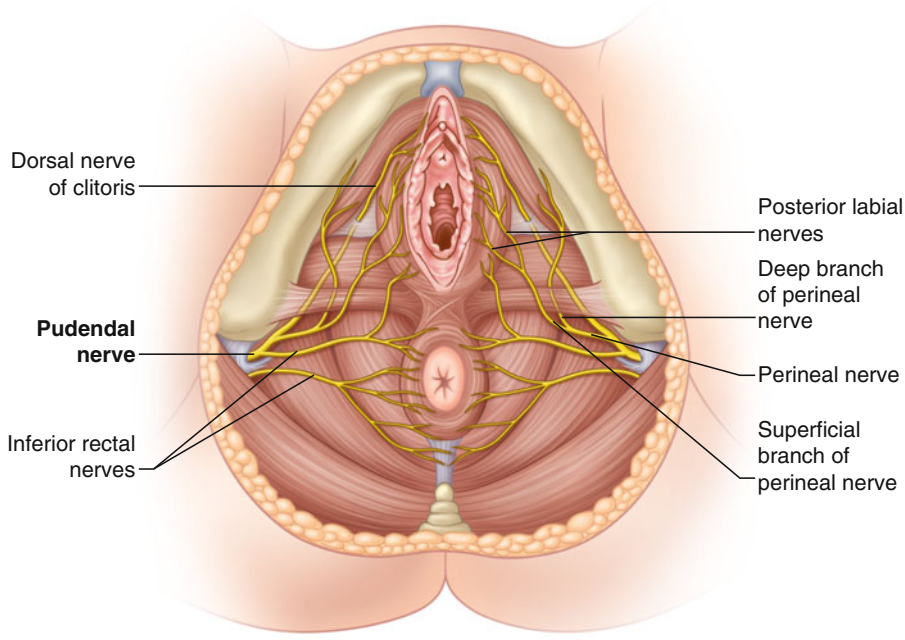


Fig. 16.1 Anatomy of pudendal nerve

defining defects in the internal and external anal sphincter muscles. The most frequently used instruments have a 360° rotating transducer and work with 7 or 10 MHz. More recently three dimensional probes have become popular. Both sphincters can be visualized and length and width can be determined. Atrophy, scar tissue, and defects in the sphincters can also be seen [18] (Fig. 16.4). This technique, similar to ultrasound in other areas of the body, is operator dependent and requires training and experience. However, when performed by an experienced clinician, this test approaches 100 % sensitivity and specificity in identifying sphincter defects [36–38].

Fig. 16.2 Probe used for pudendal nerve terminal motor latency test (*Source*: http://www.glowm.com/section_view/heading/Neurophysiologic%20Testing%20of%20the%20Pelvic%20Floor/item/57. Used with permission)

Defecography

Defecography can be performed under fluoroscopy or using Magnetic Resonance Imaging (MRI). Both techniques involve filling the rectum with either a barium paste in the case of fluoroscopic imaging or ultrasound gel in the case of MRI. Static images at rest and during squeezing and pushing allow measurement of the anorectal angle (Fig. 16.5a, b), perineal descent, and anal canal length. It has been demonstrated that the anorectal angle is increased in pelvic floor

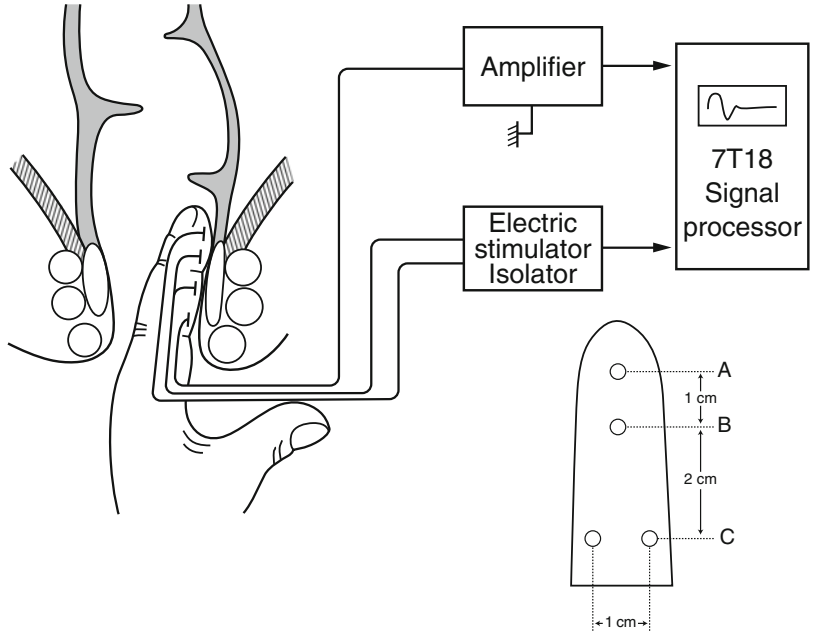


Fig. 16.3 Schematic representation of the system for measuring the pudendal nerve terminal motor latency (PNTML). Latency of the evoked muscle action responses in external anal sphincter (EAS) muscles is recorded after stimulation of both right-sided and left-sided pudendal nerve at the point of ischial spines. A. Stimulating anode. B. Stimulating cathode. C. Ground electrode. D. Recording electrodes (Used with permission from Tomita R, Igarashi S, Ikeda T, Koshinaga T, Fujisaki S, Tanjoh K. Pudendal Nerve Terminal Motor Latency in Patients with or without Soiling 5 years or more after Low Anterior Resection for Lower Rectal Cancer. World Journal of Surgery. 2007; 31(2): 403–408)

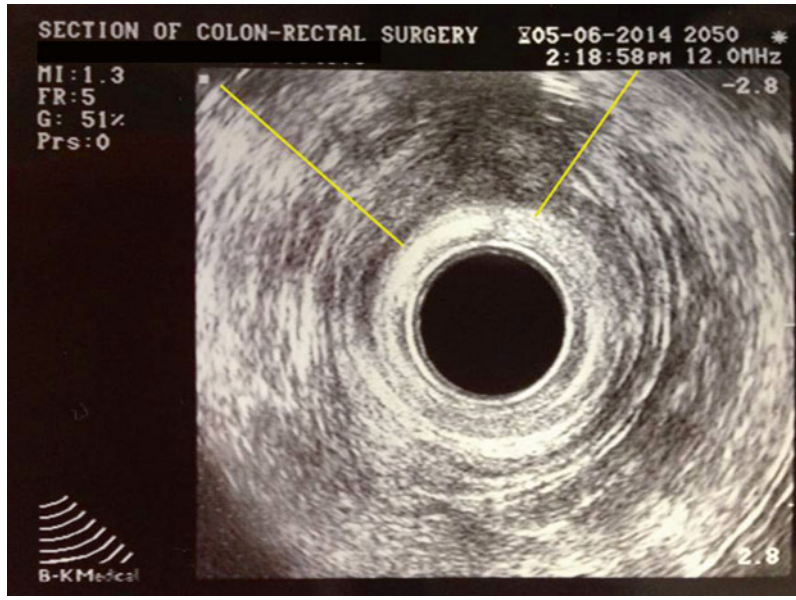


Fig. 16.4 Endorectal ultrasound demonstrating an external sphincter defect

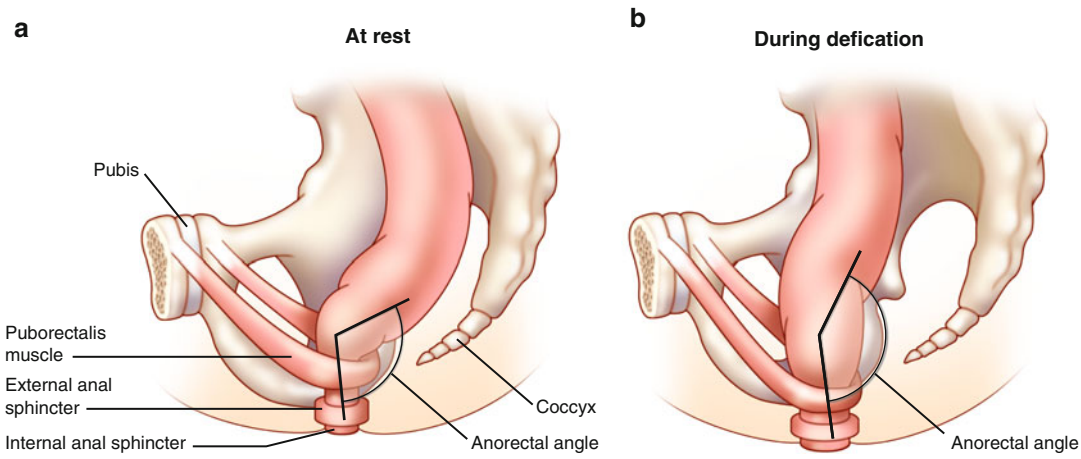


Fig. 16.5 (**a**, **b**) Normal anorectal angle at rest (**a**) and with straining (**b**)

denervation as a sign of pelvic floor weakness. However there is wide interobserver variation in the measurement of the anorectal angle which perhaps makes quantification of limited clinical value [18]. Rectal intussusception, full thickness prolapse, rectoceles, and enteroceles can also be observed. Fluoroscopic defecography tends to be a better test in some cases since the patients are sitting up in the actual position in which one normally defecates, whereas during MR defecography the patient is laying supine and it is often difficult to evacuate the gel in this non-physiologic position. In addition, although both tests can detect a number of abnormalities, these abnormalities can also be seen in otherwise asymptomatic individuals and their presence often correlates poorly with impaired evacuation [39, 40].

Treatment

Medical

After a complete history and physical examination with the addition of necessary physiologic tests, supportive measures are frequently the first approach. It is recommended for patients to keep a bowel and food diary to try and identify offending agents. For patients with diarrheal stool, one would have patients cut lactose and dairy out of the diet to evaluate for possible triggers. Trying to promote a regular ritualized bowel habit is also important. Often times, patients will not empty their rectum completely and residual stool in the rectum may seep or leak out. In these cases, bowel management programs and a regular enema may be useful to promote more complete evacuation. This type of regimen is especially helpful in patients with spinal cord injuries. In patients with loose or segmented stools, a fiber supplement is often recommended. Fiber helps to bulk the stool and promote complete emptying all at once as opposed to having to go back and forth to the bathroom several times. Unfortunately, fiber supplements can potentially worsen diarrhea by increasing colonic fermentation.

For patients with liquid or even mushy stools, Loperamide (Imodium®—McNeil Consumer & Specialty Pharmaceuticals, Fort Washington, PA) and diphenoxylate/atropine (Lomotil®—Pfizer, New York, NY) can produce modest improvement in symptoms related to fecal incontinence. A placebo controlled study of loperamide 4 mg TID has been shown to reduce the frequency of incontinence, improve stool urgency, increase colonic transit time, reduce stool weight, and interestingly, increase anal resting sphincter

Table 16.1 Classification of antidiarrheal medications

Category	Mechanism of action	Medication
Adsorbents		
Fiber supplements	Adsorb water Reduce fecal water content Increase consistency of stool	Psyllium husk (Metamucil®) Methylcellulose (Citrucel®) Guar gum Calcium polycarbophil (FiberCon®) Wheat dextrin (Benefiber®)
Bile acid sequestrant	Forms insoluble complexes with bile acid Makes bile acids osmotically inactive	Cholestyramine (Questran®)
Antispasmodics	Decreases motility Slows passage of stool Allows more time for salt and water to be absorbed	Opiods (Codeine sulfate) Diphenoxylate/atropine (Lomotil®) Diphenoxin/atropine (Motofen®) Loperamide (Imodium®)
	Inhibits hormonal secretion Decreases motility Decreases secretion	Octreotide acetate (Sandostatin®)
Anti-inflammatory	Stops expulsion of fluid into the bowel lumen by coating the mucosa Reduces inflammation/irritation of the intestinal mucosa Antibacterial	Bismuth subsalicylate (Pepto-Bismol® and Kaopectate®)

pressure [41–43]. Other medications that can be used are Codeine sulfate, which can cause drowsiness and addiction, or Cholestyramine (Questran®—Par Pharmaceuticals Inc., Spring Valley, NJ), which is a bile acid binding agent (Table 16.1).

Biofeedback

Behavioral therapy using "operant conditioning" techniques has been shown to improve bowel function and incontinence [44]. The main principle is that patients acquire new and better behaviors through a process of trial and error. The goals of biofeedback are to improve the strength of the anal sphincter muscles, improve the coordination between the abdominal, gluteal, and anal sphincter muscles, and enhance the anorectal sensory perception [1]. The benefit is variable, but improvement in as much as 64–89 % of patients has been reported [45, 46]. Careful selection of patients is crucial and includes factors such as motivation, ability to understand instruction, some rectal sensation preservation, and ability to contract the external anal sphincter voluntarily [47].

Anal Plugs

The anal plug enables controlled evacuation and helps reduce skin complications by temporarily occluding the anal canal. The plug is attached to the perineum using tape and can easily be retrieved. It is effective in controlling incontinence in a minority of patients who can tolerate its use [14] (Fig. 16.6).

Surgical Modalities

Surgery should be considered in selected highly symptomatic patients who have failed conservative measures.

Anal Encirclement Procedures

Anal encirclement was originally described by Thiersch in 1891 for the treatment of complete rectal prolapse. This was later adopted for the

Fig. 16.6 Anal plug (Courtesy of Coloplast, Minneapolis, MN)

treatment of fecal incontinence. A variety of materials have been used for this procedure including nylon, silk, strips of fascia, silver wire, silastic bands, and bioabsorbable materials [18, 48] (Fig. 16.7a–d). The goal of the procedure is to create a rigid barrier to the passage of stool. In general, the perioperative morbidity rate is high with a variety of complications described including fecal impaction, infection, breakage of the encircled material, or erosion through the skin [14, 49]. This procedure has largely been abandoned because of poor results and high postoperative complication rate.

Radiofrequency

Radiofrequency energy or the Secca® procedure (Curon Medical Inc., Sunnyvale, CA) uses heat generated by a high-frequency alternating current that flows from four electrodes causing frictional movements of ions and tissue heating. This procedure is done under sedation and local anesthetic. The device is placed under direct vision into the anal canal and needles are deployed into the tissue and into the sphincter muscles (Figs. 16.8a, b and 16.9). The generator then delivers energy (465 kHz, 2–5 W) at each needle electrode for 90 s or until the temperature reaches 85 °C. The mucosa is constantly cooled by chilled water at the base of each needle. There is constant temperature monitoring and feedback to control the amount of energy delivered to tissue. The therapeutic goal is to create thermal lesions or a controlled scar in the muscle while preserving mucosal integrity. There are variable results in the literature. In a study by Ruiz et al., of 24 patients who underwent the procedure, 16 were available for follow-up. The mean treatment time was 46 min and the number of radiofrequency lesions in the anal canal varied from 31 to 80. Four patients (25 %) experienced minor complications including bleeding, diarrhea, and constipation. Four patients (25 %) had worsening of their incontinence and 2 patients (12.5 %) had no improvement. Overall, 10 of 16 patients (62.5 %) had improvement but still had moderate incontinence at 1 year follow-up [50]. The exact mechanism of this procedure is not known. No consistent changes in anal manometry or anorectal ultrasound have been reported [51–53]. More studies are needed to determine which patients would benefit from this minimally invasive treatment.

Bulking Agents

Injection of bulking agents has emerged as a new treatment for fecal incontinence following success that has been reported in treating urinary incontinence. Many different injectable materials have been used including autologous fat, Teflon,

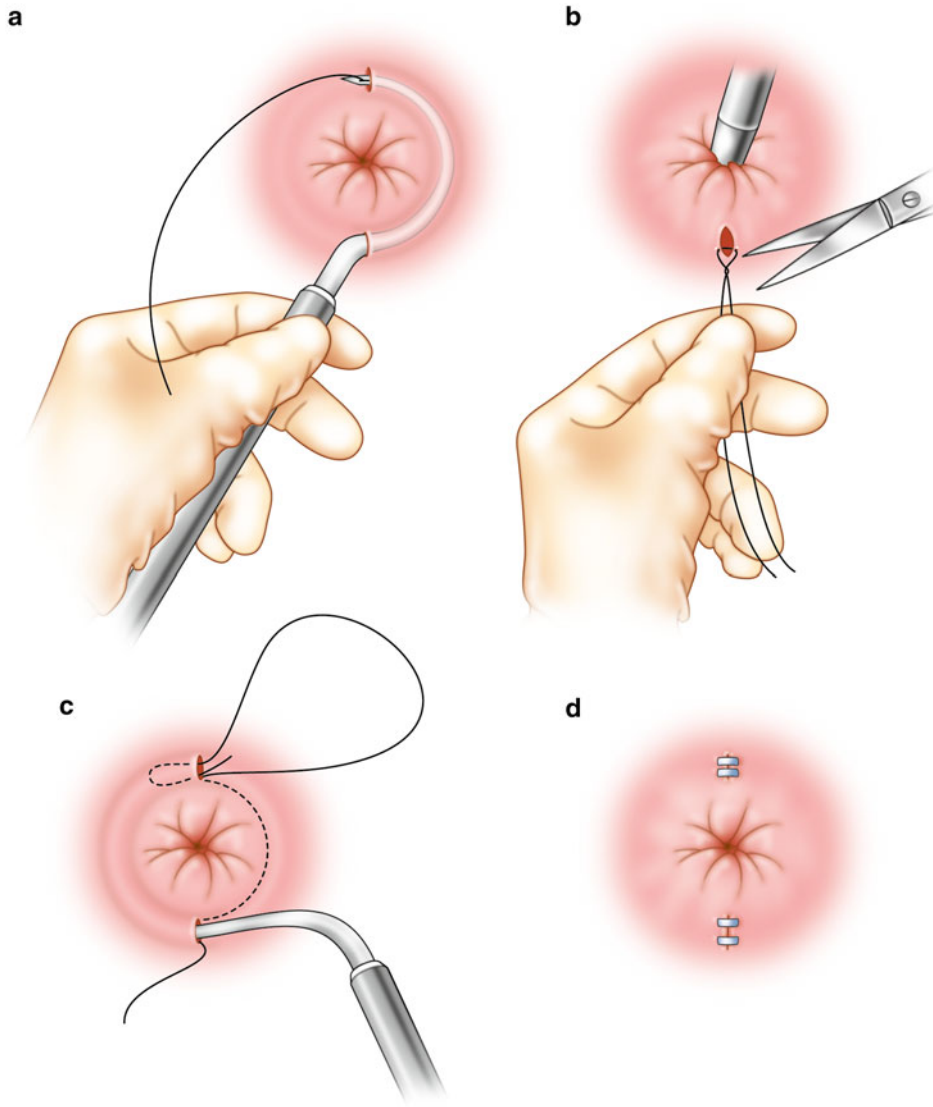


Fig. 16.7 (**a**–**d**) Anal encirclement (Thiersch) procedure

bovine glutaraldehyde cross-linked collagen, carbon-coated zirconium beads (Durasphere®), polydimethylsiloxane elastomer, dextronamer in nonanimal stabilized hyaluronic acid (NASHA™ Dx), hydrogel cross-linked with polyacrylamide (Bulkamid), porcine dermal collagen (Permacol), silicone biomateraial (PTQ™), synthetic calcium hydroxylapatite ceramic microspheres, and polyacrylonitrile in cylinder form. These materials can be injected in different ways including through the perianal skin into the intersphincteric space or through the anal mucosa into the submucosa. Injection can be guided digitally or can be done under ultrasound guidance [54].

The goal of injection is to bulk up the tissue inside the anal canal in order to approximate the anal mucosa. In doing so, this should close the anal canal or raise the pressure inside the anal

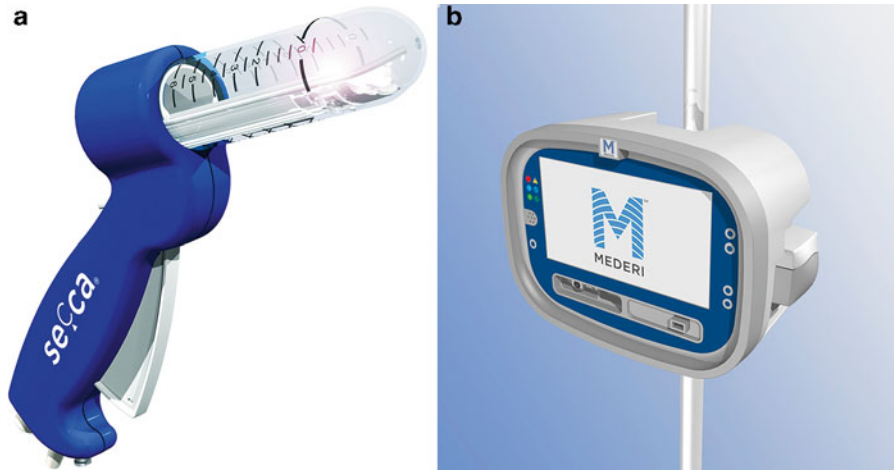


Fig. 16.8 (**a**, **b**) Secca® Radiofrequency device (Courtesy of Mederi Therapeutics, Norwalk, CT; ©2014 Mederi Therapeutics, Inc.)

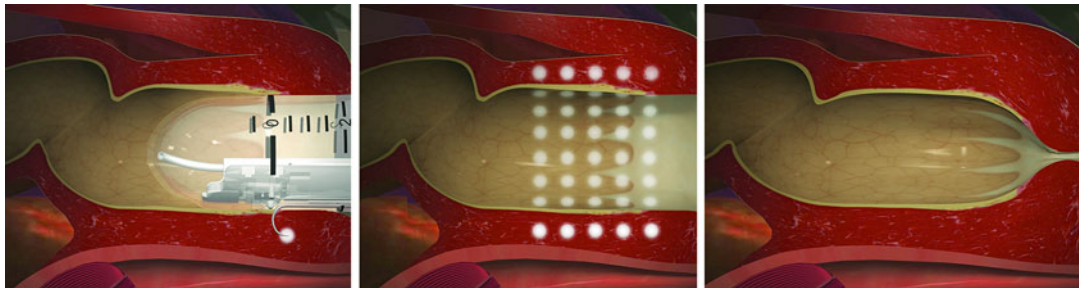


Fig. 16.9 Secca® procedure (Courtesy of Mederi Therapeutics, Norwalk, CT; ©2014 Mederi Therapeutics, Inc.)

canal to prevent leakage of stool. Studies looking at the results of this treatment are limited. There is lack of information regarding the volume of injection, ideal site of injection, and the route it should be injected. One large randomized trial comparing NASHA™ Dx to sham injections demonstrated that NASHA™ Dx is efficacious in the treatment of fecal incontinence with a follow-up of 12 months [55]. There are no studies looking at long-term benefit. In a review of all the studies published to date, the injection of bulking agents appears relatively safe; however, minor adverse events are relatively common (discomfort, pain, bleeding, abscess, and leakage of injected material) [54, 55] (Fig. 16.10).

Overlapping Sphincteroplasty

Overlapping sphincteroplasty is offered to highly symptomatic patients with an anterior external anal sphincter defect secondary to an obstetric or iatrogenic trauma. The procedure typically involves a full mechanical bowel preparation and pre-procedure intravenous antibiotics. A transverse incision is made over the perineum. Dissection is carried up to the level of anorectal ring and the anal mucosa is separated from the sphincter complex. Care is taken not to carry the dissection too far laterally as the nerve supply to the external anal sphincter enters posterolaterally. The fibrous remnant of the external anal sphincter is then divided. End-to-end repair has been

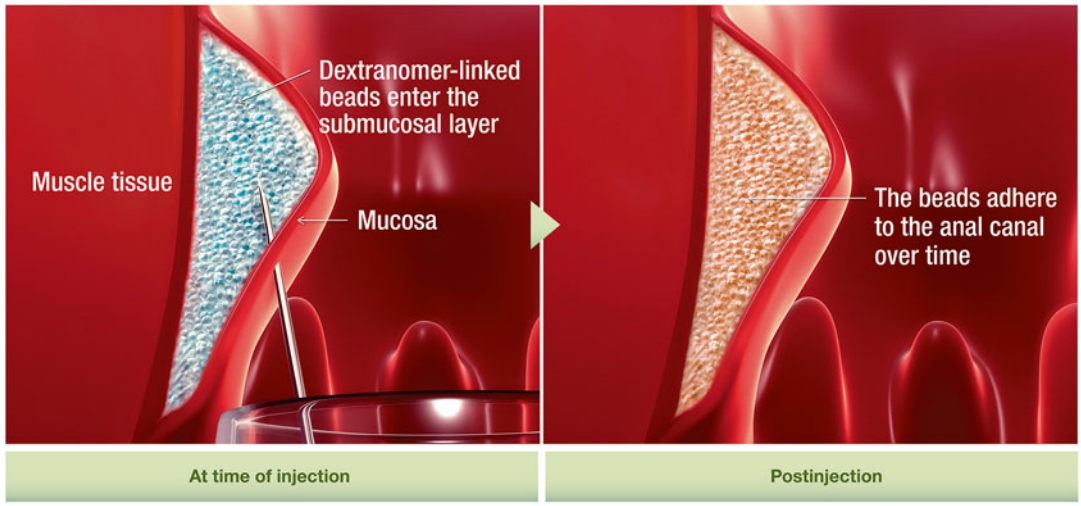


Fig. 16.10 Solesta® injection (Courtesy of Salix Pharmaceuticals, Raleigh, NC)

described but retraction of the ends of the muscle and lack of a bulking effect because of excision of the scar tissue has been implicated in the suboptimal results [56].

For overlapping repair, the scar at the ends of the sphincter is preserved to aid in anchoring the sutures. The ends of the mobilized external sphincter are overlapped and sutured together with absorbable mattress sutures. Plication of the internal anal sphincter may be concurrently performed. Anterior levatoroplasty and closure of the perineal incision in a V–Y manner can help to bulk up the perineal body and increase the anovaginal distance. Typically, the wound is left partially open to promote drainage [18] (Fig. 16.11a–d). Satisfactory results, which are defined as continence for solid and liquid stools, have been reported in 70–100 % of patients [7]. However, the majority of patients will not have perfect continence, and many patients will have residual symptoms. Some patients may even develop new evacuation problems [57]. The most important factor in the return of normal sphincter function seems to be an increase in squeeze pressures [58]. Poor outcome is usually associated with pelvic floor denervation or a residual sphincter defect [59, 60].

In a study looking at functional results of sphincter repair after a median of 10 years, zero patients were fully continent to flatus or stool [61]. Reasons for failure or decline of continence can be explained by weakening of the muscle because of normal aging, repair breakdown, or a combination of these factors [62]. Repeat sphincter repair can be performed in patients with recurrent symptoms, especially if breakdown of the repair is verified on endoanal ultrasound. It has been demonstrated that the long-term results of a repeat sphincter repair are approximately equivalent to those for primary overlapping sphincter repair [63].

Postanal Repair

Postanal repair was first described by Sir Alan G. Parks in 1975 [64]. This technique was described specifically for idiopathic or neurogenic incontinence and for incontinence following surgery for the repair of rectal prolapse. These conditions are associated with lengthening of the anorectal angle and shortening of the anal canal as a consequence of sphincter denervation [7]. The procedure is also advocated for patients with "weak" sphincters but no anatomic sphincter defect [14].

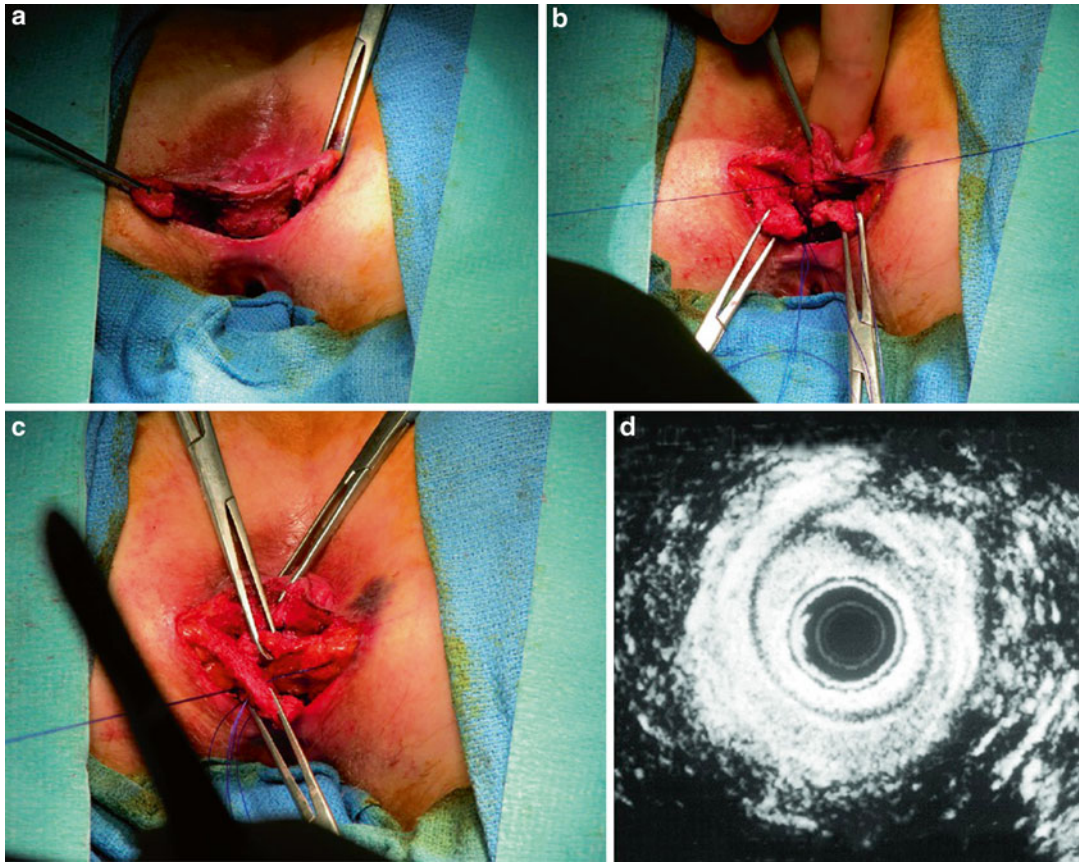


Fig. 16.11 (**a**–**d**) Overlapping sphincteroplasty (Used with permission from Seo CJ, Wexner SD, Davila GW. Reoperative Surgery for Anal Incontinence. In Billingham RP, Kobashi KC, Peters WA. Reoperative Pelvic Surgery. New York: Springer Science+Business Media: 2009)

The procedure is performed through a curved incision posterior to the anus with dissection through the intersphincteric space, through Waldeyer's fascia and into the pelvis. The ileococcygeus, pubococcygeus, and puborectalis muscles are plicated using a series of polypropylene sutures. Further plicating sutures can be placed in the deep and superficial parts of the external anal sphincter muscle using polyglactin suture [18]. The goals of the procedure are to restore the anorectal angle and to tighten the anal sphincter muscle. Although, Parks reports successful outcome in approximately 80 % of patients, these results have not been reproduced [7]. The mechanism of restoration of continence is unclear as the anorectal angle does not change significantly following this procedure and the manometric evaluation of sphincter function is variable [65, 66]. Improvement after this procedure may be caused by creation of a local stenosis or a placebo effect rather than improvement of muscle function [18].

Muscle Transposition

The most common skeletal muscle used in transposition techniques is the gracilis. Gracilis muscle transposition was first described by Pickrell in 1952 [67]. The muscle is freed from its insertion, completely mobilized, and subcutaneously tunneled to the perineum. It is then wrapped around the anus and anchored with sutures to the contralateral ischial tuberosity or the inferior ramus of

the pubic bone. The gracilis muscle is mostly composed of type two muscle fibers that are short acting and fast twitch fibers. Therefore, the muscle is fatigable and only contracts by will. Dynamic graciloplasty combines gracilis muscle transposition with an implantable electrical stimulator. This applies chronic low-frequency stimulation which functions to change the composition of the muscle to long acting, slow twitch, nonfatigable, type one muscle fibers. The procedure has a variable success rate with reports as high as 72 %. Given the steep learning curve of this technique, there is a high complication rate. Most complications are minor, but infection and rectal perforation are described [68]. Unfortunately this has not been approved for use in the United States. Other muscles that have been transposed include the gluteus maximus muscle [69], pubococcygeus [70], transverse perineal muscle [71], and even the antropylorus [72]. Free muscle transplantation has also been described [73].

Artificial Bowel Sphincter

The artificial bowel sphincter (ABS) was adapted from the artificial urinary sphincter which was introduced in 1972 by American Medical Systems (AMS). In 1987, the first description of the use of the artificial urinary sphincter was reported for fecal incontinence. The patient had an excellent result with no complications at a follow-up of 3 months [74].

Since then, modifications have been made to the artificial urinary sphincter to make it more applicable for use around the anus which culminated in the development of the Acticon Neosphincter™ (AMS, Minnetonka, Minnesota). The procedure involves encirclement of the anus with an implantable fluid-filled, silicone, elastomer cuff that is connected by tubes to a control pump and a pressure-regulating balloon. Cuff lengths range from 7 to 14 cm with three cuff widths of 2, 2.9, and 2.4 cm. The control pump is implanted in the labia or the scrotum and the balloon is implanted in the space of Retzius. The inflated cuff compresses the anus all the time. When the patient has to defecate, the fluid is manually pumped from the cuff to the balloon by using the control pump. The empty cuff allows the passage of stool and then the pressure in the balloon sends the fluid back into the cuff (Figs. 16.12 and 16.13).

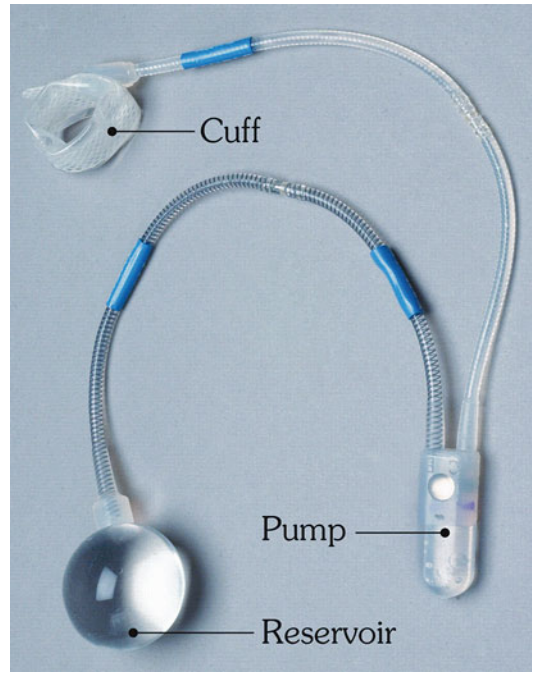


Fig. 16.12 The Acticon® artificial bowel sphincter device (American Medical Systems) (Used with permission from Goh M, Dioknow AC. Surgery for Stress Urinary Incontinence: Open Approaches. In Badlani GH, Davila GW, Michel MC, de la Rosette JJMCH, eds: Continence. London: Springer Science + Business Media, 2009)

In a multicenter, prospective, nonrandomized clinical trial looking at 115 patients, 6 patients were aborted because of perforation. Device-related complications were reported in 86 % of enrolled patients. Forty six percent of patients required device revisions to treat major adverse events including infection or erosion and 36 % required explantation. At the end of the follow-up period of 1 year, 75 of 112 patients (67 %) had functioning devices [75].

The magnetic anal sphincter (MAS) (FENIX™; Torax Medical Inc., Shoreview, MN) is a novel artificial sphincter mechanism which was recently described. This was originally used in the treatment of gastroesophageal reflux disease. This device is composed of a series of titanium beads with magnetic cores inside. The beads are interlinked with titanium wires to form

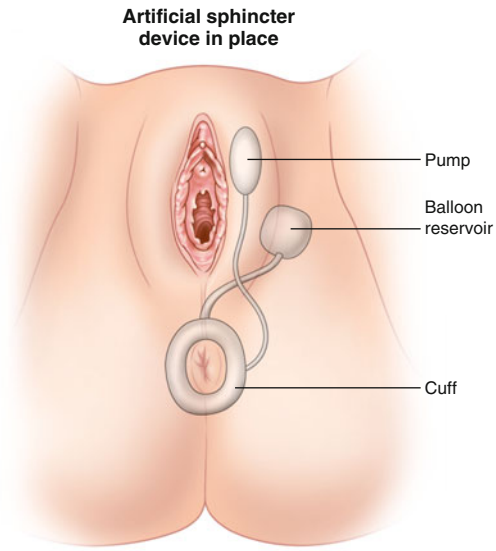


Fig. 16.13 Illustration of the implantation of the artificial bowel sphincter

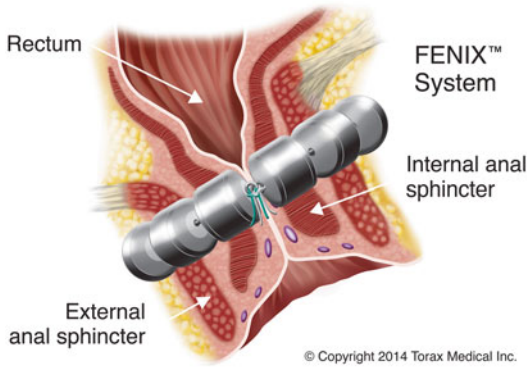


Fig. 16.14 Fenix™ magnetic anal sphincter device (Courtesy of and Copyright ©2014 Torax Medical, St. Paul, MN)

a flexible ring that is implanted around the external sphincter in a circular fashion (Fig. 16.14). The device is manufactured in different lengths based on the number of beads (14–20) [76].

One major advantage of this device in contrast to the ABS is that it works immediately once implanted without the need for further manipulation by the patient or the surgeon. The device is passively activated by the passage of stool and it automatically retracts back to its closed size after evacuation. In a study comparing 10 patients implanted with the ABS and 10 patients implanted with the MAS, there was similar 30-day complication rate but the procedure for MAS was shorter in duration with a shorter length of hospitalization [77]. Of note, this device has received European CE Mark approval for the treatment of fecal incontinence but is not available in the United States and is only limited to investigational use.

Sacral Nerve Stimulation

In 1988, Tangaho and Schmidt described the use of electrical stimulation for the treatment of neurogenic bladder [78]. Following that in 1995, Matzel et al. described its use in three patients for the treatment of fecal incontinence [79]. Since then, sacral nerve stimulation (SNS) has been advocated as a safe and effective treatment for severe fecal incontinence with minimal morbidity [80–82]. SNS has been shown to be more effective than optimal medical therapy and a placebo effect has been eliminated [83, 84]. The device has also proven to be beneficial in patients with idiopathic fecal incontinence as well as those with sphincter defects and also pudendal neuropathy [83, 85].

After a prospective multicenter study, SNS was FDA approved in the United States in 2011 for the treatment of fecal incontinence. This study looked at 120 patients that received an implant. After a mean follow-up of 28 months, 85 % of patients were improved and 40 % had perfect continence [86]. Although, no studies have been done in the United States with regards to cost, the procedure has been shown to be cost-effective in other countries [87–89].

Diversion

Although considered as the last option in the surgical strategy, construction of an end diverting colostomy may be indicated in certain patients in whom available treatments have failed, are inappropriate because of other comorbidities, or when preferred by the patient [14]. A stoma may be successful in controlling symptoms of incontinence but it may also be associated with significant psychosocial issues and stoma-related complications. As a stoma in this

instance will most likely be permanent, it is important for the patient to be marked preoperatively by an enterostomal nurse and also receive teaching and counseling prior to undergoing the procedure.

In patients with severe fecal incontinence, a stoma will improve quality of life in the majority of patients. In a survey, 83 % of patients with a permanent colostomy for incontinence reported a significant improvement in lifestyle and 84 % would choose to have the stoma again [90].

Conclusion

Fecal incontinence is an underreported condition for many reasons including embarrassment and unawareness of both physicians and patients to the available treatments. A detailed medical surgical, obstetric, and bowel history should be obtained. A thorough rectal exam combined with appropriate physiologic, endoscopic, and radiologic should be performed. Treatments are individualized to the particular patient. Emerging treatments for the treatment of fecal incontinence are promising and may avoid or even supplant traditional surgical procedures such as overlapping sphincteroplasty. The majority of patients can avoid a diverting stoma.

References

1. Rao SS, American College of Gastroenterology Practice Parameters Committee. Diagnosis and management of fecal incontinence. Am J Gastroenterol. 2004;99(8):1585–604.
2. 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(7):857–65. discussion 865–7.
3. Brown HW, Wexner SD, Lukacz ES. Factors associated with care seeking among women with accidental bowel leakage. Female Pelvic Med Reconstr Surg. 2013;19(2):66–71.
4. Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. JAMA. 1995;274(7):559–61.
5. Bharucha AE, Zinsmeister AR, Locke GR, et al. Prevalence and burden of fecal incontinence: a population-based study in women. Gastroenterology. 2005;129(1):42–9.
6. Kuehn BM. Silence masks prevalence of fecal incontinence. JAMA. 2006;295(12):1362–3.
7. Jorge JM, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum. 1993;36(1):77–97.
8. Huebner M, Gramlich NK, Rothmund R, Nappi L, Abele H, Becker S. Fecal incontinence after obstetric anal sphincter injuries. Int J Gynaecol Obstet. 2013;121(1):74–7.
9. Madoff RD, Parker SC, Varma MG, Lowry AC. Faecal incontinence in adults. Lancet. 2004;364(9434):621–32.
10. Guise JM, Morris C, Osterweil P, Li H, Rosenberg D, Greenlick M. Incidence of fecal incontinence after childbirth. Obstet Gynecol. 2007;109(2 Pt 1):281–8.
11. Donnelly V, Fynes M, Campbell D, Johnson H, O'Connell PR, O'Herlihy C. Obstetric events leading to anal sphincter damage. Obstet Gynecol. 1998;92(6):955–61.
12. Chaliha C, Kalia V, Stanton SL, Monga A, Sultan AH. Antenatal prediction of postpartum urinary and fecal incontinence. Obstet Gynecol. 1999;94(5 Pt 1):689–94.
13. Hall W, McCracken K, Osterweil P, Guise JM. Frequency and predictors for postpartum fecal incontinence. Am J Obstet Gynecol. 2003;188(5):1205–7.
14. Tjandra JJ, Dykes SL, Kumar RR, et al. Practice parameters for the treatment of fecal incontinence. Dis Colon Rectum. 2007;50(10):1497–507.
15. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. N Engl J Med. 1993;329(26):1905–11.
16. Bollard RC, Gardiner A, Duthie GS, Lindow SW. Anal sphincter injury, fecal and urinary incontinence: a 34-year follow-up after forceps delivery. Dis Colon Rectum. 2003;46(8):1083–8.
17. Burgio KL, Borello-France D, Richter HE, et al. Risk factors for fecal and urinary incontinence after childbirth: the childbirth and pelvic symptoms study. Am J Gastroenterol. 2007;102(9):1998–2004.
18. Baeten CG, Kuijpers HC. Incontinence. In: Wolff BG, Fleshman JW, Beck DE, Pemberton JH, Wexner SD, editors. The ASCRS textbook of colon and rectal surgery. 1st ed. Heidelberg: Springer; 2007. p. 653–64.
19. van den Hondel D, Sloots CE, Gischler SJ, et al. Prospective long-term follow up of children with anorectal malformation: growth and development until 5years of age. J Pediatr Surg. 2013;48(4):818–25.
20. Schmiedeke E, Zwink N, Schwarzer N, et al. Unexpected results of a nationwide, treatment-independent assessment of fecal incontinence in patients with anorectal anomalies. Pediatr Surg Int. 2012;28(8):825–30.
21. Murad-Regadas SM, Fernandes GO, Regadas FS, et al. How much of the internal sphincter may be divided during lateral sphincterotomy for chronic anal fissure in women? Morphologic and functional evaluation after sphincterotomy. Dis Colon Rectum. 2013;56(5):645–51.

22. Hamadani A, Haigh PI, Liu IL, Abbas MA. Who is at risk for developing chronic anal fistula or recurrent anal sepsis after initial perianal abscess? Dis Colon Rectum. 2009;52(2):217–21.
23. Abbas MA, Jackson CH, Haigh PI. Predictors of outcome for anal fistula surgery. Arch Surg. 2011; 146(9):1011–6.
24. Toyonaga T, Matsushima M, Kiriu T, et al. Factors affecting continence after fistulotomy for intersphincteric fistula-in-ano. Int J Colorectal Dis. 2007;22(9): 1071–5.
25. van Tets WF, Kuijpers HC. Continence disorders after anal fistulotomy. Dis Colon Rectum. 1994;37(12): 1194–7.
26. Matzel KE, Bittorf B, Gunther K, Stadelmaier U, Hohenberger W. Rectal resection with low anastomosis: functional outcome. Colorectal Dis. 2003;5(5): 458–64.
27. 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(11):664–7.
28. Krogh K, Nielsen J, Djurhuus JC, Mosdal C, Sabroe S, Laurberg S. Colorectal function in patients with spinal cord lesions. Dis Colon Rectum. 1997;40(10):1233–9.
29. Varma M, Rafferty J, Buie WD, Standards Practice Task Force of American Society of Colon and Rectal Surgeons. Practice parameters for the management of rectal prolapse. Dis Colon Rectum. 2011;54(11): 1339–46.
30. Cunin D, Siproudhis L, Desfourneaux V, et al. Incontinence in full-thickness rectal prolapse: low level of improvement after laparoscopic rectopexy. Colorectal Dis. 2013;15(4):470–6.
31. Rao SS, Hatfield R, Soffer E, Rao S, Beaty J, Conklin JL. Manometric tests of anorectal function in healthy adults. Am J Gastroenterol. 1999;94(3):773–83.
32. Rao SS, Sun WM. Current techniques of assessing defecation dynamics. Dig Dis. 1997;15 Suppl 1: 64–77.
33. Rao SS. Manometric evaluation of defecation disorders: Part II. Fecal incontinence. Gastroenterologist. 1997;5(2):99–111.
34. Jones PN, Lubowski DZ, Swash M, Henry MM. Relation between perineal descent and pudendal nerve damage in idiopathic faecal incontinence. Int J Colorectal Dis. 1987;2(2):93–5.
35. Wunderlich M, Swash M. The overlapping innervation of the two sides of the external anal sphincter by the pudendal nerves. J Neurol Sci. 1983;59(1):97–109.
36. Rieger N, Tjandra J, Solomon M. Endoanal and endorectal ultrasound: applications in colorectal surgery. ANZ J Surg. 2004;74(8):671–5.
37. Sultan AH, Kamm MA, Talbot IC, Nicholls RJ, Bartram CI. Anal endosonography for identifying external sphincter defects confirmed histologically. Br J Surg. 1994;81(3):463–5.
38. Sultan AH, Nicholls RJ, Kamm MA, Hudson CN, Beynon J, Bartram CI. Anal endosonography and correlation with in vitro and in vivo anatomy. Br J Surg. 1993;80(4):508–11.
39. Wald A. Colonic and anorectal motility testing in clinical practice. Am J Gastroenterol. 1994;89(12):2109–15.
40. Diamant NE, Kamm MA, Wald A, Whitehead WE. AGA technical review on anorectal testing techniques. Gastroenterology. 1999;116(3):735–60.
41. Hallgren T, Fasth S, Delbro DS, Nordgren S, Oresland T, Hulten L. Loperamide improves anal sphincter function and continence after restorative proctocolectomy. Dig Dis Sci. 1994;39(12):2612–8.
42. Herbst F, Kamm MA, Nicholls RJ. Effects of loperamide on ileoanal pouch function. Br J Surg. 1998;85(10):1428–32.
43. Sun WM, Read NW, Verlinden M. Effects of loperamide oxide on gastrointestinal transit time and anorectal function in patients with chronic diarrhoea and faecal incontinence. Scand J Gastroenterol. 1997; 32(1):34–8.
44. Rao SS. The technical aspects of biofeedback therapy for defecation disorders. Gastroenterologist. 1998; 6(2):96–103.
45. Jensen LL, Lowry AC. Biofeedback improves functional outcome after sphincteroplasty. Dis Colon Rectum. 1997;40(2):197–200.
46. Heymen S, Jones KR, Ringel Y, Scarlett Y, Whitehead WE. Biofeedback treatment of fecal incontinence: a critical review. Dis Colon Rectum. 2001;44(5):728–36.
47. Schiller LR. Faecal incontinence. Clin Gastroenterol. 1986;15(3):687–704.
48. Zutshi M, Hull T, Gurland B. Anal encirclement with sphincter repair (AESR procedure) using a biological graft for anal sphincter damage involving the entire circumference. Colorectal Dis. 2012;14(5):592–5.
49. Sainio AP, Halme LE, Husa AI. Anal encirclement with polypropylene mesh for rectal prolapse and incontinence. Dis Colon Rectum. 1991;34(10):905–8.
50. Ruiz D, Pinto RA, Hull TL, Efron JE, Wexner SD. Does the radiofrequency procedure for fecal incontinence improve quality of life and incontinence at 1-year follow-up? Dis Colon Rectum. 2010;53(7): 1041–6.
51. Kim DW, Yoon HM, Park JS, Kim YH, Kang SB. Radiofrequency energy delivery to the anal canal: is it a promising new approach to the treatment of fecal incontinence? Am J Surg. 2009;197(1):14–8.
52. Lefebure B, Tuech JJ, Bridoux V, et al. Temperature-controlled radio frequency energy delivery (secca procedure) for the treatment of fecal incontinence: results of a prospective study. Int J Colorectal Dis. 2008;23(10):993–7.
53. Felt-Bersma RJ, Szojda MM, Mulder CJ. Temperature-controlled radiofrequency energy (SECCA) to the anal canal for the treatment of faecal incontinence offers moderate improvement. Eur J Gastroenterol Hepatol. 2007;19(7):575–80.
54. Maeda Y, Laurberg S, Norton C. Perianal injectable bulking agents as treatment for faecal incontinence in adults. Cochrane Database Syst Rev. 2013;2, CD007959.

55. Graf W, Mellgren A, Matzel KE, et al. Efficacy of dextranomer in stabilised hyaluronic acid for treatment of faecal incontinence: a randomised, sham-controlled trial. Lancet. 2011;377(9770):997–1003.
56. Browning GG, Motson RW. Anal sphincter injury. Management and results of parks sphincter repair. Ann Surg. 1984;199(3):351–7.
57. Malouf AJ, Norton CS, Engel AF, Nicholls RJ, Kamm MA. Long-term results of overlapping anterior anal-sphincter repair for obstetric trauma. Lancet. 2000; 355(9200):260–5.
58. Ha HT, Fleshman JW, Smith M, Read TE, Kodner IJ, Birnbaum EH. Manometric squeeze pressure difference parallels functional outcome after overlapping sphincter reconstruction. Dis Colon Rectum. 2001;44(5):655–60.
59. Pinta T, Kylanpaa-Back ML, Salmi T, Jarvinen HJ, Luukkonen P. Delayed sphincter repair for obstetric ruptures: analysis of failure. Colorectal Dis. 2003; 5(1):73–8.
60. Gilliland R, Altomare DF, Moreira Jr H, Oliveira L, Gilliland JE, Wexner SD. Pudendal neuropathy is predictive of failure following anterior overlapping sphincteroplasty. Dis Colon Rectum. 1998;41(12): 1516–22.
61. Zutshi M, Tracey TH, Bast J, Halverson A, Na J. Ten-year outcome after anal sphincter repair for fecal incontinence. Dis Colon Rectum. 2009;52(6): 1089–94.
62. Halverson AL, Hull TL. Long-term outcome of overlapping anal sphincter repair. Dis Colon Rectum. 2002;45(3):345–8.
63. Vaizey CJ, Norton C, Thornton MJ, Nicholls RJ, Kamm MA. Long-term results of repeat anterior anal sphincter repair. Dis Colon Rectum. 2004;47(6):858–63.
64. Parks AG. Royal society of medicine, section of proctology; meeting 27 November 1974. President's address. Anorectal incontinence. Proc R Soc Med. 1975;68(11):681–90.
65. Orrom WJ, Miller R, Cornes H, Duthie G, Mortensen NJ, Bartolo DC. Comparison of anterior sphincteroplasty and postanal repair in the treatment of idiopathic fecal incontinence. Dis Colon Rectum. 1991; 34(4):305–10.
66. Scheuer M, Kuijpers HC, Jacobs PP. Postanal repair restores anatomy rather than function. Dis Colon Rectum. 1989;32(11):960–3.
67. Pickrell KL, Broadbent TR, Masters FW, Metzger JT. Construction of a rectal sphincter and restoration of anal continence by transplanting the gracilis muscle; a report of four cases in children. Ann Surg. 1952;135(6):853–62.
68. Rongen MJ, Uludag O, El Naggar K, Geerdes BP, Konsten J, Baeten CG. Long-term follow-up of dynamic graciloplasty for fecal incontinence. Dis Colon Rectum. 2003;46(6):716–21.
69. Devesa JM, Vicente E, Enriquez JM, et al. Total fecal incontinence–a new method of gluteus maximus transposition: preliminary results and report of previous experience with similar procedures. Dis Colon Rectum. 1992;35(4):339–49.
70. Ingelman-Sundberg A. Plastic repair of extensive defects of the anal sphincter. Acta Chir Scand. 1951;101(2):155–7.
71. State D, Katz A. The use of superficial transverse perineal muscles in the treatment of post surgical anal incontinence. Ann Surg. 1955;142(2):262–5.
72. Chandra A, Kumar A, Noushif M, et al. Perineal antropylorus transposition for end-stage fecal incontinence in humans: initial outcomes. Dis Colon Rectum. 2013;56(3):360–6.
73. Hakelius L, Gierup J, Grotte G, Jorulf H. A new treatment of anal incontinence in children: free autogenous muscle transplantation. J Pediatr Surg. 1978; 13(1):77–82.
74. Christiansen J, Lorentzen M. Implantation of artificial sphincter for anal incontinence. Lancet. 1987;2(8553): 244–5.
75. Wong WD, Congliosi SM, Spencer MP, et al. The safety and efficacy of the artificial bowel sphincter for fecal incontinence: results from a multicenter cohort study. Dis Colon Rectum. 2002;45(9):1139–53.
76. Lehur PA, McNevin S, Buntzen S, Mellgren AF, Laurberg S, Madoff RD. Magnetic anal sphincter augmentation for the treatment of fecal incontinence: a preliminary report from a feasibility study. Dis Colon Rectum. 2010;53(12):1604–10.
77. Wong MT, Meurette G, Stangherlin P, Lehur PA. The magnetic anal sphincter versus the artificial bowel sphincter: a comparison of 2 treatments for fecal incontinence. Dis Colon Rectum. 2011;54(7):773–9.
78. Tanagho EA, Schmidt RA. Electrical stimulation in the clinical management of the neurogenic bladder. J Urol. 1988;140(6):1331–9.
79. Matzel KE, Stadelmaier U, Hohenfellner M, Gall FP. Electrical stimulation of sacral spinal nerves for treatment of faecal incontinence. Lancet. 1995;346(8983): 1124–7.
80. Matzel KE, Kamm MA, Stosser M, et al. Sacral spinal nerve stimulation for faecal incontinence: multicentre study. Lancet. 2004;363(9417):1270–6.
81. Hetzer FH, Hahnloser D, Clavien PA, Demartines N. Quality of life and morbidity after permanent sacral nerve stimulation for fecal incontinence. Arch Surg. 2007;142(1):8–13.
82. Tjandra JJ, Lim JF, Matzel K. Sacral nerve stimulation: an emerging treatment for faecal incontinence. ANZ J Surg. 2004;74(12):1098–106.
83. Tjandra JJ, Chan MK, Yeh CH, Murray-Green C. Sacral nerve stimulation is more effective than optimal medical therapy for severe fecal incontinence: a randomized, controlled study. Dis Colon Rectum. 2008;51(5):494–502.
84. Vaizey CJ, Kamm MA, Roy AJ, Nicholls RJ. Double-blind crossover study of sacral nerve stimulation for fecal incontinence. Dis Colon Rectum. 2000;43(3): 298–302.

85. Chan MK, Tjandra JJ. Sacral nerve stimulation for fecal incontinence: external anal sphincter defect vs. intact anal sphincter. Dis Colon Rectum. 2008;51(7): 1015–24. discussion 1024–5.
86. Wexner SD, Coller JA, Devroede G, et al. Sacral nerve stimulation for fecal incontinence: results of a 120-patient prospective multicenter study. Ann Surg. 2010;251(3):441–9.
87. van Wunnik BP, Visschers RG, van Asselt AD, Baeten CG. Cost-effectiveness analysis of sacral neuromodulation for faecal incontinence in the Netherlands. Colorectal Dis. 2012;14(12):e807–14.
88. Munoz-Duyos A, Navarro-Luna A, Brosa M, Pando JA, Sitges-Serra A, Marco-Molina C. Clinical and cost effectiveness of sacral nerve stimulation for faecal incontinence. Br J Surg. 2008;95(8): 1037–43.
89. Leroi AM, Lenne X, Dervaux B, et al. Outcome and cost analysis of sacral nerve modulation for treating urinary and/or fecal incontinence. Ann Surg. 2011; 253(4):720–32.
90. Norton C, Burch J, Kamm MA. Patients' views of a colostomy for fecal incontinence. Dis Colon Rectum. 2005;48(5):1062–9.

Botulinum Toxin Therapy for Voiding Dysfunction and the Female Pelvic Floor

17

Michael Ingber

Background

Onabotulinum toxin A has been utilized therapeutically throughout the past century. Onabotulinum toxin is an acetylcholine release inhibitor and a neuromuscular blocking agent, and for many years was limited to fields outside of urology. Recent research has shed some light on the numerous other applications of onabotulinum toxin A, specifically within Female Pelvic Medicine and Reconstructive Surgery (FPMRS). Herein we discuss the history of onabotulinum toxin A, its use within the field of FPMRS, and potential future indications for use of this unique toxin.

History of Botulinum Toxin

The first descriptions of the actual symptoms of food-borne botulism date back to the nineteenth century, when it was discovered as a toxin found in sausages and pork. Justinius Kerner was the first to describe the effects of the toxin in two monographs in 1820 and 1822 [1]. He later began a series of animal experiments and reportedly self-ingested the toxin in order to better understand how the toxin worked and to determine if there were any potential benefits of the toxin itself.

In 1895, Van Ermengem identified the actual bacteria as *Bacillus botulinum*. Later, this was renamed *Clostridium botulinum*. This bacterium was noted to produce seven distinct antigenic toxins, notably "A, B, C, D, E, F, and G." The first to isolate onabotulinum toxin A in a stable precipitate was Herman Sommer, M.D. at the University of California, in San Francisco, USA in the early twentieth century. It was not until 1946 when Edward Schantz, Ph.D., was able to purify onabotulinum toxin A in crystalline form, allowing researchers to study the drug in greater detail. However, it was not until the 1950s when onabotulinum toxin A was noted by Vernon Brooks, M.D., to block acetylcholine release when injected into a hyperactive muscle. This specific characteristic of onabotulinum toxin A led to its early research in the field of ophthalmology.

Perhaps one of the greatest breakthroughs in medical research with onabotulinum A was with Alan B. Scott, M.D., who initiated several animal studies with the toxin. He discovered that by injecting a small amount of the purified toxin into the hyperactive ocular muscles in monkeys, he was able to realign crossed eyes due to strabismus. At the time, the only alternative to strabismus correction was surgical realignment. After this breakthrough, Dr. Scott collaborated with

M. Ingber, M.D. (✉)
Saint Clare's Health System, Denville, NJ, USA

Weill Cornell Medical College, New York, NY, USA
e-mail: inberMD@aol.com

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_17,

others, including Dr. Schantz, to develop onabotulinum toxin A into a therapy for ocular disorders.

In the early twenty-first century, the United States Food and Drug Administration (FDA) approved the drug to treat cosmetic indications such as glabellar lines (frown lines), axillary hyperhidrosis (underarm sweating), and upper limb stiffness due to muscle spasticity. The success of onabotulinum toxin A with these prior indications and the years of research in ocular and neural disease paved the way to study the drug and obtain approval in the field of FPMRS.

Mechanism of Action

Onabotulinum toxin A consists of a light and heavy chain joined by a disulfide bond. The heavy chain allows the binding to the neuron, and the light chain of the toxin inhibits vesicle-mediated neurotransmission at the synaptic cleft by cleaving the SNARE protein SNAP-25 (Fig. 17.1a, b) This prevents docking and fusion of the vesicles with the nerve terminal, thus preventing the release of acetylcholine [2, 3]. The subsequent prevention of acetylcholine release "relaxes" the detrusor muscle, preventing uninhibited bladder contractions. Because bladder instillation (as opposed to injection) of onabotulinum toxin A has also been shown to improve detrusor function, it is theorized that there may be an additional effect at the sensory, afferent level [4]. Onabotulinum toxin A is widely available under the trade names Botox (Allergan Inc, Irvine, CA, USA) and Dysport (Ipsen Ltd, Slough, Berks, UK) (Fig. 17.2).

Use in Neurogenic Detrusor Overactivity

For years, patients with neurogenic bladder were limited to only a few options for treatment of their bladder complaints. Specifically, for the patient with neurogenic detrusor overactivity (NDO), options included watchful waiting, behavioral therapy, anticholinergic therapy, clean intermittent catheterization with anticholinergic therapy, or urinary diversion. The numerous side effects of anticholinergics include dry mouth, dry eyes, constipation, nausea, amongst others, which precludes their use in a great percentage of the population [5]. Because of these side effects, the majority of patients who begin anticholinergics will stop them at some point during therapy [6, 7]. With the success of onabotulinum toxin A injection into skeletal and smooth muscle in other areas, it would seem intuitive that it would provide a benefit in patients with NDO.

Patients with spinal cord injury (SCI) and multiple sclerosis (MS) often have NDO, which can lead to urinary incontinence, and has been shown to produce high storage pressures urodynamically [8]. Schurch and colleagues performed one of the first pilot studies evaluating onabotulinum toxin A for patients with neurogenic bladder due to spinal cord injury [9]. In this prospective, nonrandomized trial, patients who were on intermittent catheterization and had leakage despite anticholinergic therapy were given between 200 and 300 units of onabotulinum toxin A. Six weeks after injection, there was improvement in several urodynamic parameters, including maximum cystometric capacity (increase from 296.3 ± 145.2 mL to 480.5 ± 134.1 mL, $p < 0.016$), and in maximum detrusor voiding pressure (decrease from 65.6 ± 29.2 cmH_2O water to 35 ± 32. 1 cmH_2O, $p < 0.016$). In this study, the effect was noted to last approximately 9 months, when patients required repeat injection. This study also noted an increase in residual volume in treated patients, with residual volume on urodynamic evaluation increasing significantly from a mean of 261.8 ± 241.3 mL to 490.5 ± 204.8 ($p < 0.016$).

The United States Food and Drug Administration approved the drug for use in neurogenic detrusor overactivity in 2011. However, dosing and tolerability of the drug remained unclear. A phase-3 placebo-controlled study evaluated both the 200 and 300 unit dosage in patients with MS or SCI. This study included 416 patients from several centers around the world. The primary endpoint measure was the change in urinary incontinence episodes from baseline to week 6. In both the 200 and 300 unit

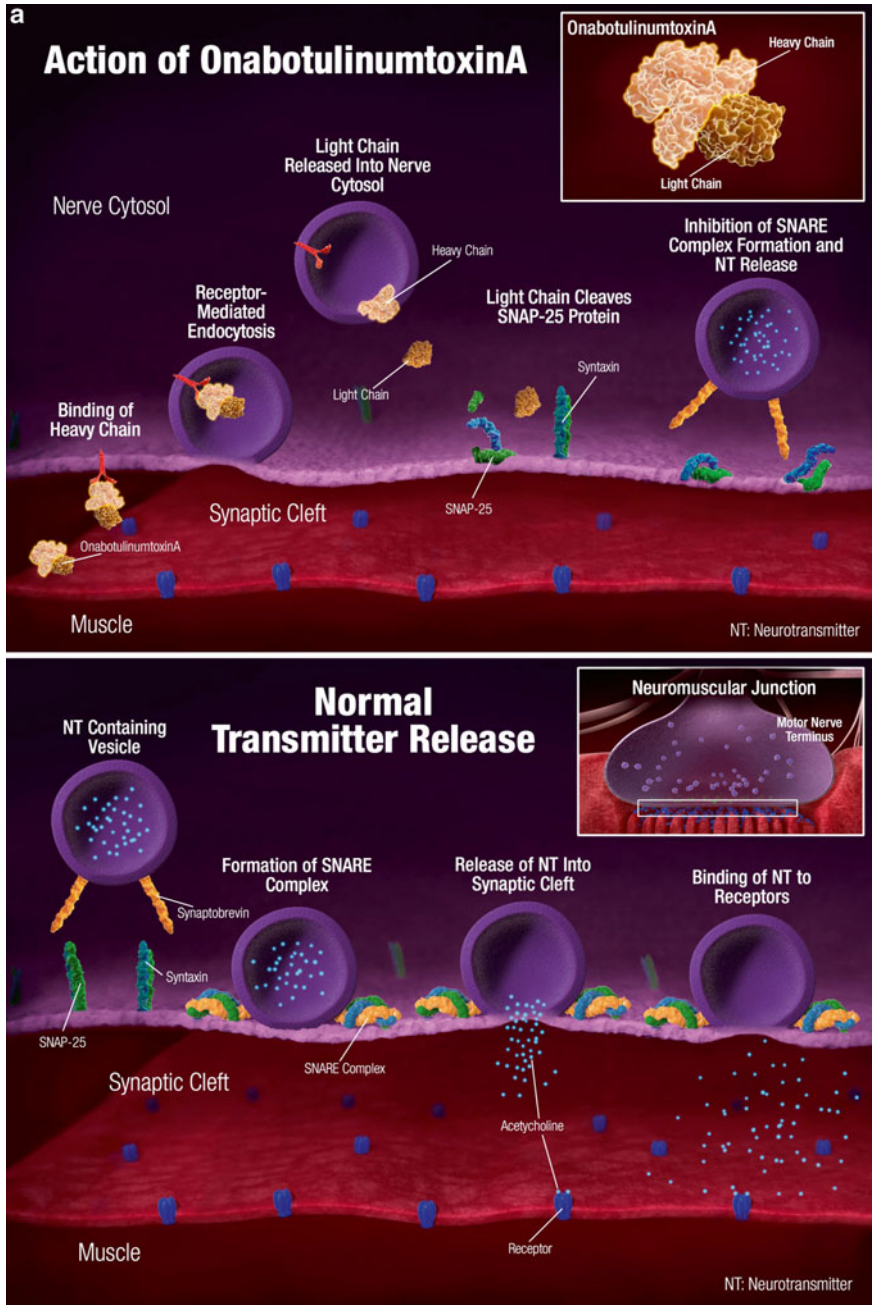


Fig. 17.1 (**a**, **b**) (**a**) Normal cholinergic transmission at the synaptic cleft. (Copyright © Allergan, Inc., Irvine, CA. Used with permission 2013.) (**b**) Onabotulinum toxin A works by inhibiting vesicle-mediated neurotransmission at the synaptic cleft

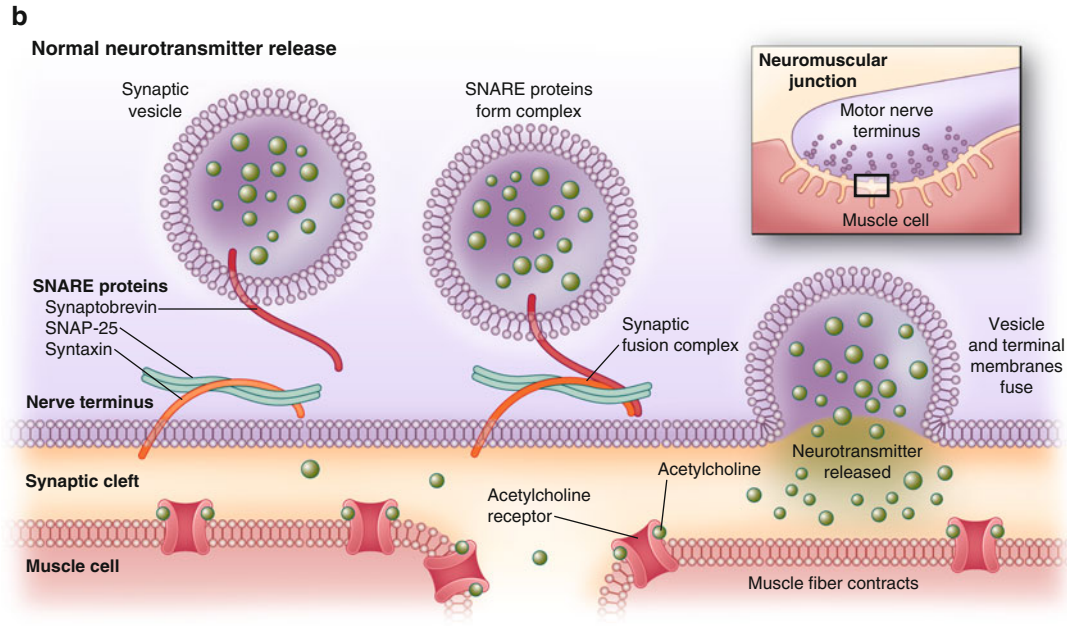


Fig. 17.1 (continued)

Fig. 17.2 Available doses of onabotulinum toxin A which are commonly used in urology applications (Botox, Copyright © Allergan, Inc., Irvine, CA. Used with permission 2013)

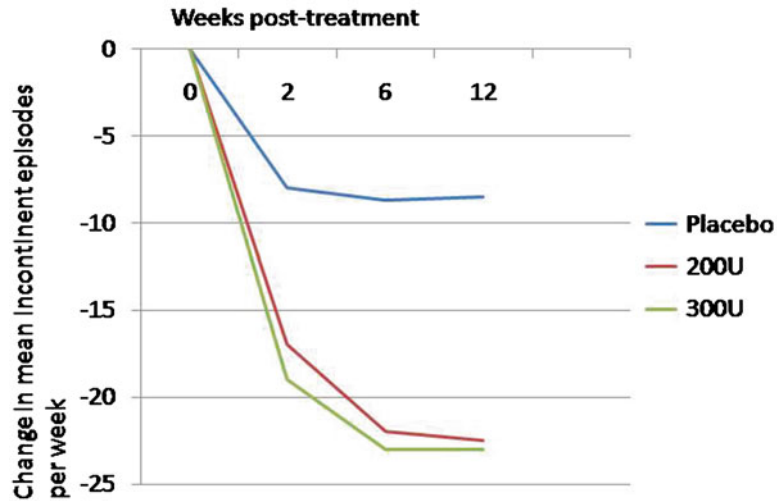


Fig. 17.3 Change from baseline in weekly incontinence episodes with 100 and 200 units of Botox compared to placebo in neurogenic patients (Adapted with permission from Ginsberg D, Gousse A, Keppenne V, Sievert KD, Thompson C, Lam W, Brin MF, Jenkins B, Haag-Molkenteller C. Phase 3 efficacy and tolerability study of onabotulinumtoxinA for urinary incontinence from neurogenic detrusor overactivity. J Urol. 2012 Jun;187(6):2131-9)

treatment arms, mean weekly incontinent episodes decreased to a greater extent than placebo, 21 and 23 vs. 9 episodes per week, respectively (Fig. 17.3). This study had a high rate of postoperative urinary retention requiring intermittent catheterization, with 35 % of patients receiving 200 units, and 42 % of patients receiving 300 units, beginning catheterization. The definition of urinary retention was investigator-dependent, which, in this study, may have led to the somewhat high rates of clean intermittent catheterization in the early data [10].

Use in Idiopathic Overactive Bladder

Approximately 16 % of the adult population has idiopathic overactive bladder (OAB), and approximately 1/3 of these patients have associated urinary incontinence [11, 12]. Patients with OAB are treated initially with behavioral therapy and pharmacologic therapy in the form of anticholinergics or beta 3-agonists. Like NDO, many patients with OAB who begin pharmacologic therapy with anticholinergics are unable to tolerate the side effects of dry mouth, constipation, and dry eyes, and therefore, the majority of patients stop these medicines within months of beginning treatment [13]. Onabotulinum toxin A represents a viable option for these patients, without the untoward side effects typically experienced by anticholinergic therapies.

Brubaker and colleagues compared 200 units of onabotulinum toxin A to placebo in a randomized trial involving female subjects [14]. These women were considered to have OAB with urge incontinence, with a minimum of six incontinent episodes over a 3 day period. Approximately 60 % of women receiving onabotulinum toxin A had a positive effect as evidenced on the Patient Global Impression of Improvement. The median duration of the response in these women was 373 days, compared to placebo, which was 62 days ($p<0.0001$). This study was halted after 43 women were randomized, however, due to increased incidence of urinary tract infection and post-void residual volume in the women receiving onabotulinum toxin A.

Dmochowski published results of a phase 2, multicenter, randomized, double-blind study evaluating multiple dosing of onabotulinum toxin A [15]. Patients with 8 or more urinary urgency

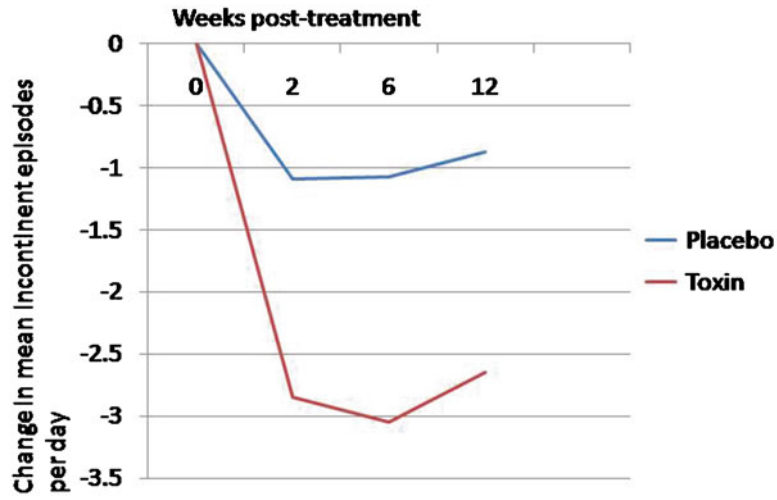


Fig. 17.4 Change from baseline in daily incontinence episodes with 100 units of Botox compared to placebo in patients with overactive bladder (Adapted with permission from Nitti VW, Dmochowski R, Herschorn S, Sand P, Thompson C, Nardo C, Yan X, Haag-Molkenteller C; EMBARK Study Group. OnabotulinumtoxinA for the Treatment of Patients with Overactive Bladder and Urinary Incontinence: Results of a Phase 3, Randomized, Placebo Controlled Trial. J Urol. 2013; 189(6):2186-2193)

incontinence episodes per week and 8 or more micturitions per day were included in the study. Patients received 50, 100, 150, 200, or 300 units of onabotulinum toxin A or placebo. The primary endpoint in this study was urinary incontinent episodes at week 12 after treatment. Efficacy was noted in all groups treated with 100 units or greater of study drug. When dosage response curves were evaluated, it was evident that doses over 150 units did not provide any additional benefit.

Nitti and colleagues reported on the first phase 3 placebo-controlled trial evaluating the 100 unit dose in patients with refractory OAB. Patients with a minimum of three or more urgency incontinent episodes over a 3 day period and with eight or more voids per day were randomized to 100 units of onabotulinum toxin A or placebo. Not surprisingly, onabotulinum toxin A reduced daily incontinent episodes at a greater frequency than placebo (2.65 vs. 0.87 fewer episodes, $p<0.001$) (Fig. 17.4). Total continence rates were 22.9 % in the onabotulinum toxin A group and 6.5 % in the placebo group. Additionally, nocturia, urgency episodes, and volume per void were improved in the study group versus placebo. Therefore, the US FDA approved the dose of 100 units of onabotulinum toxin A in patients with idiopathic OAB.

There is no doubt that reducing urinary frequency and urge urinary incontinent episodes greatly improve quality of life in patients with overactive bladder. Sahai and colleagues demonstrated such a benefit in a randomized, placebo-controlled trial evaluating a 200 unit dose of onabotulinum A [16]. Overall, patients receiving onabotulinum toxin A had a significant improvement in their quality of life when compared with placebo beginning at the 4-week endpoint (median King's Health Questionnaire Incontinence Impact domain score of 33 vs. 0, $p=0.03$). This effect on quality of life improvement appeared to extend to 24 weeks in patients receiving onabotulinum toxin A through the open-label extension study.

There have been few direct comparisons of onabotulinum toxin A to anticholinergic therapy. One study randomized patients with idiopathic urgency urinary incontinence to receive daily solifenacin or trospium (5 mg solifenacin, with escalation to 10 mg, and if necessary, subsequent switch to trospium 60 mg extended release) plus

an intradetrusor injection of saline versus intradetrusor injection of 100 units of onabotulinum toxin A plus oral placebo [17]. Two hundred forty-nine patients were randomized, and patients were evaluated at the 6-month point with voiding diaries and quality of life questionnaires. At 6 months, both groups had significant reduction in daily incontinence episodes (3.4 fewer in the anticholinergic group vs. 3.3 in the onabotulinum toxin A group, $p=0.81$). Patients receiving anticholinergics were more likely to develop dry mouth versus onabotulinum toxin A (46 vs. 31 %, $p=0.02$). Complete resolution of incontinence was noted more commonly in the onabotulinum toxin A group than placebo (27 % vs. 13 %, $p=0.003$). Not surprisingly, patients in the onabotulinum toxin A group had higher rates of urinary tract infection (33 % vs. 13 %, $p<0.001$) and need for catheter use (5 % vs. 0 %, $p=0.01$).

Evaluation, Workup, Procedure, and Post-Procedure

Pre-Procedure Considerations

Typically, patients who have failed conservative therapies such as behavioral modification and anticholinergic or beta agonist therapy or patients who have contraindications to oral anticholinergic therapies are good candidates for injection therapy. Patients should be formally evaluated prior to undergoing an injection of onabotulinum toxin A. Patients presenting with refractory overactive bladder with symptoms of urinary urgency, frequency, with or without urge urinary incontinence should always be attempted on conservative therapy prior to considering procedural therapy for their condition. A formal history to ascertain that patients have failed conservative therapy such as behavioral modification and adequate oral therapy, including dietary history, should be ascertained when evaluating patients for onabotulinum toxin A injection. A 24–48 h voiding diary can often provide insight to voiding symptoms, and daily intake of caffeine and other fluids should be recorded. A basic urologic evaluation consisting of a detailed and proper pelvic exam, residual volume measurement, and urinalysis should be performed. Patients presenting with bacteriuria should be treated appropriately prior to considering injection. Pelvic floor muscle rehabilitation or biofeedback should be attempted as first-line therapy for voiding symptoms. Typically, patients should be tried on oral anticholinergics or beta-agonist therapy prior to being scheduled for onabotulinum toxin A injection.

Once patients have been selected to undergo detrusor injection of onabotulinum toxin A, they must be counseled specifically about the risks of injection, which are discussed elsewhere in this chapter. Because the risk of urinary retention exists, patients should consider learning intermittent catheterization *prior* to injection, should they experience difficulty when the toxin exerts its effect on the detrusor. It is recommended that patients are started on a flouroquinolone antibiotic or trimethoprim-sulfamethoxazole 1–3 days prior to the procedure. Patients should avoid concurrent aminoglycoside administration, as the effects of onabotulinum toxin A have been reported to be potentiated with concomitant aminoglycoside administration in prior studies [18]. Patients should discontinue antiplatelet agents or other anticoagulants prior to injection, if possible.

Surgical Procedure

Intradetrusor injection of onabotulinum toxin A is performed through a cystoscope, in either the office or ambulatory setting. Both rigid and flexible cystoscopes can be used, and there are a variety of needles which are available for injection. There are also rigid cystoscopes designed specifically for needle injection which are small and quite tolerable to the patient with only local anesthesia. Typically, needle gauges range from 21 to 25 gauge. When using needles through a flexible cystoscope, one must be careful not to damage the scope by threading the exposed needle tip through the scope. Therefore, a variety of sheaths and retractable needle tips are available for use during flexible cystoscopy (Fig. 17.5a–d).

Anesthetic choice is surgeon-dependent, and patients should be offered no anesthesia, local

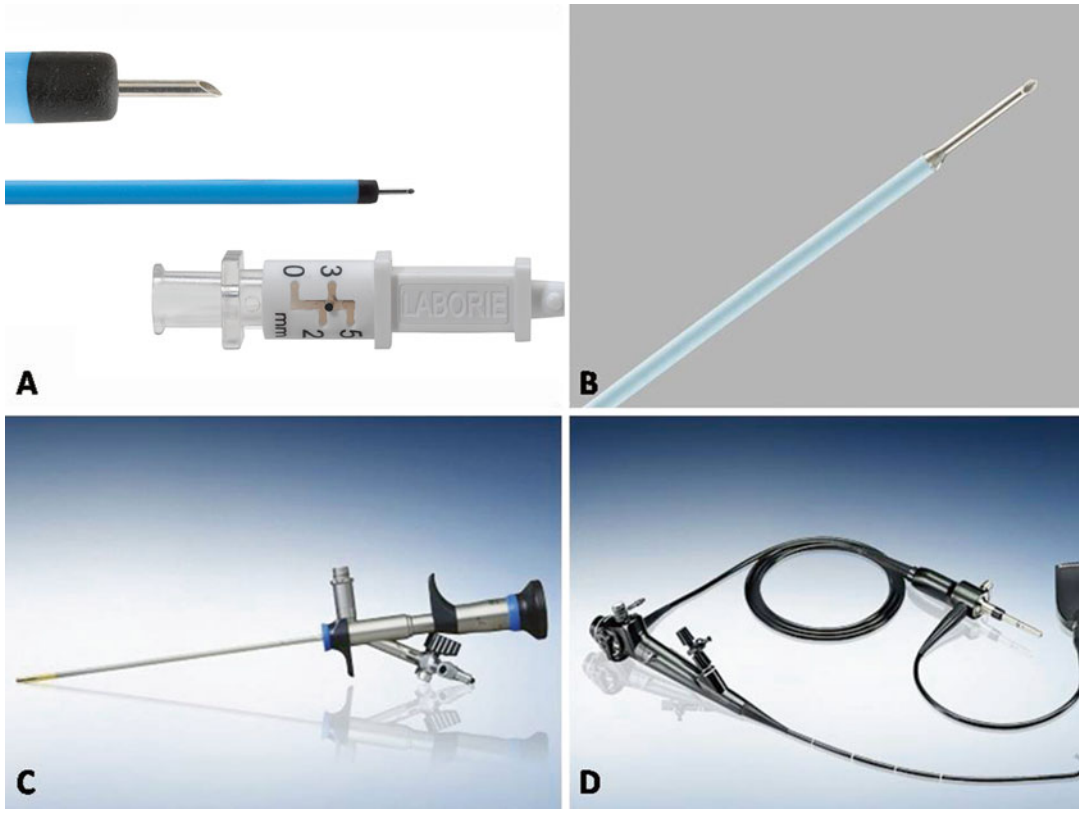


Fig. 17.5 (**a**–**d**) Commonly used instrumentation for injection. (**a**) InjeTAK needle. (**b**) Single-use flexible needle tip. (**c**) Rigid 14 French cystoscope. (**d**) Flexible cystoscope. (**a**: Courtesy of Laborie, Mississauga, Ontario; **b**, **c**, **d**: Courtesy of Olympus America, Inc, Center Valley, PA)

anesthesia, intravenous sedation, or general anesthesia. Special considerations may be made in the patient presenting with NDO and a history of autonomic dysreflexia, as patients may require preoperative blockade to prevent unopposed sympathetic efferent discharge [19]. In the author's experience, most healthy patients with idiopathic OAB tolerate injection in the office setting with simple local anesthetic. Such local anesthetic is in the form of 30–50 mL of 1 % lidocaine, which is instilled via catheter into the bladder. The solution is left for approximately 20 min in order to allow the anesthetic to exert its maximal effect. On the contrary, patients with multiple sclerosis, spinal cord injury with limited mobility may be better served under general anesthesia in order to allow for better positioning during the procedure. The patient is positioned in the lithotomy position, and genitalia should be prepped with sterile solution. The patient is draped, and anesthetic is administered if necessary.

Proper mixing onabotulinum toxin A is of utmost importance, as improper handling prior to the procedure may render the toxin ineffective. Onabotulinum toxin is stored in single-use 100 or 200 unit vials and is commercially available (BOTOX, Allergan Inc., Irvine, CA, USA). The vials themselves contain freeze-dried toxin which is in crystallized form, and is often not visible to the human eye. Botox is reconstituted by instilling 0.9 % normal saline into the vacuum-sealed vials. Total saline reconstituted depends on the total units being administered, in addition to anatomical and clinical considerations. One must remember not to shake the vial, as this can disrupt the delicate disulfide bonds within the

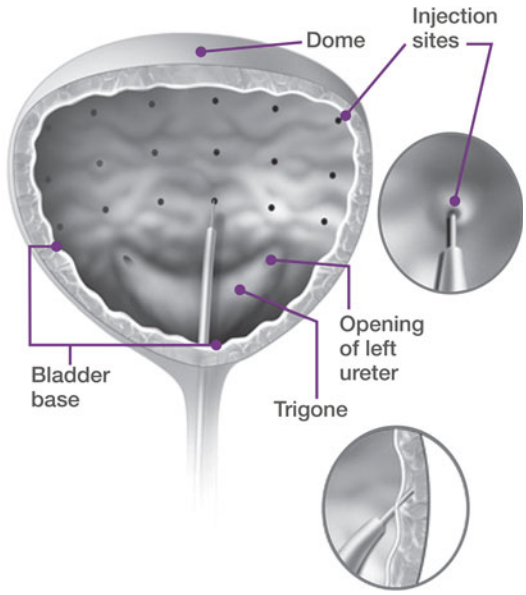


Fig. 17.6 Sites of injection for onabotulinum toxin A into the detrusor. The trigone should be spared, and sites spaced out equally to ensure uniform distribution of the toxin throughout the bladder (Copyright © Allergan, Inc., Irvine, CA. Used with permission 2013)

toxin, rendering it ineffective. Therefore, careful rotation and mixing of the vial is all that is needed for proper reconstitution.

Cystoscopy is briefly performed to map patient anatomy, visualize the trigone and ureteral orifices, and to rule out any papillary mass or lesion. The bladder should be filled with at least 100 mL of sterile water or saline. The reconstituted drug is then injected at a depth of 2 mm, under direct vision at 20–30 equally spaced sites throughout the detrusor, sparing the trigone (Fig. 17.6). The trigone is extremely sensitive especially in patients with only local anesthesia. Furthermore, there exists the theoretical risk of new-onset vesicoureteral reflux if the trigone is injected. Sites should be spaced approximately 1 cm apart. A superficial bleb occurs if the injection is done superficially in the submucosa, whereas no visual change will occur if the drug is injected too deep. A proper injection should result in a subtle visual change and rise of the mucosa under the injection site. The author's technique is to proceed from a left to right (or right to left) manner, beginning each column of injection sites at the bottom, to avoid any minor bleeding from a previous injection site seeping downward and preventing good visualization of the next injection site. For the final injection, 0.5–1 mL of sterile saline is injected in order to inject the remaining toxin, which remains within the needle sheath.

Post-procedure

In patients not already on intermittent catheterization, prior to being discharged after the injection procedure, patients should be able to void spontaneously. It is recommended that patients receive 1–3 days of antibiotics posttreatment in order to minimize the chance of urinary tract infection [Allergan PI]. Patients should be counseled that urinary tract infection is common, and they may have dysuria, or hematuria, as with any cystoscopic procedure.

Because of the mechanism of action of onabotulinum toxin A, the full effect of the procedure may not be noticed until approximately 2 weeks after the procedure. Patients should be scheduled for a visit around that time period in order to evaluate the effect, and a urinalysis, and post-void residual volume should be measured. In general, clean-intermittent catheterization should be initiated only in the setting of symptomatic urinary retention, or if adverse effects of elevated residual volume are deemed dangerous by the investigator, as elevated post-void residual volumes have been shown to be safe and tolerable in several studies in the urologic literature [20, 21]. In the author's opinion, residual volumes of under 350 mL can be treated without the use of intermittent catheterization, provided that patients are asymptomatic.

Adverse Effects of Onabotulinum Toxin A

While the clinical trials evaluating onabotulinum toxin A in both NDO and idiopathic OAB show that onabotulinum toxin A's use is relatively safe, there are important safety considerations which

Table 17.1 Adverse effects of injection of onabotulinum toxin A for neurogenic detrusor overactivity and overactive bladder

Local side effects
Urinary tract infection
Urinary retention
Hematuria
Systemic side effects
Asthenia
Generalized muscle weakness
Diplopia
Ptosis
Dysphagia
Dysphonia
Dysarthria
Urinary incontinence
Respiratory difficulty

practitioners must be aware of. In any instance when onabotulinum toxin A is used, there may be site-specific reactions, in addition to adverse effects due to distant spread of the toxin. Much of the risk lies not only in the site of injection but also in the dosing, as higher doses typically place patients at increased risk for some of the untoward side effects. Therefore, patients should be counseled about these risks specific to their indication (Table 17.1).

Risk of Intradetrusor Injection

Onabotulinum toxin A prevents the release of acetylcholine at the synaptic cleft by cleaving the SNAP protein SNARE-25. Acetylcholine is responsible for detrusor contraction, and by preventing its release, onabotulinum toxin A reduces uninhibited bladder contractions and episodes of urinary incontinence due to detrusor overactivity. However, this same effect leads to incomplete emptying in some patients, due to the decreased contractility of the detrusor after injection. When 300 units was injected in a randomized, placebo-controlled study, the most common adverse event associated with injection was urinary tract infection, with 57 and 55 % of patients developing an infection in both the study drug and placebo group, respectively [22]. In order to minimize this risk, many centers currently administer concurrent antibiotics at the time of injection, per the American Urological Association clinical guidelines on antibiotic prophylaxis with cystoscopic procedures [23].

Elevated post-void residuals are common in all doses, between 100 and 300 units of injection. However, the majority of patients are asymptomatic with respect to these elevations. Early studies on onabotulinum toxin A defined "urinary retention" based on residual volume measurements, and initiated clean intermittent catheterization independent of patient symptoms. In the phase III efficacy and tolerability study evaluating 200 and 300 units of onabotulinum toxin A in patients with neurogenic detrusor overactivity, beginning intermittent catheterization was determined based on the investigator, rather than a predefined residual volume [10]. In patients not already performing intermittent catheterization, particularly those with multiple sclerosis, approximately half (50 %) of patients receiving onabotulinum toxin A began to catheterize. Notably, a significant proportion (22 %) of placebo-injected patients also began to catheterize, which suggests that perhaps many of these patients may have benefitted from learning and performing intermittent catheterization well before being enrolled in the study. Overall, the incidence of urinary retention in NDO appears to be around 21 %.

The incidence of retention appears to be more common with larger doses, and in patients with neurogenic bladder. In the idiopathic OAB trials, investigators were able to lessen the risk of requiring patients begin intermittent catheterization by allowing patient symptoms and investigator judgement to be the determinant of starting intermittent catheterization [15, 20]. In all treatment arms, residual volumes increased significantly when compared to placebo [15]. This effect seemed to peak at week 2, with a gradual decrease between weeks 4 and 12, and the risk of patients having elevations in residual volume was dose dependent [15]. The mean duration of requiring catheterization was highest in the 200 unit group, with a median of 179 days.

By utilizing 100 unit dosing in the OAB population, and by initiating clean intermittent catheterization in patients who are symptomatic due to their retention (despite elevated post-void residual volumes) the true "retention" rate drops to around 6 % [20]. In this patient population, the fear of having to catheterize can be ameliorated by teaching patients how to catheterize before the procedure, as with proper hand function and education, this can be done easily. However, in patients with obesity, poor hand function, or those with challenging anatomy, teaching intermittent catheterization may be challenging, and therefore, indwelling catheters may be required until the effect of the onabotulinum toxin A has worn off.

Other less common side effects related to detrusor injection include hematuria (3–7 %), *increased* incontinence (7 %), and bladder pain (1–6 %) [10, 15]. However, it should be noted that many of these effects are seen in the placebo-treated arms, and therefore, are likely due to the procedure itself rather than the effects of the toxin.

Distant Non-Site Specific Effects

Distant spread of onabotulinum toxin A can occur during use in any site within the body. Death has been reported due to the distant spread of the toxin, however, as of 2013, none have been reported due to the specific toxin-related effects in any of the bladder-related studies [20]. Paralysis of distant muscle groups have been reported in both detrusor and sphincter injections [24, 25]. In a small percentage of patients, generalized muscular weakness can occur (3–7 %), in addition to fatigue (3–6 %), and headache (3–6 %) [10, 18]. These adverse effects can occur within hours to weeks after injection, and therefore patients must be counseled accordingly. In previous studies evaluating patients with NDO and compromised lung function, a small percentage (9–18 %) of patients experienced at least a 15–20 % decrease in forced vital capacity from baseline [18]. These pulmonary effects occurred between 2 and 6 weeks, and appeared to resolve by week 12. Therefore, patients with baseline muscular weakness (particularly the multiple sclerosis and spinal cord injury patient) must be counseled specifically regarding these potential distant effects and monitored closely throughout the posttreatment period.

Potential Long-Term Effects

Injection of onabotulinum toxin A appears to be safe and effective in the treatment of idiopathic OAB and detrusor overactivity due to neurological conditions such as multiple sclerosis and spinal cord injury. Because the effects of the toxin wear off over several months, the majority of patients will require repeat injections within a year of initial injection. There are limited studies evaluating long-term effects after repeated injections. Allergy has been described to onabotulinum toxin A, with the possibility of antibody-mediated degradation of the toxin leading to decreased efficacy over time [26].

Future/Off-Label Indications

Sphincter Injection

In patients with sphincter dyssynergia, onabotulinum toxin A may be injected into either the internal or external sphincter in order to provide relaxation of these muscles to allow for more coordinated voiding. Most studies evaluating sphincter injection have focused on patients with neurogenic bladder due to multiple sclerosis, spinal cord injury, or other neurologic disease with detrusor *external* sphincter dyssynergia. The toxin may also be injected at the bladder neck level, targeting patients with voiding dysfunction due to *internal* sphincter contraction, or primary bladder neck obstruction. Doses of the toxin have ranged from 50 to 200 units and the procedure can be performed in a variety of ways (transurethrally, transperineally, with and without EMG guidance) [27–29]. A recent review of the studies on urethral injection have shown that patients with obstructed flow or sphincter dysynergy can have improvements in several urodynamic parameters, including flow, residual urine

volume, maximal urethral closure pressure, and need for catheterization [28]. Optimal dosing and duration of effect has yet to be determined, although the mean duration of effect in the available studies was 4.6 months.

Pelvic Floor Muscle Dysfunction

High tone pelvic floor muscle dysfunction is characterized by hypertonus of the muscles of the levator ani complex. Pelvic floor spasm may cause chronic pelvic pain, dyspareunia, and voiding and defecatory dysfunction. This condition often exists concomitantly with other pelvic conditions but may present as the most bothersome symptom. Current literature on pelvic floor muscle dysfunction is limited to the use of behavioral therapy, and pelvic floor physical therapy, although a few recent studies have shed some light on the benefit of pharmacological intervention in these patients [30].

In patients with pelvic floor muscle spasm, onabotulinum toxin A injection has been reported to have a potential benefit. While pelvic floor physical therapy remains the mainstay of treatment, injection of onabotulinum toxin A may benefit some refractory patients. In one of the few randomized studies published, Abbott and colleagues injected sixty women with pelvic floor spasm with either 80 units of onabotulinum toxin A or placebo [31]. Patients were followed for 26 weeks, and there was a significant reduction in pain and dyspareunia in the onabotulinum toxin A group versus placebo. Manometry in these patients confirmed that injection of onabotulinum toxin A decreased vaginal resting pressures after injection.

Injections may be done transperineally or transvaginally under anesthesia or in the office setting [32]. In the author's experience, transvaginal injection is the modality of choice, as trigger points can aid the investigator in determining the proper site of injection. An Iowa Trumpet needle guide may be used, along with a 6″ small gauge spinal needle, which pierces the vaginal mucosa and enters the levator muscles. One must withdraw on the syringe in order to ensure no intravascular injection. Doses in previous studies have ranged from 20 to 450 units. In the author's experience, typically 50 units per affected muscle group are injected, with a dilution of 10–30 units per mL. As in other conditions, the optimal dosing and duration of effect is still unknown.

Interstitial Cystitis/Painful Bladder Syndrome

Only a handful of studies have evaluated injection of onabotulinum toxin A in the management of Interstitial Cystitis and Painful Bladder Syndrome. Many of the studies have focused on a dose ranging between 100 and 200 units. Because it is theorized that a defect in the urothelium exists in these patients, several of the studies evaluated submucosal injection, in an attempt to block the release of acetylcholine at the afferent nerve level [33–35]. These studies are limited in patient number with the largest study to date having only 31 patients [34, 36]. There does appear to be some effect, albeit not as impressive as the OAB or NDO trials. In the aforementioned trials, mean daytime urinary frequency improved from 12.5 to 54 %, with improvements in visual analog pain scales ranging from 6 to 79 %. Current recommendations from the American Urological Association Guidelines on the management of Interstitial Cystitis and Painful Bladder Syndrome include detrusor injection of onabotulinum toxin A as a fifth-line therapy for this disease [37]. Of course, patients choosing this therapy must be willing to catheterize or accept indwelling foley catheterization, a side effect which precludes its use in many of these patients.

Vulvodynia

While onabotulinum toxin A may improve pain in women presenting with obvious pelvic floor muscle spasm, its usefulness in treating vulvar disease is less clear. Only one randomized, placebo-controlled clinical trial had evaluated onabotulinum toxin A injection into the vestibule for provoked vulvar vestibulodynia. Interestingly, in both placebo and onabotulinum toxin A

groups, a significant reduction in pain was seen on visual analog scale at 6 months follow-up ($p < 0.001$) [38]. However, no difference was seen between the placebo or onabotulinum toxin A groups ($p = 0.635$). At this time, there appears to be no role in treating vestibulodynia in women.

Conclusions

With the advent of onabotulinum toxin A, the practitioner has an important tool in his or her armamentarium when treating lower urinary tract and pelvic dysfunctions. The early studies showing benefit in neurogenic bladder have paved the way for its use in idiopathic overactive bladder, and likely for its use in several other spastic disorders involving the male and female pelvis. Future studies will be required to better understand ideal dosing, and to identify additional indications for this unique therapy.

References

1. Erbguth FJ, Naumann M. Historical aspects of botulinum toxin: Justinus Kerner (1786-1862) and the "sausage poison". Neurology. 1999;53(8):1850–3.
2. Dolly O. Synaptic transmission: inhibition of neurotransmitter release by botulinum toxins. Headache. 2003;43 Suppl 1:S16–24.
3. Brin MF. Botulinum toxin: chemistry, pharmacology, toxicity, and immunology. Muscle Nerve. 1997; 6(Suppl):S146–68.
4. Kruht J, Zvara P. Intravesical instillation of botulinum toxin A: an in vivo murine study and pilot clinical trial. Int Urol Nephrol. 2011;43(2):337–43.
5. Chapple CR. Muscarinic receptor antagonists in the treatment of overactive bladder. Urology. 2000;55:33.
6. Wagg A, Compion G, Fahey A, Siddiqui E. Persistence with prescribed antimuscarinic therapy for overactive bladder: a UK experience. BJU Int. 2012;110(11): 1767–74.
7. D'Souza AO, Smith MJ, Miller LA, Doyle J, Ariely R. Persistence, adherence, and switch rates among extended-release and immediate-release overactive bladder medications in a regional managed care plan. J Manag Care Pharm. 2008;14(3):291–301.
8. Hollingworth W, Campbell JD, Kowalski J, et al. Exploring the impact of changes in neurogenic urinary incontinence frequency and condition specific quality of life on preference-based outcomes. Qual Life Res. 2010;19:323.
9. Schurch B, Stohrer M. Botulinum-A toxin for treating detrusor hyperreflexia in spinal cord injured patients: a new alternative to anticholinergic drugs? Preliminary results. J Urol. 2000;164(3 Pt 1):692–7.
10. Ginsberg D, Gousse A, Keppenne V, Sievert KD, Thompson C, Lam W, Brin MF, Jenkins B, Haag-Molkenteller C. Phase 3 efficacy and tolerability study of onabotulinumtoxinA for urinary incontinence from neurogenic detrusor overactivity. J Urol. 2012;187(6):2131–9.
11. Stewart WF, Van Rooyen JB, Cundiff GW, et al. Prevalence and burden of overactive bladder in the United States. World J Urol. 2003;20:327.
12. Irwin DE, Milsom I, Hunskaar S, et al. Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC study. Eur Urol. 2006;50:1306.
13. Brøstrom S, Hallas J. Persistence of antimuscarinic drug use. Eur J Clin Pharmacol. 2009;65:309.
14. Brubaker L, Richter HE, Visco A, Mahajan S, Nygaard I, Braun TM, Barber MD, Menefee S, Schaffer J, Weber AM, Wei J, Pelvic Floor Disorders Network. Refractory idiopathic urge urinary incontinence and botulinum A injection. J Urol. 2008;180(1):217–22.
15. Dmochowski R, Chapple C, Nitti VW, Chancellor M, Everaert K, Thompson C, Daniell G, Zhou J, Haag-Molkenteller C. Efficacy and safety of onabotulinumtoxinA for idiopathic overactive bladder: a double-blind, placebo controlled, randomized, dose ranging trial. J Urol. 2010;184(6):2416–22.
16. Sahai A, Dowson C, Khan MS, Dasgupta P. Improvement in quality of life after botulinum toxin-A injections for idiopathic detrusor overactivity: results from a randomized double-blind placebo-controlled trial. BJU Int. 2009;103(11):1509–15.
17. Visco AG, Brubaker L, Richter HE, Nygaard I, Paraiso MF, Menefee SA, Schaffer J, Lowder J, Khandwala S, Sirls L, Spino C, Nolen TL, Wallace D, Meikle SF, Pelvic Floor Disorders Network. Anticholinergic therapy vs. onabotulinumtoxinA for urgency urinary incontinence. N Engl J Med. 2012;367(19):1803–13.
18. Botox (botulinum toxin type A) package insert. Irvine, CA, Allergan Pharmaceuticals.
19. Shergill IS, Arya M, Hamid R, Khastgir J, Patel HR, Shah PJ. The importance of autonomic dysreflexia to the urologist. BJU Int. 2004;93(7):923–6.
20. Nitti VW, Dmochowski R, Herschorn S, Sand P, Thompson C, Nardo C, Yan X, Haag-Molkenteller C, EMBARK Study Group. OnabotulinumtoxinA for the treatment of patients with overactive bladder and urinary incontinence: results of a Phase 3, randomized placebo controlled trial. J Urol. 2013;189(6): 2186–93.
21. Ingber MS, Vasavada SP, Moore CK, Rackley RR, Firoozi F, Goldman HB. Force of stream after sling therapy: safety and efficacy of rapid discharge care pathway based on subjective patient report. J Urol. 2011;185(3):993–7.

22. Herschorn S, Gajewski J, Ethans K, Corcos J, Carlson K, Bailly G, Bard R, Valiquette L, Baverstock R, Carr L, Radomski S. Efficacy of botulinum toxin A injection for neurogenic detrusor overactivity and urinary incontinence: a randomized, double-blind trial. J Urol. 2011;185(6):2229–35.
23. Wolf Jr JS, Bennett CJ, Dmochowski RR, Hollenbeck BK, Pearle MS, Schaeffer AJ, Urologic Surgery Antimicrobial Prophylaxis Best Practice Policy Panel. Best practice policy statement on urologic surgery antimicrobial prophylaxis. J Urol. 2008;179(4):1379–90.
24. Wyndaele JJ, van Dromme SA. Muscular weakness as side effect of botulinum toxin injection for neurogenic detrusor overactivity. Spinal Cord. 2002;40:599–600.
25. De Laet K, Wyndaele JJ. Adverse events after botulinum A toxin injection for neurogenic voiding disorders. Spinal Cord. 2005;43:397–9.
26. Schulte-Baukloh H, Bigalke H, Heine G, et al. Antibodies against botulinum neurotoxin type A as a cause of treatment failure after the first detrusor injection. Urology. 2007;69(575):e13–5.
27. Kuo HC. Botulinum A toxin urethral injection for the treatment of lower urinary tract dysfunction. J Urol. 2003;170:1908–12.
28. Dykstra DD, Sidi AA, Scott AB, Pagel JM, Goldish GD. Effects of botulinum A toxin on detrusor-sphincter dyssynergia in spinal cord injury patients. J Urol. 1988;139:919–22.
29. Schulte-Baukloh H, Weiss C, Stolze T, et al. Botulinum-A toxin detrusor and sphincter injection in treatment of overactive bladder syndrome: objective outcome and patient satisfaction. Eur Urol. 2005; 48:984–90. discussion 990.
30. Rogalski MJ, Kellogg-Spadt S, Hoffmann AR, Fariello JY, Whitmore KE. Retrospective chart review of vaginal diazepam suppository use in high-tone pelvic floor dysfunction. Int Urogynecol J. 2010;21(7):895–9.
31. Abbott JA, Jarvis SK, Lyons SD, Thomson A, Vancaille TG. Botulinum toxin type A for chronic pain and pelvic floor spasm in women: a randomized controlled trial. Obstet Gynecol. 2006;108:915–23.
32. Goldstein A. Intralevator injection of botulinum toxin for hypertonic pelvic floor. J Sex Med. 2011;8: 1287–90.
33. Smith CP, Radziszewski P, Borkowski A, Somogyi GT, Boone TB, Chancellor MB. Botulinum toxin a has antinociceptive effects in treating interstitial cystitis. Urology. 2004;64(5):871–5.
34. Kuo HC. Repeated intravesical onabotulinumtoxinA injections are effective in treatment of refractory interstitial cystitis/bladder pain syndrome. Int J Clin Pract. 2013;67(5):427–34.
35. Giannantoni A, Porena M, Costantini E, Zucchi A, Mearini L, Mearini E. Botulinum A toxin intravesical injection in patients with painful bladder syndrome: 1-year follow up. J Urol. 2008;179(3):1031–4.
36. Pinto R, Lopes T, Frias B, Silva A, Silva JA, Silva CM, Cruz C, Cruz F, Dinis P. Trigonal injection of botulinum toxin A in patients with refractory bladder pain syndrome/interstitial cystitis. Eur Urol. 2010; 58(3):360–5.
37. Hanno PM, Burks DA, Clemens JQ, Dmochowski RR, Erickson D, Fitzgerald MP, Forrest JB, Gordon B, Gray M, Mayer RD, Newman D, Nyberg Jr L, Payne CK, Wesselmann U, Faraday MM, Interstitial Cystitis Guidelines Panel of the American Urological Association Education and Research, Inc. AUA guideline for the diagnosis and treatment of interstitial cystitis/bladder pain syndrome. J Urol. 2011;185(6): 2162–70.
38. Brown CS, Glazer HI, Vogt V, Menkes D, Bachmann G. Subjective and objective outcomes of botulinum toxin type A treatment in vestibulodynia: pilot data. J Reprod Med. 2006;51:635–41.

Construction of the Neovagina

18

Cecile A. Unger and Marie Fidela R. Paraiso

Introduction

Creation of a neovagina is usually necessary in the following cases: congenital absence of the vagina, gender assignment surgery, vaginal contracture and stenosis, and reconstruction following neoplastic resective surgery or radiotherapy. While there is no standard procedure for neovaginal reconstructive surgery, there exist many surgical and nonsurgical techniques that are often used to create the vagina. These techniques include vaginal dilation methods, the McIndoe vaginoplasty procedure with the use of split-thickness skin grafts, modified McIndoe procedures using full-thickness skin and mucosal grafts, transpositional skin graft techniques, laparoscopic techniques including the Davydov and Vecchietti operations, myocutaneous and fasciocutaneous pedicled flap surgeries, and intestinal flap surgeries. The ideal reconstructive method should provide a patent vaginal canal of adequate length, width, and texture that will allow for sexual intercourse, provide a cosmetically appealing appearance with minimal morbidity of both the recipient and donor surgical sites, and have a low incidence of overall complications. Construction of the neovagina can be very complex and challenging. Each method of repair has its advantages and disadvantages, which should be carefully weighed with the desired treatment goals as well as the surgeon's experience with various surgical techniques.

C.A. Unger, M.D., M.P.H. (✉)
M.F.R. Paraiso, M.D.
Department of Obstetrics and Gynecology,
Women's Health Institute, Center for Urogynecology and Pelvic Reconstructive Surgery, Cleveland Clinic, Cleveland, OH, USA
e-mail: ungerc@ccf.org; paraism@ccf.org

Indications

Creation of a neovagina is usually necessary in the following cases: congenital absence of the vagina, gender assignment surgery, vaginal contracture and stenosis, and reconstruction following neoplastic resective surgery or radiotherapy.

Approximately 1 in 4,000 to 1 in 10,000 female newborns are born with congenital absence of the vagina [1]. The most common cause of this congenital malformation is Mayer–Rokitansky–Kuster–Hauser Syndrome (MRKH). The anomaly results from hypoplasia or agenesis of the mullerian duct system. The phenotype of this anomaly exists on a wide spectrum and may include partial or total vaginal agenesis with a hypoplastic or rudimentary uterus and fallopian tubes. Patients have a normal 46,XX karyotype, normal female phenotype, and normal ovarian hormonal and oocyte function. On exam, patients have normal external genitalia with a normal-appearing hymenal ring and a small vaginal pouch with a dimple. These patients often present in the setting of primary amenorrhea or once sexual

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0_18,

intercourse proves to not be possible. Up to 10 % of patients have a functional endometrium, which can lead to cyclic pain and associated hematometra [2]. These adolescent patients most often present in the setting of primary amenorrhea and painful hematocolpos. Concomitant urologic anomalies such as unilateral renal agenesis, pelvic or horseshoe kidney, and anomalies of the collecting duct system occur in up to 50 % of patients, while 10–15 % of patients also have skeletal anomalies [3].

A shortened vagina, or blind vaginal pouch, is also found in genetic syndromes such as Morris syndrome and Turner's syndrome [4], as well as disorders of sex development including Androgen Insensitivity Syndrome (AIS) and Congenital Adrenal Hyperplasia (CAH). These conditions often require sex assignment surgery in either childhood or adolescence. AIS is an X-linked disorder caused by a mutation in the androgen receptor gene that leads to peripheral androgen resistance [5]. The complete form of AIS occurs in approximately 1:20,000 of individuals who are born 46,XY with normal-appearing female genitalia but with sparse pubic hair and a blind vaginal pouch [6]. CAH is a result of several different inherited defects of the steroid synthesis pathway. In cases of 17 α-hydroxylase deficiency, 46,XY individuals will present with normal-appearing female external genitalia, a blind short vaginal pouch, no uterus or fallopian tubes, and intra-abdominal testes [6]. This phenotype is referred to as "complete male pseudohermaphroditism." These patients frequently have abnormal-appearing external genitalia in addition to a shortened vagina, and their reconstructive surgical needs may be extensive.

An immediate partial or total vaginal reconstruction is frequently necessary in cases of extirpative or extensive radical pelvic surgery for cancer treatment. Anterior exenteration procedures are commonly performed for invasive bladder carcinoma [7], while total exenteration procedures are considered salvage operations for recurrent gynecologic cancers such as cervical, uterine, vulvar, and vaginal cancer [8]. Exenteration procedures involve removal of the pelvic organs en bloc and result in significant tissue defects that cannot be closed primarily.

While there is no standard procedure for neovaginal reconstructive surgery, there exist many surgical and nonsurgical techniques that are often used to create the vagina. These techniques include the following:

1. Vaginal dilation methods with and without physical therapy
2. McIndoe vaginoplasty procedure with the use of split-thickness skin grafts
3. Modified McIndoe procedures using full-thickness skin and mucosal grafts
4. Transpositional skin graft techniques
5. Laparoscopic techniques including the Davydov and Vecchietti operations
6. Myocutaneous and fasciocutaneous pedicled flap surgeries
7. Intestinal flap surgeries

All of these techniques will be described in detail in this chapter.

Preoperative Assessment and Planning

Patient evaluation begins with a detailed physical exam. In adolescence and young adulthood, office examination with pelvic and/or rectal exam is the first step of evaluation. In the pediatric population, vaginoscopy is the diagnostic standard for evaluating the lower reproductive tract, and is usually performed in the operating room. Patients who are suspected of having a congenital anomaly or disorder of sex differentiation should have a karyotype and hormonal evaluation. Additionally, imaging is important to help elucidate which mullerian structures are present and which are absent. T2-weighted magnetic resonance imaging (MRI) of the vagina can show a high intensity central mucosa surrounded by a low intensity submucosal layer [9]. With a marker on the perineum, the distance of an obstructed vagina from the perineum can be determined [10], delineating the length of the agenesis, which is helpful for preoperative planning. MRI is also helpful in determining the presence of the uterus, cervix, and ovaries.

MRI has been shown to have 100 % sensitivity and specificity in diagnosing MRKH syndrome in patients who are suspected of having vaginal agenesis on physical exam [9]. Transabdominal ultrasonography is useful for evaluating the presence of pelvic organs as well as detecting associated urologic anomalies. Transperineal ultrasound can be used to measure the length of the agenesis as well, especially in the setting of hematocolpos. Understanding the anatomy is the key to successful neovagina reconstruction, which is why the preoperative assessment is so important.

Goals of Therapy

Timing for nonsurgical or surgical creation of a neovagina is elective; however, it is best planned when the patient is emotionally mature. Many of these surgeries are performed in adolescence or in young adulthood. Additionally, it is important to ensure that the patient has a strong support system. The best predictor of favorable emotional outcomes after diagnosis and surgery is a positive relationship between the patient and her guardians and the patient's feeling of self-efficacy with regard to being able to share her feelings with family and friends [11].

The ideal reconstructive method should provide a durable, stable vaginal canal of adequate size and texture that will allow for sexual intercourse, provide a natural aesthetic appearance while simultaneously minimizing morbidity of both the recipient and donor sites, and have a low incidence of overall complications. The most preferable methods also preclude the need for subsequent long-term dilation or need for stents or obturators, as well as lubricants, and can be done in a single-stage fashion [6]. Conservative management is an option for most patients and should strongly be considered as first-line therapy. There are also several surgical procedures that are indicated for neovaginal reconstruction. There is currently no consensus in the literature about the best approach for this type of surgery. The approach should be based on the experience of the surgeon performing the surgery, taking into consideration that the initial surgery is the most likely to be effective and that repeat surgeries may become more challenging with less successful outcomes over time [12].

Conservative Management

Conservative approaches to the creation of a neovagina can be attempted in patients with congenital absence of the vagina. When vaginal development fails, a soft, pliable span of skin is usually left between the urethral meatus and the anus [13]. Nonoperative creation of a vagina can be achieved through progressive dilation and invagination of the perineal epithelium. To achieve this, the patient must have a shallow vaginal pouch that can be stretched to lengthen the canal [14]. Conservative therapy becomes most challenging in patients with no vagina or only a small vaginal dimple, but should still be attempted as success has been demonstrated in these patients as well [15]. The American Congress of Obstetrics and Gynecology recommends dilation as first-line therapy for vaginal lengthening in patients with agenesis of the vagina, as it is the least invasive approach, is very safe with significantly less morbidity than surgical alternatives, is not associated with disfiguring scarring, and has highly favorable outcomes [16]. In 1938, Frank [17] was the first to formally describe vaginal formation with progressive dilation by using Pyrex glass molds and applying persistent pressure to the perineum. Today, Frank's method includes the use of silicon dilators to invaginate the vaginal pouch with progression of dilator size until a functional vagina is created. Daily dilation, usually performed in lithotomy position, is required for at least 30 min each day [6]. The Ingram [18] modification of Frank's technique involves having the patient sit on a bicycle seat stool for progressive dilation, using body weight to maintain adequate dilator pressure. This method has also showed very favorable outcomes [19], but is associated with more discomfort and less patient compliance. Patients should be counseled on the need for good compliance, as well as the anatomic and functional expectations once

Fig. 18.1 Vaginal dilators. Vaginal dilators come in variable lengths and sizes (Used with permission from Ridgeway BR, Attaran M. Embryology and Congenital Anomalies of the Urinary Tract, Rectum, and Female Genital System. In Walters MD, Karram MM eds.: Urogynecology and Reconstructive Pelvic Surgery, 4th ed. Philadelphia: Elsevier; 2014)

the process is complete. Anatomic and functional success rates are as high as 90 % if there is good compliance with dilator use [20]. Roberts et al. [19] followed 51 patients, finding 92 % success in those adhering to a vaginal dilation regimen using Frank's technique. Mean time to successful dilation was 11.8 months, with a range of 3–33 months. Younger patients have been found to have the poorest compliance with dilator use [17] as the technique is time-intensive and uncomfortable, especially at the beginning of the process. These patients especially must be counseled about the commitment required for this technique, as well as the projected length of time it will take to achieve a vagina of adequate length and caliber. Figure 18.1 is an image of vaginal dilators, showing that they come in variable lengths and sizes.

Physical therapy can also be used as an adjunct to vaginal dilators in the creation of the neovagina. Vaginal dilators are widely used by physical therapists for the treatment of pelvic floor disorders such as pelvic floor hypertonicity causing pelvic pain, vaginismus, vulvodynia, and dyspareunia [21]. They are used in desensitization therapy using graded exposure with a progressive increase in size of the dilator in order to treat dyspareunia [22]. Physical therapy using various heat modalities to make the tissues more pliable in conjunction with manual stretching by a therapist while the patients continues to use dilators on her own is associated with a shorter length of treatment to attain a functional vagina [23] and may be a good option for some patients and should be considered.

Surgical Management

Surgery is indicated for patients who are unsuccessful with dilators or patients who opt for surgical management after they have been thoroughly counseled about the risks and the benefits of surgery. The patient should be counseled that surgical management with vaginoplasty is not necessarily a "quick fix" and that she will need to use vaginal dilators postoperatively to maintain her surgically created vagina. Again, the goals of therapy involve the creation of a vaginal canal that is of adequate length and caliber, in the correct axis, with some secretory capacity that will allow for sexual intercourse to occur without the need for lifelong dilation. The timing of the surgery depends on the patient and the type or procedure planned. Surgeries often are performed in late adolescence when the patient is more mature and better able to adhere to postoperative dilation or instructions [24].

McIndoe Procedure

The most common surgical procedure performed in the United States to create a neovagina is the McIndoe operation, which is commonly used to treat patients with congenital absence of the vagina. The primary goal is creation of a functional vagina. The technique involves creating a canal within the connective tissue between the bladder and the rectum and using a mold to line the vagina with a split-thickness skin graft (STSG) obtained from the patient's thigh, inguinal region, or buttocks [25] followed by progressive vaginal dilator use to achieve maximal vaginal length and caliber. In order to perform this technique, the patient is placed in dorsal

supine lithotomy position. Laparoscopy may be performed first, even in cases when an intraabdominal graft is not used, in order to delineate organs such as the bladder and rectum prior to commencing the rectovesical dissection from below. Next, the vaginal dimple or foreshortened vagina is identified and a 3 cm transverse incision is made across it. Dissection is then done, using mostly blunt technique, first creating two channels on either side of the median raphe of the perineum (Fig. 18.2a). Gentle pressure is applied cephalad during dissection to create the canal with a goal depth of approximately 10–12 cm (Fig. 18.2b). Care should be taken during the dissection to avoid entry into the bladder, rectum, and posterior cul-de-sac. An EEA sizer may be used in the rectum to help with dissection. Prior to dissection, a split-thickness graft is harvested and should be approximately 10×20 cm in size and kept moist using normal saline during the canal dissection. The graft is passed through a skin mesher, which perforates the tissue, expanding the surface area of the graft while permitting egress of blood and fluid from the surgical site postoperatively. Once dissection is complete, the graft is placed over a dilator-like mold in an "inside-out" fashion so that the external portion of the skin lies against the mold (Fig. 18.2c). Placement should be symmetric so that the tip of the mold is at the middle of the graft with the long axis of the graft draped along the long axis of the mold on both sides. The mold is then placed inside the dissected vaginal canal and the edges of the graft are then everted and sutured in an interrupted fashion to the distal opening of the neovagina using 3-0 or 4-0 delayed absorbable suture (Fig. 18.2d, e). Patients are advised about modified or complete bed rest to avoid having the mold or stent fall out and placing tension on the distal sutures. The labia majora are often sewn together over the stent to keep it in place. A foley catheter is left in place for 5–7 days postoperatively, and is removed at the time of mold or stent removal, which is done carefully during an exam under anesthesia so as to not avulse the graft from the underlying connective tissue. Patients subsequently undergo a dilation process that can last several months to a year depending upon patient and tissue compliance.

Studies have shown excellent results after the McIndoe operation. After 12 months, Seccia et al. [4] found that out of 32 patients, 90 % of patients presented with complete skin graft take and 84 % reported normal sexual activity with good sensitivity. The most common postoperative complications were anxiety (~6 % of patients) related to possible pain during insertion of the dilator and keloid scarring on the donor site of the skin grafts (~3 % of patients). Klingele et al. [20] looked at patient satisfaction with the McIndoe procedure and reported that 79 % believed that the procedure improved their quality of life, and 91 % were sexually active. Other potential complications reported in the literature include graft rejection, contraction of the graft, hematoma, infection, fistula formation, and excess granulation tissue [26–28].

Modifications of the McIndoe

A modification to the McIndoe procedure is the technique used to open the perineum prior to dissection. A triangular-shape inferiorly based flap approximately 3–4 cm in size can be created as the initial incision, which can then be sutured to the graft placed on the stent [29]. This method of opening can provide additional length to the neovagina, and also help create a tension-free reapproximation of the graft to the neointroitus.

A second modification to the standard McIndoe procedure is the addition of a laparoscopic intraperitoneal repair. Laparoscopically, the bladder is retrograde filled to facilitate visualization of its margins. The peritoneum is grasped at the superior edge of the bladder margin and opened and then dissected off of the underlying bladder muscularis. Dissection can be facilitated with injection of normal saline to create a hydrodissection plane. A retropubic dissection is sometimes necessary to further mobilize the peritoneum and to release the bladder from the pubic symphysis, which brings the peritoneum closer to the graft implant site. After the rectovesical dissection is complete and the mold is placed with the graft in its desired location, the peritoneal flap can be used to cover the mold. Bowel epiploica as

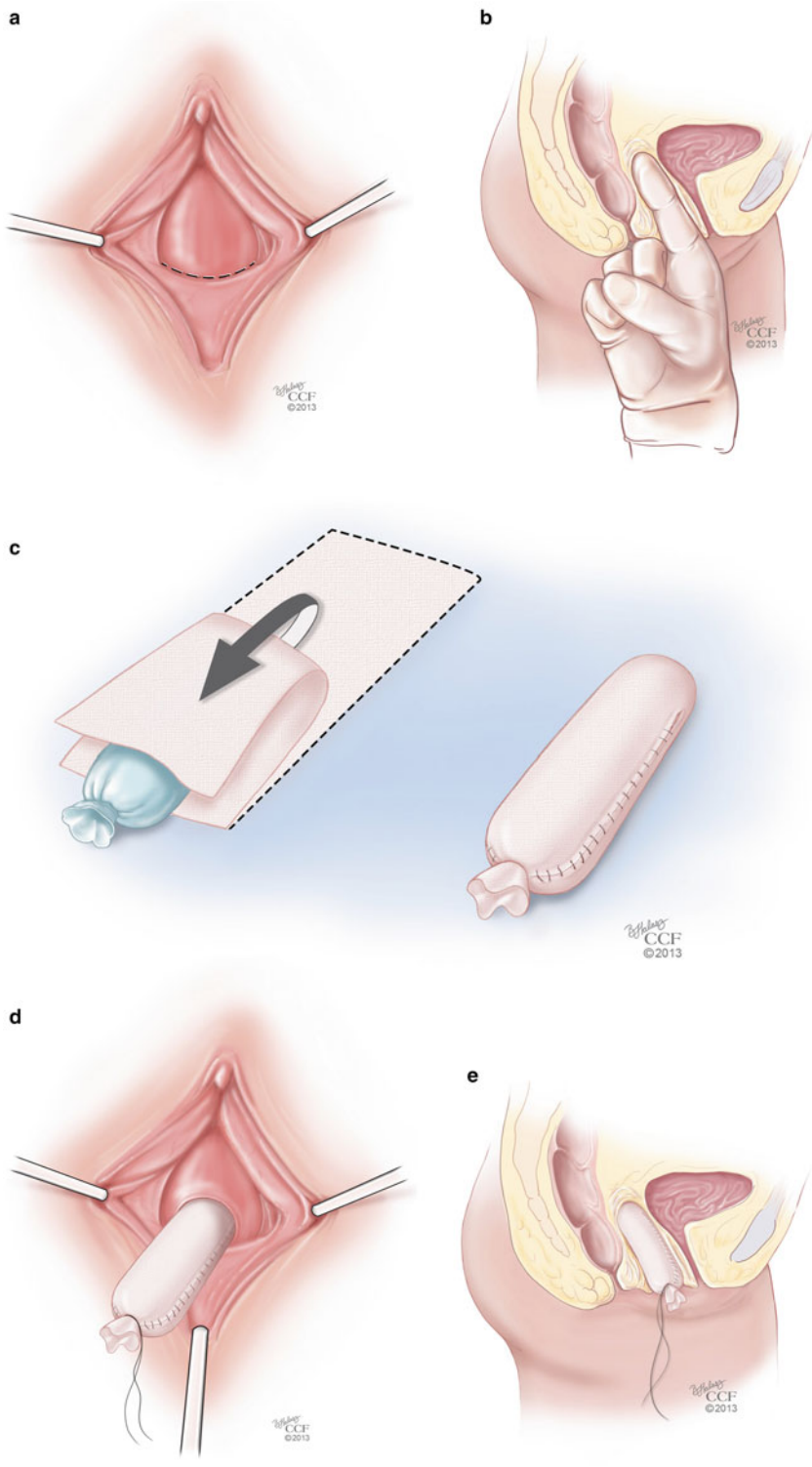


Fig. 18.2 (**a**–**e**) McIndoe procedure. (**a**) The initial step of the McIndoe procedure involves the identification of the vaginal dimple and the creation of a 3 cm transverse incision across it. (**b**) Blunt dissection can be done in the space between the rectum and the bladder in order to create the canal with a goal depth of approximately 10–12 cm. (**c**) Once dissection is complete, the graft is placed over a dilator-like mold in an "inside-out" fashion so that the external portion of the skin lies against the mold. (**d**) The mold is then placed inside the dissected vaginal canal. (**e**) Sagittal view of the mold inside the vaginal canal (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

well as the omentum, if it can be mobilized, can also be used to cover the mold.

The McIndoe procedure also has several modifications related to the type of tissue or material that is used to line the neovagina. While the split-thickness skin graft has been described as a safe and low-morbidity technique [25, 27], it has significant disadvantages. The use of a STSG can be associated with a high contracture rate, even when patients are compliant with vaginal dilation, and this can lead to inadequate vaginal length and caliber [19, 30]. Reported modifications of the McIndoe technique include replacement of the STSG with various alternatives, including autologous full-thickness skin grafts (FTSG), human amnion, peritoneum, bladder mucosal grafts, xenografts, and synthetic graft material [28, 31–33]. Techniques using FTSG are less likely to lead to neovaginal contracture and stenosis and do not require prolonged stenting [33, 34]. In addition, sebaceous and sweat glands are better preserved in these grafts, which can help with lubrication of the neovagina in some patients [35]. Akin describes a technique using a FTSG from the inner groin areas [29] (Fig. 18.3a). These grafts are used in a similar fashion to the STSG used with the traditional McIndoe procedure (Fig. 18.3b). Younger patients who require neovagina reconstruction may have limited potential graft sites for FTSG harvesting. Techniques such as tissue expander placement in sites such as the bilateral groins have been described with good outcomes and limited morbidity to the donor sites [36]. While FTSG confer many advantages when compared to STSG, the disadvantages of these grafts include skin texture mismatch and unwanted hair growth. Additionally, donor-site morbidity is slightly higher than with STSG and short-term dilation is still required with this technique.

The ideal lining for the vagina is a moist mucosa; however, there are limited donor sites for this type of graft. An option includes lining the neovaginal cavity with multiple full-thickness buccal mucosal grafts. The advantage of this type of grafting is that the neovagina is lined with mucosa, which is moist and may facilitate pleasurable intercourse. Additionally, the donor site heals well with virtually no morbidity [37]. The use of autologous buccal mucosa to reconstruct the vagina was presented in 2003 in two separate publications. Lin et al. [38] used complete pieces of full-thickness harvested mucosal grafts

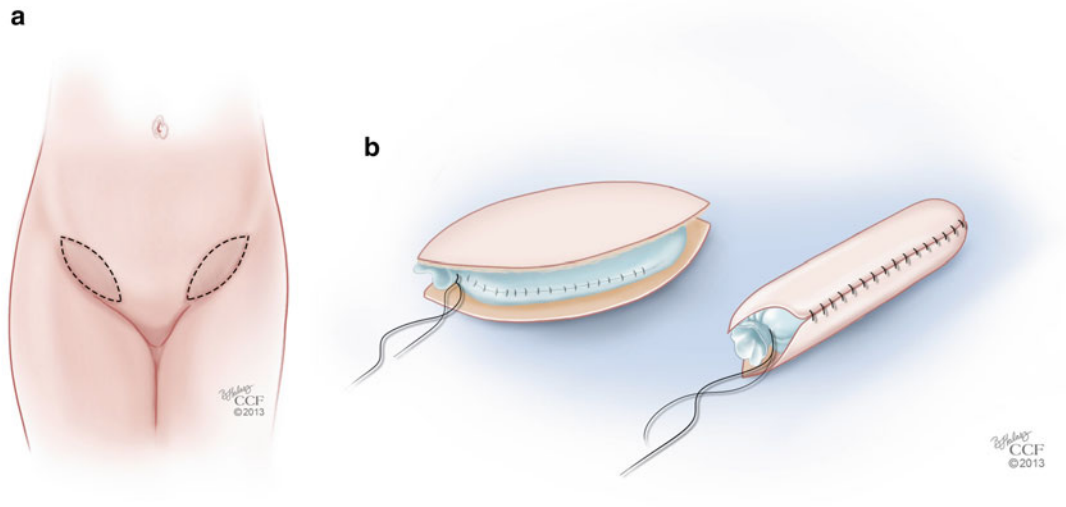


Fig. 18.3 (**a**, **b**) Modification of the McIndoe procedure. (**a**) Technique using full-thickness skin grafts (FTSG) from the inner groin. (**b**) FTSG placed over a mold (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

(approximately 6–7 cm × 2–3 cm) from both cheeks to line the neovagina; each graft was expanded in size by making stab incisions throughout the grafts, which were sutured over a stent and then placed in an inside-out fashion into the dissected vesicorectal space. The stent is removed once the graft takes to the underlying tissue. Ozgenel et al. [30] described a similar technique of harvesting the same mucosal grafts, expanding them with stab incisions, but then dividing them into several smaller pieces to cover a larger area over the stent. Another technique involves harvesting two buccal grafts, expanding them and then mincing the grafts into tiny pieces and then spreading the micromucosa graft onto the surfaces of gelatin sponge strips which are then placed over a stent and introduced into the dissected cavity, left in place until the graft takes to the underlying tissue [37]. Biologic grafts may also be used to line the neovagina and obviate the need for autologous tissue, which confers many advantages in terms of donor-site morbidity. Acellular dermal allografts such as Alloderm® (LifeCell Corps., Woodlands, TX), porcine dermal grafts such as Permacol® (Covidien, Mansfield, MA), and porcine intestinal submucosa grafts such as Surgisis® (Cook Medical Inc., Bloomington, IN) may be options in neovagina reconstruction. All these grafts are composed of an acellular collagen scaffold that provides a bridge for tissue incorporation and neovascularization. Research on the role of these materials for reconstruction is sparse, with the exception of Alloderm®, which has yielded successful outcomes in vulvovaginal reconstructive cases [39].

Tissue engineering to generate vaginal cells is being studied as an alternative approach to lining the neovagina at the time of the McIndoe procedure. Construction of a functional vagina using autologous cells expanded from a small vaginal biopsy was successful in a rabbit model [40]. And, in 2007, Panici et al. [41] reported the first case of neovaginal construction using autologous in-vitro cultured vaginal tissue. A small skin biopsy can be used to culture vaginal tissue, which can be used as a graft at the time of McIndoe vaginoplasty. Early results demonstrate a vagina with normal length and depth with vaginal tissue present on biopsy [41]. Further research is needed in tissue engineering, in its use for vaginoplasty, and long-term outcomes associated with this type of procedure.

Williams Procedure

The Williams Procedure is a vaginoplasty technique that has been employed in vaginal agenesis patients that involves creating a vulvar skin flap that is then sutured in place to create a neovagina. Williams [42] describes placing allis clamps on the vulvar tissue and applying gentle traction. A U-shaped incision is made extending across the perineum and up to the medial side of the labia (Fig. 18.4a). The upper edge of the incision is made 4 cm laterally and up to the level of the external urethral meatus. The skin flap is sharply dissected off of the underlying tissues creating a flap that can be mobilized inwards, creating a vaginal pouch (Fig. 18.4b). Once the tissues are mobilized, a first layer of sutures is placed between the inner skin margins using interrupted 0 delayed absorbable sutures, starting posteriorly and moving anteriorly (Fig. 18.4c). A second layer of sutures of the same material is used to approximate the subcutaneous fat and the perineal muscles (Fig. 18.4d). Lastly, the external skin is sutured with interrupted stitches (Fig. 18.4e). The Creatsas modification of the Williams vaginoplasty involves using electrocautery to open the hymen at the 3, 6, and 9 O'clock positions, which further opens the introitus and helps to create adequate vaginal caliber and prevents hemorrhage due to rupture of hymenal vessels during the first sexual intercourse [43]. Follow-up of these patients reveals overall subjective satisfaction with vaginal lengths of 10–12 cm and widths of 4–5 cm [44]. This procedure is considered to be superior to the McIndoe procedure as it is can be performed in less time and there is less need for postoperative vaginal dilators, which reduces the psychological impact of the treatment [43].

Laparoscopic Procedures

The Vecchietti and Davydov techniques for vaginal reconstruction were first performed as open procedures, but advances in minimally invasive

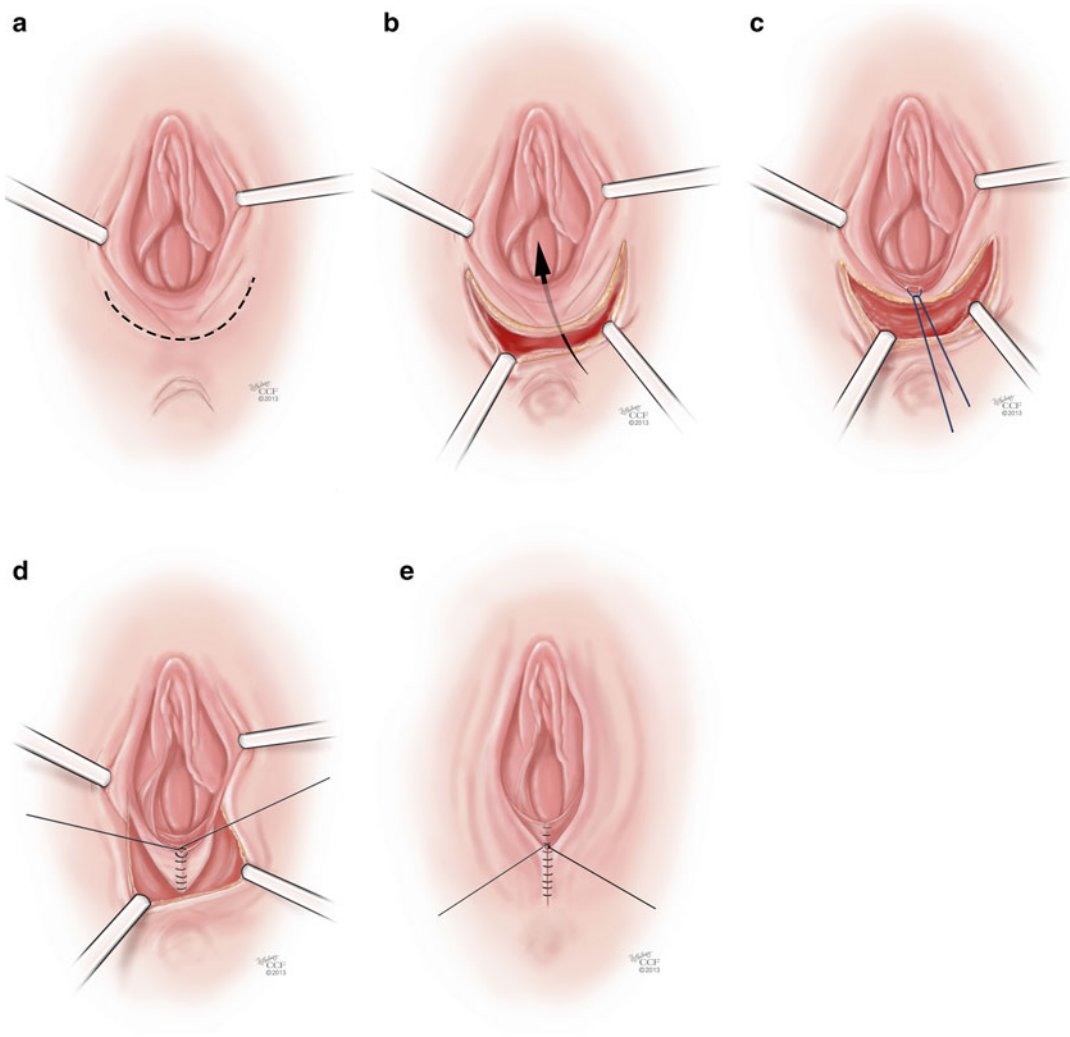


Fig. 18.4 (**a**–**e**) Williams procedure. (**a**) The initial step of the Williams procedure is a U-shaped incision extending across the perineum and up to the medial side of the labia. (**b**) The skin flap is sharply dissected off of the underlying tissues creating a flap that can be mobilized inwards, creating a vaginal pouch. (**c**) Once the tissues are mobilized, a first layer of suture is placed between the inner skin margins using interrupted 0 delayed absorbable sutures. (**d**) A second layer of sutures of the same material is used to approximate the subcutaneous fat and the perineal muscles. (**e**) The external skin is sutured with interrupted stitches (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

surgery have allowed these procedures to be performed laparoscopically. The main advantage of a laparoscopic approach is the ability to bridge the need to perform an indicated abdominal procedure with the ease of recovery for the patient. The postoperative phase of these surgeries is usually very involved, and therefore, length of hospital stay is usually not significantly shortened, as is seen with most laparoscopic operations. However, once discharged from the hospital, patient recovery is easier as postoperative pain is less and patients are able to return to their daily activities faster.

Vecchietti Procedure

The Vecchietti procedure involves gradual mechanical stretching of the patient's vaginal skin to create a full-length vagina. This procedure is

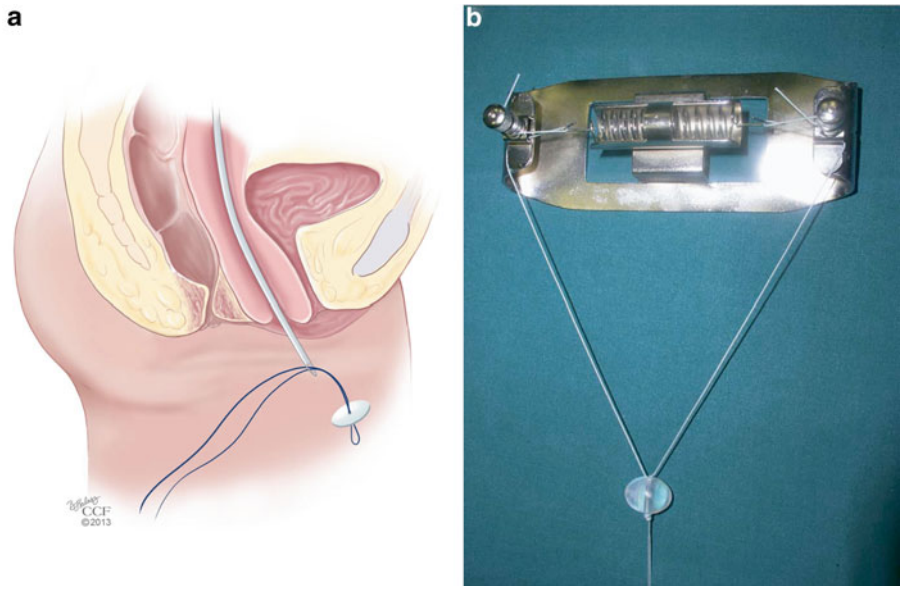


Fig. 18.5 (**a**, **b**) Vecchietti procedure. (**a**) Under direct laparoscopic visualization, a guide needle is used to pass permanent sutures through the acrylic bead and vagina and into the dissected rectovesical space in the pelvis. A guide needle is then inserted suprapubically and used to pull the sutures out of the body. (**b**) Sutures are connected to a traction device that is secured to the patient's abdomen (**a**: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved; **b**: Used with permission from Fedele L, Bianchi S, Berlanda N, Fontana E, Bulfoni A, Borruto F. Laparoscopic creation of a neovagina with the laparoscopic Vecchietti operation: comparison of two instrument sets. Fertility and Sterility 2006; 86(2):429-432)

most appropriate for patients presenting with vaginal agenesis and no prior reconstructive surgery [45]. In this procedure [46], the vesicorectal space is carefully dissected laparoscopically to reflect the bladder anteriorly. An EEA sizer can be placed inside the rectum for mobilization and visualization of the rectovesical space in order to avoid entry into the rectum at the time of dissection. A 2 cm olive-like acrylic bead is placed on the vaginal dimple and is sutured in place to the perineum. Under direct visualization with the laparoscope, a guide needle is used to pass permanent sutures through the acrylic bead and vagina and into the dissected rectovesical space in the pelvis. A guide needle is then inserted suprapubically and used to pull the sutures out of the body (Fig. 18.5a). The sutures are connected to a traction device that is secured to the patient's abdomen (Fig. 18.5b). Sutures are tightened on a regular schedule, placing traction on the vaginal epithelium, and gradually increasing the length of the vaginal canal. Once adequate vaginal length is achieved, the traction device is removed and the sutures are cut and freed from the body. Patients are advised to practice daily dilation in order to help stretch the vaginal epithelium and maintain vaginal caliber and length during the traction phase of the procedure, and for a limited amount of time once the device is removed. As with the Frank dilation method, patient compliance with vaginal dilation and routine follow-up of the traction device are paramount to the success of the surgery. Data on long-term anatomic outcomes as well as sexual health and quality of life outcomes are favorable with this procedure [47].

Davydov Procedure

The Davydov procedure [48] is a technique used to create a neovagina using the patient's own peritoneum. Good candidates for this procedure include patients with disorders of sex differentiation, such as XY females, who have undergone

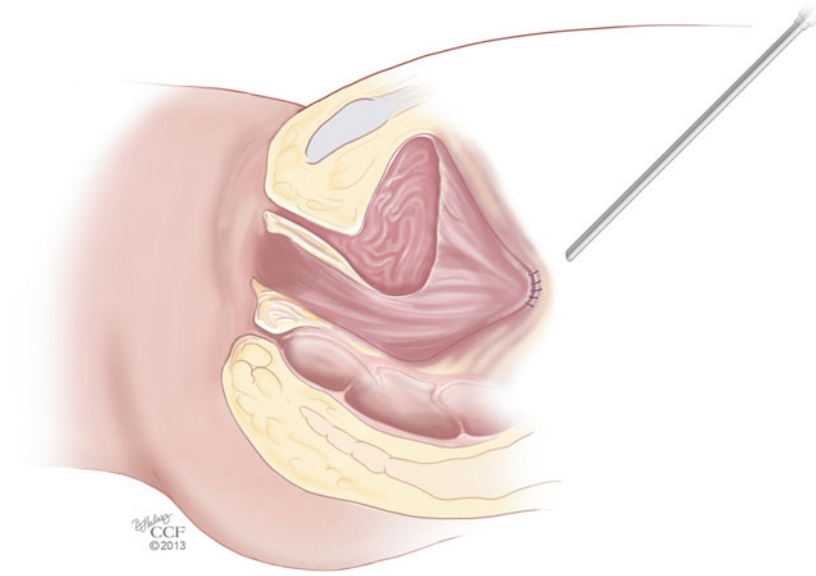


Fig. 18.6 Davydov procedure. The mobilized peritoneum is sutured to the perineal incision and closure of the abdominal end of the neovagina is done laparoscopically with a purse string suture (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

prior feminizing genitoplasty procedures, but have had poor outcomes, or are not satisfied with vaginal length or caliber. Several modifications of the procedure exist. We recommend first making a U-shaped perineal incision to serve as a landmark for where the peritoneal edges are to be sutured later in the case. Laparoscopic dissection is then done in the rectovesical space, similar to the technique described for the Vecchietti procedure. This is also done using an EEA sizer in the rectum, to delineate the correct dissection plane. Releasing peritoneal incisions in the pouch of Douglas are then made either laparoscopically or transvaginally, freeing and mobilizing the peritoneum caudally so that it can be sutured to the previously made perineal incision. Closure of the abdominal end of the neovagina is done laparoscopically with a purse string suture (Fig. 18.6). A vaginal mold is left in place for several weeks and, once removed, it is replaced with daily dilation until maximal vaginal length is created [45].

There are significant advantages to these two laparoscopic procedures when compared to non-surgical dilation methods. Lengthening of the vagina is accomplished at the time of the procedure, and does not require long-term dilation that can be very uncomfortable initially and time-consuming. The dilation that is then required postoperatively is usually much easier as the vagina has already been created and the main goal of dilation is maintenance of length and caliber. Patients tend to be very compliant with these steps. The main disadvantage is that both techniques require surgical intervention, and while they are performed in a minimally invasive fashion, require extensive dissection into the rectovesical space, which can be associated with rectal, bladder, nerve, and vascular injury. Therefore, meticulous technique is required by an experienced surgeon.

Myo- and Fascio-Cutaneous Flap Procedures

The principle of a myo- and fasciocutaneous flap is the creation of an island flap that depends on the underlying muscle or fascia for its vascular supply. The flap is made up of muscle with or without fascia or fascia alone and the overlying subcutaneous and cutaneous tissues.

Two main techniques can be described when reconstructive surgery is performed using flaps: (1) the standard local or regional flap technique which is based on a vascular pedicle that remains intact while the flap is being mobilized and (2) the more sophisticated microvascular free flap, which involves ligation of the vascular pedicle and reanastomosis to the vasculature of the recipient site. With a few exceptions, the pedicled flap is the most commonly employed flap technique for reconstruction of the neovagina and is usually used after extirpative pelvic surgery or when initial skin-graft techniques have failed in patients with congenital anomalies.

Flap orientation and dimensions of the skin paddle harvested are designed to achieve adequate perfusion through the muscular portion of the flap while achieving adequate skin for vaginal reconstruction, as well as primary donor-site closure. All pedicled flaps have their limitations in terms of the arc of rotation, the size, the tissue volume, and the restriction of mobility. These factors sometimes make it difficult to tailor the flap to the defect that needs repair [49]. Advantages of myo- and fasciocutaneous flaps include the mobilization of a substantial amount of tissue to repair pelvic dead space while providing a source for revascularization for the surrounding tissues. The disadvantages of these flaps are that they can sometimes be very bulky which can affect cosmetic outcome and make the neovaginal cavity narrow, the skin paddles that line the vagina do not provide any lubrication for intercourse, and there can be significant morbidity from the donor site [50].

Rectus Abdominis Flap

Rectus abdominis musculocutaneous flaps are based off of the deep inferior epigastric vessels and can be harvested in two different orientations: transverse (TRAM) and vertical (VRAM). The VRAM flap is usually the preferred method of harvesting for exenteration procedures when stomas are created for bowel and urologic reconstruction [49, 51]. The VRAM flap can be taken from either the patient's left or right side and is developed above the level of the arcuate line (Soper) (Fig. 18.7a). The skin paddles typically measure 12×8 cm in size, which is usually sufficient for the creation of a functional vagina [52]. The horizontal dimension is usually limited by the ability to close the skin primarily; a flap width extending 2 cm lateral to the palpable edges of the rectus can be closed easily in most cases [53]. Once the course of the deep inferior epigastric vessels is identified with Doppler, the flap is elevated from the costal margin to the level of the inguinal fold. Dissection is carried down to the rectus sheath and the lateral border is opened sharply. The skin island with the underlying subcutaneous tissue is mobilized off of the anterior rectus fascia, and the fascia is incised in a slightly smaller ellipse mirroring the skin island in order to leave a smaller fascial defect [51]. The muscle is elevated off of the posterior fascia after the intercostal neurovascular bundles are ligated. The flap is freed superiorly by dividing the muscle at the costal margin. The superior epigastric vessels are identified and ligated. The deep inferior epigastric vessels remain as the vascular supply to the flap, and are identified inferiorly on the posterolateral surface of the muscle, crossing the lateral border of the muscle at approximately the level of the arcuate line. The flap is then elevated carefully so as to not shear the underlying branches of the pedicle. Interrupted absorbable sutures can be used to secure the muscle edges to the overlying subcutaneous tissue to prevent shearing during flap transfer [53] (Fig. 18.7b, c). TRAM flaps are raised in an elliptical fashion as well, below the umbilicus, from one anterior superior iliac spine to the contralateral iliac spine (Fig. 18.8a). In a similar fashion, the dissection is made underneath the anterior rectus fascia, which is preserved in a transverse orientation. The epigastric vessels are identified, and the superior vessels are ligated to allow for mobilization of the pedicled flap in a similar method used for the VRAM (Fig. 18.8b, c).

Once the flap is raised, it is folded into a tube by approximating the edges in a 2-layer closure using absorbable sutures. VRAM flaps are folded into a tube such that the proximal and distal ends of the flap form the introitus once placed in proper position [52]. TRAM flaps are folded such that the lateral border is approximated to the medial border and the cranial edge of the flap is used to form the introitus [51]. The tube is then

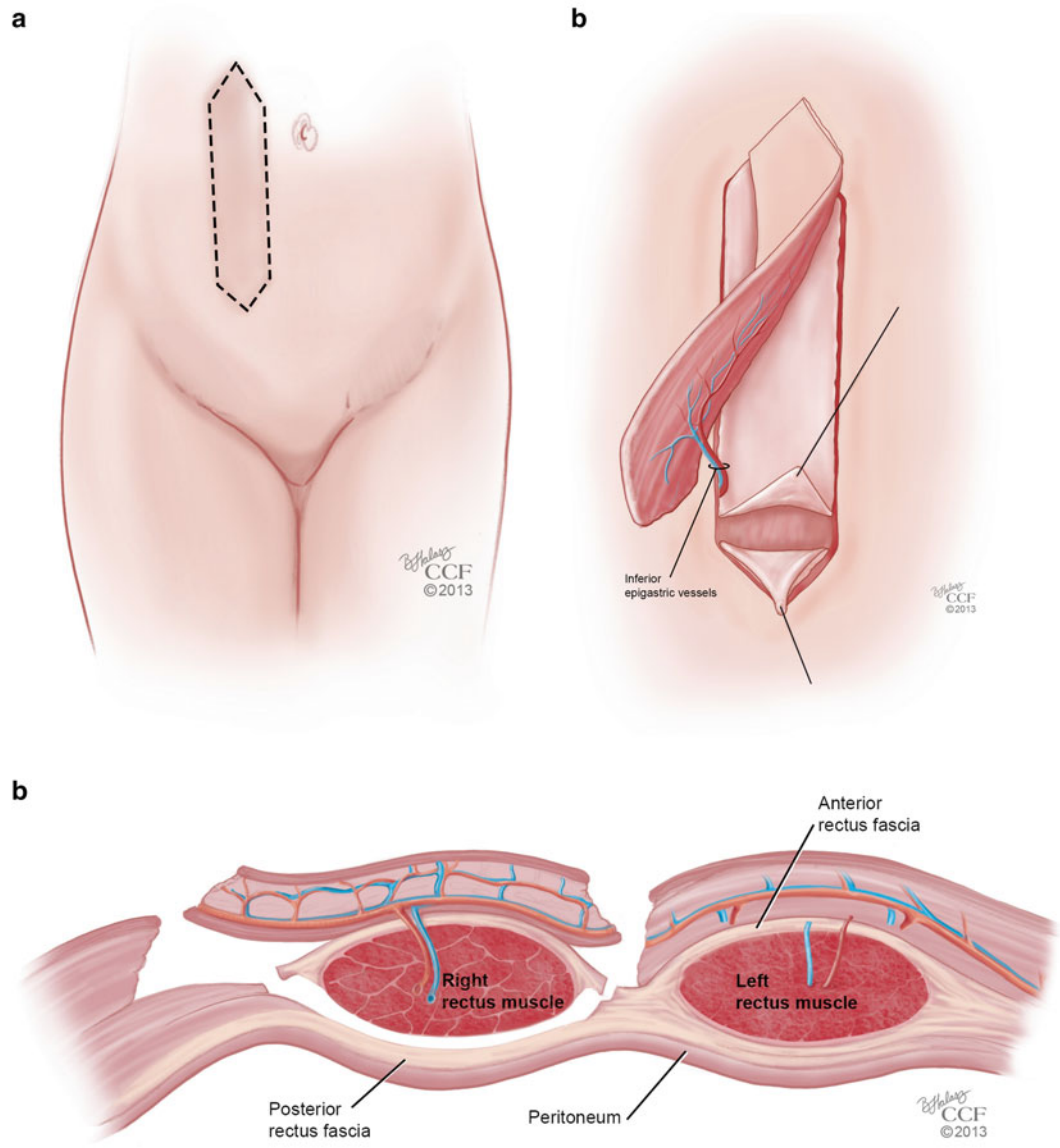


Fig. 18.7 (**a**–**c**) VRAM flap. (**a**) Orientation of the vertical rectus abdominis musculocutaneous (VRAM) flap. (**b**) Mobilization of the VRAM flap on its vascular pedicle. (**c**) Coronal view of the mobilization of the VRAM flap on its vascular pedicle (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

mobilized into the pelvis through an opening in the posterior rectus fascia, and then brought beneath the pubic ramus, without placing tension on the pedicle. Closed suction drains are placed in the abdomen and pelvis to prevent hematoma and seroma formation. The rectus fascia at the donor site is closed with heavy running sutures and the overlying skin is closed in a manner that limits the distortion of the umbilicus.

A modification to the VRAM flap is the inferior-based VRAM flap. This flap has been shown to meet reconstructive needs in cases of vulvar and perineal defects after resective surgery. Traditional myocutaneous flaps used for

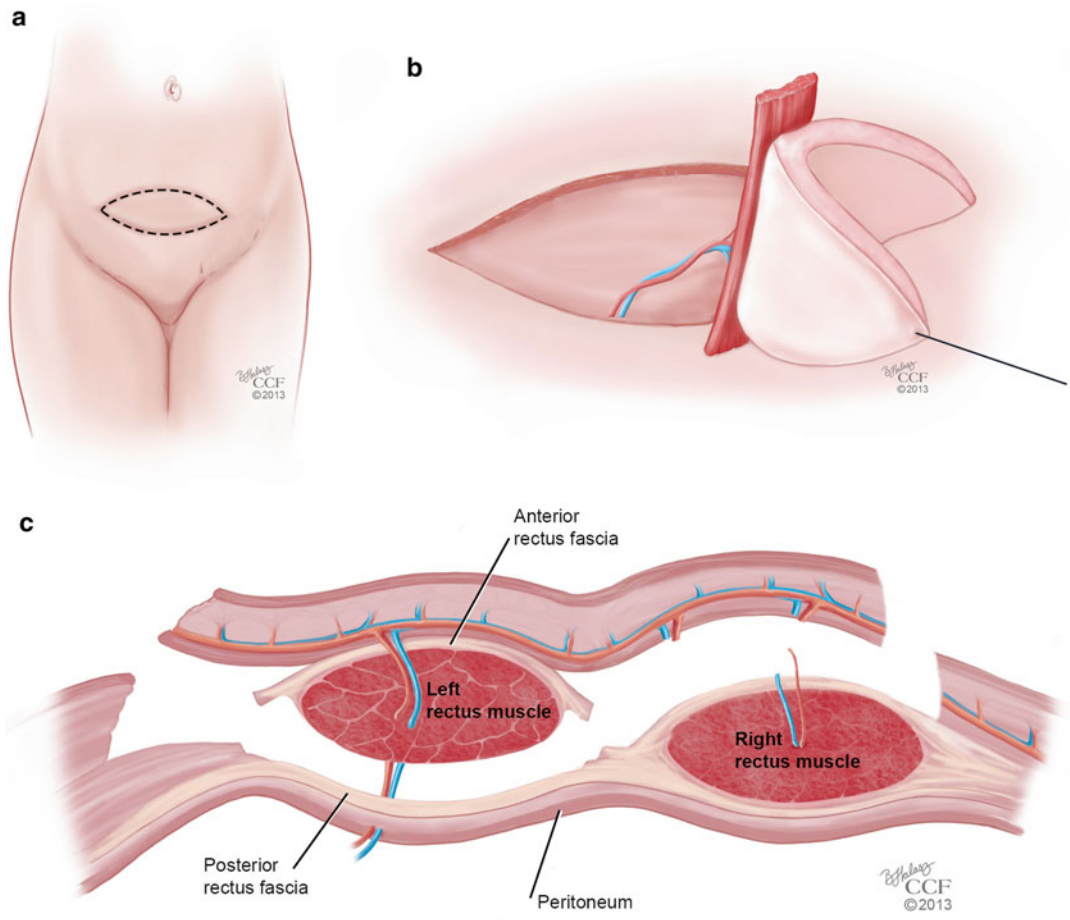


Fig. 18.8 (**a**–**c**) TRAM flap. (**a**) Orientation of the transverse rectus abdominis musculocutaneous (TRAM) flap. (**b**) Mobilization of the TRAM flap on its vascular pedicle. (**c**) Coronal view of the mobilization of the TRAM flap on its vascular pedicle (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

reconstruction following radical vulvectomy can cover the large perineal defects but do not provide a "functional" reconstruction that can preserve anal and vaginal patency. The inferior-based VRAM flap is marked and raised in a similar fashion to the standard VRAM flap. The muscle is then split distally in the midline with care taken to avoid transection of the muscular branches of the superior epigastric artery that anastomose with the deep inferior epigastric artery, and supply important perforators to the muscle and skin of the flap. These muscular branches can easily be identified and separated on the underside of the muscle by using spreading dissection, each supplying a tongue of overlying skin and subcutaneous tissue [53]. Division of the distal flap produces well-vascularized myocutaneous fork flaps that can be draped around the vaginal cuff and crossed inferiorly over the perineal body to create a fourchette. This also provides a skin edge for attachment of anal mucosa if extensive perianal dissection was performed.

Several series have looked at the complication rates and outcomes of rectus abdominis myocutaneous flaps [50, 52, 54, 55]. One of the larger series found that 38 % of patients developed

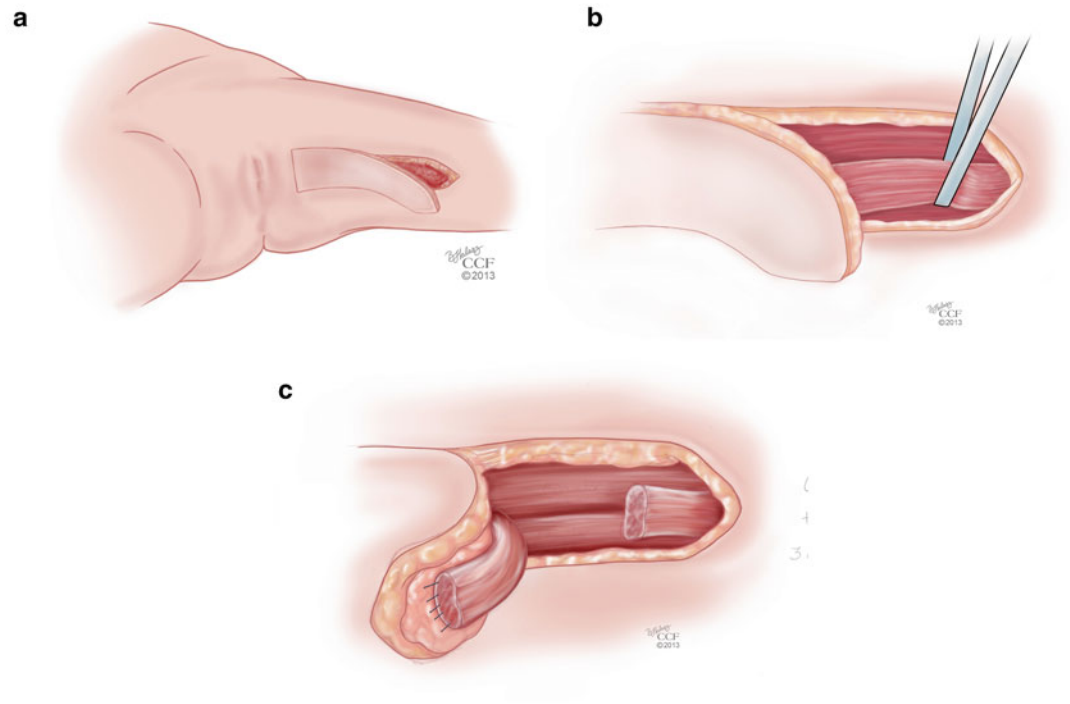


Fig. 18.9 (**a**–**c**) Gracilis flap. (**a**) Orientation of the gracilis musculocutaneous flap. (**b**) Identification of the gracilis muscle. (**c**) Mobilization of the gracilis muscle (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

flap-specific complications including stricture/stenosis (13 %), pelvic abscess or hematoma (6 %), and rectovaginal fistula (6 %), while only two (6 %) patients experienced complete flap loss [51]. Donor-site complications included fascial dehiscence (5 %) and superficial separation (13 %). A major criticism of the VRAM flap is that synthetic mesh is often necessary to repair the fascial defect to prevent the risk of ventral wall hernia [56]. However, the VRAM orientation is often preferable for certain exenteration procedures, as it allows for easier stoma and conduit creation on the contralateral side. Additionally, techniques that focus on reducing the size of the fascial defect to less than 4–6 cm in comparison to the entire flap size can significantly reduce the risk of future hernia. In their case series, Soper et al. [51] found that there were no significant differences between the TRAM and VRAM groups in the distribution of donor-site, recipient-site, or overall flap-specific complications; VRAM flaps were not more likely to be complicated by ventral wall hernia.

Gracilis Flap

Gracilis musculocutaneous flaps are based off of the medial circumflex femoral artery. A skin flap matching the dimensions of the defect is outlined on the proximal two-thirds of the inner thigh overlying the gracilis muscle, the most superficial muscle of the inner thigh (Fig. 18.9a). Once the course of the medial circumflex femoral artery has been identified with Doppler, a full-thickness elliptical island of skin and subcutaneous tissue approximately 14–20 cm in length and 8–10 cm in width is raised. The proximal skin incision and the arc of rotation are based approximately 6–8 cm from the pubic tubercle, at the point of entry of the neurovascular bundle into the gracilis muscle [57]. Using sharp and blunt dissection, the underlying gracilis muscle is

mobilized with care taken to identify and preserve the dominant neurovascular pedicle [58] (Fig. 18.9b, c). Incising the fascia over the adductor magnus muscle and dissecting this fascia medially with a blunt instrument facilitates identification of the bundle. Once the muscle is completely dissected out and separated from the surrounding inner thigh muscles, it is transected proximally close to the ischial pubic ramus and then sutured to the overlying subcutaneous tissue using 3-0 or 4-0 absorbable sutures in an interrupted fashion [58]. The raised flap is then rotated posteriorly through a subfascial perineal tunnel. If complete vaginal reconstruction is necessary, bilateral flaps are raised. Flaps are then formed into tubes by approximating the skin edges such that the distal edge of each flap becomes the apex of the neovagina. The neovaginal tube is then rotated posteriorly into the pelvic defect and the proximal skin edges approximated to the introitus. The donor site is closed in layers with synthetic absorbable suture and a closed end drain is left in place at this site.

The gracilis musculocutaneous flap is commonly used to repair vaginal defects after extirpative surgery. The main advantage of the flap is that, with proper technique, it is easy to raise and also less difficult to tunnel to the vaginal or vulvar defect. However, when compared to rectus abdominis flaps, the gracilis flap has been associated with a higher rate of flap loss (14 % versus 3 %) [57]. Casey et al. [50] performed one of the largest outcome studies evaluating myocutaneous flaps. They compared 41 VRAM, 13 gracilis, and 45 pudendal thigh flaps. They determined that the VRAM had the lowest overall and flap-related complication rates following complete vaginal reconstruction. However, the flap and donor-site complication rates for the gracilis and pudendal thigh flaps were acceptable enough to consider these flaps good alternatives if a VRAM flap is not possible. They also found that patient age and preoperative sexual activity were good predictors of postoperative sexual activity following vaginal reconstruction; therefore, this should be assessed preoperatively and considered when deciding upon vaginal reconstruction methods.

Posterior Thigh Flap (Fig. 18.10)

An alternative to the gracilis flap is the posterior thigh flap, based off of the inferior gluteal artery. The posterior femoral cutaneous nerve of the thigh is usually preserved at the time of this flap dissection; therefore, a portion of the flap is usually partially sensate. Friedman et al. [59] describe this technique. The course of the inferior gluteal artery is confirmed with Doppler and the skin island is centered over the vascular pedicle along the length of the posterior thigh. Flap dimensions are marked according to the defect needing repair. The distal most aspect of the flap should be marked a few centimeters superior to the popliteal crease, to avoid potential scar contracture deformity and wound-healing problems. Dissection is first begun at the distal aspect of the flap and carried out laterally and medially until the distal portion of the flap is reached. The flap is then elevated in continuity with the underlying fascia of the posterior compartment of the thigh, from a distal to proximal direction. Absorbable sutures are placed through the fascia and dermis to prevent shearing of the vascular pedicle and to facilitate mobilization of the flap. The flap is completely raised once it is dissected proximally to the inferior border of the gluteus maximus muscle. The flap is mobilized to the pelvis through a subcutaneous tissue tunnel, above the underlying fascia, between the posterior thigh and the adjacent defect. Bilateral flaps are usually raised and mobilized for creation of the neovagina.

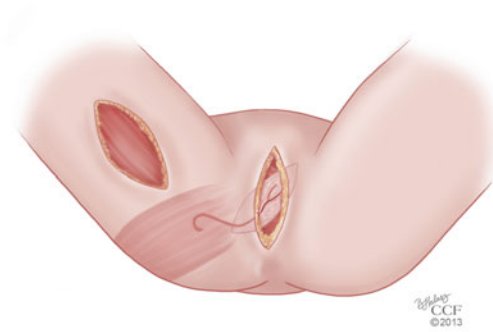


Fig. 18.10 Posterior thigh flap. Mobilization of the posterior thigh flap, based off of the inferior gluteal artery (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

Once mobilized, they are tubularized and positioned in a similar fashion as the gracilis flap. Drains are placed in the pelvis and at the donor site, which is usually closed primarily. The advantages and disadvantages of the posterior thigh flap reflect those of the gracilis and rectus abdominis flaps [59]. It remains a good alternative in cases that do not allow for the use of more common reconstructive procedures.

Pudendal Thigh Flap

The pudendal thigh flap is a vulvoperineal fasciocutaneous flap and is also known as the *Singapore* or *Malaga flap*, and can also be used for reconstruction of the vagina. The pudendal thigh flap is based off of the posterior labial arteries, which are a continuation of the perineal vessels, which are the terminal vessels of the internal pudendal artery. These arteries anastomose with branches of the deep external pudendal artery, the medial circumflex femoral, and the anterior branch of the obturator artery over the proximal portion of the adductor muscle. The posterior portion of this flap is innervated by the posterior labial branches of the pudendal nerve. As a result, the proximal portion of the flap often times maintains some degree of sensation. The superficial perineal nerve is often preserved as well with this flap, which adds additional sensation. The vulvoperineal skin island is marked vertically in a rectangular shape with the longitudinal axis overlapping the lateral limit of the labia majora; the base of the flap is inferior to the posterior border of the neovaginal introitus, extending from the labia majora across the groin crease to the medial thigh and measures up to 12 × 6 cm [60] (Fig. 18.11a). Sharp dissection is done to raise the flap starting at the superior most margins where the deep external pudendal artery branches with the superficial perineal artery.

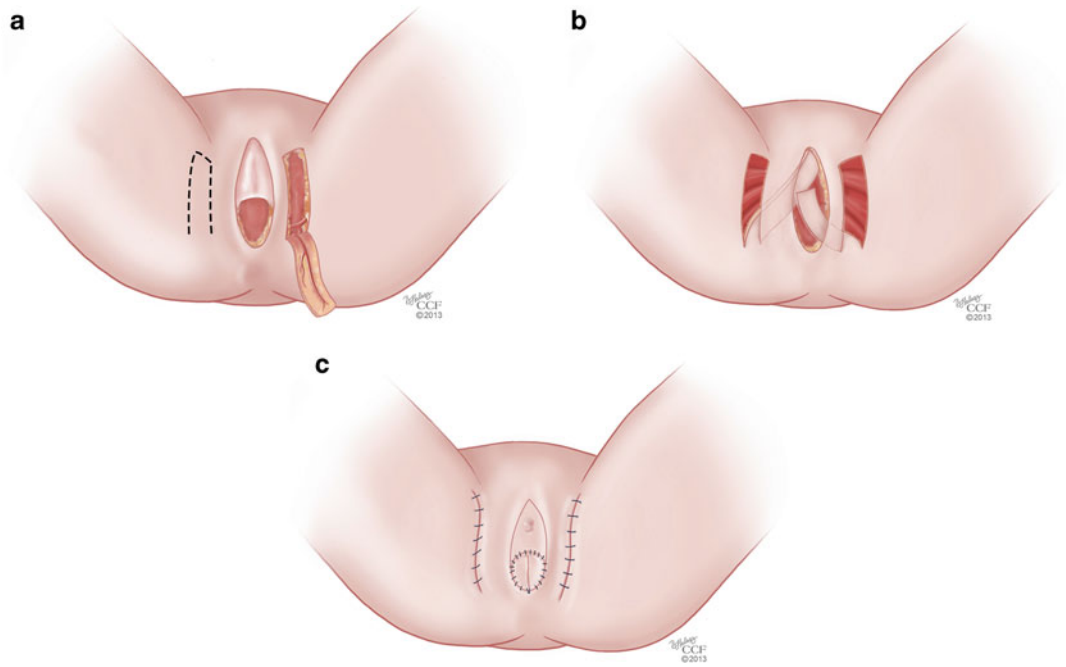


Fig. 18.11 (**a**–**c**) Pudendal thigh flap. (**a**) Pudendal thigh flap based off of the posterior labial arteries. (**b**) The strip of flap that passes under the labia is de-epithelialized and sutured to the overlying tissue. (**c**) The medial, distal, and lateral margins of the flaps are approximated, creating a tubular pouch, and the skin edges are sutured to the neointroitus and cutaneous edges of the labia majora (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

This anastomosis is ligated and the superficial perineal artery is preserved with the flap. The flap is dissected down to the level of the adductor muscles, the fascia over the muscles is raised with the flap, and the superficial perineal muscles can be identified [61]. The flap is detached medially by transecting the insertion of the adductor aponeurosis, which inserts into the ischiopubic ramus. Bilateral flaps are usually raised and then tunneled subcutaneously under the labia majora into the previously dissected rectovesical space. The strip of flap that passes under the labia is de-epithelialized and sutured to the overlying tissue. Lastly, the medial, distal, and lateral margins of the flaps are approximated, creating a tubular pouch, and the skin edges are sutured to the neo-introitus and cutaneous edges of the labia majora (Fig. 18.11b, c). The donor site is then closed primarily in several layers. Long-term follow-up of these patients shows positive anatomic and functional outcomes [61].

Martius Bulbocavernosus Flap

The Martius bulbocavernosus flap was first described in 1928 by Martius [62] and has been used frequently in the repair of complex vaginal fistulas. It can also be a useful source for the construction of a partial or complete neovagina [63–65]. For larger defects such as complete vaginectomy in the setting of total pelvic exenteration, bilateral Martius flaps can be used by tubularizing the flaps to create a complete, full-length vagina with normal caliber. When marking and mapping these flaps, the primary goal should be to create flaps that are as large as possible with the ability to close the donor site primarily without tension. This technique has been described by Green et al. [63]. The Martius flaps should extend from the level of the clitoris superiorly to a level just above the perineal body inferiorly. The medial margin is the sulci between the labia majora and minora. The lateral margin is the lateral edge of the labia majora. The flap is sharply developed and raised, preserving the vascular pedicle, which is the posterior labial branch of the internal pudendal artery. The flaps are tunneled through the paravaginal windows into the abdomen. The portion of the flap that is to become the posterior vagina is sewn to the top of the vagina. The two flaps are joined on the posterior aspect with interrupted sutures. The anterior aspects are sewn to the vaginal cuff and then joined anteriorly, creating a tube with vulvar epithelium lining the neovagina. This method of reconstruction is an excellent choice for patients who have vulvar anatomy that is conducive to creating large flaps that can be easily mobilized. In the appropriate patient, this procedure for neovaginal reconstruction is associated with minimal blood loss, short operative time, decreased pain, and less disfigurement than other types of flaps and has very favorable anatomic outcomes [63].

Omentum-Pedicled Flap

While the greater omentum-pedicled graft is not a musculocutaneous flap, it has been extensively used for coverage of perineal and other soft-tissue defects but has also been used successfully in vaginal reconstruction [66]; for these reasons, it is worth mentioning. Case series have shown that the flap can be used in conjunction with an absorbable graft such as a vicryl mesh that is first positioned into proper location using a vaginal stent and sutured to the remaining edges of either the posterior or anterior vagina. The omentum-pedicled graft is based off of the left gastro-epiploic artery, which is mobilized down along the left paracolic gutter, and then draped over all parts of the mesh and sutured to the graft [67] (Fig. 18.12). While this type of flap is not commonly used for vaginal reconstruction, it may confer important benefits. The omentum has a rich vascular supply and it unlikely to necrose, even after mobilization. Additionally, it is easily mobilized without significant morbidity. Lastly, the flap itself is much less bulky than other commonly used myocutaneous flaps and may have better cosmetic results. Its major disadvantage is that a mesh graft may be necessary for placement as there are no reported cases of omental flaps used for vaginal reconstruction without the use of a mesh graft. There are currently no studies examining the safety of synthetic nonabsorbable grafts in neovaginal reconstruction with omentum-pedicled flaps.

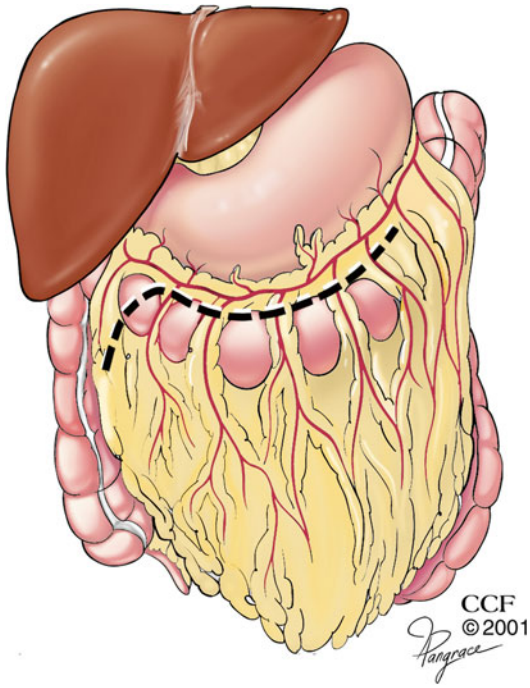


Fig. 18.12 Omentum-pedicled flap. Based off of the left gastro-epiploic artery, which is mobilized down along the left paracolic gutter to the site of reconstruction (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

Intestinal Flaps

Different bowel segments, including the ileum, jejunum, cecum, and sigmoid colon, have been used for neovaginal reconstruction. The procedure involves resecting a segment of the bowel approximately 10–12 cm in length with its vascular pedicle intact, reanastomosing the bowel, mobilizing the resected segment into the pelvis, suturing the edges to the obliterated vagina after creation of a proper space, and suturing to close the proximal end of the segment, forming a pouch [68] (Fig. 18.13a–d). The main advantages of intestinal flaps are that the risk of contracture and stenosis is significantly reduced, molds or stents are not required to ensure patency, and lubrication is not a problem, facilitating intercourse [69]. Procedures are usually performed via a laparotomy; however, there are some case reports and series describing laparoscopic techniques [70]. The most significant disadvantages of intestinal flaps include the morbidity of laparotomy such as infection and wound dehiscence, shrinkage with intestinal stenosis, anastomotic dehiscence, possible need for colostomy, and persistent copious secretion of colonic mucus. These procedures are more complicated than other neovaginal procedures and are limited to surgeons with a very unique skill set.

Sigmoid Flap

The sigmoid is the most commonly used piece of colon for neovaginal reconstruction because of its location in the pelvis and ease of mobilization [6]. Its vascular supply consists of the sigmoidal arteries that branch off of the inferior mesenteric artery. In a large series, Kwun Kim et al. [71] showed that the sigmoid neovagina achieved a very low contraction rate, was able to maintain adequate vaginal length and width without the use of stenting, mucous secretion helped with sexual intercourse, there was low incidence of malodor, and patients were satisfied with the cosmetic appearance of the reconstruction. Additionally, sexual satisfaction has been reported to be as high as 78 % after sigmoid vaginoplasty [72]. While these cases have been done laparoscopically [70], the vast majority are performed via laparotomy, which has its risks and associated comorbidities. In addition, there are reports of diversion colitis, ulcerative colitis, patient dissatisfaction with copious malodorous neovaginal discharge, prolapse, flap failure, and defecatory dysfunction [70]. While very rare, primary adenocarcinoma of the colon has been reported in the sigmoid neovagina as well [73].

Ileocecal Flap

The ileum and cecum have both been used successfully in the creation of the neovagina and are based off of the ileal branches of the superior mensenteric artery (SMA). The main advantage of these flaps is that there is excellent blood supply to those portions of the bowel and the vascular pedicle is long enough to be mobilized to the pelvis. Additionally, reports show that there is the least mucous production of all of the intestinal flaps and therefore, there is less vaginal discharge [69]. The main disadvantage of the ileal flap is

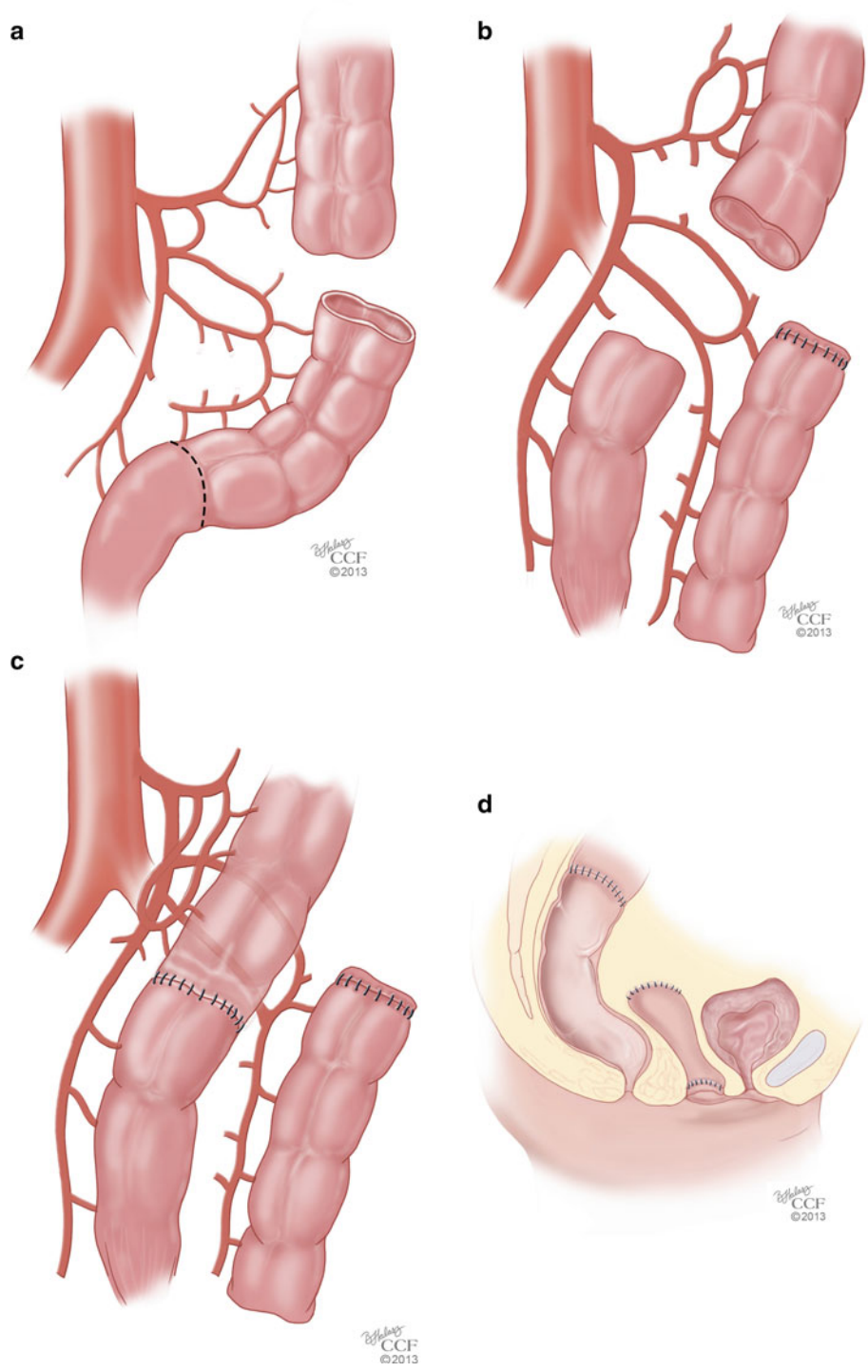


Fig. 18.13 (**a**–**d**) Creation of a neovagina with an intestinal flap. (**a**) A segment of the bowel is resected with its vascular pedicle intact. (**b**) A segment of the bowel is resected with its vascular pedicle intact. (**c**) The bowel is reanastomosed and the resected portion is mobilized into the pelvis. (**d**) A sagittal view of the bowel flap sutured to the obliterated vagina, creating a neovaginal pouch (All: Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2013. All Rights Reserved)

that compared to the jejunum and sigmoid, the ileum wall is much more fragile and delicate and more likely to sustain trauma with subsequent bleeding at the time of mobilization and is also associated with a higher rate of stenosis and intestinal obstruction [6]. Similar to the sigmoid flaps, there are reported cases of laparoscopic ileal vaginoplasty, which have been successful and confer the advantage of less morbidity [69].

Jejunal Flap

This pedicled flap is also based off of a branch of the SMA. The jejunum has a smaller lumen than the rectosigmoid and can provide favorable cosmetic and functional outcomes [6]. Another significant advantage over the sigmoid flap is that there is less dissection needed for mobilization and patients report significantly less defecatory dysfunction [74]. A disadvantage of the flap is copious intestinal secretion but this can be rectified by performing the surgery as a free flap procedure with anastomosis rather than as a pedicled flap procedure, as temporary ischemia of the flap during mobilization leads to decreased mucous production [6]. When a free flap procedure is performed, the SMA branches that are mobilized with the flap are anastomosed using a microvascular technique to the inferior epigastric vessels. There are fewer laparoscopic cases that have been reported, as mobilization of the jejunum is done most easily via a laparotomy, and is obligatory when a free flap technique is employed.

Prolapse of the Neovagina

There are few reported cases of vaginal vault prolapse in women who have undergone neovaginal reconstruction for vaginal agenesis [75–78]. In seven previous case reports, four cases of vaginal vault prolapse occurred after mechanical dilation and three occurred after surgical management with either the McIndoe or Williams procedures. The vagina is composed of three segments: upper and middle and lower. The upper and middle segments are derived from the mullerian ducts and are suspended to the surrounding structures by connective tissue fibers to the sacrum and fascia of the pelvic sidewall, respectively. The lower segment of the vagina is derived from the urogenital sinus and is fused to the perineum as well as the fascia of the levator ani muscles and perineal body. Patients with mullerian agenesis are born without the fibrous connections of the upper and middle segments of the vagina. In these patients, the vagina is a blind pouch that opens at the introitus. A mechanically created neovagina or a surgically constructed one lacks the fibrous supports that suspend the vagina to the bony pelvis, and as a result, are at risk for prolapse of the vaginal apex and its lateral supports. Based on existing published case reports, successful outcomes can be achieved with vaginal vault prolapse surgeries. Abdominal sacrocolpopexy with synthetic mesh and paravaginal repair has been performed successfully on patients with prolapse of the neovagina [77]. There is also a case report showing a successful sacrospinous ligament colpopexy used in the case of vaginal vault prolapse in a MRKH patient [78]. In this particular case, the vagina was not long enough to be directly sutured to the sacrospinous ligament, and so, a fascia lata allograft was sutured to the anterior and posterior vaginal walls and then secured to the right sacrospinous ligament to help bridge the gap between the vaginal apex and ligament. The prevalence of vaginal vault prolapse in vaginal agenesis patients who have undergone neovaginal construction is not known. However, as we continue to perform reconstructive neovaginal procedures for these patients, prolapse procedures for some patients will continue to be necessary.

Conclusion

Patients with congenital anomalies of the vagina usually present in adolescence while children with sex differentiation disorders present with an intersex phenotype in the early period of life. Once these disorders are identified, proper evaluation is required in order to identify all anatomic abnormalities and to help with accurate diagnosis. Evaluation includes all or some of the following: office physical exam and exam under anesthesia if necessary, karyotype, hormonal panel, and

imaging with MRI and/or ultrasonography. Once this information is obtained, proper management planning can be done. Treatment takes place once the patient is mature enough to understand their disorder, to commit to the treatment plan, and as long as adequate social supports are in place. First-line management for patients with vaginal agenesis is any version of Frank's method of progressive vaginal dilation. It should be the first therapeutic procedure because it is the least invasive and has fewer serious complications.

If this method fails to create a proper vagina, or patients decline this method, surgical neovaginal construction can be performed. The surgical option often depends on the experience and preference of the surgical team, and sometimes requires a multidisciplinary approach with gynecologists, pediatric surgeons, urologists, and plastic surgeons. The technique most often employed for patients with vaginal agenesis or disorders of sex differentiation is the McIndoe operation, a modification of this surgery, or the Williams vaginoplasty. Laparoscopic techniques such as the Vecchietti and Davydov operations are also minimally invasive options for patients and have yielded favorable outcomes. For failed procedures or more complex reconstruction, various flap procedures can be performed. Many factors direct the type of flap that is used. These include the size and type of defect that needs to be repaired, the availability of certain flaps, the morbidity associated with flap harvesting and repair of the donor site, and the number and types of prior reconstructive procedures the patient has already had. Patients with vaginal agenesis or disorders of sex differentiation require construction of a patent vaginal tube that is functional and cosmetically appealing. Patients who have undergone resective surgeries or have undergone radiation therapy for malignancy have these same requirements, in addition to larger vulvar and perineal defects that require repair. Many different flaps have been described in this chapter, including those that are musculo- and fasciocutaneous and intestinal. The majority of these flaps are pedicle-based and rotational in nature and do not require microvascular surgery. Additionally, the use of biologic allografts and tissue engineering to create the neovagina was briefly discussed and may be promising minimally invasive techniques for the future, but need to be investigated further. The ideal reconstructive method should provide a patent vaginal canal of adequate length, width, and texture that will allow for sexual intercourse, provide a cosmetically appealing appearance with minimal morbidity of both the recipient and donor surgical sites with a low incidence of overall complications.

Reconstruction of the neovagina can be very complex and challenging. Each method of repair has its advantages and disadvantages, which should be carefully weighed with the desired treatment goals as well as the surgeon's experience with various surgical techniques.

References

1. Evans TN, Poland ML, Boving RL. Vaginal malformations. AJOG. 1981;141(8):910–7.
2. Silber CG, Magness RL, Farber M. Duplication of uterus with non-communicating functional horn. Mt Sinai J Med. 1990;57:374–7.
3. Griffen JE, Edwards C, Madden JD, Harrod MJ, Wilson JD. Congenital absence of the vagina. The Mayer-Rokitansky-Kuster-Hauser syndrome. Ann Intern Med. 1976;85(2):224.
4. Seccia A, Salgarello M, Sturla M, Loreti A, Latoree S, Farallo E. Neovaginal reconstruction with a modified McIndoe technique: a review of 32 cases. Ann Plast Surg. 2002;49(4):379–84.
5. Burgu B, Duffy PG, Cuckow P, Ransley P, Wilcox DT. Long-term outcome of vaginal reconstruction: comparing techniques and timing. J Pediatr Urol. 2007;3:316–20.
6. Ozkan O, Akar EM, Ozkan O, Dogan NU. Reconstruction of vaginal agenesis. Ann Plast Surg. 2011;66(6):673–8.
7. Marshall FF. Radical cystectomy in the female. AUR Update Ser. 1997;27:1–8.
8. Eifel PJ, Berek JS, Thigpen JT. Cancer of the cervix, vagina and vulva. In: DeVita VT, Hellman S, Rosenberg SA, editors. Cancer principles and practice of oncology. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 1997. p. 1433–78.
9. Economy KE, Barnewolt C, Laufer MR. A comparison of MRI and laparoscopy in detecting pelvic structures in cases of vaginal agenesis. J Pediatr Adolesc Gynecol. 2002;15:101–4.
10. Church DG, Vancil JM, Vasanawala SS. Magnetic resonance imaging for uterine and vaginal anomalies. Curr Opin Obstet Gynecol. 2009;21:379–89.
11. Poland ML, Evans TN. Psychologic aspects of vaginal agenesis. J Reprod Med. 1985;30:340–4.

12. Laufer MR. Congenital absence of the vagina: in search of the perfect solution. When, and by what technique, should a vagina be created? Curr Opin Obstet Gynecol. 2002;14:441–4.
13. Lappohn RE. Congenital absence of the vagina: results of conservative treatment. Eur J Obstet Gynecol Reprod Biol. 1995;59:183–6.
14. Williams EA. Congenital absence of the vagina. J Obstet Gynaecol Br Commonw. 1972;79:1147–8.
15. Gargollo PC, Cannon GM, Diamond DA, Thomas P, Burke V, Laufer MR. Should progressive perineal dilation be considered first line therapy for vaginal agenesis. J Urol. 2009;182:1882–91.
16. Nonsurgical diagnosis and management of vaginal agenesis. ACOG Committee Opinion. No. 274, July 2002. Obstet Gynecol. 2002;100(1):213–7.
17. Frank RT. The formation of an artificial vagina without operation. AJOG. 1938;35:1053–5.
18. Ingram JM. The bicycle seat stool in the treatment of vaginal agenesis and stenosis: a preliminary report. AJOG. 1981;140:867–73.
19. Roberts CP, Haber MJ, Rock JA. Vaginal creation for mullerian agenesis. AJOG. 2001;185:1349–52.
20. Klingele CJ, Gebhart JB, Croak AJ, DiMarco CS, Lesnick TG, Lee RA. McIndoe procedure for vaginal agenesis: long-term outcomes and effect on quality of life. AJOG. 2003;189:1569–73.
21. Bo K, Berghmans B, Morkved S, Van Kampen M. Evidence-based physical therapy for the pelvic floor: bridging science and clinical practice. 1st ed. Philadelphia, PA: Elsevier; 2007. p. 263–4.
22. McGuire H, Hawton KKE. Interventions for vaginismus. Cochrane Database Systematic Reviews [Internet] 2003 [cited 12 Dec 2012] Available from http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001760/frame.html
23. McVearry ME, Warner WB. Use of physical therapy to augment dilator treatment for vaginal agenesis. FPMRS. 2011;17(3):153–6.
24. Vaginal agenesis: diagnosis, management, and routine care. ACOG Committee Opinion No. 355. Obstet Gynecol. 2006;108:1605–9.
25. McIndoe AH, Banister JB. An operation for the cure of congenital absence of the vagina. J Obstet Gynecol Br Emp. 1938;45:490–6.
26. Farber M, Mitchell GW. Surgery for congenital absence of the vagina. Obstet Gynecol. 1978;51:364–6.
27. Buss JG, Lee RA. McIndoe procedure for vaginal agenesis: results and complications. Mayo Clin Proc. 1989;64:758–62.
28. Martinez-Mora R, Isnard R, Castellvi A, Lopez OP. Neovagina in vaginal agensis: surgical methods and long-term results. J Pediatr Surg. 1992;27:10–4.
29. Akin S. Experience with neovaginal construction using the full-thickness skin graft in vaginal agenesis. Ann Plast Surg. 2004;52(4):391–6.
30. Ozgenel GY, Ozcan M. Neovaginal construction with buccal mucosal grafts. Plast Reconstr Surg. 2003; 111(7):2250–4.
31. Ashworth MF, Morton KE, Dewhurst J, Lilford RJ, Bates RG. Vaginoplasty using amnion. Obstet Gynecol. 1986;67:443–6.
32. Jackson ND, Rosenblatt PL. Use of interceed absorbable barrier for vaginoplasty. Obstet Gynecol. 1994;84:1048–50.
33. Sadove RC, Horton CE. Utilizing full-thickness skin grafts for vaginal reconstruction. Clin Plast Surg. 1988;15:443–8.
34. Chen YB, Cheng T, Lin HH, Yang YS. Spatial W-plasty full-thickness skin graft for neovaginal reconstruction. Plast Reconstr Surg. 1994;94:727–31.
35. Alessandrescu D, Peltecu GC, Buhimschi CS, Buhimschi IA. Neocolpopoiesis with split-thickness skin graft as a surgical treatment of vaginal agenesis: retrospective review of 201 cases. AJOG. 1996;175: 131–8.
36. Loveless M, Gutman R, Cundiff G. Tissue expanders optimize harvesting full-thickness skin grafts in select patients undergoing McIndoe neovagina. J Pelvic Med Surg. 2008;14(3):185–90.
37. Zhao M, Li P, Li S, Li Q. Use of autologous micromucosa graft for vaginoplasty in vaginal agenesis. Plast Reconstr Surg. 2009;63(6):645–9.
38. Lin WC, Chang CY, Shen YY, Tsai HD. Use of autologous buccal mucosa for vaginoplasty: a study of eight cases. Hum Reprod. 2003;18:604–7.
39. Stany MP, Sunde J, Bidus MA, Rose GS, Elkas JC. The use of acellular dermal allograft for vulvovaginal reconstruction. Int J Gynecol Cancer. 2010;20:1079–81.
40. DePhilippo RE, Bishop CE, Filho LF, Yoo JJ, Atala A. Tissue engineering a complete vaginal replacement from a small biopsy of autologous tissue. Transplantation. 2008;86:208–14.
41. Panici PB, Bellati F, Boni T, Francescangeli F, Frati L, Marchese C. Vaginoplasty using autologous in vitro cultured vaginal tissue in a patient with Mayer-von-Rokitansky-Kuster-Hause syndrome. Hum Reprod. 2007;22:2025–8.
42. Williams EA. Congenital absence of the vagina; a simple operation for its relief. J Obstet Gynecol Commonw. 1964;71:511–4.
43. Creatsas G, Deligeoroglou E, Makrakis E, Kontoravdis A, Papadimitriou L. Creation of a neovagina following Williams vaginoplasty and the Creatsas modification in 111 patients with Mayer-Rokitansy-Kuster-Hauser syndrome. Fertil Steril. 2001;76(5):1036–40.
44. Deligeoroglou E, Makrakis E, Papagianni V. The Creatsas modification of Williams vaginoplasty. J Gynecol Surg. 2003;19(3):121–7.
45. Ismail IS, Cutner AS, Creighton SM. Laparoscopic vaginoplasty: alternative techniques in vaginal reconstruction. BJOG. 2006;113:340–3.
46. Vecchietti G, Ardillo C. La sindrome di Rokitanski-Kuster-Hauser fisiologia e clinica. Aplasia Vaginale. Corni uterini rudimentali. SEU Roma. 1970;320–7.
47. Fedele L, Bianchi S, Frontino G, Fontana E, Restelli E, Bruni V. The laparoscopic Vecchietti's modified technique in Rokitansky syndrome: anatomic,

functional, and sexual longterm results. AJOG. 2008; 198:377.e1–6.
48. Crouch NS, Creighton SM. Minimal surgical intervention in the management of intersex conditions. J Pediatr Endocrinol Metab. 2004;17:1591–6.
49. Ninkovic M, Dabernig W. Flap technology for reconstructions of urogenital organs. Curr Opin Urol. 2003;13(6):483–8.
50. Casey III WJ, Tran NV, Petty PM, Stulak JM, Woods JE. A comparison of 99 consecutive vaginal reconstructions: an outcome study. Ann Plast Surg. 2004; 52:27–30.
51. Soper JT, Havrilesky LJ, Secord AA, Berchuck A, Clarke-Pearson DL. Rectus abdominis myocutaneous flaps for neovaginal reconstruction after radical pelvic surgery. Int J Gynecol Cancer. 2005;15:542–8.
52. Smith H, Genesen M, Runowicz C, Goldberg GL. The rectus abdominis myocutaneous flap. Cancer. 1998; 83:510–20.
53. Hui K, Zheng F, Pickus E. Modification of the vertical rectus abdominis muscylocutaenous (VRAM) flap for functional reconstruction of complex vulvoperineal defects. Ann Plast Surg. 2003;51(6):556–60.
54. Pursell SH, Day RG, Tobin GR. Distally based rectus abdominis flap for reconstruction in radical gynecologic procedures. Gynecol Oncol. 1990;37:234–8.
55. Carlson JW, Carter JR, Saltzman AK, Carson LF, Fowler JM, Twiggs LB. Gynecologic reconstruction with a rectus abdominis myocutaneous flap: an update. Gynecol Oncol. 1996;61:364–8.
56. Bakx R, Van Lanschot JJB, Zoetmulder FAN. Inferiorly based rectus abdominis flaps in surgical oncology: indications, technique and experience in 37 patients. J Surg Oncol. 2004;85:93–7.
57. Soper JT, Secord AA, Havrilesky LJ, Berchuck A, Clarke-Pearson DL. Comparison of gracilis and rectus abdominis myocutaenous flap neovaginal reconstruction performed during radical pelvic surgery: flap-specific morbidity. Int J Gynecol Cancer. 2007; 17:298–303.
58. McGraw JB, Massey FB, Shanklin KD, Horton CE. Vaginal reconstruction with gracilis myocutaneous flaps. Plast Reconstr Surg. 1976;58:176–83.
59. Friedman JD, Reece GR, Eldor L. The utility of the posterior thigh flap for complex pelvic and perineal reconstruction. Plast Reconstr Surg. 2010;126(1): 146–55.
60. Giraldo F, Solano A, Mora M, Abehsera M, Gonzalez C, Rus JA. The Malaga flap for vaginoplasty in the Mayer-Rokitansy-Kuster-Hauser syndrome: experience and early-term results. Plast Reconstr Surg. 1996;98(2):305–12.
61. Ganatra MA, Ansari NR. Pudendal thigh flap for congenital absence of vagina. J Pak Med Assoc. 2005; 55(4):143–5.
62. Martius J. Operations for urinary incontinence. In: McCall M, Bolten KA, editors. Operative gynecology. Boston: Little, Brown; 1956.
63. Green AE, Escobar AF, Neubaurer N. The martius flap revisited. Int J Gynecol Cancer. 2005;15:964–6.
64. Crawley WA, Grumbine FC, Dorsey JH. Flay reconstruction of the stenotic vagina. J Am Coll Surg. 1994;178:47–8.
65. Hatch KD. Construction of a neovagina after exenteration using the vulvobulbocavernosus myocutaneous graft. Obstet Gynecol. 1984;63:110–4.
66. Esrig D, Freeman JA, Stein JP, Elmajian DA, Lytton B, Skinner DG. New technique of vaginal reconstruction following anterior exenteration. Urology. 1997;49:768–71.
67. Ellaffandi AH, Khalil HH, Aboul Kassem HA, El Sherbiny M, El Gemeie EH. Vaginal reconstruction with greater omentum-graft combined with a vicryl mesh after anterior pelvic exenteration. Surgical approach with long-term follow-up. Int J Gynecol Cancer. 2007;17:536–42.
68. Davies MC, Creighton SM. Vaginoplasty. Curr Opin Urol. 2007;17:415–8.
69. Wu JX, Li B, Liu T, Li WZ, Jiang YG, Liang JX, Wei CS, Hu HO, Zhong CX. Eighty-six cases of laparoscopic vaginoplasty using an ileal segment. Chin Med J. 2009;122:1862–6.
70. Li B, Wang J, Wu JX, Wang LY. Clinical analysis of vaginoplasty with sigmoid colon by laparoscopic surgery. Zhonghua Fu Chan Ke Za Zhi. 2009;44:673–5.
71. Kwun Kim S, Hoon Park J, Cheol Lee K, Min Park J, Tae Kim J, Chan KM. Long-term results in patients after rectosigmoid vaginoplasty. Plast Reconstr Surg. 2003;112:143–51.
72. Hensle TW, Shabsigh A, Shabsigh R, Reiley EA, Meyer-Bahlburg HF. Sexual function following bowel vaginoplasty. J Urol. 2006;175:2283–6.
73. Hiroi H, Yasugi T, Matsumoto K, Fujii T, Watanabe T, Yoshikawa H, Taketani Y. Mucinous adenocarcinoma arising in a neovagina using the sigmoid colon thirty years after operation: a case report. J Surg Oncol. 2001;77:61–4.
74. Chen HC, Chana JS, Feng GM. A new method for vaginal reconstruction using a pedicled jejunal flap. Ann Plast Surg. 2003;51(4):429–31.
75. Ramaswamy S. Mullerian agenesis with vaginal prolapse. Case report. BJOG. 1986;93:640–1.
76. Peters WA, Uhlir JK. Prolapse of a neovagina created through self-dilatation. Obstet Gynecol. 1990;76:904–6.
77. Schaffer J, Fabricant C, Carr B. Vaginal vault prolapse after surgical and nonsurgical treatment of Mullerian agenesis. Obstet Gynecol. 2002;99(5):947–9.
78. Muir TW, Walters MD. Surgical management of vaignal vault prolapse in a woman with a neovagina and pelvic kidneys. Obstet Gynecol. 2004;104(5,2):1199–201.

Index

F. Firoozi (ed.), *Female Pelvic Surgery*, DOI 10.1007/978-1-4939-1504-0,