Herand Abcarian · Jose Cintron
Richard Nelson *Editors*

Complications of Anorectal Surgery

Prevention and Management

Springer

Complications of Anorectal Surgery

Herand Abcarian · Jose Cintron
Richard Nelson
Editors

Complications of Anorectal Surgery

Prevention and Management

Springer

Editors
Herand Abcarian
Division of Colon and Rectal Surgery
University of Illinois at Chicago
Chicago, IL
USA

Jose Cintron
Division of Colon and Rectal Surgery
John H. Stroger Jr. Hospital of Cook County
Chicago, IL
USA

Richard Nelson
Epidemiology/Biometry Division, School of Public Health
University of Illinois at Chicago
Chicago, IL
USA

ISBN 978-3-319-48404-4 ISBN 978-3-319-48406-8 (eBook)
DOI 10.1007/978-3-319-48406-8

Library of Congress Control Number: 2016954713

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

We would like to dedicate this book to a dear friend and recently deceased colleague, Leela M Prasad, whose wisdom was seldom withheld, spot on and always greatly appreciated, and also one of the finest surgeons we all have ever seen.

Preface

There are many texts in which we can find techniques of various anorectal procedures. The interested surgeon can easily find descriptions and illustrations of each surgical technique. However, what is more difficult to find is the prevention and treatment of many complications following anorectal operations.

Complications of surgical procedures are without a doubt the greatest source of disquiet for every surgeon. We never feel more alone when one of our patients has suffered a complication that has caused them suffering and never more comforted when we find that our mates have experience with the same complication. It is one of the major purposes of our annual clinical congresses: conversation, commiseration, consolation, a.k.a., GOBAGSATT (good old boys—and girls—sitting around talking trash) and ultimately education.

We present in this book the complications that have been suffered by our own patients and/or by those of our closest friends. Some are common and can only be avoided most of the time. Others are rare, and in some cases just weird. But if it has happened once, it will happen twice. Many can be prevented, which we hope to facilitate with this book.

Chicago, IL, USA — Herand Abcarian, M.D.
Chicago, IL, USA — Jose Cintron, M.D.
Chicago, IL, USA — Richard Nelson, M.D.

Contents

Contributors

Ariane M. Abcarian Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, Chicago, IL, USA

Herand Abcarian Division of Colon and Rectal Surgery, University of Illinois at Chicago, Chicago, IL, USA

Jennifer Blumetti Colon and Rectal Surgery Residency Program, Stroger Hospital of Cook County, Chicago, IL, USA

Marc I. Brand Section of Colon and Rectal Surgery, Department of General Surgery, Rush University Medical Center, Chicago, IL, USA

Steven Brown Thornbury Hospital, Sheffield, South Yorkshire, UK

Vivek Chaudhry Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, Chicago, IL, USA

Jose Cintron Division of Colon and Rectal Surgery, John H. Stoger Hospital of Cook County, Chicago, IL, USA

Francois Dagbert Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, Chicago, IL, USA

Kristen Donohue Department of Surgery, Rutgers: Robert Wood Johnson Medical School, New Brunswick, NJ, USA

Linda M. Farkas Division of Colon and Rectal Surgery, Department of Surgery, University of California, Davis, Sacramento, CA, USA

Timothy F. Feldmann Capital Medical Center, Olympia, WA, USA

Salvador G. Guevara Division of Colon and Rectal Surgery, Department of Surgery, University of California, Davis, Sacramento, CA, USA

Jacqueline Harrison Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, Chicago, IL, USA

Torbjörn Holm Department of Molecular Medicine and Surgery, Karolinska Institutet, Stockholm, Sweden; Karolinska University Hospital, Stockholm, Sweden

Kunal Kochar Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, Chicago, IL, USA

Kristine Makiewicz Section of Colon and Rectal Surgery, Department of General Surgery, Rush University Medical Center, Chicago, IL, USA

Nell Maloney Patel Department of Surgery, Rutgers: Robert Wood Johnson Medical School, New Brunswick, NJ, USA

Slawomir Marecik Division of Colon and Rectal Surgery, Advocate Lutheran General Hospital, Park Ridge, IL, USA; Division of Colon and Rectal Surgery, University of Illinois at Chicago, Chicago, IL, USA

Anders Mellgren Division of Colon and Rectal Surgery, Department of General Surgery, University of Illinois at Chicago, Chicago, IL, USA

Richard Nelson Epidemiology/Biometry Division, School of Public Health, University of Illinois at Chicago, Chicago, IL, USA

Johan Nordenstam Division of Colon and Rectal Surgery, Department of Surgery, University of Illinois at Chicago, Chicago, IL, USA

Adrian E. Ortega LAC+USC Medical Center, University of Southern California, Los Angeles, CA, USA

Leela M. Prasad Division of Colon and Rectal Surgery, Advocate Lutheran General Hospital, Park Ridge, IL, USA; Division of Colon and Rectal Surgery, University of Illinois at Chicago, Chicago, IL, USA

Marc Singer Division of Colon and Rectal Surgery, Rush University Medical Center, Chicago, IL, USA

Sany Thomas Division of Colon and Rectal Surgery, Department of Surgery, University of Illinois at Chicago, Chicago, IL, USA

Kristin Vercillo Colon & Rectal Surgery Resident, Stroger Hospital of Cook County, Chicago, USA

Christina Warner Department of General Surgery, University of Illinois at Chicago, Chicago, IL, USA

Surgery for Anorectal Abscess

1

Adrian E. Ortega, Timothy F. Feldmann, Ariane M. Abcarian and Herand Abcarian

Complications of Surgery for Cryptoglandular Anorectal Infections

Adrian E. Ortega and Timothy F. Feldmann

Introduction

Like many of the writers since antiquities, Hippocrates believed that anorectal infections resulted from traumatic activities including horseback riding and rowing. He referred to them as "tubercles," and wrote: "Whenever you observe any such tubercle forming, incise this as soon as possible during the *unconcocted* state before suppuration into the rectum occurs." [1].

Late nineteenth and early twentieth century surgical scholars codified numerous elements pervasive in the current day practice of proctology. Namely, drainage is directed over the area where the abscess points. Cruciate incision with resection of

A.E. Ortega (✉)
LAC+USC Medical Center, University of Southern California,
Clinic Tower Room, 6A231-A, 1200N. State Street, Los Angeles, CA 90033, USA
e-mail: sccowboy78@gmail.com

T.F. Feldmann
Capital Medical Center, 3900 Mall Dr. SW, Olympia, WA 98502, USA

A.M. Abcarian
Division of Colon and Rectal Surgery, John H. Stroger Hospital
of Cook County, 1900W. Polk Street, Chicago, IL 60612, USA

H. Abcarian
Division of Colon and Rectal Surgery, University of Illinois at Chicago,
840 S. Wood Street, Chicago, IL 60612, USA

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_1

the skin edges is required to prevent premature wound closure and persistent sepsis. Loculations within the abscess cavity should be broken up aggressively for the same reason. Packing is indicated in order to establish hemostasis within the wound. Primary fistulotomy at the time of incision and drainage is ill advised since most acute abscesses do not result in chronic fistula and may be harmful vis-à-vis iatrogenic fistula and incontinence. Finally, fistula is a non-preventable component of the natural history of anorectal infections in a small proportion of patients.

The current understanding of anorectal infection suggests that *all the aforementioned* concepts are associated with their own untoward consequences and should be reconsidered. This chapter identifies common and uncommon complications associated with the surgical treatment of acute cryptoglandular anorectal infections (Part I). The authors offer evidence-based as well as novel perspectives focusing on the clinical strategies required for the prevention of surgical misadventures and suboptimal outcomes (Part II).

Part I: Complications

Neurovascular Injuries

While bleeding may occur with any surgical procedure, hemorrhage is distinctly uncommon in incision and drainage of anorectal abscesses. Generalized oozing occurs particularly in deep-seated abscesses. It also usually resolves following evacuation of the purulent material and irrigation. However, the practice of breaking up loculations with a clamp typically in the ischioanal fossa or higher may be associated with injury to either or both inferior rectal or inferior pudendal vessels and nerves. Gentle exploration with the surgeon's index finger is preferred. Frank hemorrhage may be associated with a pseudoaneurysmal rupture of an injured blood vessel within an abscess cavity, but is an exceedingly rare event. The incidence of postoperative bleeding in one large series was only 0.6% [2]. Similarly aggressive packing of the wound is associated with delayed healing and potential sphincteric injury probably secondary to nerve injury. Light packing or no packing is generally preferred [3].

Persistent Sepsis

Persistent sepsis is the failure to resolve an acute infection following a surgical intervention. The largest reported series of 500 consecutive cases examined early reoperations for perirectal abscess. Onaka et al. cites an overall incidence of 8.2% of persistent sepsis. In their estimation, these failures were secondary to incomplete drainage (4.6%), missed loculations within the abscess (3.0%), and missed abscesses (0.6%). Albeit infrequent, persistent sepsis is a preventable complication [2] (Fig. 1.1).

The root cause for persistent sepsis following a surgical drainage procedure is multifactorial. In the series above, infections classified as perianal had the highest rate of reoperations followed by ischiorectal, intersphincteric and supralevator abscesses. The observation is counter-intuitive. However, it is not difficult to understand. Many surgeons, particularly the less experienced focus much attention

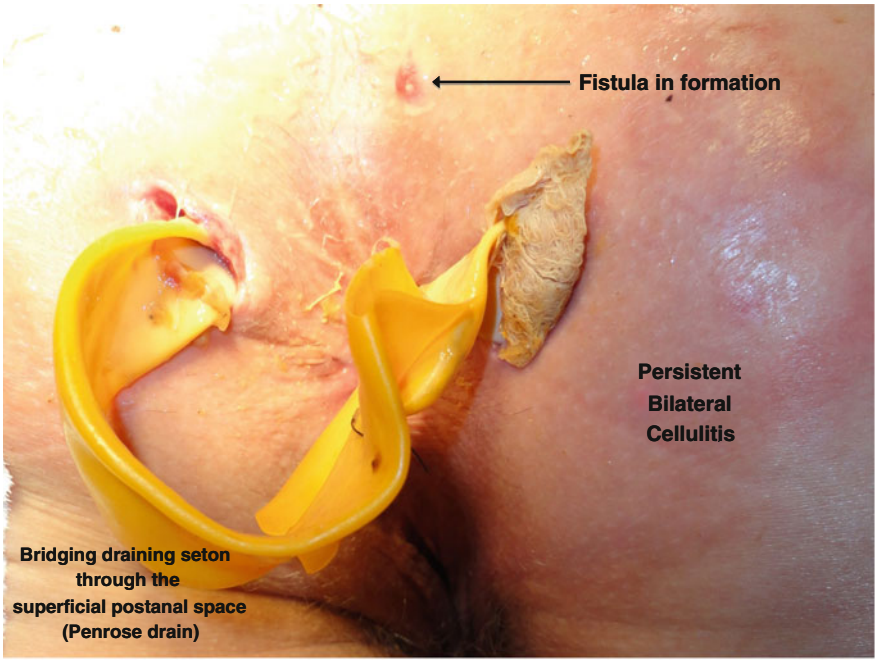


Fig. 1.1 Persistent sepsis and a fistula in formation are in evidence. While both ischioanal fossae were incised and connected with a draining penrose seton communicated through the superficial postanal space, the deep postanal infection was not addressed by this intervention

on the obvious topographic appearance of the perianal region. This tendency facilitates overlooking important elements in the history and physical examination of the patient. Most anorectal infections appear simple. However, they may represent the "tip of the iceberg" of a more complex infection.

The authors speculate on the legitimacy of the concept of loculated abscesses. It is a difficult concept to prove objectively. Multiple space infections, however, are well documented. Examples include secondary ischioanal infections from a deep post anal space or supralevator space primary. Supralevators may also appear as a perianal infection on topographic inspection.

While the percentage of persistent infections is relatively small, it is not a diminutive topic in clinical practice. It is seen commonly in diabetic patients even when appropriately drained. These patients often require a tincture of time on antibiotics, glucose control and local wound care (sits baths). Ongoing sepsis is also common in patients who undergo drainage of a secondary infection leaving the primary septic focus undrained. When in doubt, it is best to image the patient and proceed to the anatomically indicated drainage procedure in a formal operative setting.

Progression of Sepsis

The concept of "pus under pressure" allows dissection along tissue planes and into potential spaces. Cryptoglandular infections can also disseminate into the supralevator space through various routes as discussed in Part B. Once seeded, large collections can propagate to the psoas muscle or along the paraspinal musculature. The treatment of these complications must be control of the primary site of infection and drainage of the infection itself. Necrotizing fasciitis is a relatively uncommon complication of cryptoglandular disease seen in the setting of immunologically challenged individuals [3].

Recurrent Abscess

Recurrent abscess is a common finding with cryptoglandular disease. 25–50% of patients may experience recurrence of an abscess. Recurrence should be differentiated from persistent disease through a thorough history and delineation of the patient's symptoms. Without a symptom-free interval, the abscess was likely not resolved from the initial treatment. True recurrent abscesses may or may not have an associated anal fistula. Recurrent infection is distinctly less common following definitive treatment of the abscess addressing the primary fistulous tract as compared to simple incision and drainage alone [4]. Definitive surgery also results in fewer surgeries overall vis-a-vis simple drainage [5].

Delayed Wound Healing

The perineum is normally a very well vascularized field. Redundant blood supply through the branches of the internal iliac arteries allow for excellent healing in most circumstances. Despite the possible frequent contamination from stool, these wounds will often heal quickly. Nutritional status and immune competence may mitigate wound healing. Robust packing of wounds has been demonstrated to delay wound healing [6]. Wounds rarely heal in the setting of previous radiation. Occasionally, non-healing wounds may be seen in the context of an underlying malignancy or osteomyelitis of a pelvic bone may be seen. Poorly optimized patients with human immune deficiency virus may demonstrate poor wound healing. However, they are generally symptomatically benefited from anorectal surgical interventions.

Wound Contraction Deformities

Contraction deformities or "step-offs" are the result of cruciate or vertical drainage incisions. Neither of the previous has been show to prevent premature wound closure. However, the effectiveness of radial incisions was reported to be 99.6% in one large series [7]. Radial incisional drainage was first described by Ayers in 1886 specifically emphasizing the prevention of contraction deformities [8].

Iatrogenic Fistula

Fistula may form following an anorectal abscess in 5–85% of cases [9]. An iatrogenic fistula can form following incorrect drainage of a complex abscess. Examples include a suprasphincteric fistula secondary to external drainage of an

intersphincteric supralevator abscess. Conversely, extrasphincteric fistula result from internal drainage of an extrasphincteric supralevator abscess. Theoretically, iatrogenic fistula may result from definitive treatment of the abscess and primary fistulous trajectory simultaneously. However, the incidence of this latter complication is unknown. It is also important to note that complex fistula may form when simple drainage is performed on multi-space infections. Spontaneous drainage of an abscess can also produce complex fistulas.

Chronic Fistula

Conventional wisdom dictates that a certain proportion of patients with an acute anorectal infection will progress to the chronic phase with an anal fistula. To what degree a chronic fistula is preventable is not known. However, it is clear that drainage with definitive treatment of its primary fistulous component is associated with fewer operations subsequently for recurrent abscesses and fistula. The incidence of incontinence does not appear to increase with combined treatment [10]. Definitive treatment of infections involving the deep postanal space abscesses can prevent horseshoe, hemi-horseshoe, suprasphincteric, and extrasphincteric fistulas in most cases.

Incontinence

Anorectal infections and their treatment continue to put patients at risk for incontinence. Knoefel also note that recurrent infections are more likely to alter sphincter function than fistulotomy with drainage than simple drainage alone [11]. Prior to any intervention that may involve the sphincters, a surgeon should assess sphincter function in both a subjective and objective fashion. The patient should be queried as to their current function with control of stool and gas. Objectively the resting pressure and squeeze pressure of the anorectal ring should be assessed. This does not need to involve specific manometric testing but rather a digital exam by an experienced practitioner. In the acute setting this may be impossible due to pain from the infection. The physician can then document the perceived continence prior to the infection. If treatment for fistula is to be undertaken, preoperative assessment is essential prior to operative decision-making. The subjective portion of the patient's continence may then be less reliable due to leakage from the fistula. Many patients may be unable to distinguish incontinence from fistula drainage in this setting. In the current state of the art, alterations in continence may result from both simple drainage as well as definitive treatment of abscesses with the fistulous components. Fortunately, it is an uncommon problem (1–2%) [10]. Moreover, these alterations are generally limited to gas and liquid incontinence of the majority. Full disclosure of the inherent risks, albeit small, is paramount in all cases.

Part II: Prevention Strategies

The prevention of untoward sequelae in the treatment of acute anorectal infections begins with a systematic clinical approach. Pain is the most common symptom. Its time in evolution and duration are important considerations. Rapid onset over a short duration is commonly seen with perianal infections. This feature is thought to

be secondary to the dissection of pus through the tight fascial compartments of the corrugator cutis extensions of the sphincter muscles out toward the skin at the anal margin. Pain augmented on defecation is compatible with an intersphincteric infection secondary to the dissection of pus between the internal and external sphincters. Longer durations of symptoms favor infections occupying large spaces like the ischioanal fossa or multiple spaces (e.g., supralevator abscesses). Tenesmus and pain augmented with defecation are also characteristic of supralevator processes.

Visual inspection of the perianal region is important. However, it needs to be considered in the context of acute cryptoglandular obstruction as a chronologic sequence of inflammatory changes. Cellulitis generally precedes lymphedema and lymphatic obstruction. Fluctuance secondary to a frank abscess is a relatively late event in this sequence. Therefore, clinicians should not over interpret obvious topographic signs, as they may be harbingers of deeper seated infections. The absence of obvious clinical signs suggests an occult anorectal infection. Submucosal, intersphincteric, deep post anal space and supralevator abscesses fall into this category.

Digital rectal examination should also be systematic. Palpation of a boggy mass above the sphincters is the sine qua non of supralevator processes. Submucosal abscess begin in the anal canal extending proximally a short distance. Intersphincteric supralevators are palpable above puborectalis muscle. Bi-digital examination with the examiner's index finger within the rectum and thumb over the ischioanal fossa identifies involvement of the latter space. Palpation of the anal canal may reveal an area of induration or a divot associated with the site of the primary cryptoglandular obstruction. Purulent discharge per anus has three possible explanations: (1) decompression of the abscess at the primary crypt of origin; (2) spontaneous decompression of a supralevator abscess into the rectum; and (3) proctocolitis. When formal exploration of an abscess is contemplated, the abscess should not be compressed until evaluation under anesthesia in order to optimize one's ability to identify the primary crypt and fistulous trajectory.

The natural history of anorectal infections is mucocutaneous necrosis and spontaneous drainage by Providence unless intervened first by a competent physician. Longstanding neglected infections upon spontaneous drainage may produce undesirable consequences including complex fistula. Therefore, the time honored concept of early drainage remains as an operant fundamental concept.

The tetralogy of occult anorectal sepsis consists of pain, evidence of infection, a paucity of obvious physical findings and hyperesthesia often limiting the physical examination. It is commonly associated with submucosal, intersphincteric, isolated deep post anal and supralevator abscess. Imaging is invaluable in this setting with the intent to treat formally in the operative room setting.

A Practical Approach to Anorectal Infections

Cryptoglandular infection propagates by three routes: (1) superficial, (2) intersphincteric, and (3) transsphincteric. Extrasphincteric refers to a supralevator transsphincteric process. Superficial infections result in submucosal or subcutaneous

abscess. They pose few problems if correctly diagnosed. While submucosal abscess are generally associated with normal perianal topography, they are palpable within the anal canal as a walnut-size soft and tender mass. Intersphincteric trajectories may present as perianal, an abscesses confined to the anal canal or with proximal extension as a supralevator abscess. Combinations in all three spaces are seen with an intersphincteric trajectory. Transsphincteric trajectories are the least common, but the most problematic. In its simplest configuration, it results in a primary unilateral primary ischioanal abscess. A posterior transsphincteric fistulous trajectory results in a deep post anal space abscess. Abscesses in this location may be isolated with negligible physical signs if seen early. However, they may propagate anterolaterally to produce a secondary ischioanal abscess. Bilateral propagation from the deep post anal space results in one important variant of a "horseshoe" presentation. Deep post anal space infections can dissect between the leaves of the pubococcygeus muscles that insert onto the sides of the coccyx. This trajectory produces a posterior extrasphincteric supralevator abscess. In its most unusual form, a deep post anal space abscess can propagate to both the supralevator and ischioanal spaces. The secondary ischioanal component can independently and simultaneously disseminate into the supralevator space along the medial aspect of the fascia of the obturator internus muscle. This scenario produces a combined extrasphincteric supralevator abscess from a deep post anal primary and ischioanal secondary infection. Ortega and colleagues have proposed a novel classification of supralevator abscesses based on these principles: type I intersphincteric, type II anterolateral extrasphincteric via the ischioanal fossa, type III posterior extrasphincteric from the deep post anal space, and type IV combined posterior and anterolateral extrasphincteric via the deep post anal and ischioanal spaces simultaneously [12] (Fig. 1.2).

The correct diagnosis and treatment of supralevator abscesses require the evaluation of three spaces: (1) supralevator, (2) ischioanal, and (3) the deep postanal. It is important to point out that more than half of supralevator abscesses of cryptoglandular origin have the deep postanal space as their primary site of infection. Figure 1.3 demonstrates the diagnostic and treatment algorithm required for successful management of supralevator infections.

Simple superficial low infections pose few problems in the short and long term. Deeper seated as well as infections occupying multiple spaces are distinctly different. The latter offer opportunities for the prevention of persistent, recurrent, and chronic sepsis (i.e., complex anal fistula). Therefore, horseshoe presentations, ischioanal, deep postanal, supralevator abscesses, and horseshoe presentations merit special consideration.

Ischioanal infections are the second most common type of infection following perianal abscesses. In contrast, their presentation is longer in evolution and their size is greater in lateral extension. Few topographic signs may be present early in their evolution. However, a palpable fullness between the examiner's index finger and thumb may be appreciated. In their most common later presentation, a large area of cellulitis often extending several centimeters outside the anal margin extending over the gluteal tissues may be seen. They are often drained as a bedside procedure. This common practice merits reconsideration. Primary ischioanal

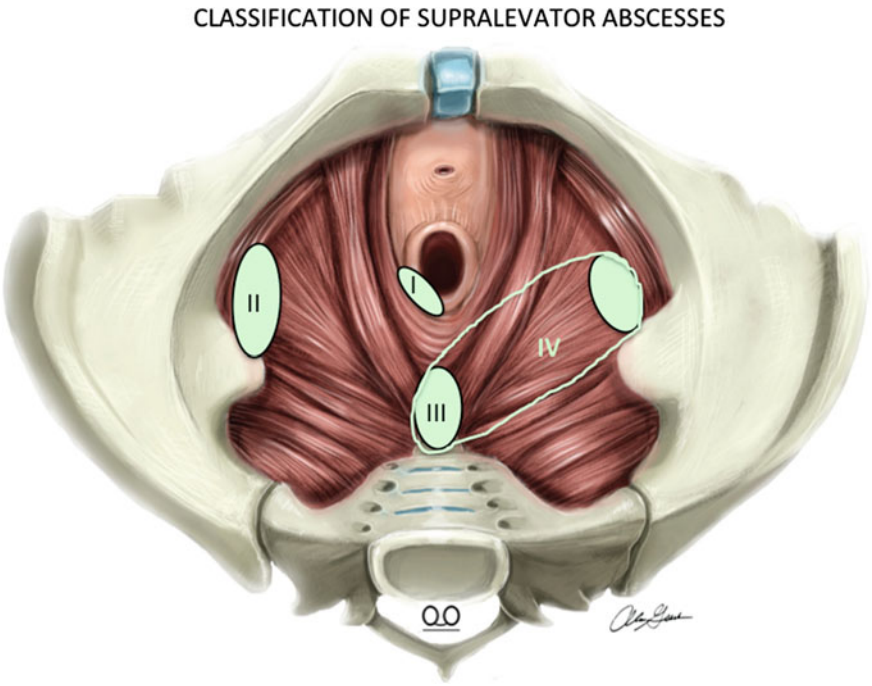


Fig. 1.2 Novel classification scheme for supralevator abscesses. Type I—Intersphincteric. Type II—Extrasphincteric supralevator extension of a primary transsphincteric ischioanal abscess. Type III—Posterior extrasphincteric supralevator extension from a primary deep post anal space abscess. Type IV—Extrasphincteric supralevator extension from both the deep post anal space and the ischioanal space simultaneously (unilateral or bilateral)

abscesses represent a direct transsphincteric process to one isolated fossa. Secondary ischioanal infections originate in the deep postanal space and then propagate to one or both ischioanal fossae. This bilaterally may be symmetrical, asymmetrical, or delayed. For all these reasons, the authors recommend imaging of ischioanal abscesses in order to facilitate definitive management. This strategy appears to be most reasonable for the prevention of persistent and recurrent sepsis as well as difficult chronic fistula in this context.

Primary ischioanal abscesses are approached either as a bedside or formal operative procedure. Secondary ischioanal infections offer a unique opportunity for definitive management in which persistent and recurrent infection can be minimized. The surgical approach to the deep postanal space entails a crypt to coccyx midline incision. The anococcygeal ligament is sectioned vertically in its midline. A transsphincteric seton may be placed through the primary posterior fistulous trajectory.

The belief that supralevator abscesses are rare needs to be reconsidered. Supralevator abscesses were identified in one large series in 9.1% of cases. Therefore, while less common they comprise nearly one in ten cryptoglandular

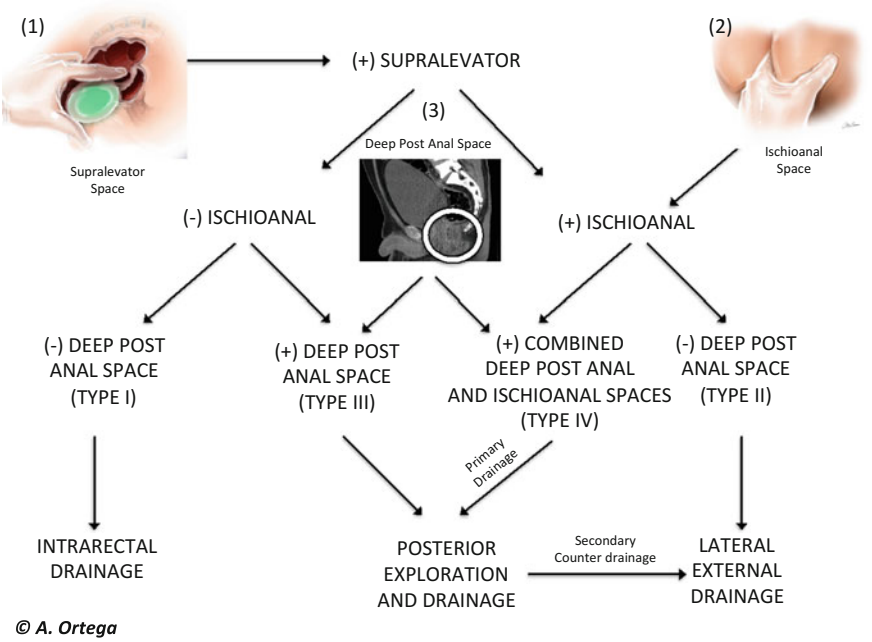


Fig. 1.3 Algorithm for treatment of supralevator abscesses. Evaluation of three spaces is required: (*1*) supralevator, (*2*) ischioanal, and (*3*) deep postanal. Their inclusion or exclusion determines the correct intervention required to prevent recurrent or persistent sepsis as well complex fistula

infections [13]. More than half have no obvious physical signs on visual inspection. Palpation of a boggy mass within the rectum is the diagnostic sine qua non. More than half of supralevator abscesses have the deep postanal space as their primary site of infection.

Intrarectal drainage of a type I intersphincteric supralevator abscess is definitive treatment. If the intersphincteric primary fistulous trajectory is identified, an internal sphincterotomy incorporating it may be performed. It may be preferable to place an intersphincteric seton in special cases including an anterior trajectory in a female or a posterior midline trajectory in either gender. The latter diminishes the risk of forming a posterior keyhole deformity that enables seepage of mucous subsequently (Fig. 1.4).

Anterolateral type II extrasphincteric abscesses are drained external via the ischioanal fossa with or without a transsphincteric seton through the primary fistulous trajectory. Because of the depth of the drainage tract, most surgeons place a supralevator drainage catheter in the space. Once the space is collapsed several weeks later, this catheter is withdrawn. Some surgeons make no effort in localizing the primary fistulous trajectory thereby simplifying wound care with the single catheter drain (Fig. 1.5).

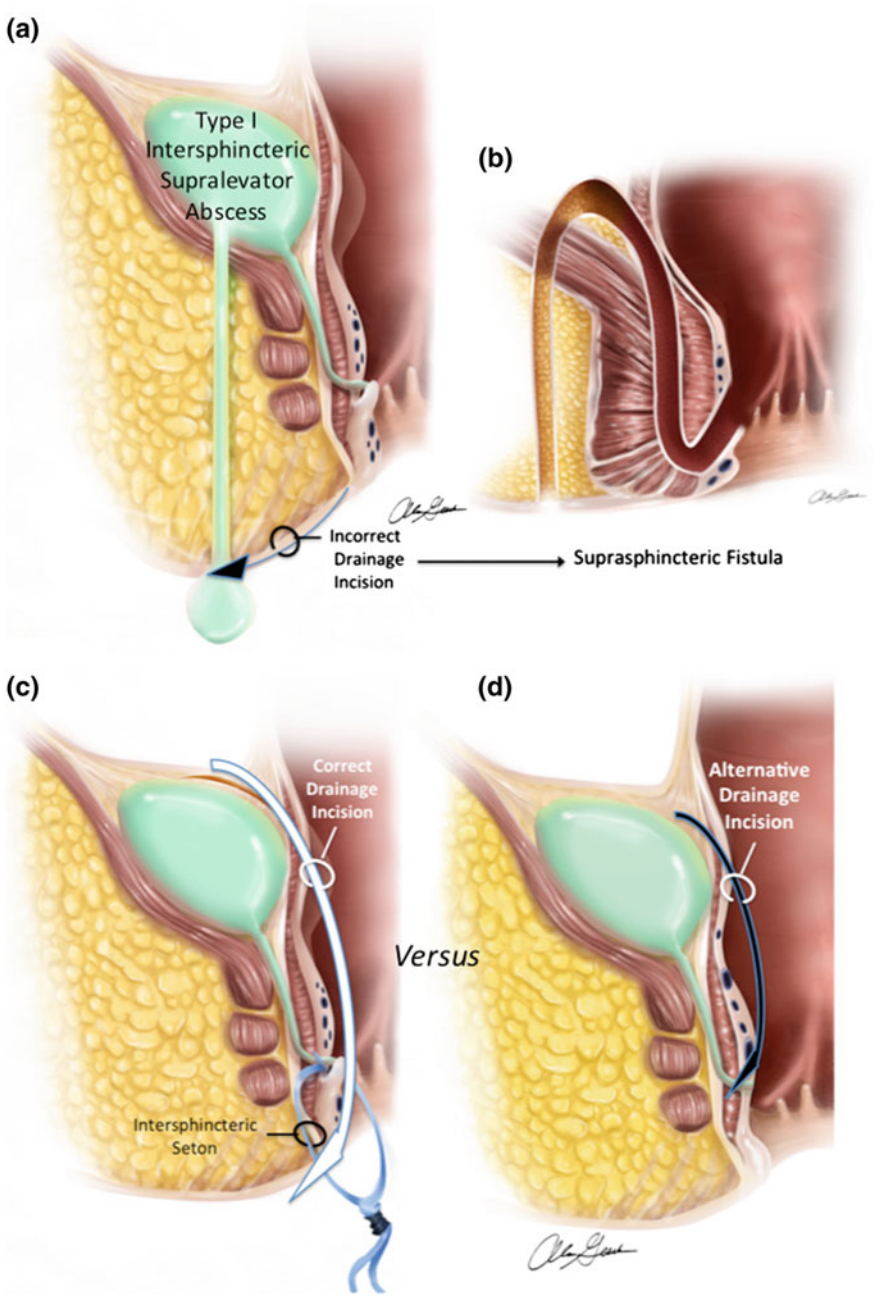


Fig. 1.4 Type I intersphincteric supralevator abscess. **a** Incorrect external drainage through the ischioanal fossa results in **b** a suprasphincteric fistula. **c** Correct internal drainage of the abscess with an intersphincteric seton shown. **d** Alternative internal drainage in continuity with an internal sphincterotomy demonstrated

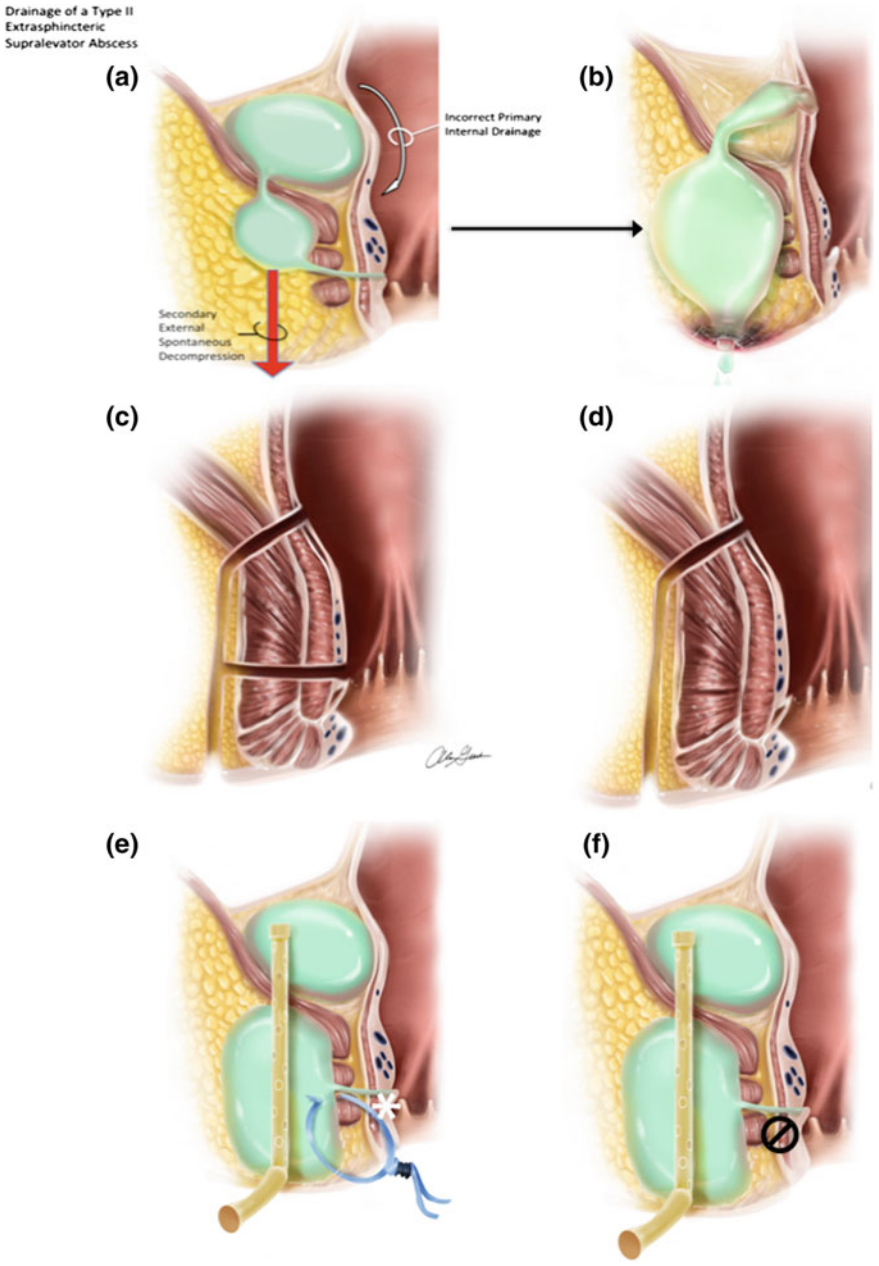


Fig. 1.5 Type II extrasphincteric supralevator abscess from primary transsphincteric ischioanal process. **a** Incorrect drainage internally can lead to **b** spontaneous decompression of the ischioanal fossa. This intervention may produce either an F-type fistula as depicted (**c**) or an extrasphincteric fistula (**d**). If the primary fistulous component is identified, a transsphincteric seton and drainage catheter are employed. **e** If not identified, only a drainage catheter is placed (**f**)

Posterior type III extrasphincteric abscesses are managed similarly to a deep postanal space infection with vertical sectioning of the anococcygeal ligament. A supralevator catheter may be placed in the setting of a large retrorectal abscess. A transsphincteric seton through the primary fistulous trajectory is also an option in this scenario (Fig. 1.6).

A type IV supralevator abscess has the deep postanal space as its primary focus of infection. There is a secondary extension of the deep postanal space infection to the ischioanal fossa. Both the deep postanal space and ischioanal infections contribute to the supralevator abscess simultaneously. Surgical treatment is directed at the primary site of infection in the deep postanal space with counter drainage of the involved ischioanal fossa (Fig. 1.7).

Abscesses presenting as a distinct fullness on the posterior wall of the rectum also merit special consideration. Both posterior type I and type III abscesses should be considered. The key to this differential diagnosis is inclusion or exclusion of the deep postanal space as the primary infection (Fig. 1.8).

Preoperative imaging optimizes effective management of posterior supralevator abscesses. Failure to include or exclude the deep postanal space as the primary nidus of infection preoperatively compromises the surgical intervention. Albeit less than ideal, this situation may be salvaged by the selective percutaneous aspiration of the deep postanal space. The authors recommend a limited posterior midline cut down to identify the anococcygeal ligament. A large bore needle is introduced in the axis of the anal canal. It is important not to aspirate in the direction of the coccyx. The latter scenario only confirms that a posterior retrorectal supralevator abscess is in evidence. It does not identify involvement of the deep postanal space (Fig. 1.9). A positive aspiration of purulence from the deep postanal space implies a type III posterior extrasphincteric abscess requiring posterior exploration and drainage. A negative aspiration is evidence of a type I posterior intersphincteric abscess that requires internal (intrarectal) drainage.

Horseshoe presentations of anorectal abscesses are an important and pleomorphic group of infections. Circumanal inflammatory changes including cellulitis, peau d' orange, and fluctuance are characteristics. An anterior midline primary gland infection is far less common than posterior processes. Anterior infection may appear as horseshoes but tend to extend into the scrotal or labial tissues.

Posterior infections are far more common [14]. Treatment failures are as common as 50% [2]. Surgeons need to consider that any of the four posterior spaces may be responsible for a horseshoe presentation: (1) superficial postanal, (2) deep postanal, (3) supralevator, and (4) retrorectal. The authors use the term horseshoe presentation rather than abscess because often the changes observed inflammatory, i.e., cellulitis rather that frank fluctuance. These changes often reflect a deeper seated infection. The most important feature of treatment of horseshoe presentations

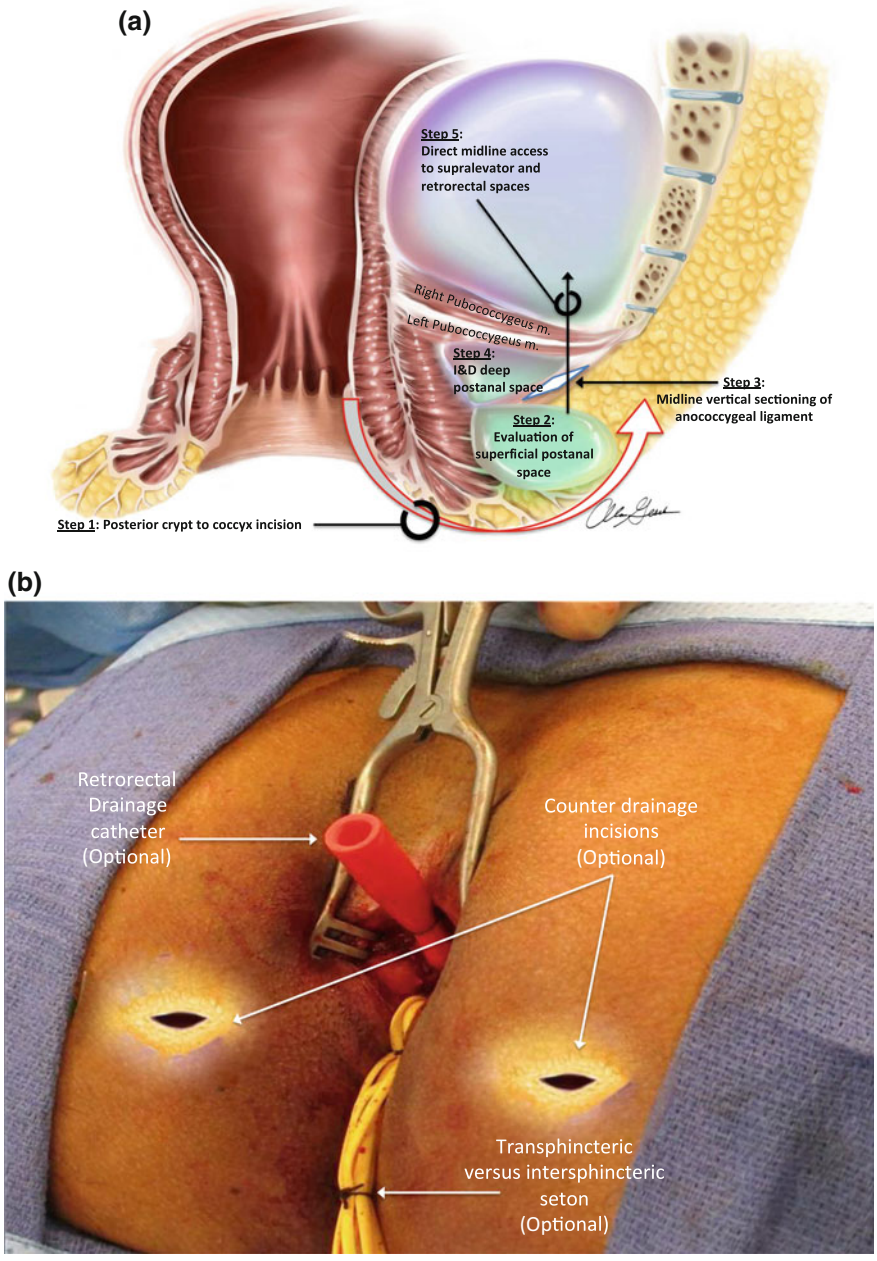


Fig. 1.6 Type III extrasphincteric supralevator abscess from a posterior midline process. **a** A posterior midline incision is used to explore the postanal spaces. The superficial postanal space is evaluated and then the anococcygeal ligament sectioned to enter the deep postanal space. Passage through the pubococcygeus muscles allows evacuation of the supralevator component. **b** A primary seton is placed through the internal opening if identified. Counter incisions can be made over the ischioanal fossa bilaterally if needed. A retrorectal catheter can be inserted to decompress the supralevator abscess

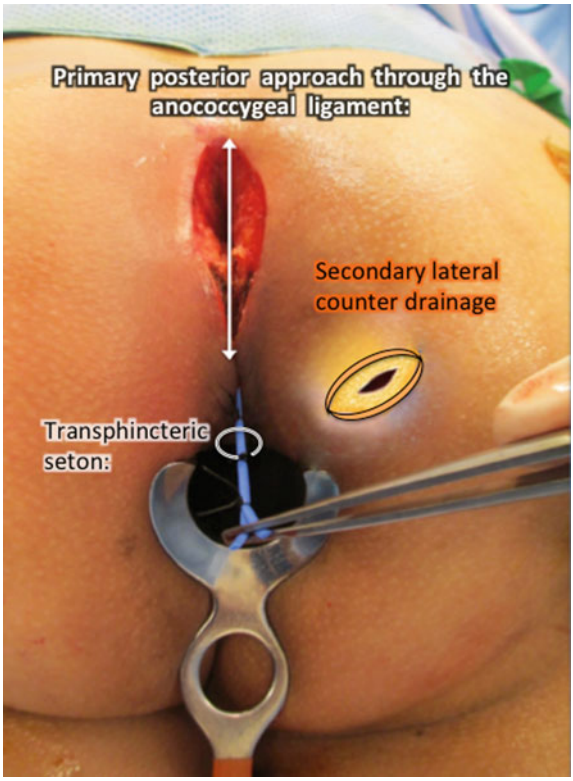


Fig. 1.7 Type IV extrasphincteric supralevator abscess from a primary posterior midline process with ischioanal component. A posterior exploration (crypt to coccyx) is required to evaluate the postanal and the supralevator spaces. The posterior process can be treated with catheter drainage with or without a seton. A counter incision is made to drain the ischioanal component externally. Catheter drainage may be implemented to address the supralevator component in this infection

is treatment of the primary focus of infection a priori. Moreover, not all inflammatory changes over the ischioanal fossae require counter drainage. The most common mistake encountered in the treatment of horseshoe abscesses is the bilateral treatment of the ischioanal fossae without addressing the primary midline infection. For all these reasons, surgeons should consider preoperative imaging of ischioanal infection as well as definitive treatment of the primary fistulous component (Fig. 1.10).

The Role of Adjunctive Imaging

Imaging plays an important role in most areas of surgery. Its utility in the evaluation and treatment of acute anorectal infections has largely been unrecognized. However, four groups of patients have a clear benefit: (1) tetralogy of occult anorectal sepsis, (2) differentiation of primary versus secondary ischioanal infections, (3) supralevator abscesses, and (4) horseshoe presentations (Fig. 1.11). The tetralogy of occult anorectal sepsis consists on the constellation of pain, sepsis, paucity of physical finding, and/or a physical examination limited by hyperesthesia. Identification of the primary site of an ischioanal abscess is relevant to its surgical management. Identification of the deep postanal space as the primary focus of infection can be a game

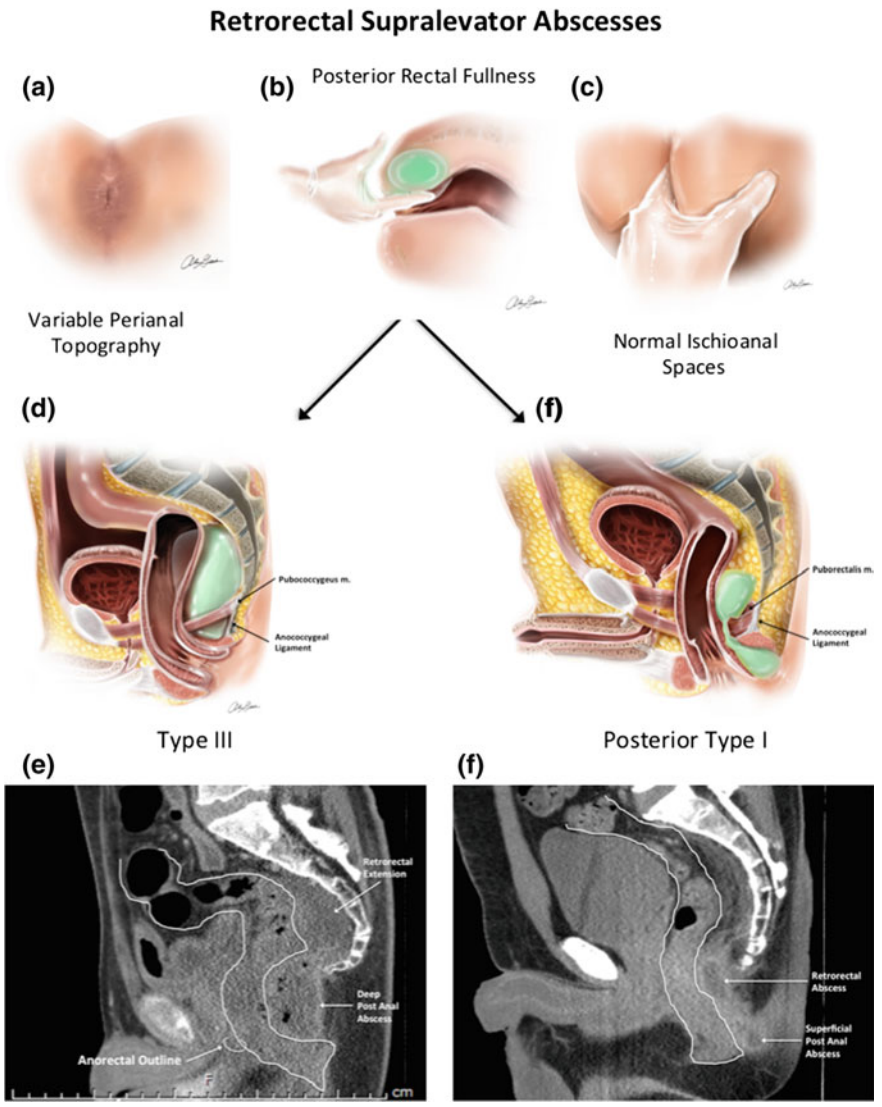


Fig. 1.8 Retrorectal supralevator (Type I or III). **a** These abscesses may have normal topography. **b** Fullness is encountered within the rectum posteriorly. **c** The ischioanal fossae are normal. This clinical scenario may represent either a type III configuration (**d**) or type I configuration (**f**). Computed tomography can assist in delineating the difference between type III (**e**) or type I (**f**) to guide drainage

changer. Similarly, horseshoe presentations are benefited by identification of the primary infection as well as differentiation between secondary abscesses versus fat stranding (cellulitis) and phlegmon (lymphedema/lymphatic obstruction). The former require surgical drainage a priori while the latter observations may not. These

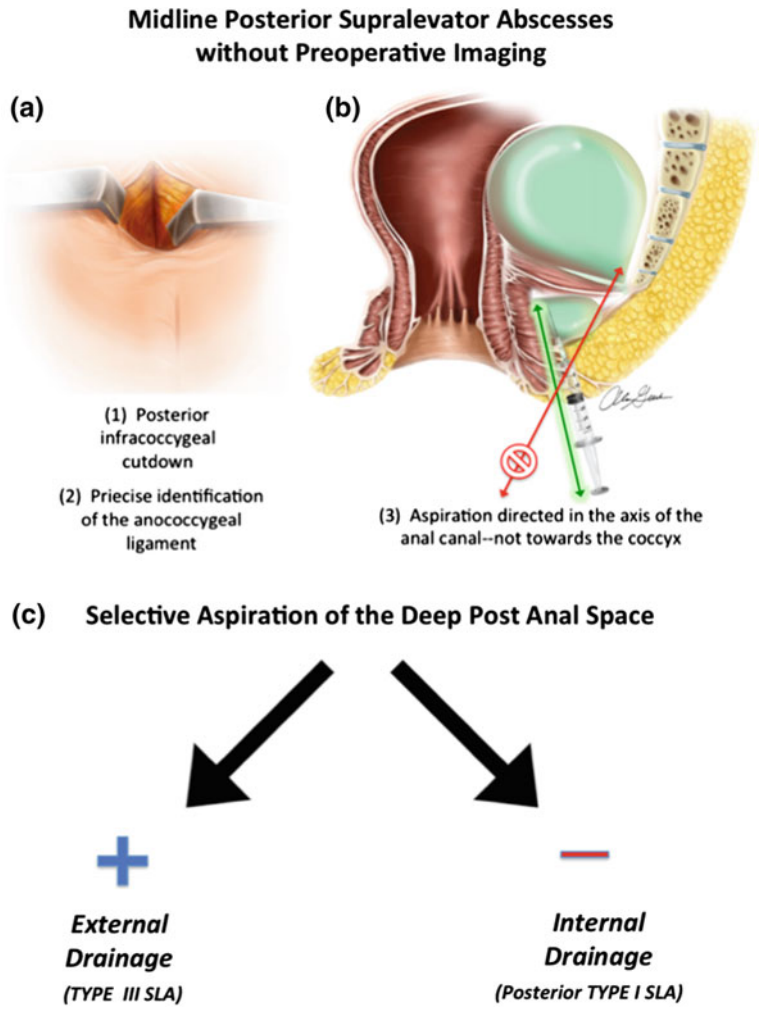


Fig. 1.9 Evaluation of retrorectal abscesses without preoperative imaging. **a** A posterior incision is made with identification of the anococcygeal ligament. **b** A needle is inserted through the ligament into the deep postanal space. Care should be taken to advance in a trajectory parallel to the anal canal. **c** Purulence from the deep post anal space confirms a type III abscess and further external exploration and drainage should be undertaken. A negative aspiration implies a type I intersphincteric abscess and intrarectal drainage should be performed

are difficult distinctions in the purely clinical context. Finally, supralevator abscesses require the evaluation of three spaces: (1) supralevator, (2) ischioanal, and (3) deep postanal. Misdiagnosis of a primary deep postanal abscess associated with a supralevator component is the most important cause of treatment failures in this setting.

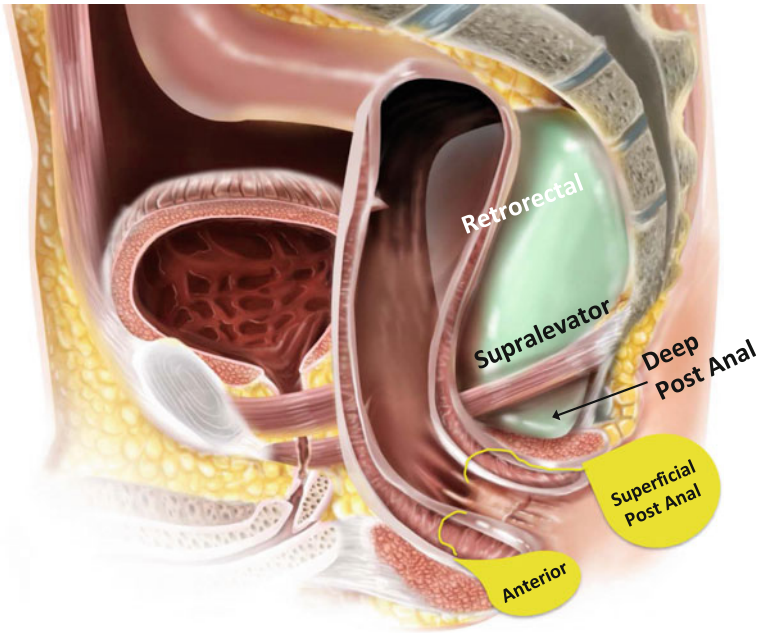


Fig. 1.10 Horseshoe presentations may occur in multiple potential spaces including anterior, superficial postanal, deep postanal, supralevator, and retrorectal. Preoperative imaging can assist in identifying the spaces involved

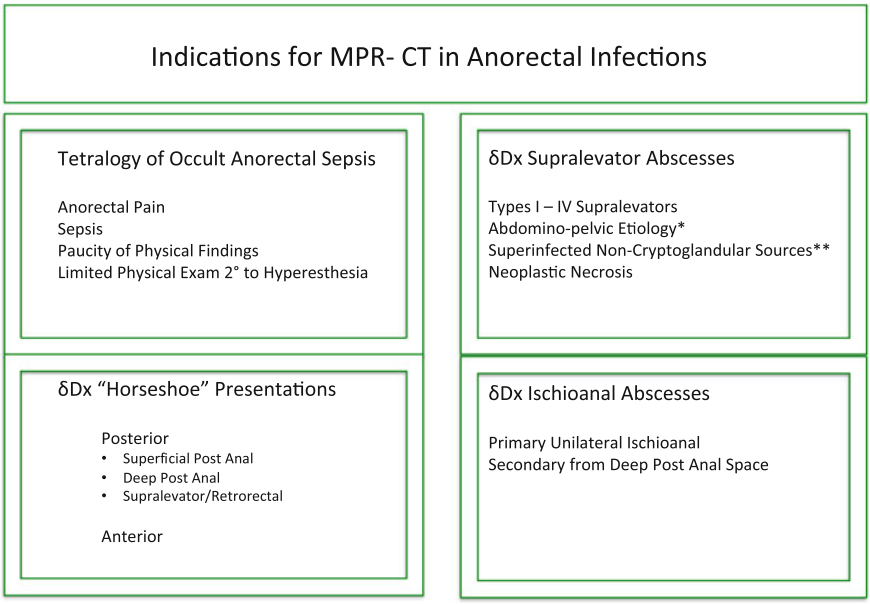


Fig. 1.11 Indications for multi-plane reconstruction imaging with computed tomography in anorectal infections

Each of the predominant imaging modalities has its proponents. Ultrasound is interesting because of its cost and portability. The discomfort associated with a transanal transducer is limiting. Magnetic resonance imaging is useful in the evaluation of deep-seated and multiple space infections. It is relatively limited by its availability. Multi-plane reconstruction computer tomography (MPR CT) is emerging as an important modality for potentially complex anorectal infections. It reliably identifies deep postanal space abscesses. Moreover, it is useful in discriminating between abscess and pro-inflammatory changes observed on physical examination (cellulitis and lymphatic alterations).

Preoperative imaging should be considered indispensible in the setting of suspected abdominopelvic processes and supralevator presentations. The authors do not support imaging all patients. However, it is clear that a systematic approach to the history and physical examination helps identify patients harboring complex, multi-space infections who may benefit from adjunctive imaging.

Conclusions

Anorectal infections are a common problem treated by a variety of medical practitioners since antiquities. Historically based dogma is still pervasive. Optimal outcomes rely on a thorough systematic history and physical examination. Adjunctive imaging has an increasingly important role in selected clinical settings. The natural history of acute anorectal infections inevitably forming chronic anal fistula requires scrutiny. Fortunately, complex fistulas are avoidable when complex abscesses are appropriately evaluated and treated surgically.

Fournier's Gangrene Complications, Prevention, and Treatment

Etiology

Ariane M. Abcarian and Herand Abcarian

Fournier's gangrene is a necrotizing soft tissue infection involving the perianal and ischiorectal areas, perineum, genitalia, and groin with occasional spread to the lower abdominal wall, thighs, and lower back. The disease was originally described by Jean Alfred Fournier in 1883 and was considered uniformly fatal [15]. In the recent years, two alternative terms have been employed:

"Synergistic gangrene" refers to multiple species of bacteria involved in the pathology of the disease and "necrotizing fasciitis" refers to common involvement of various muscular fascias in the necrotizing process. Fournier's gangrene must be differentiated from myocutaneous necrosis caused by Clostridium species, which requires different treatment.

Anorectal and urinary tract infections are very common, however, it is unclear why in some patients a seemingly minor septic process progresses to a life-threatening necrotizing infection. It is thought that obliteration of distal arterial disease caused by diabetes mellitus or an immunocompromized host (e.g., AIDS, chemotherapy, or immunosuppressive therapy) allow the rapid growth and spread of multiple bacterial species leading to tissue necrosis hence the term "synergistic gangrene." Understanding the contributory or the underlying disease process is critical in the choice of appropriate treatment to prevent a fatal outcome.

The point of entry of the invasive bacteria may be in the lower gastrointestinal tract or in the urogenital system. Anorectal infections of cryptoglandular origin have been implicated and a common finding of a fistula in Fournier's gangrene lends credence to this theory [16]. Pelvic sepsis secondary to rubber band ligation of hemorrhoids are forms of the same pathologic process [17, 18]. Necrotizing sepsis following conventional hemorrhoidectomy is rare unless the patient is severely immunocompromized [19].

Urogenital tract is the other important point of entry. Instrumentation such as urethral catheterization, dilation, cystoscopy, and at times a simple urinary tract infection may be the initiating factor [20, 21]. Immune compromise whether due to acquired immunodeficiency disease (AIDS), chemotherapy or immunosuppression from transplantation may be a contributing factor in the rapid spread of the septic process [18, 22, 23].

A very important extensive case series was reported by Stephens et al. [24]. They compared the difference in etiology and clinical presentation of 449 cases of Fournier's gangrene in historical (1964–1978) era versus the "current" (1979–1988) period. The average age was 50 years and gender strongly favored men (M/F ratio of 86% vs. 14%). The most common etiologic factor was colorectal (33%) and genitourinary (21%). In 26% no source could be identified and were labeled idiopathic. The morbidity was 22% with significant unfavorable results seen in colorectal disease (mortality 33%) [24].

Symptoms and Signs

The presenting symptoms of Fournier's gangrene are pain, swelling, and discharge of pus. Appearance of a "black spot" indicates the presence of gangrene and impending spread along anatomic (Fig. 1.12) planes [25].

Cellulitis and fluctuation may be palpable to a variable distance from the main swelling (Fig. 1.13).

Additional findings include fever, elevated WBC with left shift, and severe hyperglycemia signifying uncontrolled diabetes mellitus.

Culture of pus or biopsy of margins of infected tissue usually yields mixed flora including streptococcus, staphylococcus aureus, bacteroides, klebsiella, E. coli, proteus, enterococcus, peptostreptococcus, and citrobacter with the highest concentrations seen in diabetics [20]. In addition to the usual aerobes and anaerobes, Clostridium species may also be found in bacteriologic specimens [25].

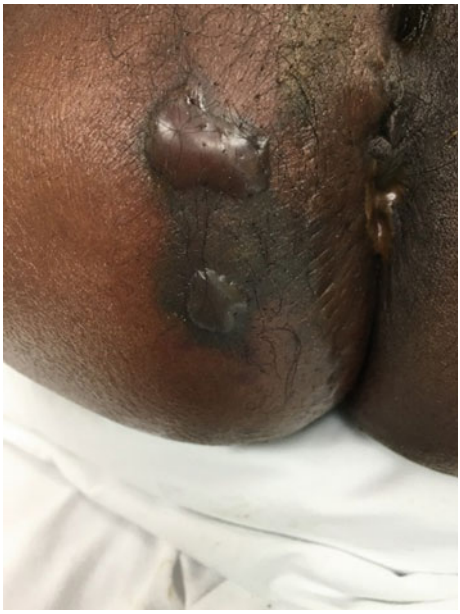


Fig. 1.12 Typical Fournier's gangrene with central "black spot" and bullae

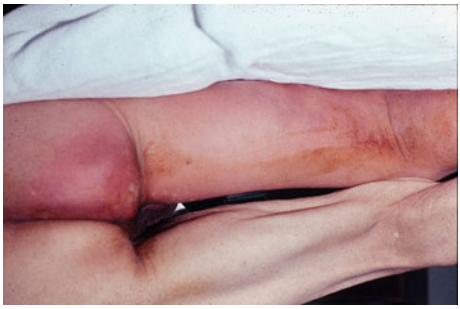


Fig. 1.13 Extension of Fournier's gangrene along tissue planes to the buttock and thigh

Principals or Treatment, Prevention of Complications:

1. Frequent Surgical debridement
2. Broad spectrum antibiotics
3. Control of associated disease
4. Fecal/Urinary Diversion
5. Supportive care (Nutrition)
6. Hyperbaric Oxygen
7. Delayed wound care/reconstructive procedures.

1. Surgical debridement of all necrotic tissue down to normal bleeding tissue is essential in the treatment of Fournier's gangrene. Extensive debridement should be undertaken without consideration to form or function or subsequent reconstruction. Attempts to salvage questionably viable tissue is to be condemned. The

surgical wound must be inspected on a daily basis and if there is any evidence of purulence, the patient must return to the operating room for evaluation under anesthesia and further debridement. The same must be said for presence of fever or leukocytosis [15, 25–27]. The vulvar skin and the scrotum must be excised if needed. The tough fascial layer of Dartos prevents testicular involvement and orchiectomy is never indicated. If the scrotal skin has been excised the testes can be implanted in a thigh pouch for subsequent reconstruction (Fig. 1.14).
Pulsed saline irrigation assists with tissue debridement. The surgical wounds are left open for ease of inspection and return to the operating room if needed. Packing of the surgical wound in the operating room using dilute Dakins or peroxide solution is a good technique to assist in debridement of residual necrotic tissue.

2. Broad spectrum antibiotics are essential in the treatment of this polymicrobial disease. Treatment with antibiotic agents against aerobes, anaerobes, and clostridial species should be started immediately until definitive culture and sensitivity results become available. In the operating room a biopsy from the margins of the infected tissue is valuable in the choice or change in antibiotic therapy and to assure the adequacy of the extent of the surgical debridement. Despite the current concerns regarding C. difficile super infection, antibiotics should be continued until the patient's fever and leucocytosis abate and the wound is clean enough as not requiring return to the operating room [28, 29].

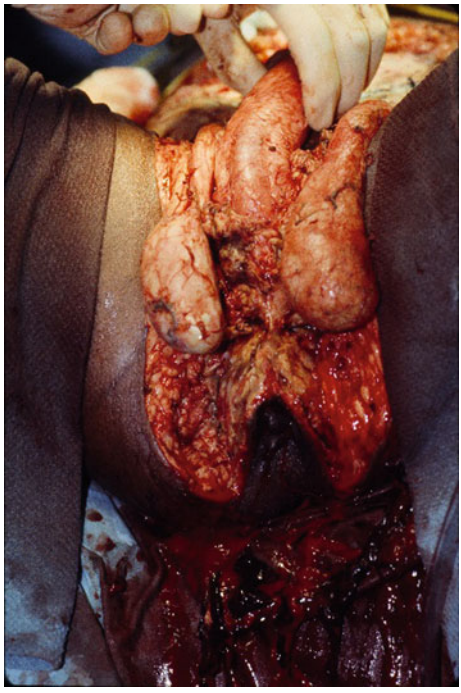


Fig. 1.14 Extensive debridement of perineum, ischiorectal fossa, scrotum, and penis. Note preservation of testes

3. Control of Associated Diseases
   Diabetes mellitus remains the main associated illness seen in 50–70% of case series of Fournier's gangrene. The diabetic patients are admitted in a state of extreme hyperglycemia and worsening diabetic control. Rarely Fournier's gangrene may be the initial disease leading to a diagnosis of diabetes mellitus. In any case control of blood sugar, even resorting to insulin drip is mandatory.
   In cases of AIDS, the treatment of Fournier's gangrene must be associated with an attempt to treat AIDS with HAART as the best method of affecting the course of HIV/AIDS. When immune compromise is a result of immunosuppression for transplantation, the transplant physician should be consulted to alter or reduce the dose of the immunosuppressive regimen in conjunction with the surgical treatment of Fournier's gangrene and broad spectrum antibiotic administration. If cancer chemotherapy is the source of immune comprise, the medical oncologist should be consulted to alter or reduce the dose of chemotherapeutic agents and in cases of severe neutropenia to administer appropriate agents to raise the leukocyte count and if necessary platelet transfusion to raise the platelet counts [30].
4. Fecal/Urinary Diversions
   In general, patients with Fournier's gangrene do not require fecal diversion. The anal sphincter mechanism is usually spared despite destruction of the surrounding soft tissue and often remains as a viable island of tissue floating into the midst of necrotic tissue [31] (Fig). It is important, therefore, that the anal sphincter is not indiscriminately divided [31]. If an anal fistula is found, a seton should be placed and definitive treatment postponed to a later date. In a personal series of 83 cases of Fournier's gangrene only two patients had diverting colostomy and both were transferred from an outside hospital after the colostomy procedure. The important fact is that colostomy is not a substitute for meticulous debridement of necrotic tissue, often in multiple stages.
   Urinary diversion is employed by urologists when there is a definite urethral injury precluding insertion of a Foley catheter. Clayton et al. reported a series of 57 men with male genital necrotizing fasciitis [21] (Fig. 1.15).

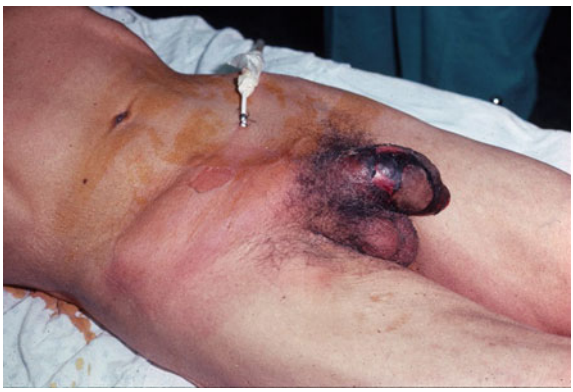


Fig. 1.15 Urinary diversion (suprapubic catheter cystotomy) in Fournier's gangrene originating from urethral injury

Forty-seven patients (87%) survived. Better prognosis was associated with younger age, BUN < 50 mg/d L on admission and fewer major complications after initial debridement. In this series survival of patients with localized process was not better than those in whom the necrosis extended to the abdominal wall or thighs [21].

5. Supportive therapy
   Almost all patients with Fournier's gangrene suffer from debilitating conditions. Therefore it is unreasonable to expect that with the addition of surgical stress and worsening of the patient's catabolic state, surgical wounds will granulate and heal by secondary intention. Therefore, it is imperative that the patient's nutritional status be addressed immediately following initial debridement. Obviously the oral nutrition route is preferable but if the patient cannot manage to consume enough calories, nasogastric administration of nutritional products should be considered. If this fails, TPN through a PICC line should be initiated to supply sufficient caloric intake. The aid of a registered dietitian and a nutritional support team is invaluable.
6. Hyperbaric Oxygen Therapy
   Some centers advocated hyperbaric oxygen therapy (HbO) not only to combat the predominate anaerobic bacteria but also to aid in healing of large granulating wounds by providing greater oxygen tension in perfused body tissues [19]. Recommendations for the use of HbO are based on experimental and human studies that show inhabitation of endotoxin production by Clostridia species [32]. However HbO therapy is not available in most hospitals and transporting a critically ill patient back and forth, which may very well take the better part of a day, is unadvisable. Also a recent retrospective comparison of outcomes of necrotizing soft tissue infection in two hospitals failed to demonstrate survival advantage and there were actually a greater number of operative debridement done in the patients receiving HbO therapy [33].
7. Delay wound care and reconstructive procedures
   Traditionally, the large wounds after extensive debridement of Fournier's gangrene are left open, inspected regularly and packed with wet dressings soaked either in saline or an antimicrobial agent (e.g., peroxide, Dakins) (Fig. 1.16).
   Recently the advent of vacuum-assisted wound closure has been reported in the management of these large wounds. Of course the wound VAC can only be applied after elimination of all necrotic tissues and on healthy granulating wound if a complete seal for vacuum can be accomplished [34]. The plastic and reconstructive surgical team can provide coverage when the wound base is clean enough to allow surgical attempts (Fig. 1.17).

Outcomes

The classic literature reported uniformly fatal outcomes [15]. Multidisciplinary team management of Fournier's gangrene has resulted in much improved survival. Stephens and colleagues reported high mortality associated with colorectal cases (33%) while the overall morbidity for the 11 cases was 22% [24]. Recently a

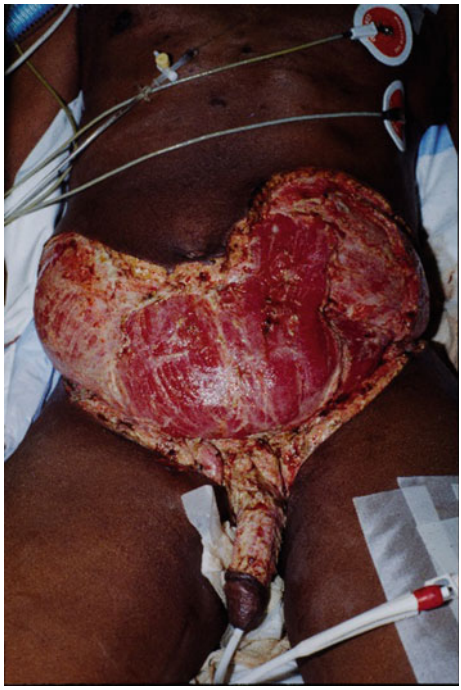


Fig. 1.16 Clean post debridement Fournier's gangrene

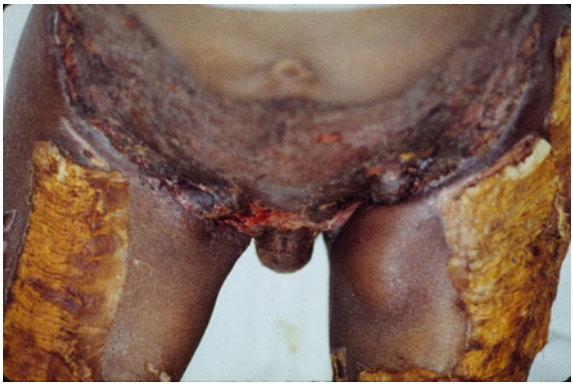


Fig. 1.17 Patient in Fig. 1.16 after extensive coverage with split thickness skin grafts

Fournier's gangrene severity index score (FGSI) has been proposed as an aid to predict outcome. A score greater than 9 predicted a 75% mortality while a score of 9 or less was associated with 78% probability of survival [35, 36]. In the past decade, there were further publications in the Urological Surgery journals referring to FGSI score and its value in predicting treatment outcomes [37, 38]. Other authors have published on LRINE (Laboratory Risk Indicators for Necrotizing fasciitis) as a tool to distinguish between, necrotizing fasciitis and other soft tissue infections [39]. The same methodology has been used to predict outcome [40]. In these

multiple scoring systems, points are assigned to level of C reactive protein, white blood cell count, hemoglobin, sodium, creatinine, and glucose. While the positive and negative predictive values were initially found to be of prognostic value, subsequent analysis have failed to confirm the initial results and therefore they are no longer used extensively [41].

Light and colleagues have reported a large consecutive single intuition series of long-term outcomes of patients with necrotizing soft tissue infection compared with the population-based mortality rates [42]. Among the patients who survived at least 30 days after initial admission to the hospital, 25% died over a mean follow-up of 3.3 years and the median survival was 10.0 years. The most common causes of mortality were cardiopulmonary disease, diabetes mellitus, malignancies, and infectious causes. Deaths related to infectious causes were considerably higher in survivors of necrotizing soft tissue infection compared with the population at large, suggesting that these patients may have an inherent defect in host defenses [42].

Personal Experience and View

In my view the most important factor for a potentially favorable outcome is early diagnosis. This takes an experienced surgeon's physical examination. Hours wasted on unnecessary CT scans, admission to the wrong (medical) service, delay in obtaining the initial surgical consultation may seal the fate of the patient.

Once the patient is seen, vigorous resuscitation should begin on the way to the operating room. Extensive debridement of all visibly nonviable tissue and pulse irrigation should be carried out without concern to form or function. The sphincter mechanism and the testes should be preserved. The wounds are left open and packed with saline-soaked gauze and inspected regularly. The patient must return to the operating room if there is any hint of further pus or tissue necrosis or if he/she has elevated temperature or white blood cell count. Often three to four trips to the operating room is needed to get "ahead of the infection." Broad spectrum antibiotics against aerobes, anaerobes, and clostridial species should be started immediately and can be adjusted or changed when the results of culture of pus or tissue biopsy becomes available usually in 48 h. HbO therapy does not offer any advantage and the desperately ill patient should not be transported to another hospital where HbO is available. Urinary diversion is usually decided by the urologist caring for the patient. Fecal diversion is rarely indicated with the exception of patients who are so sick and debilitated that they cannot be moved to sitz baths or burn care tubs on daily basis. Associated diseases, usually diabetes mellitus must be controlled and if there is severe decrease in leukocytes and platelets due to chemotherapy or immunosuppression, medical oncology or transplant team should be involved in adjusting the chemotherapeutic or immunosuppressive agents. Finally, the state of nutrition of the patient must be addressed as soon as the first debridement is done. Oral or nasogastric routes are preferable, but if not adequate, central line (PICC) should be inserted and total parenteral nutrition started to optimize the patient's nutritional status. Registered dietitians and nutritional support teams are of great assistance in this matter.

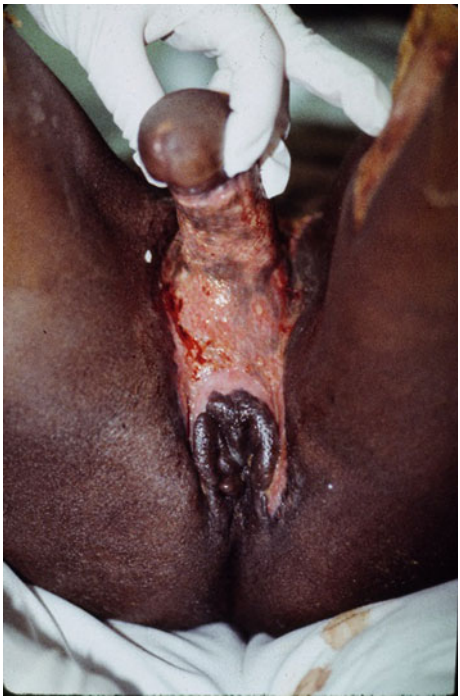


Fig. 1.18 Excellent healing by secondary intension (patient in Fig. 1.14). Note the testes are protected in upper inner thigh pockets. The wound can be skin grafted at this stage. It is important to always be reminded that in this particular disease process, failure of optimal surgery will result in the ultimate complication, i.e., death of the patient

The multidisciplinary approach with colorectal surgeons, urologists, internist, infectious disease specialists, and nutritionists have resulted in favorable outcomes in many patients in recent years, allowing the plastic and reconstructive team to get involved in the ultimate care of the patients with extensive Fournier's gangrene (Fig. 1.18).

References

1. Hippocrates d' Fistulis (4th–3rd Century B.C.) In: Potter P (translator) Hippocrates Volume VIII (1995) Harvard University Press, Cambridge, p. 387.
2. Onaca N, Hirshberg A, Adar R. Early reoperation for perirectal abscess: A preventable complication. Dis Colon Rectum. 2001;44(10):1469–72.
3. Bode WE, Ramos R, Page CP (1982) Invasive necrotizing infection secondary to anorectal abscess. Dis Colon Rectum 25: 416–419.
4. Bizos DB. Evaluation of a policy of primary fistulotomy for acute anorectal abscesses. Johannesburg, South Africa: University of Witwatersrand; 1997. p. 65.
5. Tang CL, Chew SP, Seow-Choen F. Prospective randomized trial of drainage alone vs. drainage and fistulotomy for acute perianal abscess with proven internal opening. Dis Colon Rectum. 1996;39:1415–7.
6. Kyle S, Isbister WH. Management of anorectal abscesses: comparison between traditional incision and packing and de pezzer catheter drainage. 1990;60:129–31.

7. Read DR, Abcarian H. A prospective survey 474 patients with anorectal abscess. Dis Colon Rectum. 1979;22(8):566–8.
8. Ayres M, Abscess of the rectum. In: Some of the diseases of the rectum and their homeopathic surgical treatment. Chicago: Duncan Brothers; 1884. p. 62.
9. Rickard MJFX. Anal abscesses and fistulas. ANZ J Surg 2005;75a:64–72.
10. Malik AI, Nelson RL, Tou S (2010) Incision and drainage of perianal abscess with or without treatment of anal fistula. Cochrane database Syst Rev (7):CD006827.
11. Knoefel WT, Hosch SB, Hoyer B, Izbicki JR. The initial approach to anorectal abscesses: fistulotomy is safe and reduces the chance of recurrence. Dig Surg. 2000;17:274–8.
12. Ortega AE, Bubbers E, Liu W, Cologne KG, Ault GT. A novel classification, evaluation, and treatment strategy for supralevator abscesses. Dis Colon Rectum. 2015;58:1109–10.
13. Prasad ML, Read DR, Abcarian H. Supralevator abscess: diagnosis and treatment. Dis Colon Rectum. 1981;24:456–61.
14. Held D, Khubchandani I, Sheets J, Stasik J, Rosen L, Riether R. Management of anorectal horseshoe abscess and fistula. Dis Colon Rectum. 1986;29:793–7.
15. Fournier JA. Gangrène foudroyante de la verge (overwhelming gangrene). Med Pract. 1883;4:589–97.
16. Difalco G, Gucciona C, D'Annibale A, et al. Fournier's gangrene following a perianal abscess. Dis Colon Rectum. 1986;29(9):582–5.
17. Clay LD III, White JJ, Davidson JJ, et al. Early recognition and successful management of pelvic cellulitis following hemorrhoidal banding. Dis Colon Rectum. 1986;29(9):367–70.
18. Quevedo-Bonilla G, Farkas AM, Abcarian H, Hambrick E, Orsay CP. Septic Complications of hemorrhoidal Banding. Arch Surg. 1988;123:650–1.
19. Cihan A, Mentes BB, Sucak G, et al. Fournier's gangrene after hemorroidectomy: Association with drug-induced agranulocytosis. Report of a Case. Dis Colon Rectum. 1999;42(12):1644–8.
20. Jones RB, Hischmann JV, Brown GS, et al. Fournier's syndrome: Necrotizing subcutaneous infection of the male genetalia. J Urol. 1979;112(3):279–82.
21. Clayton MD, Fowler JE Jr, Sharifi R, et al. Cases, presentation and survival of 57 patients with necrotizing fasciitis of the male gentalia. Surg Gynec Obstet. 1990;170(1):49–55.
22. Rajbhandam SM, Wilson RM. Classical infections in Diabetes. Diabetes Res Clin Pract. 1988;39(2):123–8.
23. Barkel DC, Villaba MR: A reappraisal of surgical management of necrotizing perianal infection. Am Surg 1986;5(7)395–397.
24. Elk N. Fournier's gangrene: A review of 1726 cases. Lancet. 2000;87(6):718–28.
25. Hejas MR, Simonin JE, Bihrle R, Coogan CL. Genital Fournier's gangrene: Experience with 38 patients. Urology. 1996;47(5):734–9.
26. Lamerton AJ. Fournier's gangrene: Non-clostridial gas gangrene of the perineum and diabetes mellitus. J Soc Med. 1986;79(4):212–5.
27. Stephens BR, Lathrop JC, Rice WT, et al. Fournier's gangrene: Historic (1764-1978) versus current (1979-1988) differences in etiology and clinical importance. Am Surg. 1993;59 (3):149–54.
28. Khan SE, Smith NL, Gonder M et al: Gangrene of male external genitalia a patient with colorectal disease. Dis Colon Rec 1985;l28(7)519–522.
29. Enriquez S, Moreno M, Devesa V. Fournier's syndrome of urogenital and Anorectal origin a retrospective comparative study. Dis Colon Rectum. 1987;30(1):33–7.
30. Koralcik DJ, Jones J. Necrotizing perianal infection. Am Surg. 1983;49(3):163–6.
31. Diettrich N. Mason: Fournier's gangrene: A general surgery problem. World J Surg. 1983;7 (2):288–94.
32. Abcarian H, Eftaiha M. Floating free-standing anus: A complication of massive anorectal infection. Dis Colon Rectum. 1983;25:516.
33. Jallali N, Withey S, Butler PE. Hyperbaric oxygen as adjuvant therapy in the management of necrotizing fasciitis. Am J Surg. 2005;189:462–6.

34. Kay D: Effect of hyperbaric oxygen on clostridia in vitas and in vivo. Proc Soc Exp Biolog Med 19687;124:360–366.
35. Huang WS, Hsieh SC, Hsieh CS, et al. Case of vacuum-assisted wound closure to manage limb wounds in patients suffering from acute necrotizing fasciitis. Asian J Surg. 2006;29 (3):135–9.
36. Yenijoy CO, Seulozgen T, Arslan M, et al. Fournier's gangrene: Experience with 25 patients and use of Fournier's gangrene severity index score. Urol. 2004;64(2):218–22.
37. Unalp HR, Kamer E, Derici H, et al. Fournier's gangrene: Evaluation of 68 patients and analysis of prognostic variables. J Postgrad Med. 2008;54(2):102–5.
38. Corcoran AT, Smaldone MC, Gibbons EP, et al. Validation of the Fournier's gangrene severity index in a large contemporary series. J Urol. 2008;180(3):944–8.
39. Kabay S, Yucel M, Yaylak F, Algin MC, et al. The clinical features of Fournier's gangrene and the predictivity of the Fournier's gangrene severity index on the outcomes. Int Urol Nephrol. 2008;40(4):997–1004.
40. Wong CH, Khin LW, Heng KS, Tan KC, et al. The LRINEC (Laboratory risk indicators for necrotizing fasciitis) score: A Tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 2004;32(7):1535–41.
41. Su YC, Chen HW, Hong YC, Chen CT, et al. Laboratory risk indicator for necrotizing fasciitis score and outcomes. ANZ J Surg. 2008;74(11):968–72.
42. Ustin JS, Malangoni MA. Necrotizing soft-tissue infections. Crit Care Med. 2011;30 (9):2156–62.
43. George Me, Rueth NM, Strada DE, et al: Hyperbaric oxygen has not improved outcomes in patients with necrotizing soft tissue infection. Surg Infec 2009;10(1)21–28.
44. Light TD, Choi KC, Thomas TA, et al. Long-term outcomes of patients with necrotizing fasciitis. J Burn Care Res. 2010;31:93–9.
45. Abcarian H: Fournier's Gangrene—Unpublished Data.

Fistulotomy

2

Richard Nelson

Introduction

R. Nelson (✉)
Epidemiology/Biometry Division, School of Public Health, University of Illinois at Chicago, 1603 West Ashland, Room 956, Chicago, IL 60612, USA
e-mail: altohorn@uic.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_2

1398 Trevisa Barth. de P.R. vii. lix. (1495) 274 Fistula, the fester is a postume that. rootyth wythin.]

1563 T. Gale Antidot. ii. 25 This vnguent. doeth also profyte muche in Fistulays.

c1570 Sir H. Gilbert Q. Eliz. Acad. (1869) 5 Towching all kinds of Vlcers, Sores, Phistiloes, wowndes, &c.

1579 Langham Gard. Health (1633) 12. It is good for all wounds, fistilaes, and sores of the mouth.

1671 Salmon Syn. Med. iii. xxii. 423 It cools Feavers and cures Ulcers, Fistulas, Cancers.

1732 Arbuthnot Rules of Diet 360. It happens sometimes to end in a Fistula.

1879 Green Read. Eng. Hist. xviii. 89 H, notwithstanding his fistula and his fever, was able to sit on horseback.

It is surprising that none of these quotes about the early use of the term fistula (Oxford English Dictionary CDROM, Oxford, 1995) come from John of Arderne (1307–1392), whose classic work on the treatment of anal fistula is still in print, and who pre-dates them all. For many colorectal surgeons, the Queen disease of the specialty is anal fistula and the gold standard treatment for it is fistulotomy. Like fissure and hemorrhoids, anal fistula should be one of life's little problems, one apparently of less consequence, rubbing its shoulders against the ankles of giants like colorectal cancer, ulcerative colitis or diverticulitis. So, why?

It is possible that no condition has been written about more by surgeons over the expanse of time and world geography than anorectal abscess and fistula. There is hardly a major author that has not written about it: Sushruta, who probably first described fistulotomy [1], Hippocrates, who used a cutting seton, Celsius, Galen [2], al Razi, describing curettage and cauterization [3], John of Ardern—who wrote a whole book on the topic [4, 5] Boyer, Brodie, Bodenhamer, and frequently to the present day [6].

Many of their treatments are still in common use. This broad concern is a testament both to the prevalent nature of this disorder and the difficulty it presents to the surgeon in basic decision making. The reasons for these difficult decisions are obvious. On the one hand it is necessary to resolve sepsis and symptoms associated with fistula, principally pain, and yet the procedures that one employs must also preserve function in the anal canal, that is, prevent incontinence.

In addition, the history of surgery is very much influenced by anal fistula. Henry the fifth of England, the victor at Agincourt, died of sepsis from an anal fistula at age 34 (or 36) [2]. Louis the XIV, the Sun King was troubled by the pain and odor of his fistula beginning in 1684. His physicians had irrigated the track with various fluids known to cause good health elsewhere with no effect. Some time had been spent on the local residents with fistulas to see what was most efficacious, often sending them to other regions of France for specific waters, again with no effect. Finally a surgeon was consulted. M. Charles Antoine Felix was in fact not a known expert in the field and had not operated before on an anal fistula. So he begged the king to give him some time to learn the procedures and practice, which he did on various residents, again with fistulas, in prisons and poor houses around Paris. Once he felt sure of himself, he returned to Versailles and on November 18, 1686, the

King had his surgery, descriptions of which sound like fistulotomy and somewhat more, including curettage and caustic irrigations.

The king, fortunately for all, was cured and he rewarded M Felix with several benefits, including a house in Bougival, where Louis kept his mistresses, a fee that adjusted for inflation is almost beyond calculation, and the French academy of surgery was founded. Surgeons could at last wear white coats and attend medical school. In addition, on a trip to a convent on the outskirts of Paris in January 1687, the King received a poem from the abbess celebrating his miraculous recovery, which was set to music by the court composer, Jean Baptiste Lulli, and with some modification became the British national anthem [2, 7].

Complications of Fistulotomy

If one were to list the top ten complications of fistulotomy for anal fistula, the first seven would be incontinence. The problem is dealing with the numbers. This is best illustrated by the data from the practice parameters of the American Society of Colon and Rectal Surgeons. It is stated in the introduction that reported incontinence rates of postoperative incontinence vary from 0 to 70% after fistulotomy [8]. What? How can one possibly resolve that in discussing surgery with a patient?

A somewhat narrower, but still very broad range of possibilities had been published for sphincterotomy for anal fissure. In the chapter in this book on surgery for anal fissure, this disparity in numbers was resolved by reference to randomized trials of partial lateral internal sphincterotomy. The published range of incontinence risk declines from 0% to over 40% to a fairly secure level of just less than 5% [FISSURE CHAPTER]. The larger numbers were mostly due to measuring continence much too soon after the surgery, when pain and an open wound results in discharge that is easily mistaken for incontinence. Full recovery of function as with any surgical wound takes time. More importantly, randomized trials do not just, minimize selection bias, in allocating patients to treatment groups, but ethics committees in hospitals and universities require a protocol for the performance of the trial before patients are recruited, unlike retrospective chart reviews. In the protocol there must be awareness of patient welfare including prospective recording of side effects of the intervention. In a retrospective mail or phone survey, only those who had a tough time may respond.

So, there are several sources. There are five randomized trials comparing fistulectomy to fistulotomy. Three of the five reported no anal incontinence in either treatment group [9–11]. One reported minor incontinence in two of 32 patients having fistulotomy (which means in most publications incontinence to flatus only or some wound discharge) and five cases in 44 patients having fistulectomy [12]. The other reported one case of minor incontinence in 26 patients in the fistulotomy group and 3 cases in 21 patients in the fistulectomy group [13].

There are five randomized trials of incision and drainage alone versus a "cutting procedure" meaning either fistulotomy, or cutting seton (essentially a slow fistulotomy—see below) for higher fistulas. In a systematic review and meta-analysis of these trials, of 204 patients having the cutting procedure, 23 developed incontinence to flatus or fecal soiling only (common with fresh rectal wounds), and 7 of 201 having I&D only also developed similar minor incontinence. The odds ratio for incontinence is 2.46, obviously favoring I&D, but with a 95% confidence interval of 0.75–8.06, not statistically significant [14].

There are four trials that have compared fistulotomy alone to fistulotomy with marsupialization of the open wound—a technique to narrow the spread of the wound and perhaps speed healing. This is to be differentiated from primary repair of the divided sphincter muscle in fistulotomy (discussed below). Here incised skin only is anchored to the base of the open wound. Two trials reported no incontinence in either treatment group [9, 15]. One reported improved continence results with marsupialization, with only one of 52 patients developing incontinence post fistulotomy with marsupialization and six of 52 developing incontinence with fistulotomy alone [16]. One study reported worse minor incontinence in 9% of each group [17].

There are five trials comparing surgery to fibrin glue injection into the fistula track. They are quite a mixed bag and only two of them yield data to this discussion, one comparing glue to fistulotomy and one to cutting seton [18, 19]. In those having the cutting procedure 15 of 48 developed incontinence and 17 of 58 in the glue group developed incontinence (How could this be?). The odds ratio for incontinence for both studies was 1.00 with 95% confidence intervals of 0.43–2.34 [20].

There are two randomized trials of fistulotomy compared to a cutting seton infused with Indian spices called an Ayurvedic seton. One case of incontinence to solid feces was reported in the fistulotomy group in one study along with two cases of minor incontinence in 24 patients and one case of minor incontinence in the seton group in 26 patients [21]. In the second study two cases of minor incontinence in 46 seton patients and one case of minor incontinence in the 54 patients of the fistulotomy group were reported [22].

There are just a few more randomized trials with at least one fistulotomy arm. Of those that had incontinence data, one used a radiofrequency scalpel compared to traditional instruments and no incontinence was found in either group [23]. One included in the fistulotomy group immediate repair of the divided muscle and found four new cases of minor incontinence in 28 patients [24].

All in all, this is an unusual group of publications. A surprising number reported no incontinence after fistulotomy, or even fistulectomy. None reported an incidence of new onset incontinence anywhere approaching what has been reported in retrospective observational studies. Only one mentioned a single patient with incontinence to solid stool. There are more publications of case series and nonrandomized trials providing data on incontinence with fistulotomy than can be counted and the numbers they provide are dizzying. None provide more valid data than those discussed above. The retrospective case series are to prone to selection bias. The few prospective reports have too high a rate of attrition [25].

Setons—A Method to Prevent Incontinence with Fistulotomy?

A cutting seton is essentially a slow fistulotomy, a procedure to lay open the fistula gradually without excising it. By cutting the muscle slowly, it is hoped that the scarring as it forms would fix the two sides of the divided external (and internal) sphincter in close proximity before they fall widely apart. Several problems exist when comparing cutting seton to surgical fistulotomy. First there are no direct randomized trials to provide data to support this hope. Second, I have never met any two surgeons who use a seton in exactly the same way. There is the fast seton, dividing the muscle in 2 weeks, and the slow seton extending that period to over a year, and everything in between. Each surgeon is very adherent to a specific seton material with tremendous variation from horse hair to silk to silastic to threads impregnated with Indian spices. How it is tightened is also very individual. There are studies comparing division of the internal sphincter prior to placement of the seton to no sphincter division. One systematic review showed a higher incidence of incontinence associated with such division (25.2 from 5.6% undivided) [26]. The included studies were predominantly nonrandomized trials, whereas one subsequent randomized trial showed no difference in continence between the two groups [27]. Looking at the huge number of published case series on this topic there does not seem to be much difference in incontinence risk between fistulotomy, cutting seton, or even staged fistulotomy with an intervening seton [28].

One intriguing procedure that has yet to be adequately evaluated is the loose seton. This is a loose seton that is left in until it essentially falls out, so very like the slow cutting seton [29, 30]. Again there are no randomized trials and each publication presents a very different technique.

High Versus Low Fistulotomy

This is an area of publication wherein the border between high and low is hard to define. How can one develop a scoring method for high and low fistula within ones own practice that is precise, repeatable and valid, much less across practices? It is intuitive that cutting a greater length of sphincters would increase the risk of postoperative incontinence and that has generally been found to be true [31]. How is incontinence to be avoided in high fistula surgery? Seton was one attempt in use for over 2000 years. Caustics and curettage were common in the ancient world as well , and now [2]. There are a number of reports in contemporary literature of immediate sphincter repair after fistulotomy or fistulectomy which often offer fair results but not better than those described above for fistulotomy alone [32–34]. The newer technologies such as LIFT, VAAFT, MAFT, PERFACT, FIPS, flaps, glue, or plugs are all interventions present in many randomized trials but almost never in comparison to fistulotomy, because of concern over the ethics of allocation of participants to the fistulotomy group. Yet fistulotomy is the gold standard therapy

with the best chance of curing a disease that is at least unpleasant, and, in the case of Henry V, fatal. There are those that, against the flow, have advocated fistulotomy for high fistula [35]. I must admit that I have been impressed with the results when my patients that have failed my flaps and glue, went on to fistulotomy for high fistula.

The elephant in the room is that in all this literature there is no discussion of the treatment of incontinence after fistulotomy. This incontinence is, in almost all cases, related to an otherwise uninjured sphincter, neurologically speaking. So unlike sphincter injury during childbirth (wherein the most serious injury occurs in the third trimester [36]), the results of a delayed sphincter repair should be excellent. What I am suggesting to solve this ancient problem is a two stage fistulotomy, not in the traditional sense, but a complete fistulotomy in the first stage and, for what may be a small minority of those patients, a delayed sphincter repair after complete healing. I must admit I have never done this, as I have never done an internal sphincter repair after lateral internal sphincterotomy for fissure, since I have not yet encountered such a patient.

Enough said about incontinence.

Other Complications of Fistulotomy

Finding the Internal Opening

Successful fistulotomy is dependent on accurate location of the internal opening. It usually follows Goodsall's Rule (which really states that most fistulas originate in the posterior midline), but often does not. It is a mistake to do a fistulotomy until this opening is found and this can be very difficult. Peroxide and or methylene blue injection in the fistula track, and repeated surgical exploration are all useful techniques [37]. In recent years magnetic resonance imaging has attained the status of the gold standard in this regard, both to assist in finding the opening, and to locate occult septic foci that need drainage [38–41]. Its use is routine in some centers and more selected in others. Of course it is a guide and not replacement of surgical exploration.

Cancer

It may seem far fetched but there are data that show an increased risk of cancer in neglected anorectal disease [42, 43]. The reason to mention this in the context of fistulotomy is a publication reporting anorectal cancers found in abscesses and fistulas, in which three cases were unsuspected even at the time of fistulotomy and came as a surprise when the pathology report was received 3 days later [44]. It was my habit at that time in my career to biopsy all fistulas and the appearance of these three fistulas was entirely normal.

Crohn's Disease

Nobody has done fistulotmies in patients with Crohn's disease though there are not a lot of data to suggest why they should not, and in fact that timidity seems to be waning [30, 45, 46]. With a very edematous inflamed anal region a seton drainage certainly seems a wiser course, though that often does not do much good.

The Non-healing Wound

Any anal wound may be slow to heal. Several things need to be ruled out, including Crohn's, cancer, duplication cysts, viral infections, and tuberculosis [47–49]. Skin grafts have been proposed but I have never seen it done [50].

References

1. Kunja K, Bhishgratna L. English translation of the sushruta samhita based on the original sanskrit text. Chapter VIII. U of Toronto (off copyright). https://archive.org/details/englishtranslati00susruoft. Calcutta, 1907;338–45.
2. Bodenhamer W. Practical observations of some of the diseases of the rectum, anus and contiguous structures giving their nature, seat, causes, symptoms, consequences and prevention especially addressed tot he non-medical reader. New York: JD Redfield; 1855.
3. al-Humadi. Treatment of anorectal disease by al-Razi. JIMA. 2009;41(3):122–133.
4. Power D. Treatises of fistula in uno, hemorrhoids and clysters by John Arderne: From early 15th century manuscript translation. 1910. Early English text society of the Oxford University Press.
5. Beyond J, Carr N. Master John of Arderne—surgeon of Newark. J R Soc Med. 1988;81:43–4.
6. Dudukgian H, Abcarian H. Why do we have so much trouble treating anal fistula? World J Gastroenterol. 2011;17(28):3292–6.
7. Bernard L. Medicine at the court of Louis XIV. Med Hist. 1962;6:201–13 PubMed PMID: 13868046; Pub Med Central PMCID: PMC1034724.
8. Steele SR, Kumar R, Feingold DL, Rafferty JL, Buie WD. Standards practice task force of the american society of colon and rectal surgeons. Practice parameters for the management of perianal abscess and fistula-in-ano. Dis Colon Rectum. 2011;54(12):1465–74.
9. Chalya PL, Mabula JB. Fistulectomy versus fistulotomy with marsupialisation in the treatment of low fistula-in- ano: a prospective randomized controlled trial. Tanzan J Health Res. 2013;15(3):193–8.
10. Jain BK, Vaibhaw K, Garg PK, Gupta S, Mohanty D. Comparison of a fistulectomy and a fistulotomy with marsupialization in the management of a simple anal. Fistula: a randomized, controlled pilot trial. J Korean Soc Coloproctol. 2012;28(2):78–82.
11. Ahmed MA, Gafar A. Fistulotomy versus fistulectomy as a treatment for low anal fistula in infants: a comparative study. J Ped Surg. 2013;9(3):103–7.
12. Kamal ZB. Fistulotomy versus fistulectomy as a primary treatment for low fistula in ano. The Iraqi Postgrad Med J. 2012;11(9):1–10.
13. Kronborg O. To lay open or excise a fistula-in-ano: a randomized trial. Br J Surg. 1985;72(12):970.
14. Quah HM, Tang CL, Eu KW, Chan SY, Samuel M. Meta-analysis of randomized clinical trials comparing drainage alone versus primary sphincter-cutting procedures for anorectal abscess-fistula. Int J Colorectal Dis. 2006;21(6):602–9.

15. Sahakitrungruang C, Pattana-Arun J, Khomvilai S, Tantiphlachiva K, Atittharnsakul P, Rojanasakul A. Marsupialization for simple fistula in ano: a randomized controlled trial. J Med Assoc Thai. 2011;94(6):699–703.
16. Ho YH, Tan M, Leong AF, Seow-Choen F. Marsupialization of fistulotomy wounds improves healing: a randomized controlled trial. Br J Surg. 1998;85(1):105–7.
17. Pescatori M, Ayabaca SM, Cafaro D, Iannello A, Magrini S. Marsupialization of fistulotomy and fistulectomy wounds improves healing and decreases bleeding: a randomized controlled trial. Colorectal Dis. 2006;8(1):11–4.
18. Altomare DF, Greco VJ, Tricomi N, Arcanà F, Mancini S, Rinaldi M, Pulvirentd'Urso A, La Torre F. Seton or glue for trans-sphincteric anal fistulae: a prospective randomized crossover clinical trial. Colorectal Dis. 2011;13(1):82–6.
19. Lindsey I, Smilgin-Humphreys MM, Cunningham C, Mortensen NJ, George BD. A randomized, controlled trial of fibrin glue versus conventional treatment for anal fistula. Dis Colon Rectum. 2002;45(12):1608–15.
20. Cirocchi R, Santoro A, Trastulli S, Farinella E, Di Rocco G, Vendettuali D, Giannotti D, Redler A, Coccetta M, Gullà N, Boselli C, Avenia N, Sciannameo F, Basoli A. meta-analysis of fibrin glue versus surgery for treatment of fistula-in-ano. Ann Ital Chir. 2010;81(5):349–56.
21. Ho KS, Tsang C, Seow-Choen F, Ho YH, Tang CL, Heah SM, Eu KW. Prospective randomised trial comparing Ayurvedic cutting seton and fistulotomy for low fistula-in-ano. Tech Coloproctol. 2001;5(3):137–41.
22. Dutta G, Bain J, Ray AK, Dey S, Das N, Das B. Comparing ksharasutra (Ayurvedic seton) and open fistulotomy in the management of fistula-in-ano. J Nat Sci Biol Med. 2015;6 (2):406–10.
23. Filingeri V, Gravante G, Baldessari E, Casciani CU. Radiofrequency fistulectomy versus diathermic fistulotomy for submucosal fistulas: a randomized trial. Eur Rev Med Pharmacol Sci. 2004;8(3):111–6.
24. Gupta PJ, Heda PS, Shrirao SA, Kalaskar SS. Topical sucralfate treatment of anal fistulotomy wounds: a randomized placebo-controlled trial. Dis Colon Rectum. 2011;54(6):699–704.
25. Abramowitz L, Soudan D, Souffran M, Bouchard D, Castinel A, Suduca JM, Staumont G, Devulder F, Pigot F, Ganansia R, Varastet M. Groupe de Recherche en Proctologie de la Société Nationale Française de Colo-Proctologie and the Club de Réflexion des Cabinets et Groupe d'Hépato-Gastroentérologie; The outcome of fistulotomy for anal fistula at 1 year: a prospective multicentre french study. Colorectal Dis. 2016;18(3):279–85.
26. Vial M, Parés D, Pera M, Grande L. Faecal incontinence after seton treatment for anal fistulae with and without surgical division of internal anal sphincter: a systematic review. Colorectal Dis. 2010;12(3):172–8.
27. Zbar AP, Khaikin M. Should we care about the internal anal sphincter? Dis Colon Rectum. 2012;55(1):105–8.
28. Ritchie RD, Sackier JM, Hodde JP. Incontinence rates after cutting seton treatment for anal fistula. Colorectal Dis. 2009;11(6):564–71.
29. Kelly ME, Heneghan HM, McDermott FD, Nason GJ, Freeman C, Martin ST, Winter DC. The role of loose seton in the management of anal fistula: a multicenter study of 200 patients. Tech Coloproctol. 2014;18:915–919.
30. Galis-Rozen E, Tulchinsky H, Rosen A, Eldar S, Rabau M, Stepanski A, Klausner JM, Ziv Y. Long-term outcome of loose seton for complex anal fistula: a two-centre study of patients with and without Crohn's disease. Colorectal Dis. 2010;12(4):358–62.
31. Garcés-Albir M, García-Botello SA, Esclapez-Valero P, Sanahuja-Santafé A, Raga-Vázquez J, Espi-Macías A, Ortega-Serrano J. Quantifying the extent of fistulotomy. How much sphincter can we safely divide? a three-dimensional endosonographic study. Int J Colorectal Dis. 2012;27(8):1109–16.
32. Ratto C, Litta F, Parello A, Zaccone G, Donisi L, De Simone V. Fistulotomy with end-to-end primary sphincteroplasty for anal fistula: results from a prospective study. Dis Colon Rectum. 2013;56(2):226–33.

33. Perez F, Arroyo A, Serrano P, Sánchez A, Candela F, Perez MT, Calpena R. Randomized clinical and manometric study of advancement flap versus fistulotomy with sphincter reconstruction in the management of complex fistula-in-ano. Am J Surg. 2006;192(1):34–40.
34. Arroyo A, Pérez-Legaz J, Moya P, Armañanzas L, Lacueva J, Pérez-Vicente F, Candela F, Calpena R. Fistulotomy and sphincter reconstruction in the treatment of complex fistula-in-ano: long-term clinical and manometric results. Ann Surg. 2012;255(5):935–9.
35. Atkin GK, Martins J, Tozer P, Ranchod P, Phillips RK. For many high anal fistulas, lay open is still a good option. Tech Coloproctol. 2011;15(2):143–50.
36. Nelson RL, Furner SE, Westercamp M, Farquhar C. Cesarean delivery for the prevention of anal incontinence. Cochrane Database Syst Rev. 2010;(2).
37. Gaj F[1], Andreuccetti J, Trecca A, Crispino P. Identification of internal fistolous orifice: evolution of methylene blue technique with a mini-probe. Clin Ter. 2012;163(2):e57–60.
38. Lunniss PJ, Barker PG, Sultan AH, Armstrong P, Reznek RH, Bartram CI, Cottam KS, Phillips RK. Magnetic resonance imaging of fistula-in-ano. Dis Colon Rectum. 1994;37 (7):708–18.
39. Chapple KS, Spencer JA, Windsor AC, Wilson D, Ward J, Ambrose NS. Prognostic value of magnetic resonance imaging in the management of fistula-in-ano. DisColon Rectum. 2000;43 (4):511–6.
40. Siddiqui MR, Ashrafian H, Tozer P, Daulatzai N, Burling D, Hart A, Athanasiou T, Phillips RK. A diagnostic accuracy meta-analysis of endoanal ultrasound and MRI for perianal fistula assessment. Dis Colon Rectum. 2012;55(5):576–85.
41. Liang C, Lu Y, Zhao B, Du Y, Wang C, Jiang W. Imaging of anal fistulas: comparison of computed tomographic fistulography and magnetic resonance imaging. Korean J Radiol. 2014;15(6):712–23.
42. Nelson RL, Abcarian H. Do hemorrhoids cause cancer? Seminars in Colon and Rectal Surgery. 1995;6:178–81.
43. Murata A, Takatsuka S, Shinkawa H, Kaizaki R, Hori T, Ikehara T. A case report of metastatic anal fistula cancer treated with neoadjuvant chemotherapy. Gan To Kagaku Ryoho. 2014;41(12):1869–71.
44. Nelson RL, Prasad ML, Abcarian H. Anal carcinoma presenting as a perirectal abscess or fistula. Arch Surg. 1985;120:632–8.
45. Papaconstantinou I, Kontis E, Koutoulidis V, Mantzaris G, Vassiliou I. Surgical management of fistula-in-ano among patients with crohn's disease: analysis of outcomes after fistulotomy or seton placement-single-centerexperience. Scand J Surg. 2016.
46. Lee MJ, Heywood N, Sagar PM, Brown SR, Fearnhead NS; Pcd collaborators. surgical management of fistulating perianal crohn's disease—a UK survey. Colorectal Dis. 2016 Jul 16.
47. Khushbakht S, ul Haq A. Rectal duplication cyst: a rare cause of rectal prolapse in a toddler. J Coll Physicians Surg Pak. 2015;25(12):909–10.
48. Diaconescu IB, Bergamaschi R. Rectal duplication. Tech Coloproctol. 2015;19(11):711–2.
49. Molloy D, Sayana MK, Keane J, Mehigan B. Anal fistula: an unusual presentation of tuberculosis in a migrant health care professional. Ir J Med Sci. 2009;178(4):527–9.
50. Binda GA, Trizi F. Treatment of unhealed wound after anal fistulotomy with full-thickness skin graft. Tech Coloproctol. 2007;11(3):294.

Anorectal Fistula Surgery: Sphincter Sparing Operations

3

Marc Singer

Fistulotomy remains the most commonly performed operation to treat anal fistula. It has remained the most popular choice for decades primarily due to the high rate of successful fistula closure. Healing rates are commonly documented to be greater than 90–95%. In addition, fistulotomy is technically straightforward to perform, does not require specialized instrumentation, and is usually performed on an outpatient basis. However, fistulotomy is widely recognized to cause postoperative fecal incontinence. There are many risk factors, which have been associated with fecal incontinence including Crohn's disease, HIV, diabetes, obstetric injuries, chronic diarrhea, multiple fistulas, etc. Perhaps most importantly, the anatomy of the fistula itself influences the risk of postoperative incontinence. Clearly the amount of sphincter divided by a fistulotomy will influence postoperative function. However, this relationship is neither direct nor predictable. There are no firm guidelines regarding the amount of sphincter that is safe to divide in a particular patient. Intersphincteric and low transsphincteric fistulas are generally safe to treat with simple fistulotomy, however, higher transsphincteric, suprasphincteric, and extrasphincteric fistulas present a risk for postoperative incontinence due to the quantity of sphincter muscle divided with fistulotomy.

The rate of incontinence after fistulotomy is quite variable, with reports ranging from 0 to 50% [1–6]. Quality of reporting is inconstant, with some authors not commenting on incontinence or simply reporting rates of zero, and some offering validated survey instruments or objective measurements such as anal manometry.

In patients that have undergone fistula treatments, postoperative fecal incontinence is often minimized or ignored by surgeons. In recent years, there has been greater attention to the assessment of postoperative incontinence with formal survey

M. Singer (✉)
Division of Colon and Rectal Surgery, Rush University Medical Center,
1725 W. Harrison Street, Suite 1138, Chicago, IL 60612, USA
e-mail: Marc_Singer@rush.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_3

instruments, such as the fecal incontinence severity index (FISI) or the fecal incontinence quality of life instrument (FIQL). This has revealed significant impact on patients' quality of life. It is no longer considered acceptable to heal a fistula at the expense of continence or quality of life. Patients are well informed, and will not tolerate postoperative incontinence. The patients, like surgeons, are actively seeking alternatives.

Surgeons have embraced a variety of non-fistulotomy operations as alternatives to dividing sphincter muscle in the treatment of anal fistula. Unfortunately, none of these operations are as effective as fistulotomy. Investigators continue to search for an operation that offers the ideal balance between a high rate of fistula healing and a low rate of incontinence. These new procedures include filling of the fistula tract, local tissue transfers, and closure of the tract itself. These newer strategies are associated with some of the same risks as fistulotomy, and also some risks unique to the specific procedure or technology. Results, recurrence rates, and complications of these alternatives to fistulotomy are reviewed.

Fibrin Sealant

Brief Description

The concept of fibrin sealant used to treat anal fistulas was first introduced by Hjortrup in 1991. Traditional dogma suggested that fistula tracts were contaminated, if not infected, and often epithelized. Treatments therefore focused on laying open of the fistula (fistulotomy or cutting setons), to allow granulation of the open wound or tissue transfer onto the pathologic internal opening (flaps). Insertion of foreign material into a contaminated anal fisulta was a novel concept. Certainly the introduction of a permanent material would result in chronic infection. However, the availability of biologic materials dramatically altered this approach. Fibrin sealant was one such biologic material. It was initially derived from patient plasma, and later commercially available. Fibrin sealant is essentially a mixture of fibrinogen and thrombin, with calcium and aprotinin to stabilize the clot. The components combine and react to form a fibrin matrix, which immediately halts the flow of stool through the fistula, and then functions as a biologic scaffolding. This framework facilitates the ingrowth of fibroblasts, which ultimately yields native scar tissue. The benefit of this strategy is the induction of scar closure of the fistula, without division of any muscle fibers. In fact, there is only minimal debridement of the fistula, without any disruption of the muscle fibers.

The procedure is technically straightforward. The fistula is carefully examined and probed in order to exclude side branches or undrained sepsis. The fistula is then flushed with peroxide in order to clear stool, debris, and identify additional tracts. Gentle debridement may be performed with a fistula bush, cytology brush, curette, or gauze if the diameter of the fistula permits. The flexible application catheter is introduced through the external opening and pulled through the fistula towards the

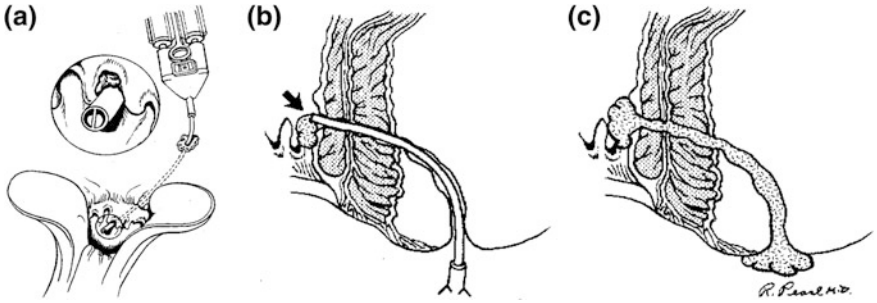


Fig. 3.1 Surgical technique for fibrin sealant injection. **a** The dual lumen catheter is inserted through the external opening towards the internal opening. **b** The fibrin sealant is injected at the internal fistula opening. **c** The catheter is withdrawn through the fistula as the fibrin sealant fills the entire space. Adapted from Singer et al. [21]

internal opening. Once the catheter is identified at the internal opening, then the two components are injected through the dual chamber catheter, and mixed at the catheter tip. The catheter is slowly withdrawn through the fistula as the sealant is injected, thus filling the entirety of the tract. Patients are instructed to avoid sitz baths and maintain light activity postoperatively (Fig. 3.1).

Results

A variety of retrospective and prospective trials have been conducted examining the success of fibrin sealant to treat anal fistula. Table 3.1 includes a data from a sampling of trials. Nearly all trials are relatively small, and most are retrospective, institutional experiences. It is clear that the success rates of fibrin sealant injection are highly variable, with success ranging from 14 to 84% in this group. Although not a formal weighted average, it can be seen that the largest number of trials suggest success rates in the range of 50–70%.

Incontinence

Although most of the retrospective and prospective trials have failed to formally assess continence with validated survey instruments or anal physiology, there are no reports, even anecdotal, of postoperative incontinence. This in fact, is consistent with clinical experience. There is minimal manipulation of the tissues, and no injury to the sphincter fibers or pudendal nerves, therefore incontinence would be unlikely. Aggressive debridement of the fistula could potentially injure muscle fibers, however, careful manipulation of the curette or fistula brush should avoid this complication. Another potential source of incontinence would be a robust fibrotic reaction to the fibrin sealant. Fortunately, the inflammatory reaction to the sealant is

Table 3.1 Results of selected trials of fibrin sealant injection for anal fistula

Author	Number patients	Healing rate (%)	Incontinence rate	Complications
Jurczak [7]	31	84	0	0 complications
Tinay [8]	19	78	NR	0 complications
Haim [9]	60	74	0	0 complications
Vitton [10]	14	71	NR	0 complications
Mishra [11]	30	70	0	1 itching
Sentovich [12]	48	69	0	1 new fistula
De Oca [13]	28	68	0	0 complications
Lindsey [14]	19	63	0	1 abscess
Maralcan [15]	46	63	0	0 complications
Adams [16]	36	61	0	10% abscess
Cintron [17]	79	61	NR	NR
Zmora [18]	60	57	0	4 pruritus, 1 rash, 1 draining sinus, 1 abscess
Hjortrup [19]	23	52	NR	0 complications
Yeung [20]	40	50	NR	0 complications
Singer [21]	75	35	0	1 abscess
Lougnarath [22]	42	31	0	0 complications
Buchanan [23]	22	14	NR	NR

NR Not reported

minimal. Many authors do not report incontinence rates, although most report a zero rate (see Table 3.1).

Abscess

Postoperative abscess is a known complication of nearly all anorectal operations, including fibrin sealant injection. Most trials report zero infections, and some trials report a rate <5% [14, 18, 21]. One trial [16] reports a 10% rate of abscess. It should be noted the external opening was closed in most of these patients. This practice is generally not shared by other authors and the external opening is specifically left open to drain. New fistulas have been reported after fibrin sealant injection [12]. This may be related to a postoperative abscess, or possibly to overzealous probing of the existing fistula.

Unique Complications

There are several potential complications unique to fibrin sealant injection. Early expulsion of the clot is one suspected cause of failure. The exact incidence of early expulsion of the clot is unknown, as this specific complication is difficult to capture in clinical trials. Patients may not identify the fibrin clot on the dressings or after bowel movements.

Another unique complication of this procedure is a reaction to one or more of the specific components of the sealant. Currently available commercial products such as Evicel® (Ethicon, Somerville, NJ) or Tisseel® (Baxter Healthcare Corporation, Deerfield, IL), contain human products. Hypersensitivity to aprotinin is a potential complication, although there are no reports of patients having sustained this complication related to treatment of anal fistula. Another potential complication includes transmission of infectious agents, such as prions causing Creutzfeldt–Jakob disease. Most manufacturers of fibrin sealant have removed bovine components due to concern for hypersensitivity to bovine proteins or transmission of infectious diseases such as bovine spongiform encephalopathy. Again, these have never been reported as consequences of the treatment of anal fistula, but remain potential complications of the fibrin sealant itself.

Implications for Further Treatment

As fibrin sealant injection does not require any incisions or tissue transfer, there is almost no deformity of the anus, injury to the sphincter, or fibrosis of the soft tissues. This means the procedure is repeatable, although rates of healing with repeat treatment are diminished, except in one series, [21] compared to initial treatment. Additionally, there is little or no effect on subsequent treatment by other techniques. This makes it a safe and reasonable choice as an initial treatment option. This is especially true in high risk patients, such as those with Crohn's disease or impaired continence. Fibrin sealant injection can be performed with essentially no risk of incontinence or negative effects on subsequent treatments if necessary.

Anal Fistula Plugs

Brief Description

The success of fibrin sealant, even if modest, to treat anal fistulas established the concept of treating anal fistula by insertion of a material into the fistula in order to promote healing. Fibrin glue was used to provide a biologic scaffolding, which promoted fibroblast ingrowth and scar formation. This treatment strategy became the basis for application of newer materials, such as anal fistula plugs. The first commercially available product was the Biodesign® anal fistula plug, formerly

Surgisis® anal fistula plug (Cook Medical, Bloomington, IN). The Biodesign® material is made from porcine small intestine submucosa. The manufacturing process removes the cells from the material, leaving a collagen rich extracellular matrix which facilitates ingrowth of vascularized tissue. Biodesign® had been used for abdominal wall reconstruction in the setting of contaminated wounds. It appeared to be relatively resistant to infection, which suggested that it would be a suitable choice for obviously contaminated anal fistula. The plug is simply inserted into the fistula, secured at the internal opening, and it then functions as a biologic scaffolding for scar ingrowth. The Biodesign® is replaced by scar tissue in the months following implantation.

The successful application of the Biodesign® anal fistula plug confirmed the viability of the fistula plug strategy. However, its success was not uniform. Surgeons believed that some of the failures were due to early plug extrusions, mismatch between the size of the plug and the diameter of the fistula, and performance of the Biodesign® material in the setting of heavy bacterial contamination. In order to address these specific concerns, a second anal fistula plug was developed. The GORE® BIO-A® Fistula Plug (W.L Gore & Associates, Newark, DE) is made from a non-woven web of polyglycolic acid: trimethylene carbonate fibers that form a 3D matrix of open, highly interconnected pores. This entirely synthetic material is hydrolyzed within 3–6 months following implantation. After the material is hydrolyzed, no prosthetic material remains in the fistula. In addition, rather than a simple conical design, the BIO-A® fistula plug was designed with two significant changes. First, a disk of the material was placed at the internal opening. This facilitated a more secure anchoring of the internal aspect of the plug to the internal sphincter. Second, the body of the plug is composed of six tubes of material. This allows the surgeon to remove a number of tubes so as to approximate the overall girth of the plug to the diameter of the fistula tract.

Fistulas treated with plugs are most often drained with a loose seton for 6–12 weeks preoperatively. The fistula is managed as with fibrin sealant injection: careful probing, gentle debridement, and flushing. The Biodesign® plug is dragged with a suture from the internal opening to the external opening. The body is conical, and so gentle traction is applied until the plug fits snugly within the fistula. It is secured with absorbable sutures at the internal sphincter. The BIO-A® plug is also pulled through the internal opening with a suture. The disk of material at the internal aspect can either be secured with simple suturing, or buried in a small submucosal pocket. Patients are typically discharged home postoperatively with instructions for light activity. Sitz baths avoided so as not to promote hydrolysis of the BIO-A® material.

Results

Results of treatment with anal fistula plugs have been variable. The publications referenced in Table 3.2 reveal healing rates from 14 to 80% for the Biodesign® fistula plug, and 16–73% for the GORE BIO-A® fistula plug. Initial reports were

quite promising, [25, 26, 28] however these data were generated by a single institution, and subsequent results have been less successful. O'Riordan [24] published a systematic review of 20 trials, including 530 patients, demonstrating healing with the Biodesign® fistula plug to be from 20 to 86%. The weighted mean rate of healing in non-Crohn's patients was 54% and in patients with Crohn's disease was 55%. As it is relatively newer, there are fewer publications documenting the success rate with the BIO-A® fistula plug. Most publications suggest the efficacy of the BIO-A® plug is similar to the Biodesign® plug [40–45].

Incontinence

As the fistula plug procedures do not involve division of sphincter fibers, the risks of incontinence should be minimal. In fact, there is little evidence that either type of fistula plug causes postoperative incontinence. As with fibrin sealant, most publications do not document formal evaluation with validated survey instruments or physiologic testing, but the number of patients with postoperative incontinence is minimal. O'Riordan et al. identified 0 patients with worsening continence in their review of 8 trials including 196 patients. Narang [47] performed a systematic review of 6 trials including 221 patients treated with BIO-A® fistula plug and identified worsening continence in 5.8% of patients. Interestingly, Stamos [45] et al. evaluated patients with the Wexner Score, and found that postoperative scores improved after treatment with the BIO-A® plug in the overall population. However, 11% of patients documented a worsening score. The authors speculate that patients may not be capable of accurately differentiating true incontinence from fistula drainage.

Abscess

Fistula plugs have been associated with postoperative abscess. The external fistula opening is specifically left open to drain; in fact, should be further opened to promote drainage of the contaminated tract. Trials included in Table 3.2 report abscesses to occur 5–14% [30, 35, 39, 42, 45, 46] Garg [48] conducted a systematic review of 25 publications including 317 patients, which identified 10% rate of abscess in trials reporting complications.

Unique Complications

Both types of anal fistula plugs are at risk for early postoperative extrusion of the plug itself. Part or the entire plug may expel prior to adequate tissue ingrowth. The exact time required for the plugs to remain in place is unknown, however patients presenting with part or the entire plug within the first 2 weeks postoperatively seem very unlikely to obtain the full benefit of the operation. In the case of partial plug

Table 3.2 Results of selected trials of anal fistula plug to treat anal fistula

Author	Number patients	Healing rate (%)	Incontinence rate (%)	Other complications
Biodesign®				
Johnson [25]	15	87	NR	
Champagne [26]	46	83	NR	9% early extrusion
Ellis [27]	63	81	0	1% early extrusion
O'connor [28]	20	80	NR	0
Schwander [29]	60	62	0	3% early extrusion
Ky [30]	45	55	0	11% abscess
Han [31]	114	54	NR	10% early extrusion
Thekkinkattil [32]	43	44	NR	22% early extrusion
McGee [33]	42	43	0	0
Van Koperen [34]	17	41	0	41% early extrusion
Cintron [35]	73	38	0	9% early extrusion, 5% abscess
Hyman [36]	43	32	NR	NR
Christoforidis [37]	37	32	0	19% early extrusion, 14% antibiotics for pain and drainage
El-Gazzaz [38]	33	25	0	9% early extrusion
Safar [39]	35	14	0	9% early extrusion; 14% abscess
GORE BIO-A®				
Ratto [40]	11	73	0	0 early extrusion
Heydari [41]	48	69	0	0 early extrusion
Ommer [42]	40	58	0	5% early extrusion, 3% abscess
Buchberg [43]	10	55	0	NR
Herold [44]	60	52	0	10% early extrusion
Stamos [45]	93	49	11	9% early extrusion, 12% infections, 2% new fistula
De la Portilla [46]	19	16	5	5% early extrusion, 5% infection

NR Not reported

fallout, it is unclear if the benefit of the operation persists if the plug remains at the internal opening. This is likely to be the pathologic part of the fistula, the abnormality that keeps the fistula patent, however, so few such cases are reported that no conclusions may be drawn. Early fallout may be related to inadequate techniques of securing the plug at the internal opening. A consensus conference was convened in order to standardize best practices related to the Biodesign® plug [49]. The BIO-A®

plug may be directly sutured or buried in a submucosal pocket. Inadequate evidence exists to recommend one technique over the other. Most of the trials represented in Table 3.2 suggest a 5–10% rate of early plug extrusion. Van Koperen [34] reports a fallout rate of 41%, however this was in a small number of patients. The trial was prematurely closed due to unacceptable results. The systematic reviews of patients treated with Biodesign® document an extrusion rate of 19% in 432 patients [48] and 8.7% in 530 patients [46]. The systematic review of patients treated with BIO-A® reveals an extrusion rate of 5% in 221 patients [47].

Some patients experience a significant inflammatory response to the plug material. This is a risk factor related to any foreign material implantation. This may be an allergy in the case of porcine tissue, or a simple inflammatory response to the BIO-A® material. This has not been formally characterized enough to understand the relationship to healing rates.

Implications for Further Treatment

Similar to fibrin sealant, the insertion of an anal fistula plug is a low risk procedure. Typically, if the plug fails, the wound simply continues to drain. There is some degree of inflammatory response to either material, and this may be variable between patients, but there is not significant disruption of the sphincter muscle fibers or the nerves. There is minimal disfigurement of the normal anatomy of the anus. Therefore, insertion of anal fistula plugs may be considered a first line therapy, provided the surgeon appropriately counsels the patient regarding the expected healing rate. It is also an excellent first line therapy in patients with marginal or impaired continence, chronic diarrhea, or Crohn's disease.

Flaps (Endorectal Advancement Flap and Dermal Advancement Flap)

Brief Description

Flap coverage of a chronic wound is a commonly employed wound closure strategy. This principle of transfer of normal tissue to an abnormal, nonhealing tissue bed has also been applied to anal fistula. The internal fistula opening is believed to be the pathological site. If healthy tissue can be transferred onto and subsequently heal the internal opening, then the remainder of the tract should eventually heal. This may be accomplished by transfer of tissue from within the rectum, as endorectal advancement flap, or from the perianal skin, as a dermal flap. Flaps are technically more challenging to perform than fibrin sealant or fistula plugs. Thorough knowledge of the relevant anatomy, and precise dissection of the appropriate tissue plans are critical to perform a safe procedure. This can be particularly difficult

in patients that have undergone previous operative procedures. Fibrosis surrounding the fistula can make dissection of the flap quite challenging.

Endorectal advancement flaps were the initial flaps described to treat anal fistula, and have been widely adopted. Clinical experience, and therefore published data, are extensive. The endorectal advancement flap is designed as a broad based flap originating well proximal to the internal fistula opening. The endorectal advancement flap is often called a mucosal flap, which is somewhat of a misnomer, as the flap is generally thicker than mucosa alone. A purely mucosal flap is at high risk to become ischemic and retract. The flap should in fact incorporate the submucosa and even a small amount of muscle. Injection of an epinephrine containing solution into the submucosal space can facilitate this dissection and aid hemostasis. A broad based flap is prepared and generously mobilized. The internal opening should be debrided, and sutured closed with absorbable sutures. The distal aspect of the flap is debrided and finally secured just distal to the internal opening.

The dermal flap (also called dermal island flap, island flap, or anocutaneous flap), described by Nelson et al. [73], also transfers normal tissue to the internal opening. This flap however utilizes perianal skin. The fistula is flushed, and the internal opening closed as with endorectal advancement flaps. Either a "U" shaped, or teardrop-shaped incision is then created at the perianal skin in order to create a broad based flap of skin and subcutaneous fat, including the fistula tract itself. This tissue is then advanced into the anal canal, and sutured overlying the internal opening. The external wound may be closed, or left open to avoid tension on the flap.

Results

The results of flaps are more consistent than the results of fibrin sealant or anal fistula plugs. Table 3.3 details selected publications with healing rates mostly in the range of 60–80% for both endorectal advancement flaps and dermal flaps. Soltani [50] published a systematic review of 35 studies including 2065 patients who underwent mucosal advancement flaps. Successful closure was achieved in 76% (37–99%) of patients. Recurrences were noted up to 2 years postoperatively.

Incontinence

The flap procedures have been designed to avoid division of the sphincter muscle fibers. However, there is most often some degree of disruption of the muscle. This may be the cause of incontinence in postoperative patients. Alternatively, the flap may alter the contour of the anal canal, impair sensation, or even injure fibers of the pudendal nerve. It may also be possible that the conduct of operations require significant retraction and manipulation of the anus, causing stretch injuries to the muscle and nerve fibers. The exact mechanisms of postoperative incontinence are unclear. As with other the procedures, the methods of evaluation of continence are

Table 3.3 Results of selected trials of endorectal advancement flaps and dermal flaps

Author	Number patients	Healing rate (%)	Incontinence rate (%)	Other complications
Endorectal advancement flap				
Aguilar [51]	189	99	10	1% anal stenosis, 1% bleeding
Golub [52]	164	97	15	8% urinary retention, 1% bleeding
Wedell [53]	30	97	28	
Uribe [54]	60	93	20	
Ortiz [55]	103	93	8	
Ortiz [56]	91	82	12	
Roig [57]	71	82	28	
Dubsky [58]	54	76	29	
Schouten [59]	44	75	35	
Van Koperen [60]	80	74	NR	
Koehler [61]	42	74	32	
Jarrar [62]	98	72	43	
Mitalas [63]	87	69	3	
Ellis [64]	95	67	0	
Sonoda [65]	105	64	NR	
Christoforidis [37]	43	63	5	5% bleeding
Mizrahi [66]	94	60	9	
Van Koperen [67]	29	48	NR	
Van der Hagen [68]	103	37	10	
Dermal flap				
Hossack [69]	16	94	0	
Sungurtekin [70]	65	91	0	5% wound dehiscence
Amin [71]	18	83	0	
Ho [72]	10	80	0	
Nelson [73]	65	80	NR	
Robertson [74]	20	80	NR	
Ellis [75]	12	75	NR	
Zimmerman [76]	26	46	30	

NR Not reported

poorly detailed in the literature, if reported at all. The trials reported in Table 3.3 suggest a postoperative incontinence rate from 0 to 43%. It should be noted that it is not always clear in these publications if the postoperative incontinence is entirely new, or how many patients experienced preoperative incontinence. The Soltani [50]

systematic review reported 13.3% incontinence as a weighted average for those publications which detailed incontinence rates. Anal manometry only reported in four trials (44 patients), with only one reporting reduction of resting and squeeze pressures [54], and the others without differences [77–79]. Those studies which do report incontinence, only follow patients for less than 2 years. Incontinence is truly a lifetime risk, therefore longer term follow-up is required.

Unique Complications

Both endorectal or dermal flaps are at risk of ischemia. If the flap is constructed too thin, containing mucosal only, it will be at a high risk for ischemia, nonhealing, and retraction. This can also occur if the base of the flap is too narrow. If either type of flap is not adequately mobilized, then excess tension on the flap may cause retraction. Retraction of the flap will leave the internal opening exposed, and will generally not heal. Flap necrosis is known to occur, although documented reports are rare [53].

If the internal opening is relatively distal, or the distance between the dentate line and anal verge is short, then the distal aspect of an endorectal advancement flap may protrude from the anus. In essence, the flap creates an ectropion. Although not dangerous, this may cause the patient to have a chronically wet anus, itching, perianal irritation, etc. If symptoms are significant enough, this may require operative revision [52].

The dermal flap is designed to contain the fistula (Fig. 3.2). Mobilization of the soft tissue flap may cause a violation of the fistula itself. This may potentially lead to drainage from a new external opening, thus creating a new fistula.

Implications for Further Treatment

If an endorectal advancement flap retracts, there may be a large defect overlying the internal opening. A repeat flap is possible, but scarring in the submucosal plane will make subsequent mobilization of the flap progressively more difficult. Alternative approaches such as a dermal flap or LIFT may be technically less difficult after a failed endorectal advancement flap. Repeating a dermal flap is also possible, but the degree of fibrosis may limit the feasibility.

LIFT Procedure

Brief Description

The most recent fistula treatment that has been widely adopted is the ligation of the intersphincteric fistula tract (LIFT) procedure. This operation was first described by

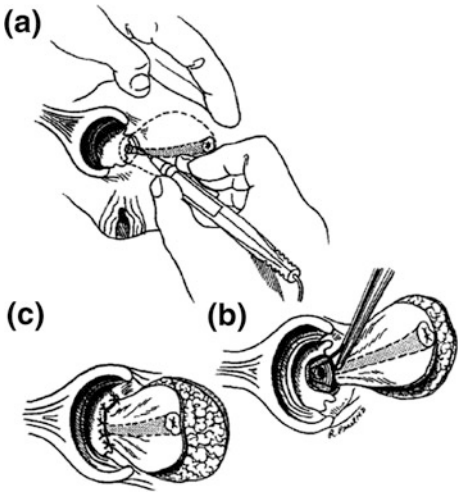


Fig. 3.2 Surgical technique for dermal flap. **a** A teardrop incision is made to include the fistula tract within the flap. **b** The flap is mobilized without undermining and the internal opening is excised. **c** The internal sphincter defect is closed and the flap secured with absorbable sutures. Adapted from Nelson et al. [73]

Rojanasakul [83] in Thailand, and subsequently popularized in the United States [94]. Although multiple surgical options were available to surgeons by this time, none of the procedures achieved the optimal balance of high rates of healing and low rates of incontinence. Frustration with the failure of non-fistulotomy options to achieve similar rates of healing as fistulotomy likely contributed to the enthusiastic adoption of LIFT.

The concept of LIFT was novel. The premise involves suture ligation and division of the fistula tract within the intersphincteric space. Ligation of the internal opening alone has been attempted with little success. Closure of the internal opening with flaps or sealant was met with only moderate success. Closure of the external opening was likely to precipitate an abscess. LIFT introduced the concept of closure of the fistula at a different location, the intersphincteric portion. This anatomic space can be entered without disruption of the sphincters, achieving access to the fistula. Division of the tract isolates the pathologic internal opening from the remainder of the fistula.

A skin incision is made overlying the fistula at the intersphincteric groove. The internal and external sphincters are bluntly separated as the intersphincteric space is developed. The fistula is isolated from surrounding sphincter fibers, and then suture ligated proximally (near the internal sphincter) and distally (near the external sphincter). The fistula is then divided between the sutures. The sphincters are reapproximated and the skin loosely closed (Fig. 3.3).

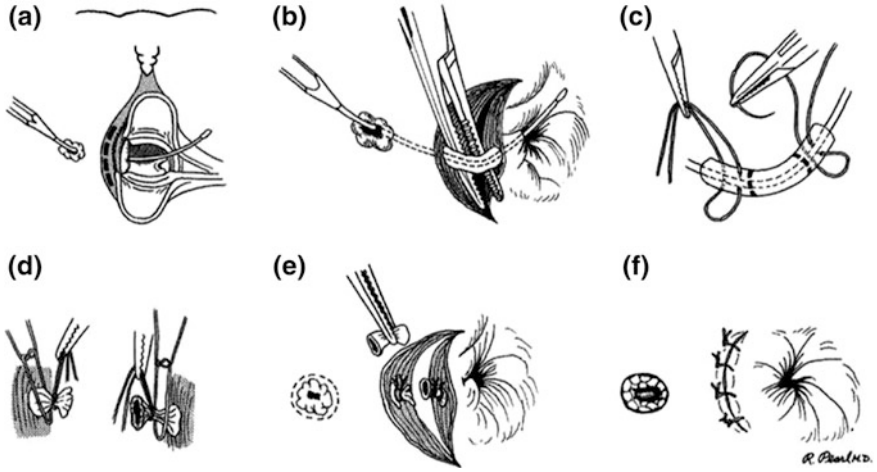


Fig. 3.3 Surgical technique of the ligation of the intersphincteric tract (LIFT) procedure. **a** Incision overlying the intersphincteric groove. **b** Isolation of the intersphincteric portion of the fistula tract. **c** Suture ligation of the fistula tract proximally and distally. **d** Additional ligation of the tract. **e** Division of the fistula tract, with excision of a segment. **f** Loose closure of incision and debridement of external opening. Adapted from Abcarian et al. [88]

Results

Selected publications are detailed in Table 3.4. Most authors report success rates in the range of 60–80%. It should be noted, that most of these trials were published in the last 2–4 years, therefore longer term results are unknown. Several systematic reviews which can give further perspective on larger data sets have been recently published. Yassin et al. [80] reviewed 29 papers including 498 patients. Success rates ranged from 40 to 95% with weighted average 71% healing. Alasari [81] reviewed 13 trials including 435 patients. This group enjoyed 81% success rate. And most recently, Hong [82] reviewed 24 papers including 1,110 patients. This largest review to date calculated an overall 76% healing. Although the number and quality of the trials included were variable, the final healing rates were quite similar.

Incontinence

The selected trials detailed in Table 3.4 reveal postoperative incontinence to be quite rare. As with the other procedures however, the quality of reporting is highly variable and many trials do not use validated instruments or anal physiology studies. The systematic reviews confirm very low rates of incontinence (Yassin [80] 6% minor incontinence, Alasari [81] and Hong [82] 0 incontinence). Although the newest choice, the short term rate of incontinence appears to be extremely low. As incontinence may be a lifetime risk, long term data are necessary.

Table 3.4 Results of selected trials of LIFT procedure

LIFT	Number of patients	Healing rates (%)	Incontinence rates (%)	Other complications
Rojanasakul [83]	17	94	0	
Schulze [84]	75	88	1.3	
Shanwani [85]	45	82	0	
Van Onkelen [86]	22	82	0	
Tan [87]	93	78	0	
Abcarian [88]	40	74	0	
Sileri [89]	26	73	0	
Aboulian [90]	25	68	0	8% vaginal fungal infections
Ooi [91]	25	68	0	
Lehmann [92]	17	65	0	6% hematoma, 6% wound infection
Chew [93]	33	63	0	
Bleier [94]	39	57	0	3% anal fissure, 3% persistent pain
Wallin [95]	93	40	0	

Unique Complications

Dehiscence of the surgical incision is a complication unique to LIFT. Dehiscense can also occur with flaps, but the consequences are different. If the flap suture line opens, the flap is likely to retract to its normal anatomic position, and will likely result in a failure. If the LIFT incision opens, this does not necessarily mean that the suture ligature of the fistula tract itself will also fail. If the wound is treated with appropriate wound care, then the wound may granulate and fistula healing may occur. The patient may experience pain, drainage, or bleeding, but should be encouraged to be patient and attend to the wound.

In addition, the dissection of the intersphincteric space can be difficult. This may be encountered if the patient has had a very chronically inflamed fistula or if previous LIFT or flap were performed. Also, the posterior midline may be technically challenging location to isolate the tract in the intersphincteric space. A difficult dissection for any of these reasons may lead to deviation from the intersphincteric space, and perforation of the rectal mucosa. This could potentially lead to a new fistula tract onto the perineum or even vagina.

Implications for Further Treatment

The pattern of failure of LIFT should be considered. Persistent fistulas after LIFT generally occur either at the original external opening or through the surgical

incision. The two ends of the fistula tract may simply re-fistulize. This results in recurrence of the transsphincteric fistula, with resultant drainage through the original external opening. However, some recurrences will occur at the surgical incision, meaning through the intersphincteric space. This occurs when the proximal portion of the fistula fails to heal, and drains through the newly opened intersphincteric space, and through the surgical incision. This may happen because the proximal portion fails to heal, or may be due to the technical error of failing to completely close the tract. Sutures may not encompass the deep aspect of the fistula, or slip off the tract. This converts a transsphincteric fistula to an intersphincteric fistula. This specific pattern of recurrence is documented in some publications [87], but not most. These patients, with an intersphincteric fistula, can be easily treated with a fistulotomy of the persistent, proximal portion of the tract, as it now involves only the internal sphincter. This two stage approach of a LIFT, followed by an internal sphincterotomy, can successfully treat most fistulas while sparing the external sphincter.

Endorectal advancement flap after LIFT should not be significantly more difficult as the dissection occurs at the skin and intersphincteric space. The mucosa/submucosal planes inside of the anal canal should remain relatively undisturbed. A dermal flap may be more difficult due to the previous dissection in the immediate vicinity of the tract.

As LIFT is the newest surgical option, there is not yet significant experience treating the failures by other methods. Firm recommendations can not be made. Publication of these experiences would be helpful in the decision-making process.

References

1. Garcia-Aguilar J, Belmonte C, Wong WD, Goldberg SM. Mad- off RD. Anal fistula surgery: factors associated with recurrence and incontinence. Dis Colon Rectum. 1996;39:723–9.
2. Marks CG, Ritchie JK. Anal fistulas at St Mark's Hospital. Br J Surg. 1977;64:84–91.
3. Lunniss PJ, Kamm MA, Phillips RK. Factors affecting continence after surgery for anal fistula. Br J Surg. 1994;81(9):1382–5.
4. Sainio P, Husa A. Fistula-in-ano. Clinical features and long-term results of surgery in 199 adults. Acta Chir Scand. 1984;151(2):169–76.
5. Jordán J, Roig JV, García-Armengol J, García-Granero E, Solana A, Lledó S. Risk factors for recurrence and incontinence after anal fistula surgery. Colorectal Dis. 2010;12(3):254–60.
6. van Koperen PJ, Wind J, Bemelman WA, Bakx R, Reitsma JB, Slors JF. Long-term functional outcome and risk factors for recurrence after surgical treatment for low and high perianal fistulas of cryptoglandular origin. Dis Colon Rectum. 2008;51(10):1475–81.
7. Jurczak F, Laridon JY, Raffaitin P, Pousset JP. Biological fibrin used in anal fistulas: 31 patients. Ann Chir. 2004;129:286–9.
8. Tinay EY, El-Bakry AA. Treatment of chronic fistula-in-ano using commercial fibrin glue. Saudi Med J. 2003;24(10):1116–7.
9. Haim N, Neufeld D, Ziv Y, et al. Long-term results of fibrin glue treatment for cryptogenic perianal fistulas: a multicenter study. Dis Colon Rectum. 2011;54:1279–83.
10. Vitton V, Gasmi M, Barthet M, Desjeux A, Orsoni P, Grimaud JC. Long-term healing of Crohn's anal fistulas with fibrin glue injection. Aliment Pharmacol Ther. 2005;21:1453–7.

11. Mishra A, Shah S, Nar AS, Bawa A. The role of fibrin glue in the treatment of high and low fistulas in ano. J Clin Diagn Res. 2013;7(5):876–9.
12. Sentovich SM. Fibrin glue for anal fistulas. Dis Colon Rectum. 2003;46(4):498–502.
13. De Oca J, Millán M, Jiménez A, Golda T, Biondo S. Long-term results of surgery plus fibrin sealant for anal fistula. Colorectal Dis. 2012;14(1):e12–5.
14. Lindsey I, Smilgin-Humphreys MM, Cunningham C, Mortensen NJ, George BD. A randomized, controlled trial of fibrin glue versus conventional treatment for anal fistula. Dis Colon Rectum. 2002;45:1608–15.
15. Maralcan G, Başkonuş İ, Gökalp A, Borazan E, Balk A. Long-term results in the treatment of fistula-in-ano with fibrin glue: a prospective study. J Korean Surg Soc. 2011;81(3):169–75.
16. Adams T, Yang J, Kondylis LA, Kondylis PD. Long-term outlook after successful fibrin glue ablation of cryptoglandular transsphincteric fistula-in-ano. Dis Colon Rectum. 2008;51 (10):1488–90.
17. Cintron JR, Park JJ, Orsay CP, Pearl RK, Nelson RL, Sone JH, Song R, Abcarian H. Repair of fistulas-in-ano using fibrin adhesive. Dis Colon Rectum. 2000;43(7):944–9.
18. Zmora O, Neufeld D, Ziv Y, et al. Prospective, multicenter evaluation of highly concentrated fibrin glue in the treatment of complex cryptogenic perianal fistulas. Dis Colon Rectum. 2005;48:2167–72.
19. Hjortrup A, Moesgaard F, Kjærgård J. Fibrin adhesive in the treatment of perineal fistulas. Dis Colon Rectum. 1991;34(9):752–4.
20. Yeung JM, Simpson JA, Tang SW, Armitage NC, Maxwell-Armstrong C. Fibrin glue for the treatment of fistulae in ano–a method worth sticking to? Colorectal Dis. 2010;12:363–6.
21. Singer M, Cintron J, Nelson R, et al. Treatment of fistulas-in-ano with fibrin sealant in combination with intra-adhesive antibiotics and/or surgical closure of the internal fistula opening. Dis Colon Rectum. 2005;48(4):799–808.
22. Loungnarath R, Dietz DW, Mutch MG, Birnbaum EH, Kodner IJ, Fleshman JW. Fibrin glue treatment of complex anal fistulas has low success rate. Dis Colon Rectum. 2004;47:432–6.
23. Buchanan GN, Bartram CI, Phillips RK, Gould SW, Halligan S, Rockall TA, Sibbons P, Cohen RG. Efficacy of fibrin sealant in the management of complex anal fistula: a prospective trial. Dis Colon Rectum. 2003;46:1167–74.
24. O'Riordan JM, Datta I, Johnston C, Baxter NN. A systematic review of the anal fistula plug for patients with Crohn's and non-Crohn's related fistula-in-ano. Dis Colon Rectum. 2012;55 (3):351–8.
25. Johnson EK, Gaw JU, Armstrong DN. Efficacy of anal fistula plug vs fibrin glue in closure of anorectal fistulas. Dis Colon Rectum. 2006;49:371–6.
26. Champagne BJ, O'Connor LM, Ferguson M, Orangio GR, Schertzer ME, Armstrong DN. Efficacy of anal fistula plug in closure of cryptoglandular fistulas: long term follow-up. Dis Colon Rectum. 2006;49:1817–29.
27. Ellis CN, Rostas JW, Greiner FG. Long-term outcomes with the use of bioprosthetic plugs for the management of complex anal fistulas. Dis Colon Rectum. 2010;53(5):798–802.
28. O'Connor L, Champagne BJ, Ferguson MA, Orangio GR, Schertzer ME, Armstrong DN. Efficacy of anal fistula plug in closure of Crohn's anorectal fistulas. Dis Colon Rectum. 2006;49(10):1569–73.
29. Schwandner T, Roblick MH, Kierer W, Brom A, Padberg W, Hirschburger M. Surgical treatment of complex anal fistulas with the anal fistula plug: a prospective, multicenter study. Dis Colon Rectum. 2009;52:1578–83.
30. Ky AJ, Sylla P, Steinhagen R, Steinhagen E, Khaitov S, Ly EK. Collagen fistula plug for the treatment of anal fistulas. Dis Colon Rectum. 2008;51:838–43.
31. Han JG, Wang ZJ, Zhao BC, et al. Long-term outcomes of human acellular dermal matrix plug in closure of complex anal fistulas with a single tract. Dis Colon Rectum. 2011;54:1412–8.
32. Thekkinkattil DK, Botterill I, Ambrose NS, et al. Efficacy of the anal fistula plug in complex anorectal fistulae. Colorectal Dis. 2009;11:584–7.

33. McGee MF, Champagne BJ, Stulberg JJ, Reynolds H, Marderstein E, Delaney CP. Tract length predicts successful closure with anal fistula plug in cryptoglandular fistulas. Dis Colon Rectum. 2010;53:1116–20.
34. van Koperen PJ, D'Hoore A, Wolthuis AM, Bemelman WA, Slors JF. Anal fistula plug for closure of difficult anorectal fistula: a prospective study. Dis Colon Rectum. 2007;50:2168–72.
35. Cintron JR, Abcarian H, Chaudhry V, Singer M, Hunt S, Birnbaum E, Mutch MG, Fleshman J. Treatment of fistula-in-ano using a porcine small intestinal submucosa anal fistula plug. Tech Coloproctol. 2013;17:187–91.
36. Hyman N, O'Brien S, Osler T. Outcomes after fistulotomy: results of a prospective, multicenter regional study. Dis Colon Rectum. 2009;52:2022–7.
37. Christoforidis D, Pieh MC, Madoff RD, Mellgren AF. Treatment of transsphincteric anal fistulas by endorectal advancement flap or collagen fistula plug: a comparative study. Dis Colon Rectum. 2009;52:18–22.
38. El-Gazzaz G, Zutshi M, Hull T. A retrospective review of chronic anal fistulae treated by anal istulae plug. Colorectal Dis. 2010;12:442–7.
39. Safar B, Jobanputra S, Sands D, Weiss EG, Nogueras JJ, Wexner SD. Anal fistula plug: initial experience and outcomes. Dis Colon Rectum. 2009;52:248–52.
40. Ratto C, Litta F, Parello A, Donisi L, Zaccone G, De Simone V. Gore Bio-A® Fistula Plug: a new sphincter-sparing procedure for complex anal fistula. Colorectal Dis. 2012;14:e264–9.
41. Heydari A, Attinà GM, Merolla E, Piccoli M, Fazlalizadeh R, Melotti G. Bioabsorbable synthetic plug in the treatment of anal fistulas. Dis Colon Rectum. 2013;56(6):774–9.
42. Ommer A, Herold A, Joos A, Schmidt C, Weyand G, Bussen D. Gore BioA fistula plug in the treatment of high anal fistulas–initial results from a german multicenter-study. Ger Med Sci. 2012;10:Doc13.
43. Buchberg B, Masoomi H, Choi J, Bergman H, Mills S, Stamos MJ. A tale of two (anal fistula) plugs: is there a difference in short-term outcomes? Am Surg. 2010;76:1150–3.
44. Herold A, Ommer A, Fürst A, Pakravan F, Hahnloser D, Strittmatter B, Schiedeck T, Hetzer F, Aigner F, Berg E, Roblick M. Results of the gore Bio-A fistula plug implantation in the treatment of anal fistula: a multicentre study. Tech Coloproctol. 2016;20(8):585–90.
45. Stamos MJ, Snyder M, Robb BW, Ky A, Singer M, Stewart DB, Sonoda T, Abcarian H. Prospective multicenter study of a synthetic bioabsorbable anal fistula plug to treat cryptoglandular transsphincteric anal fistulas. Dis Colon Rectum. 2015;58(3):344–51.
46. de la Portilla F, Rada R, Jiménez-Rodríguez R, Díaz-Pavón JM, Sánchez-Gil JM. Evaluation of a new synthetic plug in the treatment of anal fistulas: results of a pilot study. Dis Colon Rectum. 2011;54:1419–22.
47. Narang SK, Jones C, Alam NN, Daniels IR, Smart NJ. Delayed absorbable synthetic plug (GORE® BIO-A®) for the treatment of fistula-in-ano: a systematic review. Colorectal Dis. 2016;18(1):37–44.
48. Garg P, Song J, Bhatia A, Kalia H, Menon GR. The efficacy of anal fistula plug in fistula-in-ano: a systematic review. Colorectal Dis. 2010;12(10):965–70.
49. The Surgisis AFP anal fistula plug. report of a consensus conference. Colorectal Dis. 2008;10:17–20.
50. Soltani A, Kaiser AM. Endorectal advancement flap for cryptoglandular or Crohn's fistula-in-ano. Dis Colon Rectum. 2010;53(4):486–95.
51. Aguilar PS, Plasencia G, Hardy TG Jr, Hartmann RF, Stewart WR. Mucosal advancement in the treatment of anal fistula. Dis Colon Rectum. 1985;28:496–8.
52. Golub RW, Wise WE Jr, Kerner BA, Khanduja KS, Aguilar PS. Endorectal mucosal advancement flap: the preferred method for complex cryptoglandular fistula-in-ano. J Gastrointest Surg. 1997;1:487–91.
53. Wedell J, Meier zu Eissen P, Banzhaf G, Kleine L. Sliding flap advancement for the treatment of high level fistulae. Br J Surg. 1987;74:390–391.

54. Uribe N, Millan M, Minguez M, et al. Clinical and manometric results of endorectal advancement flaps for complex anal fistula. Int J Colorectal Dis. 2007;22:259–64.
55. Ortiz H, Marzo J. Endorectal flap advancement repair and fistulectomy for high transsphincteric and suprasphincteric fistulas. Br J Surg. 2000;87:1680–3.
56. Ortiz H, Marzo M, de Miguel M, Ciga MA, Oteiza F, Armendariz P. Length of follow-up after fistulotomy and fistulectomy associated with endorectal advancement flap repair for fistula in ano. Br J Surg. 2008;95:484–7.
57. Roig JV, Garcia-Armengol J, Jordan JC, Moro D, Garcia-Granero E, Alos R. Fistulectomy and sphincteric reconstruction for complex cryptoglandular fistulas. Colorectal Dis. 2010;12: e145–52.
58. Dubsky PC, Stift A, Friedl J, Teleky B, Herbst F. Endorectal advancement flaps in the treatment of high anal fistula of cryptoglandular origin: full-thickness versus mucosal-rectum flaps. Dis Colon Rectum. 2008;51:852–7.
59. Schouten WR, Zimmerman DD, Briel JW. Transanal advancement flap repair of transsphincteric fistulas. Dis Colon Rectum. 1999;42:1419–22.
60. van Koperen PJ, Wind J, Bemelman WA, Slors JF. Fibrin glue and transanal rectal advancement flap for high transsphincteric perianal fistulas; is there any advantage? Int J Colorectal Dis. 2008; 23: 697–701.
61. Koehler A, Risse-Schaaf A, Athanasiadis S. Treatment for horse- shoe fistulas-in-ano with primary closure of the internal fistula opening: a clinical and manometric study. Dis Colon Rectum. 2004;47:1874–82.
62. Jarrar A, Church J. Advancement flap repair: a good option for complex anorectal fistulas. Dis Colon Rectum. 2011;54:1537–41.
63. Mitalas LE, Gosselink MP, Zimmerman DDE, Schouten WR. Repeat transanal advancement flap repair: impact on the overall healing rate of high transsphincteric fistulas and on fecal continence. Dis Colon Rectum. 2007;50:1508–11.
64. Ellis CN. Bioprosthetic plugs for complex anal fistulas: an early experience. J Surg Educ. 2007;64:36–40.
65. Sonoda T, Hull T, Piedmonte MR, Fazio VW. Outcomes of primary repair of anorectal and rectovaginal fistulas using the endorectal advancement flap. Dis Colon Rectum. 2002;45 (12):1622–8.
66. Mizrahi N, Wexner SD, Zmora O, et al. Endorectal advancement flap: are there predictors of failure? Dis Colon Rectum. 2002;45:1616–21.
67. van Koperen PJ, Bemelman WA, Gerhards MF, et al. The anal fistula plug treatment compared with the mucosal advancement flap for cryptoglandular high transsphincteric perianal fistula: a double-blinded multicenter randomized trial. Dis Colon Rectum. 2011;54:387–93.
68. van der Hagen SJ, Baeten CG, Soeters PB, van Gemert WG. Long-term outcome following mucosal advancement flap for high perianal fistulas and fistulotomy for low perianal fistulas: recurrent perianal fistulas: failure of treatment or recurrent patient disease? Int J Colorectal Dis. 2006;21:784–90.
69. Hossack T, Solomon MJ, Young JM. Ano-cutaneous flap repair for complex and recurrent supra–sphincteric anal fistula. Colorectal Dis. 2005;7(2):187–92.
70. Sungurtekin U, Sungurtekin H, Kabay B, Tekin K, Aytekin F, Erdem E, Ozden A. Anocutaneous VY advancement flap for the treatment of complex perianal fistula. Dis Colon Rectum. 2004;47(12):2178–83.
71. Amin SN, Tierney GM, Lund JN, Armitage NC. VY advancement flap for treatment of fistula-in-ano. Dis Colon Rectum. 2003;46(4):540–3.
72. Ho KS, Ho YH. Controlled, randomized trial of island flap anoplasty for treatment of trans–sphincteric fistula–in-ano: early results. Tech Coloproctol. 2005;9(2):166–8.
73. Nelson RL, Cintron J, Abcarian H. Dermal island-flap anoplasty for transsphincteric fistula-in-ano. Dis Colon Rectum. 2000;43(5):681–4.

74. Robertson WG, Mangione JS. Cutaneous advancement flap closure. Dis Colon Rectum. 1998;41(7):884–6.
75. Ellis CN, Clark S. Fibrin glue as an adjunct to flap repair of anal fistulas: a randomized, controlled study. Dis Colon Rectum. 2006;49(11):1736–40.
76. Zimmerman DD, Briel JW, Gosselink MP, Schouten WR. Anocutaneous advancement flap repair of transsphincteric fistulas. Dis Colon Rectum. 2001;44(10):1474–7.
77. Lewis WG, Finan PJ, Holdsworth PJ, Sagar PM, Stephenson BM. Clinical results and manometric studies after rectal flap advancement for infra-levator trans-sphincteric fistula-in-ano. Int J Colorectal Dis. 1995;10:189–92.
78. Kreis ME, Jehle EC, Ohlemann M, Becker HD, Starlinger MJ. Functional results after transanal rectal advancement flap repair of trans-sphincteric fistula. Br J Surg. 1998;85:240–2.
79. Perez F, Arroyo A, Serrano P, et al. Randomized clinical and manometric study of advancement flap versus fistulotomy with sphincter reconstruction in the management of complex fistula-in-ano. Am J Surg. 2006;192:34–40.
80. Yassin NA, Hammond TM, Lunniss PJ, Phillips RK. Ligation of the intersphincteric fistula tract in the management of anal fistula. A systematic review. Colorectal Dis. 2013;15(5):527–35.
81. Alasari S, Kim NK. Overview of anal fistula and systematic review of ligation of the intersphincteric fistula tract (LIFT). Tech Coloproctol. 2014;18(1):13–22.
82. Hong KD, Kang S, Kalaskar S, Wexner SD. Ligation of intersphincteric fistula tract (LIFT) to treat anal fistula: systematic review and meta-analysis. Tech Coloproctol. 2014;18(8):685–91.
83. Rojanasakul A, Pattanaarun J, Sahakitrungruang C, Tantiphlachiva K. Total anal sphincter saving technique for fistula-in-ano; the ligation of intersphincteric fistula tract. J Med Assoc Thai Chotmaihet Thangphaet. 2007;90(3):581–6.
84. Schulze B, Ho YH. Management of complex anorectal fistulas with seton drainage plus partial fistulotomy and subsequent ligation of intersphincteric fistula tract (LIFT). Tech Coloproctol. 2015;19:89–95.
85. Shanwani A, Nor AM, Amri N. Ligation of the intersphincteric fistula tract (LIFT): a sphincter-saving technique for fistula-in-ano. Dis Colon Rectum. 2010;53:39–42.
86. Van Onkelen RS, Gosselink MP, Schouten WR. Ligation of the intersphincteric fistula tract in low transsphincteric fistulae: a new technique to avoid fistulotomy. Colorectal Dis. 2013;15(5):587–91.
87. Tan KK, Tan IJ, Lim FS, Koh DC, Tsang CB. The anatomy of failures following the ligation of intersphincteric tract technique for anal fistula: a review of 93 patients over 4 years. Dis Colon Rectum. 2011;54:1368–72.
88. Abcarian AM, Estrada JJ, Park J, Corning C, Chaudhry V, Cintron J, Prasad L, Abcarian H. Ligation of intersphincteric fistula tract: early results of a pilot study. Dis Colon Rectum. 2012;55(7):778–82.
89. Sileri P, Giarratano G, Franceschilli L, Limura E, Perrone F, Stazi A, Toscana C, Gaspari AL. Ligation of the intersphincteric fistula tract (LIFT): a minimally invasive procedure for complex anal fistula Two-Year results of a prospective multicentric study. Surg Innov. 2014;21(5):476–80.
90. Aboulian A, Kaji AH, Kumar RR. Early result of ligation of the intersphincteric fistula tract for fistula-in-ano. Dis Colon Rectum. 2011;54:289–92.
91. Ooi K, Skinner I, Croxford M, Faragher I, McLaughlin S. Managing fistula-in-ano with ligation of the intersphincteric fistula tract procedure: the Western Hospital experience. Colorectal Dis. 2012;14(5):599–603.
92. Lehmann JP, Graf W. Efficacy of LIFT for recurrent anal fistula. Colorectal Dis. 2013;15:592–5.
93. Chew MH, Lee PJ, Koh CE, Chew HE. Appraisal of the LIFT and BIOLIFT procedure: initial experience and short-term outcomes of 33 consecutive patients. Int J Colorectal Dis. 2013;28(11):1489–96.

94. Bleier JI, Moloo H, Goldberg SM. Ligation of the intersphincteric fistula tract: an effective new technique for complex fistulas. Dis Colon Rectum. 2010;53:43–6.
95. Wallin UG, Mellgren AF, Madoff RD, Goldberg SM. Does ligation of the intersphincteric fistula tract raise the bar in fistula surgery? Dis Colon Rectum. 2012;55(11):1173–8.

Hemorrhoids

4

Jose Cintron, Ariane M. Abcarian, Herand Abcarian, Kristine Makiewicz and Marc I. Brand

Complications of Excisional Hemorrhoidectomy

Jose Cintron

Although symptoms of hemorrhoids are quite common, only about 5–10% of patients with symptomatic hemorrhoids will require surgical treatment. This is due in large part to the success of conservative management in conjunction with office-based procedures in the majority of patients.

J. Cintron (✉)
Division of Colon and Rectal Surgery, John H. Stroger Jr. Hospital of Cook County, 1900 W. Polk Street, Chicago, IL 60612, USA
e-mail: cintron2@gmail.com

A.M. Abcarian
Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, 1900 W. Polk Street, Chicago, IL 60612, USA

H. Abcarian
Division of Colon and Rectal Surgery, University of Illinois at Chicago, 840 S. Wood Street, MC 958, Chicago, IL 60612, USA
e-mail: abcarian@uic.edu

K. Makiewicz · M.I. Brand
Section of Colon and Rectal Surgery, Department of General Surgery, Rush University Medical Center, Chicago, IL, USA
e-mail: kristine_makiewicz@rush.edu

M.I. Brand
e-mail: Mbrand1@rush.edu; Marc_i_brand@rush.edu

M.I. Brand
Department of General Surgery, Rush University Medical Center, Professional Office Building, Suite 1138, 1725 West Harrison Street, Chicago, IL 60612, USA

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_4

Operative treatment is usually reserved for patients with grade III/IV hemorrhoids. Although surgery is under most circumstances superior to office-based procedures, it does come at a price. Surgery is associated with increased complications including, but not limited to pain, urinary retention, bleeding, anal stenosis, infection, and incontinence. This chapter will focus primarily on the prevention and management of complications associated with excisional hemorrhoidectomy which are illustrated in the following three figures (Figs. 4.1, 4.2 and 4.3) (Milligan–Morgan open hemorrhoidectomy Fig. 11.7 p. 166 and p. 167 ASCRS [1] textbook 1st ed, Ferguson closed hemorrhoidectomy Fig. 11.8, Whitehead hemorrhoidectomy Fig. 11.9).

Excisional hemorrhoidectomy is usually reserved for patients with symptomatic mixed component (internal and external) hemorrhoids who have either failed or are not candidates for nonoperative treatment.

Pain

One of the most significant consequences of undergoing excisional hemorrhoidectomy is postoperative pain. Although one can argue as to whether pain is a complication versus an expected consequence of excisional hemorrhoidectomy, it is nonetheless, probably the most significant factor that patients have to contend with postoperatively. Unfortunately, despite newer alternatives and multimodal approaches, pain control postoperatively remains one of the major challenges for patient and surgeon. Despite the widespread adoption of performing excisional hemorrhoidectomy as an ambulatory procedure, it still remains the most common reason for delaying discharge after ambulatory surgery [2]. Additionally, increased pain can also contribute to the development of urinary retention in patients that is another cause of unplanned admission to the hospital after day case surgery. In a study looking at predictive factors for postoperative pain in the ambulatory setting Gramke [3] found that the presence of pain preoperatively, the age of the patient, the patient's fear of their surgery, and the expectations of patient and physician regarding postoperative pain were the strongest predictors in the outpatient setting.

The process of dealing with postsurgical pain management for excisional hemorrhoidectomy begins preoperatively. When the patient is seen in the office a candid discussion regarding pain expectations and strategies to control it are essential to the outcome and satisfaction of the procedure. Unfortunately, obtaining a balance of adequate pain control while preventing opioid-related side effects can sometimes be easier said than done. Nevertheless, when the patient is educated preoperatively as to the multimodal approach to analgesia they realize that the physician can target their pain from a number of different angles. This stepwise multidrug approach to pain management will lead to fewer intolerable adverse events, increased efficacy, and improved patient satisfaction.

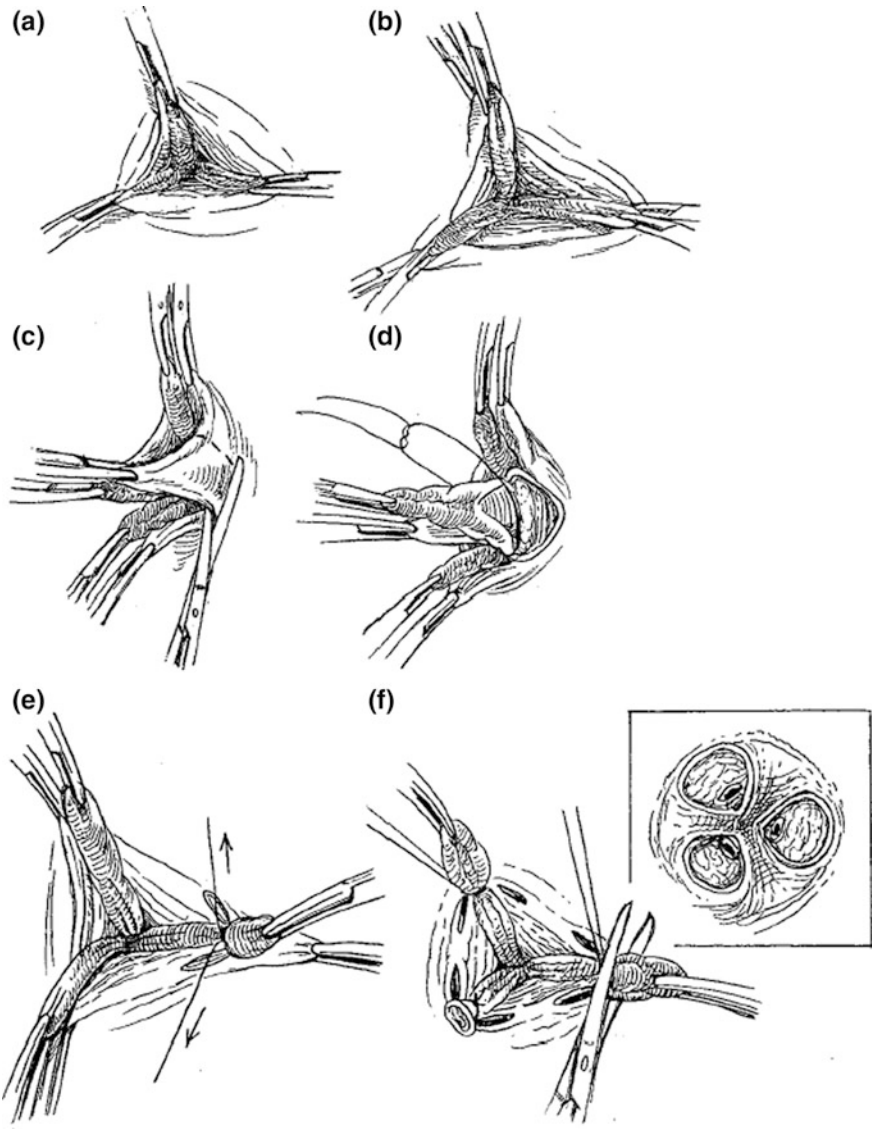


Fig. 4.1 Open (Milligan–Morgan) hemorrhoidectomy. **a** External hemorrhoids grasped with forceps and retracted outward. **b** Internal hemorrhoids grasped with forceps and retracted outward with external hemorrhoids. **c** External skin and hemorrhoid excised with scissors. **d** Suture placed through proximal internal hemorrhoid and vascular bundle. **e** Ligature tied. **f** Tissue distal to ligature is excised. *Insert* depicts completed three bundle hemorrhoidectomy

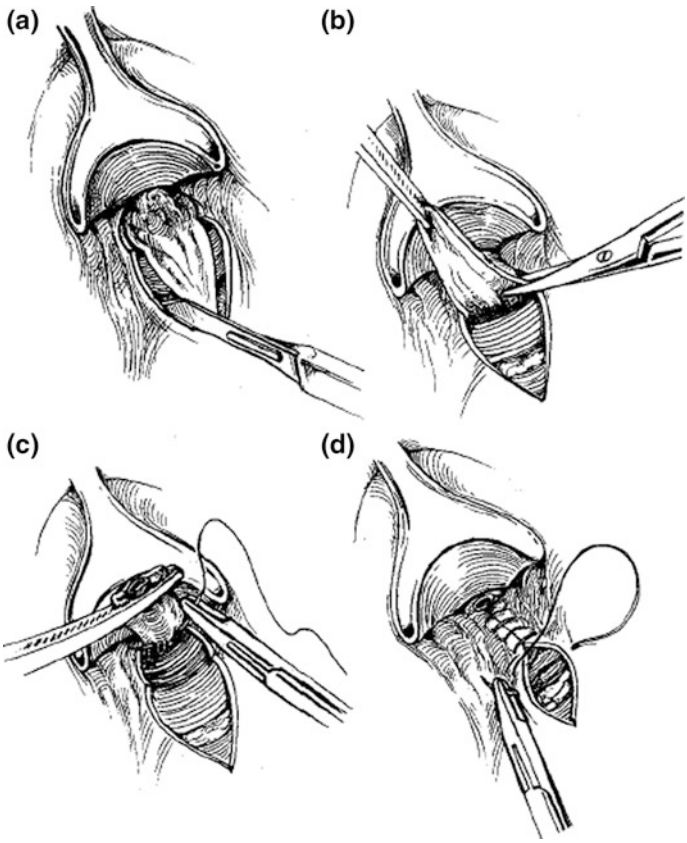


Fig. 4.2 Modified Ferguson excisional hemorrhoidectomy. **a** Double elliptical incision made in mucosa and anoderm around hemorrhoidal bundle with a scalpel. **b** The hemorrhoid dissection is carefully continued cephalad by dissecting the sphincter away from the hemorrhoid. **c** After dissection of the hemorrhoid to its pedicle, it is either clamped, secured, or excised. The pedicle is suture ligated. **d** The wound is closed with a running stitch. Excessive traction on the suture is avoided to prevent forming dog ears or displacing the anoderm caudally

Perianal Infiltration of Local Anesthetics

Probably one of the most important pillars in the multimodal approach is the intraoperative administration of local analgesics. The use of locally infiltrated anesthetics in conjunction with intravenous sedation is safe and may even have fewer complications than other anesthetic techniques for excisional hemorrhoidectomy. This technique has been shown to be the most cost effective anesthetic technique when compared to spinal anesthesia and general anesthesia [4]. This randomized clinical trial compared these three anesthetic techniques in 93 patients undergoing ambulatory anorectal surgery. The local anesthetic infiltration consisted of a mixture of 15 mL of 2% lidocaine and 15 mL of 0.5% bupivacaine

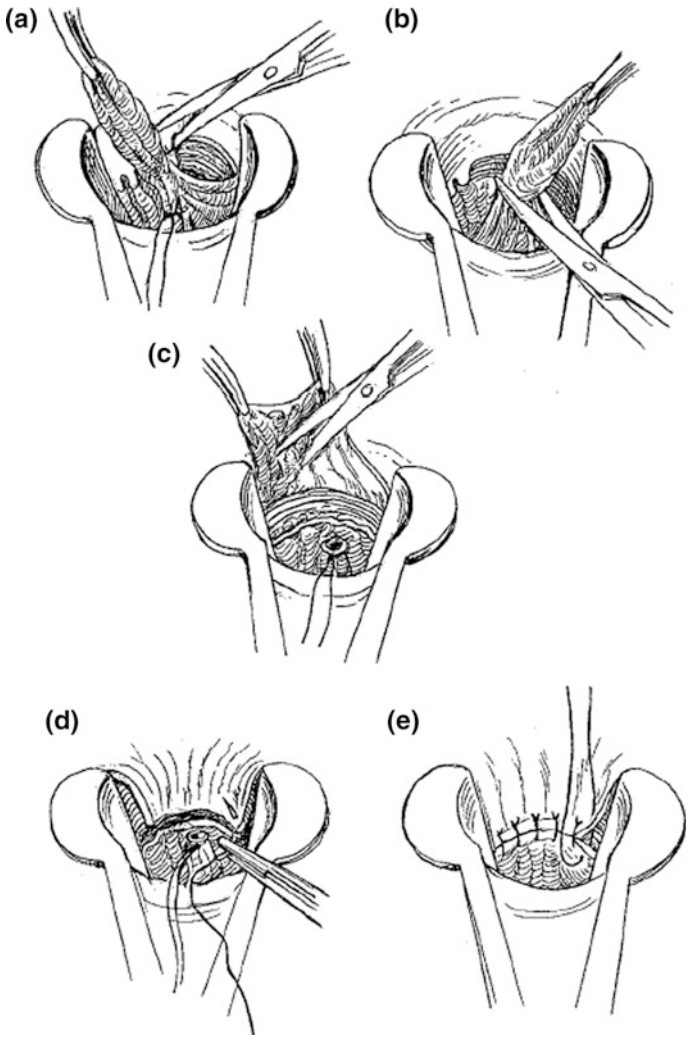


Fig. 4.3 Whitehead hemorrhoidectomy. **a** Suture placed through proximal internal hemorrhoid for orientation. Excision started at dentate line and continued to proximal bundle. **b** Internal hemorrhoidal tissue excised above ligated bundle. **c** Vascular tissue excised from underside of elevated anoderm. **d** End of anoderm reapproximated with sutures to original location of dentate line. (E) Completed procedure

with 1:200,000 of epinephrine. The intravenous sedation consisted of propofol. There was a 30–50% cost savings over the other anesthetic techniques in the ambulatory setting. In addition to the increased cost savings with this technique, there was also no difference in postoperative side effects and unanticipated hospitalizations when utilizing local perianal infiltration. The need for pain medication was also less in comparison to general anesthesia. An anal block is performed by

infiltrating circumferentially either in a field block subcutaneously and submucosally or by infiltrating into the intersphincteric groove in a four- or eight-quadrant manner. Additionally, local infiltration to the proposed site of excision is needed in order to ensure adequate anesthesia during the case. Since pain is the most common reason for delaying discharge after ambulatory surgery, significant attention must be given to good analgesia for effective postsurgical pain management. Most of these agents are short acting however, and can block noxious stimuli and pain intensity for at most 8–12 h after which the patient must take other analgesics to control pain.

Liposomal Bupivacaine

Approved by the FDA in 2011, Exparel® is a liposome injection of the amide local anesthetic bupivacaine encapsulated in a proprietary DepoFoam® delivery technology that is infiltrated into the local site for postoperative analgesia. The DepoFoam® consists of multivesicular liposomes that encapsulate the bupivacaine and release it over an approximately 96-h period. These properties are beneficial in prolonging the time to first narcotic use and in decreasing overall narcotic use. The drug is infiltrated locally at the end of surgery. Gorfine et al. [5] published a randomized multicenter, double-blind placebo controlled trial in 189 adults undergoing excisional hemorrhoidectomy (2- or 3-column). Those patients who received a bupivacaine liposome injectable suspension had a 30% statistically significant reduction in pain scores at 72 h. In addition, this led to a significant reduction in opioid consumption. Haas et al. reported a randomized clinical trial using liposomal bupivacaine for post-hemorrhoidectomy pain management compared to standard bupivacaine HCL [6]. They found that the liposomal bupivacaine significantly reduced postsurgical pain and opioid consumption in comparison to bupivacaine HCL. This in turn led to decreased opioid-related adverse events.

Of note, the injection of bupivacaine liposomal injectable suspension should not be admixed with lidocaine or other non-bupivacaine-based local anesthetics, which can lead to the immediate release of bupivacaine from the suspension.

Catheter Delivery Systems

These pumps were developed to provide a continuous infusion of nonnarcotic pain relief in the form of local anesthetics directly to or near the surgical site through specially designed catheters. This delivery method can provide patients with days of targeted pain relief after surgery thereby minimizing narcotic usage and the side effects that go along with increased narcotic usage such as nausea, emesis, constipation, and over sedation. In theory, the use of catheter delivery systems, which provide a continuous administration of local anesthetics to the site of surgery through a locally placed catheter, seems intuitive. However, this has been significantly limited by difficulty in maintaining correct catheter position, the cost of the

delivery device, and the resources needed to manage patients in the outpatient setting. These catheter delivery systems (STA cath®, On-Q®) are not used widely for excisional hemorrhoidectomy for the reasons listed above.

NSAIDS and Cox-2 Inhibitors

Nonsteroidal anti-inflammatory drugs (NSAIDS) are peripherally acting analgesics utilized worldwide. These drugs provide pain relief and an alternative to opioid-based analgesia. They play a key role in the multimodal approach to pain relief in the perioperative setting providing analgesic, anti-inflammatory, and antipyretic benefits. The use of NSAIDS in the perioperative period has been shown to provide improved analgesia, lower rates of urinary retention, and decreased narcotic usage [7–9]. Additionally, it is not associated with excessive sedation, respiratory depression, or cognitive dysfunction. This drug is indicated for the management of moderate to moderately severe postsurgical pain. NSAIDS can be delivered orally, transdermally, intramuscularly, intravenously, and through direct local infiltration at the surgical site. Ketoralac tromethamine was the first injectable NSAID approved for use in the USA. The combined duration of oral, intramuscular, and intravenous administration should not exceed 5 days. When utilized in a multimodal fashion a 30 mg loading dose followed by 15–30 mg doses every 6 h can be a potent adjunct and opioid sparing modality to control postoperative pain. However, analgesic effects must be balanced and weighed against the potential for adverse effects especially GI bleeding, platelet dysfunction, and renal failure [7, 8].

Another injectable NSAID formulation approved in the USA in 2006 was injectable ibuprofen (Caldolor®). This medication can be used to treat mild to moderate pain by itself or as an adjunct to opioid analgesics (Ibuprofen Injection (Caldolor®). Nashville, TN: Cumberland Pharmaceuticals, Inc. [10]. Recommended dosing for ibuprofen injection are 800 mg every 6 h with a maximum dose of 3200 mg over a 24 h period. Patients weighing less than 50 kg and elderly patients may achieve effective analgesia with 400 mg doses. Its lower selectivity for Cox-1 isoenzymes in comparison to Ketoralac may reduce the risk of adverse side effects such as GI bleeding or platelet dysfunction. Unlike Ketoralac, this drug should be diluted with 250 mL of sterile saline or lactated Ringer's solution and infused slowly over 7–15 min in order to achieve maximal plasma concentrations more rapidly and at the site of tissue injury.

Cox-2 inhibitors were developed to improve GI safety while providing effective analgesia. Approved by the FDA in 1998, the only Cox-2 inhibitor available for perioperative pain management is celecoxib (Celebrex®). This subclass of NSAIDS is more selective for the Cox-2 isoenzyme, which is induced following tissue injury. Unfortunately, other Cox-2 inhibitors were withdrawn from the US market and the FDA mandated a black box warning for celecoxib with respect to its risks regarding cardiovascular and cerebrovascular thrombosis with long-term use.

Acetaminophen

This is a centrally acting analgesic for mild to moderate acute as well as chronic pain that is one of the most widely administered over the counter analgesics. Since it does not act peripherally it has no anti-inflammatory effects locally at the site of surgery. Modes of administration include oral, rectal, and intravenous. It has been shown to significantly reduce postsurgical pain versus placebo [11]. Additionally, acetaminophen lacks the adverse side effects of NSAIDS and opioids. The intravenous formulation (Ofirmev®) gained FDA approval in the United States in 2010. In addition to its antipyretic benefits, it is indicated for the treatment of mild to moderate pain by itself or as an adjunct to opioid analgesics in the treatment of moderate to severe pain. The touted benefits over the oral or rectal formulations are a higher analgesic efficacy, a higher maximum plasma concentration, and a more rapid onset of action. The medication comes in a 1000 mg solution that should be infused over a 15-min period every 4–6 h not to exceed 4 g/day in adults less than 70 years of age. Dosing should be adjusted for children, adolescents, and the elderly. There are some data to suggest that the analgesic effectiveness of intravenous acetaminophen is enhanced when administered prior to making the surgical incision although this was in patients undergoing abdominal hysterectomy and not excisional hemorrhoidectomy [12]. Because of acetaminophen's narrow therapeutic window great care should be taken to adhere to recommended dosing by the manufacturer in order to minimize potential hepatotoxicity. Additionally, acetaminophen should not be used in patients with severe hepatic impairment or severe active liver disease.

Metronidazole

Orally administered metronidazole has been shown to improve postoperative pain after excisional hemorrhoidectomy [13]. Published results, however, have been somewhat variable and can either support or not support the use of metronidazole [14, 15].

Glyceryl-Tri-Nitrate (GTN)

Nitroglycerin ointment has been studied looking at its analgesic efficacy, its effect on wound healing, and its adverse effects with respect to headache. Glyceryl trinitrate has been shown to decrease muscle spasm and increase anodermal blood flow. A meta-analysis looking at 333 patients from 5 randomized trials demonstrated analgesic efficacy on days three and seven compared to placebo [16]. Additionally, wound healing was reported to be better at 3 weeks compared to placebo and the side effect of headache was not statistically increased over placebo.

Another meta-analysis of 12 randomized controlled trials by Liu and colleagues looked at a total of 1095 patients. That meta-analysis found that there was a significant pain reduction on days 1, 3, 7, and 14. There also seemed to be a benefit with respect to improved wound healing three weeks postoperatively but this came at a cost of increased headache [17].

Joshi and Neugebauer reported a study on behalf of the PROSPECT Collaboration working group evaluating the available literature on the management of pain after hemorrhoid surgery [18]. The collaborative group was formulated to provide evidence-based recommendations for specific surgical procedures. Of 207 randomized studies identified, only 106 met inclusion criteria and of these 41 were excluded leaving a total of 65 studies for evaluation. Although quantitative analyses were not performed, the conclusion was that local anesthetic infiltration either as a sole technique or in conjunction with a multimodal approach to pain (NSAIDS, Acetaminophen, Opiates) is recommended in the management of pain after hemorrhoidectomy. The following algorithm can be utilized in the management of postoperative pain after excisional hemorrhoidectomy (Fig. 4.4) (Pain management algorithm).

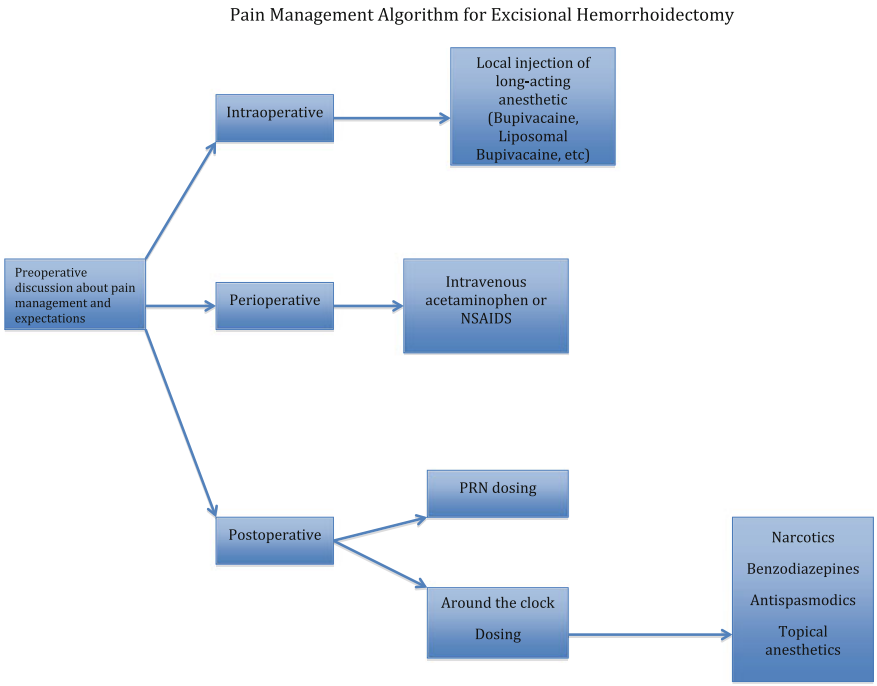


Fig. 4.4 Pain management algorithm for excisional hemorrhoidectomy

Urinary Retention

Postoperative urinary retention after excisional hemorrhoidectomy is the most frequent complication after pain. This is especially evident after multiple-quadrant excisions or the performance of other concomitant anorectal procedures performed at the time of excisional hemorrhoidectomy. A number of studies have shown that limiting perioperative fluids to less than 1000 mL can lower the incidence of urinary retention from approximately 20% to less than 10% [19, 20]. Bailey lowered the incidence of urinary catheterization from 14.9 to 3.5% with fluid restriction [19]. Additionally, multimodal analgesia has been shown to decrease urinary retention from 25 to 8% [21]. Toyonaga et al. found that female sex, presence of preoperative urinary symptoms, diabetes mellitus, need for postoperative analgesics, and more than three hemorrhoids resected were independent risk factors for urinary retention as assessed by multivariate analysis [21]. The most recent practice parameters published by the American Society of Colon and Rectal Surgeons gives a strong recommendation with level 1B evidence stating that "urinary retention after ambulatory surgery may be reduced by limiting perioperative fluid intake." [22] Since the vast majority of hemorrhoidectomies are performed on an ambulatory basis it is not very practical to mandate voiding prior to discharge. Patients are encouraged to minimize fluid intake until voiding. Warm sitz baths or warm showers the following day usually promote voiding.

Postoperative Hemorrhage

Massive hemorrhage after excisional hemorrhoidectomy requiring operative treatment occurs in less than 1–2% of patients. Hemorrhage occurs either early (immediate in PACU or within 48 h) or late (72 h or greater). Although it is usually not difficult to identify bleeding internally, it can be potentially masked if there is an anal pack in place and some surgeons advocate not placing an anal pack after hemorrhoidectomy for this reason.

Early Hemorrhage

Hemorrhage in the early postoperative period is almost always secondary to a technical issue likely from inadequate ligation of the internal hemorrhoid pedicle. Although bleeding from the external portion of the wound is unusual, it can occur and can potentially be managed at the bedside in the PACU. Bleeding from the external wound may be managed with simple injection of 1% lidocaine with 1:100,000 epinephrine. This may control the bleeding alone and at the very least allows the surgeon the ability to assess the outer wound carefully. Occasionally, suture ligation of a bleeding point on the external skin may be needed. If there is severe bleeding from the anal canal while the patient is in the post anesthesia care unit (PACU) or within 48 h they should be brought back to the operating room immediately. This will allow optimal visualization and management with suture ligation.

Late or Delayed Hemorrhage

Late or delayed hemorrhage can occur up to several weeks after excisional hemorrhoidectomy although the majority occurs within the first week. The delayed hemorrhage frequently requires admission to the hospital for observation and management. Delayed hemorrhage is usually due to bleeding from the sloughed hemorrhoidectomy wound where there is a granulating tissue base possibly with an exposed vessel. The incidence of late bleeding is similar in open Milligan–Morgan hemorrhoidectomies versus closed Ferguson hemorrhoidectomies [23]. Delayed hemorrhage can be managed through a variety of treatments. Once the patient is resuscitated in the emergency department, an assessment is made to determine the extent of bleeding through a thorough history and physical examination. If the patient is hemodynamically unstable despite resuscitation then they should be brought to the operating room immediately for an examination under anesthesia and possible suture ligation. If the patient is stable, then an attempt can be made to perform rectal tap water irrigation with a large three-way foley catheter. This usually does not require anesthetics or narcotics but can be used on a selective basis. If fresh blood is persistent throughout the rectal irrigation then the patient should be brought to the operating room for an examination under anesthesia. If the rectal irrigation becomes clear then the patient can be admitted for close observation. Chen et al. performed a prospective study comparing rectal irrigation with immediate examination under anesthesia. They found that rectal irrigation was well tolerated and bleeding stopped in 88% of patients. In comparison to surgery patients undergoing rectal irrigation had a higher satisfaction, lower length of stay and more cost effective treatment [24].

Another minimally invasive approach is proctoscopic or anoscopic inspection followed by injection of 1% lidocaine with 1:100,000 epinephrine. This may require the use of local anesthesia and narcotics for patient tolerance. Additionally, good lighting is essential for appropriate visualization [25].

Anal packing has been described with a variety of materials including Surgicel, and gelfoam soaked with thrombin or epinephrine. This usually requires anesthesia and narcotics for patient tolerance and may lead to other complications such urinary retention [26]. An alternative to packing is Foley catheter tamponade of the bleeding. Once the catheter is inserted the balloon is inflated with 20–40 mL of fluid and placed on gentle traction. The balloon can then be deflated and removed within 24 h. This technique can also be utilized as a temporizing procedure prior to going to the operating room if it is not immediately available [27, 28]. Lastly, in patients with significant ongoing bleeding, suture ligation in the operating room provides the best means for a thorough and painless examination so that suture ligation can be performed.

Infection

Although the rate of bacteremia has been reported to be as high as 8.5% following sigmoidoscopic examination, the incidence of local infectious complications and or systemic sepsis is surprisingly low following excisional hemorrhoidectomy. This has been attributed in part to the excellent blood supply of the anorectal region as

well as effective clearance of portal bacteremia by the reticuloendothelial system of the liver [29]. There is definitely a paucity of reported wound infections following hemorrhoidectomy. The reported rate of local infection following excisional hemorrhoidectomy in most cases is less than 1–2%. In a report by Bouchard et al. in over 600 patients undergoing hemorrhoidectomy the incidence of local infection was 1.4% [30]. Chen et al. reported 1 infection in 666 patients undergoing excisional hemorrhoidectomy with the LigaSure device [31]. Qarabaki et al. reported zero wound infections in a comparative study looking at 688 patients undergoing either circumferential excisional hemorrhoidectomy versus three-quadrant Ferguson hemorrhoidectomy [32].

Anal Stenosis

Anal stenosis following excisional hemorrhoidectomy is usually a preventable complication that results from excessive excision of perianal skin and or anoderm. Its incidence is typically less than 5% but has been reported as high as 10%. The best treatment for anal stenosis is prevention. If adequate skin bridges and anoderm are preserved during excisional hemorrhoidectomy, the risk of anal stenosis will be decreased. Excisional hemorrhoidectomy is best performed with a large Hill-Ferguson retractor in place during the entire procedure. If disease is circumferentially extensive then you are better off leaving enough skin/anoderm bridges in situ even though the patient may complain about some residual disease. This can be taken care of at a subsequent operation if needed once there is complete healing of the initial wounds. Although the surgeon has the option of performing more extensive excision with a concomitant anoplasty, it is my preference to leave sufficient skin bridges and anoderm in situ to avoid postoperative anal stenosis. The timeline for presentation of anal stenosis may be anywhere from weeks to several months after excisional hemorrhoidectomy [33]. Medical and or surgical treatment should be tailored to the severity of anal stenosis. Patients usually report painful or difficult bowel movements, rectal bleeding, and or narrow caliber stools. Visual inspection and attempted digital rectal examination usually establishes the diagnosis of anal stenosis. However, some patients may require examination under anesthesia in order to make an adequate assessment. If the etiology of the stenosis is unclear then the patient should undergo endoscopy to rule out malignancy and or inflammatory bowel disease. Crohn's disease must also be in the differential diagnosis. Stenoses can be classified as either mild, moderate or severe [34]. A mild stenosis is characterized by the ability to perform a digital rectal examination on the patient or to be able to insert a medium Hill-Ferguson retractor into the anus without forceful dilatation. A moderate stenosis requires forceful dilatation in order to perform a digital rectal examination or to insert a medium Hill-Ferguson retractor. A severe stenosis is defined as one in which the 5th digit or a small Hill-Ferguson retractor can only be inserted with forceful dilatation. Stenoses can also be classified as to their level of involvement as low, middle, and high [35]. Low involves at least 0.5 cm distal to the dentate line, middle 0.5 cm distal and proximal to the dentate

line and high as involving 0.5 cm above the dentate line. Mild stenosis can frequently be treated with stool softeners or bulking agents [34]. Some patients may require daily self-digital dilation or mechanical dilation with dilators 2–3 times per day. Mechanical dilators can be quite costly and may be replaced with a well-lubricated tapered candle for significantly less money. The patient is instructed to bring the candle to the office and then observed in its use for dilation. The candle can also be marked to provide limits of insertion for the patient. These patients are best seen in the office on a weekly basis to assess progress and to perform dilation or digital rectal examination. Moderate stenosis should be treated initially with conservative management with fiber supplements and dilation. If adequate results are not obtained patients may benefit from incision of a constricting band and concomitant lateral internal sphincterotomy. The lateral internal sphincterotomy should be performed in an open fashion in order to incise the scarred anoderm at the same time. The sphincterotomy wound should be left open to heal by secondary intention and then the patient placed on bulk forming fiber immediately after surgery. Some patients may require more than one sphincterotomy in order to allow appropriate dilation. In Milsom's series of 212 patients greater than 50% were treated with a sphincterotomy [34]. More severe stenosis usually requires surgical intervention in the form of anoplasty. Figure 4.5 is a simplified algorithm for the treatment of anal stenosis post-hemorrhoidectomy. Anoplasty essentially treats the

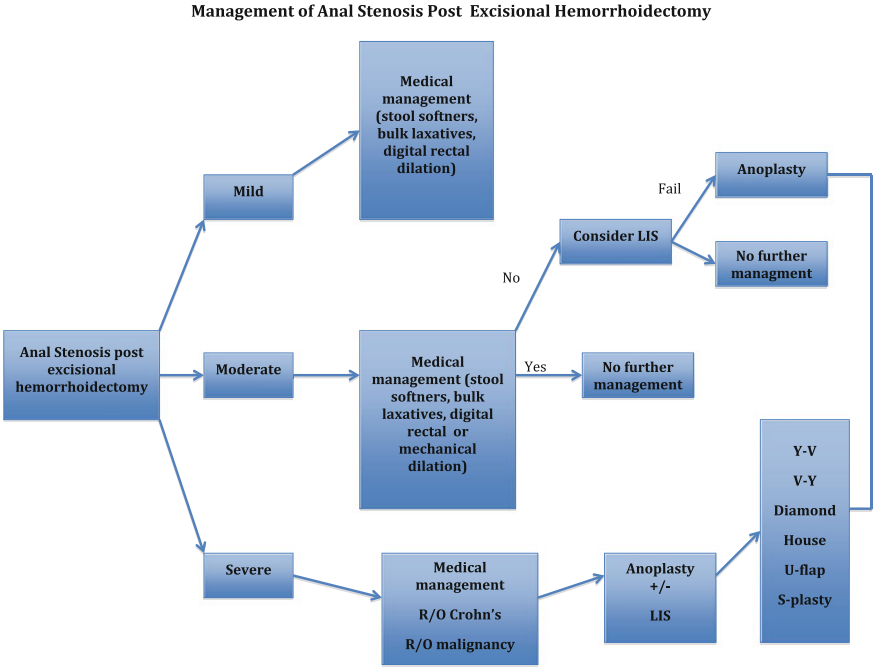


Fig. 4.5 Management of anal stenosis post excisional hemorrhoidectomy

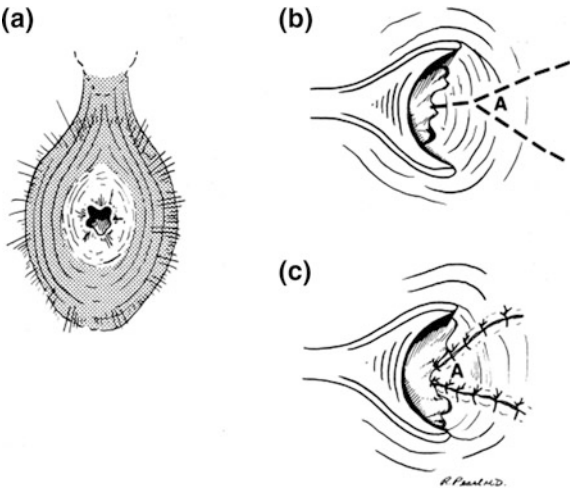


Fig. 4.6 Y-V Anoplasty. **a** Anal canal with stenosis. **b**. Line of incision for Y-V anoplasty. Note that the base of the incision from superior to inferior (i.e., the distance between the arms of the Y) should be equal or greater to the length of the Y. **c** Completed Y-V anoplasty with all wounds closed. Adapted from Blumetti and Abcarian [36]

loss of anoderm that resulted from the excisional hemorrhoidectomy. A variety of advancement flaps have been developed in order to deliver new healthy, pliable tissue that replaces the scarred and missing anal canal tissue. Occasionally, more than one flap may be needed in order to correct the deformity. In common to virtually almost all flaps for the correction of anal stenosis secondary to excisional hemorrhoidectomy is that they should be based laterally in either the left or right lateral positions away from the midline where there is more tension and where healing can be impaired. Although not all-inclusive the following is a list of the more common anoplasty procedures utilized for treating severe anal stenosis secondary to excisional hemorrhoidectomy (Figs. 4.6, 4.7, 4.8 and 4.9).

Patients usually undergo full mechanical bowel preparation as well as intravenous antibiotics preoperatively. In order to facilitate sphincter muscle relaxation, patients should have either regional or general anesthesia. Infiltration with local anesthetics is also utilized in order to help with postoperative pain. Flaps are created in full thickness with its underlying adipose tissue that includes its blood supply. Care needs to be taken not to undermine the flap in order to prevent ischemia. Of the flaps listed all include closure of the primary donor site except the U-flap that leaves the donor site partially open to heal by secondary intention [37].

In a 1-year follow-up in 488 patients, Bouchard et al. reported a 4.7% incidence of anal stenosis [30]. Nienhuijs and de Hingh performed a Cochrane review looking at conventional versus LigaSure hemorrhoidectomy [38]. Twelve randomized controlled studies with 1142 patients met the inclusion criteria. Data for anal stenosis was reported in only 931 patients. Of the 931 patients undergoing either LigaSure or conventional hemorrhoidectomy, the reported incidence of anal stenosis was 0.86% (8/931).

In a literature review by Brisinda et al. they reported an overall healing rate ranging from 60 to 100% in 29 reports with a total of more than 700 patients. In 26

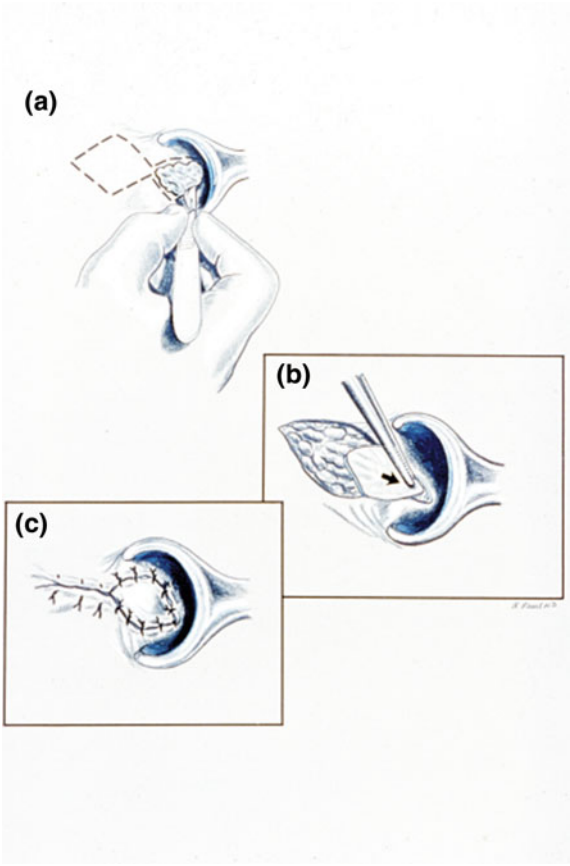


Fig. 4.7 Diamond flap anoplasty. **a** Line of incision for diamond flap anoplasty. The leading edge of the flap should be the same size as the defect in the anal canal. **b** The fully mobilized flap is brought into the wound. Adapted from Blumetti and Abcarian [36]

of the 29 reports the healing rate was greater than or equal to 90% [39]. Of note however, some of these anoplasty procedures were not performed for anal stenosis and some patients underwent bilateral anoplasties. Unfortunately, it is quite difficult to compare results of anoplasty procedures as there are no prospective randomized trials available. Nonetheless, results have been reported to be successful in the majority of patients.

Mucosal Ectropion

This condition may arise occasionally when the mucosa is incorrectly sutured distal to the dentate line. The defect results in mucosa visible at the level of the anal verge. This can lead to a "wet anus" secondary to mucous discharge where the patient complains of persistent moisture and irritation in the anal region. This complication

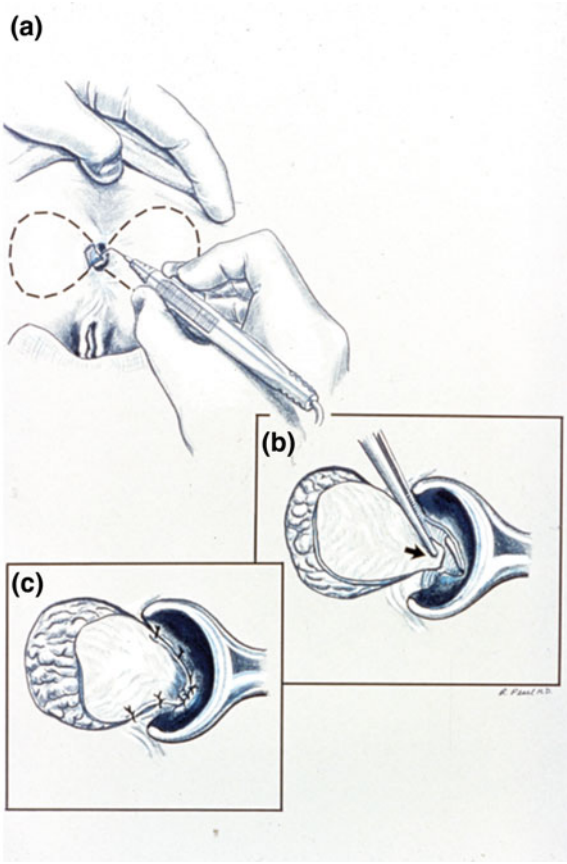


Fig. 4.8 U-Flap anoplasty. **a** Outline of incision for bilateral U-flap anoplasty. **b** The fully mobilized flap is brought into the wound. **c** The flap sutured in place. Note the lateral donor site is *left* open to heal by secondary intention. Adapted from Blumetti and Abcarian [36]

more often occurs after an improperly performed Whitehead hemorrhoidectomy [40] (Fig. 4.3). If there is no associated anal stenosis and the ectropion is confined to a small quadrant of the anus then local excision can be performed. After excising the ectropion, the rectal mucosa is sutured to the internal sphincter in a transverse fashion at the level of the dentate line and the skin is allowed to heal by secondary intention. If anal stenosis is present or if the mucosal ectropion is extensive or a Whitehead deformity exists then an anoplastic procedure is indicated.

Incontinence

Patients being considered for hemorrhoidectomy must undergo a thorough history that includes episodes of fecal soiling, gross incontinence, or incontinence to flatus. This is particularly important in the elderly patient who may have impaired continence. This may be particularly difficult to sort out when minor fecal soiling or

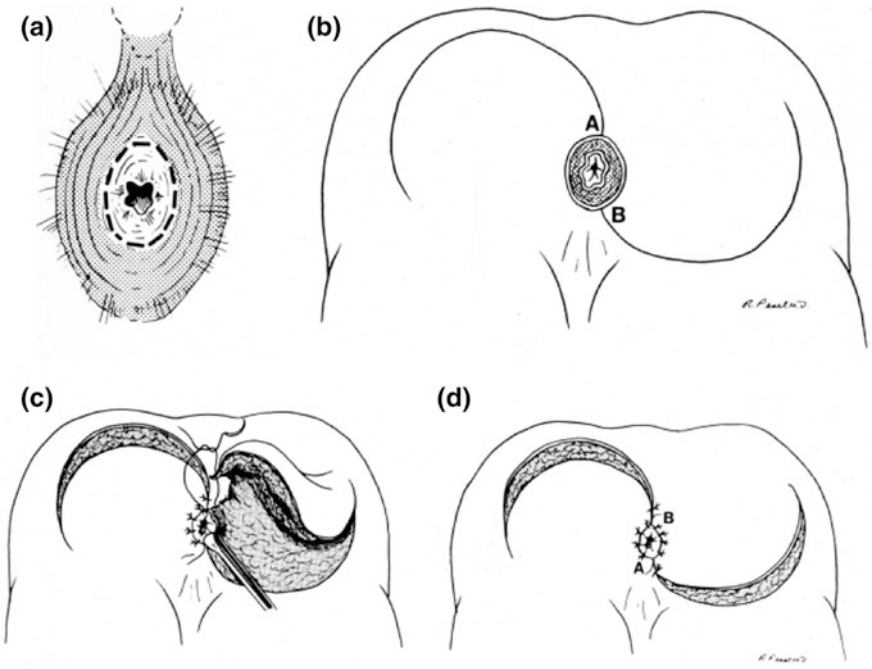


Fig. 4.9 S-Plasty. **a** Line of excision of stenosis and ectropion. **b** Line of incisions for S-Plasty. The distance from A to the *left* lateral edge is the base of the superior flap. Note that this distance is longer than the height of the flap from superior to inferior. **c** Mobilization of the inferior flap is demonstrated. The superior flap has already been completed **d** final appearance after completion. Note that the tip of the superior flap (**a**) has been rotated and sutured to the inferior aspect of the wound, and the tip of the inferior flap (**b**) now lies at the superior aspect. The donor sites are *left* open, but may also be closed primarily. Adapted from Blumetti and Abcarian [36]

incontinence is present in patients complaining of mucosal prolapse. Although the presence of new postoperative incontinence is rare, it is not unusual for patients to experience temporary difficulty controlling flatus for several weeks postoperatively [41]. Whether this is secondary to removal of internal hemorrhoidal cushions that contribute to flatus continence versus removal of transitional zone tissue that contributes to sensation is unclear. This problem may be of particular concern in women. Anal incontinence after hemorrhoidectomy has been reported in up to 12% of patients [42]. Additionally, concomitant internal sphincterotomy must be avoided in patients with disturbed continence. It has been reported that anal retractors can also disturb continence in patients after anorectal surgery [43, 44]. As in many complications prevention is the best treatment. One should avoid excisional hemorrhoidectomy in patients with disturbed continence. In addition to appropriate patient selection, careful use of the anal retractor and avoidance of concomitant internal sphincterotomy will minimize this postoperative complication.

Constipation

Although constipation is not uncommon after excisional hemorrhoidectomy, it is best prevented by placing the patient on an appropriate bowel management program postoperatively. Patients should be instructed on the use of bulk fiber laxatives immediately postoperatively as well as stimulant laxatives should they not have a proper bowel movement by postoperative day three. Fecal impaction occurring in approximately 1–3% of patients should be avoided at all costs as this will typically require a trip to the operating room for correction [30].

Bulk laxatives have been shown to decrease pain with bowel movements as well as lead to decreased soiling [45].

Complications of Stapled Hemorrhoidopexy

Ariane M. Abcarian and Herand Abcarian

Stapled hemorrhoidopexy or PPH (Procedure of Prolapse and Hemorrhoids) was originally designed by Antonio Longo in 1995 and reported as a new procedure to the 6th World Congress of Endoscopic Surgery in Rome, Italy in 1998 [46]. In this novel concept and operation, Longo proposed that excision of all hemorrhoidal tissue practiced in Europe (Milligan–Morgan procedure) or in the US (Ferguson procedure) is unnecessary, and all that is needed is elevation and fixation of the prolapsing hemorrhoid and rectal mucosa to the rectal wall at the level of the anorectal ring. This could be accomplished by a circular stapler which he designed and Ethicon Endosurgery (Cincinnati, Ohio) produced and marketed [46]. In essence, this was the ultimate extension of all "Nonoperative treatments" of hemorrhoids, e.g., injection sclerotherapy, rubber band ligation, infrared coagulation, etc., which succeeded in alleviating hemorrhoidal symptoms of prolapse and bleeding by promoting fixation of the tissue to the anorectal wall. It is important to note at the onset that many European surgeons continued to use the term "stapled hemorrhoidectomy" instead of hemorrhoidopexy.

This operation gained rapid popularity in Europe especially in Italy and Germany due to the simple nature of the procedure and minimal pain experienced by the patient due to avoidance of incising the anoderm and placement of the staple line well (2–3 cm) above the dentate line. As any new procedure, its rapid expansion in the hands of surgeons, many of whom were noncolorectal specialists, led to a series of complications some new and others common to all previous hemorrhoidectomies. Ultimately, an international working party was assembled in France with representation from many countries (including the US) to establish guidelines for the use of this new instrument and procedure recommending lectures, videos, application in animal models, etc., leading to a formal credentialing by each practitioner's surgical department [47].

The early experience with stapled hemorrhoidopexy in the US was quite favorable and the results of this procedure performed at the University of Illinois at Chicago and Washington University in St. Louis were reported by Singer and colleagues [48]. Since then numerous publications have confirmed significant reduction in postoperative pain, early resumption of normal activity and greater patient satisfaction [48, 49]. Randomized controlled trials comparing "stapled hemorrhoidectomy" with other conventional techniques all favored the former in regards to postoperative pain and patient satisfaction [50–53]. With further experience, long-term results of stapled hemorrhoidectomy confirmed the earlier favorable results [54, 55].

In an attempt to validate the results of the European studies, a prospective randomized controlled multicenter trial comparing stapled hemorrhoidopexy and Ferguson hemorrhoidectomy was carried out in the United States [56]. Perioperative and 1-year results confirmed the advantage of hemorrhoidopexy in decreasing postoperative pain, time off work and similar if not better results at the end of 1-year follow-up [56]. Among the detailed data collected were the postoperative complications which are summarized in Table 4.1 under Adverse Events (AE).

Table 4.1 Adverse events (AE)

AE	PPH	Ferguson	P value
Pts ≥ 1AE	27 (35.1%)	32 (40.5%)	0.367
Return to OR due to AE	0	6 (7.6%)	0.007
Urinary retention	9 (11.7%)	6 (7.6%)	0.382
Constipation	4 (5.2%)	10 (12.7%)	0.102
Postoperative hemorrhage	7 (9.1%)	4 (5.2%)	0.193
Micturition disorder (dysuria)	2 (2.6%)	6 (7.6%)	0.154
Temporary fecal incontinence	3 (3.9%)	4 (5.2%)	0.667
Wound complications	0	6 (7.6%)	0.103
Perianal Itching	3 (3.9%)	2 (2.5%)	0.610
Emesis	2 (2.5%)	2 (2.5%)	0
Fever	0	4 (5.2%)	
Anal fissure	0	2 (2.5%)	
Anal stricture	2 (2.5%)	0	
Fistula-in-ano	0	2 (2.5%)	
Pruritus	0	2 (2.5%)	
Rectal Pain	2 (2.5%)	0	
Abscess (perianal)	0	1 (1.3%)	
Abdominal distention	0	1 (1.3%)	
Chills	1 (1.3%)	0	
Perianal burning	1 (1.3%)	0	
Perianal inflammation	1 (1.3%)	0	
Postoperative wound infection	0	1 (1.3%)	
Sexual dysfunction	0	1 (1.3%)	
Temporary flatus incontinence	0	1 (1.3%)	

The authors concluded that: (a) PPH and Ferguson have similar safety profile, (b) PPH results in less postoperative pain, a faster recovery and fewer patients requiring analgesics and (c) PPH and Ferguson had similar rate of control of hemorrhoidal symptoms. Also, PPH patients required fewer additional anorectal procedures within the first postoperative year [56].

The complications of stapled hemorrhoidopexy and their management will be grouped as best as possible and discussed under the following headings.

Pain

1. *Usual postoperative pain* is best managed with nonnarcotic analgesics to prevent added side effects of opioid-related constipation. Narcotics are rarely prescribed in Europe for postoperative pain. Administration of 1000 mg intravenous acetaminophen in the operating room is a valuable adjunct for postoperative pain control. Warm sitz baths 10–15 min t.i.d or q.i.d is very helpful. Compared with Ferguson hemorrhoidectomy, pain after stapled hemorrhoidopexy is much less in severity, lasts for shorter period, and requires lower doses of analgesics [56]. "Persistent pain" or fecal urgency of unknown etiology has been reported [57]. If severe pain persists beyond 3–4 weeks an examination under anesthesia is advisable.
2. *Pelvic Floor Spasm* May cause intense deep burning pain in the rectum which may radiate to the pubis. This is usually a brief postoperative episode, lasting no more than a few days and can be managed by addition of a nightly dose of striated muscle relaxant (e.g., 10 mg oral cyclobenzaprine) in addition to frequent sitz baths.
3. *Low staple line* It is essential that the purse string for stapled hemorrhoidopexy be placed 3–4 cm cephalad to the dentate line to prevent irritation of the somatic sensory nerves which stop at the dentate line but may diffuse at least 5–6 mm cephalad (analogous preventing pain during rubber band ligation of hemorrhoids). Generally, if the staple line can be visualized with simple eversion of the buttocks, it is safe to conclude that it is too low, abutting the dentate line.
4. *Chronic pain* is poorly understood but could be related to low staple line. After 3–4 weeks, the patient may be returned to the operating room and if no other causes for pain can be identified, the surgeon may attempt to remove as many readily easily visible staples as possible with no risk of staple line dehiscence or bleeding. If an anal stenosis is found, it should be managed appropriately with simple dilation, division of scar, or even partial lateral internal sphincterotomy.
5. *Anal Fissure* may occur after dilation of the anal canal for insertion of the circular anal dilator (CAD). Manual dilation of the anus should be avoided. The lubricated obturator of the CAD should be inserted 2–3 times to allow safe placement of CAD. If the patients complain of typical postcibal pain of anal fissure, a simple eversion of the buttocks without digital examination will allow

visualization of the fissure. This can be managed with topical application of nitroglycerine (NTG) or calcium channel blockers (CCB), but may require lateral internal sphincterotomy if the fissure does not respond to topical medications.

6. *Thrombosed External Hemorrhoids* may occur if the external hemorrhoids are large preoperatively and are not excised during the procedure. Prolonged "conservative" treatment with analgesics, anti-inflammatory, steroid topical analgesic creams should be condemned. Excision of the hemorrhoid with its thrombosis under local anesthesia provides immediate relief.
7. *Perianal Abscess/Fistula* is rarely seen after stapled hemorrhoidopexy due to avoidance of incision through the anoderm. If a deep (intraanal) abscess is suspected (pain, fever, swelling, tenesmus), an urgent examination under anesthesia will allow prompt diagnosis and treatment (drainage).
8. *Perianal Burning, Itching, and Irritation* are all mild forms of pain expressed as troublesome symptoms. Proper hygiene, mild steroid creams, and maintaining dryness will help resolve the symptoms quickly.

Infectious Complications

1. *Bacteremia* may occur after all rectal operations. This is usually inconsequential and causes temporary low grade fever and chills lasting less than 24 h [58]. The incidence is low and conservative treatment with sitz baths and antipyretics suffice [56–58]. It is unclear whether preoperative use of antibiotic prophylaxis decreases the incidence of bacteremia due to rarity of the condition and lack of evidence-based data or prospective randomized trails.
2. *Retroperitoneal Sepsis* Secondary to breakdown of the staple line has been reported. Seow-Choen and colleagues published in a case report and review of literature [59]. Other reports of septic complications resulting from hemorrhoidectomy has been reported [60, 61]. If the patient develops fever, leukocytosis, and severe pain or dysuria, immediate workup including CT scan should be initiated. Due to the potential lethal nature of this complication, emergency surgery, external drainage of sepsis, and fecal diversion must be undertaken without delay. After the patient recovers a careful endoscopy and contrast enema will guide the surgeon toward appropriate surgical intervention addressing the abscess cavity, stricture and ultimately utilizing coloanal anastomosis if indicated.
3. *Anorectal Abscess fistula* has already been addressed (vide supra)

Genitourinary Complication

1. *Dysuria* after stapled hemorrhoidopexy is more often seen in male patients. Although the etiology is not clearly understood, it may be related to pelvic floor spasm and or reflex bladder neck contraction secondary to postoperative pain. Empiric use of cyclobenzaprine, 10 mg and tamsulosin HcL (Flomax®) 0.4 mg qhs for 3–4 nights after surgery is greatly helpful.
2. *Urinary Retention* Urinary retention is the most common postoperative complication after any type of hemorrhoidectomy. Overzealous intravenous fluid administration during and immediately after surgery especially when regional anesthesia is used is most often at fault. In an old study of 610 patients with anorectal surgery, hemorrhoidectomy stood out among all other operations for benign anorectal disease as a risk factor or urinary retention [20]. In the study by Senagore and colleagues, the incidence of urinary retention after stapled hemorrhoidopexy was 11.7% versus 7.6% for Ferguson hemorrhoidectomy ($p = 0.382$ ns) [56]. Bladder decompression, fluid restriction, warm sitz baths, and use of tamsulosin HcL (Flomax®) 0.4 mg daily help prevent need for repeat catherization.
3. *Sexual Dysfunction* Temporary impotence in men almost always resolves in time. Occasional dyspareunia has been reported by women. The very close proximity of the staple line to the posterior vaginal wall is a possible etiologic factor. I have personal experience with a case of small hematoma in the rectovaginal septum which caused deep vaginal pain and dyspareunia. One month after surgery, the hematoma began draining spontaneously through a minute defect in the staple line. Under local anesthesia, the opening was enlarged with a hemostat resulting in further drainage of "old blood" and resolution of symptoms in 2 weeks.
4. *Penile Laceration* in sexual partner of an individual after stapled hemorrhodiopexy has been reported [62]. It is safe to say that a careful history especially in men, should alert the surgeon to counsel the patient against opting for stapled hemorrhoidopexy for the treatment of symptomatic hemorrhoidal disease. Women can be counseled to avoid anal receptive intercourse for 6–12 moths until all staples are extruded.

Defecatory Complications

1. *Constipation and fecal urgency* constipation is related to dehydration, decreased physical activity, dietary change, and most often injudicious use of narcotic analgesics. This was reported in 5.2% of patients with stapled hemorrhoidopexy

and 12.7% of the patients following Ferguson hemorrhoidectomy [56]. Patients should be placed on high fiber diet, increased oral fluid/water intake and stool softeners. Patients with history of chronic constipation should be started on this regimen plus daily dose of polyethylene glycol (PEG) powder for 1–2 weeks before surgery. Fecal urgency and sense of incomplete evacuation is related to the inverting type of staple line which does functionally somewhat narrow the anorectal outlet. With proper bowel management and reassurance, the urgency abates gradually and resolves in 3–4 weeks postoperatively.

2. *Fecal Impaction* after stapled hemorrhoidopexy is usually related to overuse of narcotic analgesic. After 5–6 days of "constipation," patients have tenesmus and pass liquid stool which many mistake for diarrhea. Using Loperamide or other constipating agents at this stage greatly aggravate the condition. Even though this is rare, patients with postoperative fecal impaction should be disimpacted under anesthesia or deep sedation followed by use of PEG or lactulose laxatives.
3. *Fecal Incontinence* Temporary fecal incontinence was reported in 3.9% of patients following stapled hemorrhoidopexy and 5.2% of the patients after Ferguson hemorrhoidectomy [56]. It is more commonly seen in elderly patients who do not tolerate anal stretch for any rectal surgery. In general the fecal incontinence is temporary and resolves in 2–4 weeks. If the patient complains of prolonged periods of fecal incontinence, EAUS can be useful to pinpoint a sphincter injury, even though this could have been present for years prior to the operation in an occult asymptomatic state. If a sphincter defect is found, a course of biofeedback should be recommended, and if this fails, an overlapping sphincter repair should be attempted.
4. *Rectal Obstruction* manifesting a severe constipation or obstipation has been reported [63]. Workup for rectal obstruction must include an early return to the operating room for examination and endoscopy under anesthesia. The obstruction may be amenable to local dilation, irrigation, and placement of a mushroom catheter for subsequent irrigation. However, if local therapy is unsuccessful, diverting colostomy should be performed to get the patient over the acute obstruction and allow subsequent workup and elective procedure for restoration of continuity with or without proctectomy.
5. *Rectal Stricture* Low staple line contributes to painful and difficult defecation and may result in anal stenosis. Pescatori reported post anopexy (stapled hemorrhoidopexy) rectal stricture and discussed its management [64].
6. *Obstructed Defecation Syndrome (ODS)* Dowden and colleagues reported on four cases of obstruction defecation disorder after stapled hemorhoidopexy [65]. ODS is difficult to manage anyway and postoperative ODS leaves the patient with a significant functional and psychological problems. Biofeedback, pelvic relaxation exercises and physical therapy may be of help. There is no recommended surgical procedure for this complication.

Bleeding

1. *Postoperative bleeding* using the 1st generation of staples (PPH33-01)® it was not unusual to see bleeding points at the staple line at the conclusion of the stapling procedure. This was easily controlled with 3/0 absorbable sutures placed across the staple line at the bleeding point. Postoperative bleeding (in recovery room, at home, the first 24–48 h) is considered as a technical error due to the same etiology (not diagnosed and treated). The height of the staples was shortened in subsequent productions (PPH33-03®) and this resulted in tighter staple lines and reduction of incidence of staple line bleeding [66]. In our CRS unit, if a patient with stapled hemorrhoidopexy must be restarted on anticoagulants postoperatively, the entire staple line is oversewn with a continuous running 3/0 absorbable suture.
2. *Submucosal Intramucosal Hematoma* causes fullness, tenesmus, and pain in the rectum. Most often the pressure from hematoma results in its partial decompression through the staple line and this can be easily visualized in the office or under sedation. Hematoma in rectovaginal septum results in pain and dyspareunia. The hematoma can be safely drained transrectally by removing a few staples and enlarging the opening gently with the tip of a hemostat.
3. *Rectal Laceration and Perforation* results from incorrect use of the stapler, excessive force in insertion of the anvil or opening and reclosing and firing the stapler. The perforation may be small and manifest as pelvic hematoma and peritonitis or could be overt and large with excessive bleeding and preumo-hemoperitoneum [67, 68]. These emergencies need immediate resuscitation, return to the operating room, transrectal or transpelvic control of bleeding, repair of laceration/perforation, and diverting sigmoid colostomy. Even though most colon and rectal surgeons prefer ileostomy for diversion, following colorectal trauma principles, a sigmoid colostomy is preferable due to the proximity to the injury and not leaving a long column of stool potentially decompressing into the pelvis. Hemoperitoneum is treated with thorough washout and closed external drainage, which can also be performed laparoscopically as in the cases of perforated sigmoid diverticulitis.

"Air Leaks"

Pneumoperitoneum, pneumoretroperitorum, and pneuomomediastinum have been reported in association with rectal perforation following stapled hemorrhoidopexy [69]. When rectal perforation is large, laparotomy and repair of leaks and colostomy are mandatory. However on occasion, air is seen in retroperitoneum and mediastinums without significant clinical signs and symptoms. After diagnosis is confirmed by CT, the patient may be placed on IV antibiotics, kept NPO and under

close observation. If fever, leukoytosis, abdominal or pelvic tenderness occurs timely intervention is indicated. The staple line should always be visualized under anesthesia and if possible a small defect may be amenable to transanal closure.

Rectovaginal Fistula

This is arguably the most dreaded complications of stapled hemorrhoidopexy. It can be prevented by closing the stapler mostly outside the anus before advancing it intraanally to complete the closure. After the stapler is closed, with a finger in the vagina palpating the posterior vaginal wall, the stapler should be rotated gently to the right and left axially. Once it is ascertained that the posterior vaginal wall is free, then the stapler is fired and removed. It is imperative that the surgeon inspects the doughnut of the resected tissue carefully. The rectal mucosa and the submucosa have a distinct pink and red appearance while the vaginal wall, in stark contrast, is whitish in color. If a piece of white tissue is seen in the hemorrhoidal specimen, the posterior vaginal wall must be carefully examined and visualized using good light source and retractors, including Lone Star®.

If despite all precautions, a small segment of vaginal wall is entrapped in the staple line, the staples causing the vaginal wall defect must be removed, separating the rectal and vaginal walls. After careful debridement, the vaginal wall is closed with interrupted 3/0 absorbable sutures. Then the defect in the circular staple line is closed with absorbable sutures as well.

In the unfortunate circumstances of undiagnosed vaginal wall entrapment where the patient returns to the surgeon or the emergency room with fecal discharge from the vagina 5–7 days later, the management is the same as low RVF following EEA for rectal cancer. The patient should be diverted allowing the infection to subside. Then in the operating room, the staples at the RVF site are carefully removed, both the vaginal and rectal wall defects debribed and closed separately with interrupted absorbable sutures. The diverting stoma is closed 6–12 weeks later after endoscopy and contrast enemas confirm successful closure of the RVF.

Staple Line Dehiscence

Is the result of faulty technique where the stapler handle has not been squeezed satisfactorily to fire and close the staples, but the knife has already cut the tissue. The result is a gap between the proximal rectal and distal anal mucosa with loose staples in the lumen and significant to massive hemorrhage. This is best handled immediately by grasping the rectal mucosa with noncrushing clamps, approximating to the distal mucosa with circumferential suturing with running 3/0 absorbable suture and if needed by a second layer of reinforcing running suture.

Although there are anecdotal reports of small bowel prolapse due to staple line dehiscence, this should never occur because unlike the EEA stapler, the PPH stapler is designed to resect only the mucosa and submucosa and not the full thickness of

the rectal wall. Erroneous placement of the staple line (too high) in women with a deep pouch of Douglas may predispose to this rare and unusual complication. Immediate laparotomy, reduction of the prolapsed small bowel, closure of the rectal defect or Hartmann's procedure with proximal end colostomy is mandatory.

The long litany of complications after stapled hemorrhoidopexy is significant for two reasons. First, there are many complications which did not exist during decades of Milligan–Morgan or Ferguson hemorhoidectomies. Second, it affirms clearly that the operation must be performed only by surgeons experienced with this technique and capable of dealing with potential complications. Review of the vast literature dealing with complications of stapled hemorrhodiopexy is a testament that majority of the complications have occurred in the hands of a less experienced surgeon during their "learning curve."

To avoid complications, the surgeon must pay attention to details, adhere to strict operative indications and technique, be familiar with all potential complications during and after surgery and whenever possible learn from others' mistakes. The famous quote of Danish surgeon Søren Laurberg from Arhus, "A fool with a tool is still a fool" is quite appropriate in stapled hemorrhoidopexy.

Complication of Sutured Hemorrhoidopexy

Sutured hemorrhoidopexy is an operative technique based on the principal of caudad sliding of hemorrhoidal cushions during defecation demonstrated by anatomic and radiographic studies [70, 71]. Microscopically, hemorrhoids are submucosal arteriovenous cushions in the anal canal which are suspended to the muscularis propria with the muscular and elastic fibers seen at the typical anatomic location of hemorrhoids [72, 73]. Gradual deterioration and degeneration of the suspensory muscles and elastic fibers allows for downward displacement of hemorrhoidal cushions resulting in protrusion. Bleeding is caused by rupture of the hemorrhoidal cushions or overlying mucosal ulceration due to hard stools [74]. Ultimately, 10% of the patients with symptomatic hemorrhoids will need surgical treatment [75].

The traditional hemorrhoidectomy, whether closed (Ferguson) or open (Milligan–Morgan) are based on excision of hemorrhoids and this includes all subsequent variations using banding, electrocautery, laser, freezing. In 1996, Morinaga and colleagues described a novel technique of ligation of hemorrhoidal arteries with the aid of Doppler flow meter and without actual excision of the arteries [76]. The stapled hemorrhoidopexy or PPH proposed by Longo essentially accomplished the same procedure, i.e., elevation and fixation of hemorrhoidal complex at the level of the anorectal ring without resection of hemorrhoidal cushions [77].

Stapled hemorrhoidopexy has one major advantage over excisional hemorrhoidectomy, i.e., significantly less postoperative pain, allowing for surgery to be performed on outpatient basis, reducing postoperative sick days, morbidity, and time off work. The results of stapled hemorrhoidopexy were compared with

Milligan–Morgan hemorrhoidectomy in the UK and in a randomized, controlled trial with long-term follow-up supporting the above-mentioned benefits [78]. A similar study in the US comparing early and late results of multicenter, post-operatively randomized, controlled trial of stapled hemorrhoidopexy with Ferguson hemorrhoidectomy validated similar results, i.e., less postoperative pain, early return to work, and equivalent short and long-term results [56].

Hemorrhoids as a disease afflicts patients in every country, among them many third world or lesser affluent countries, where hemorrhoidopexy staplers or trans-anal hemorrhoidal **dearterialization** devices are simply unaffordable. Therefore, attempts have been made to replicate the hemorrhoidal preserving, elevation, and fixation procedures using sutures and without the need for special costly devices. The early results of the reported case series have been encouraging and the complications have been quite low [79, 80].

One of the earliest reports of sutured hemorrhoidopexy was published by Pakravan in 2009 [80]. In this report, they presented a z stitch placed above the dentate line in multiple quadrants elevating and fixing the hemorrhoidal cushions without actual hemorrhoidectomy. A small mucosal window was removed in order to enhance fixation. Eighty-four percent of their patients (32/38) were free of

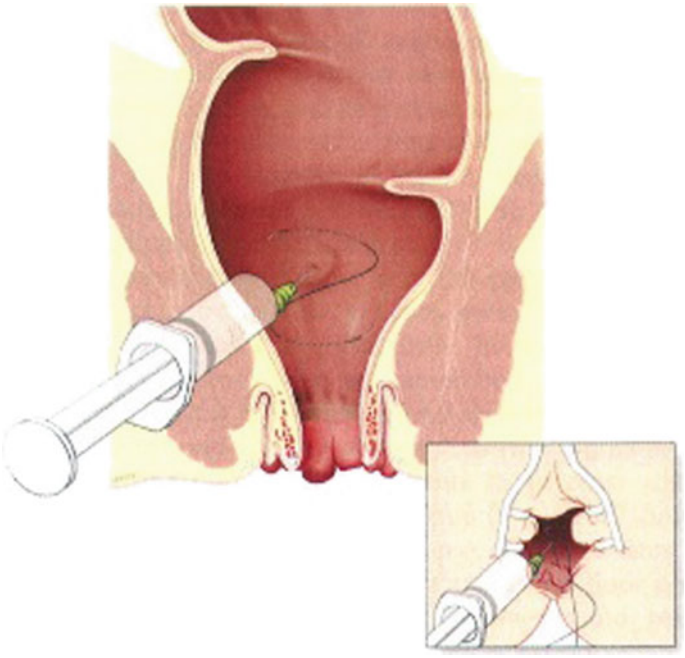


Fig. 4.10 Z-shaped suture approximately 4 cm above the dentate line. Submucosal injection of adrenaline solution (1:100,000)

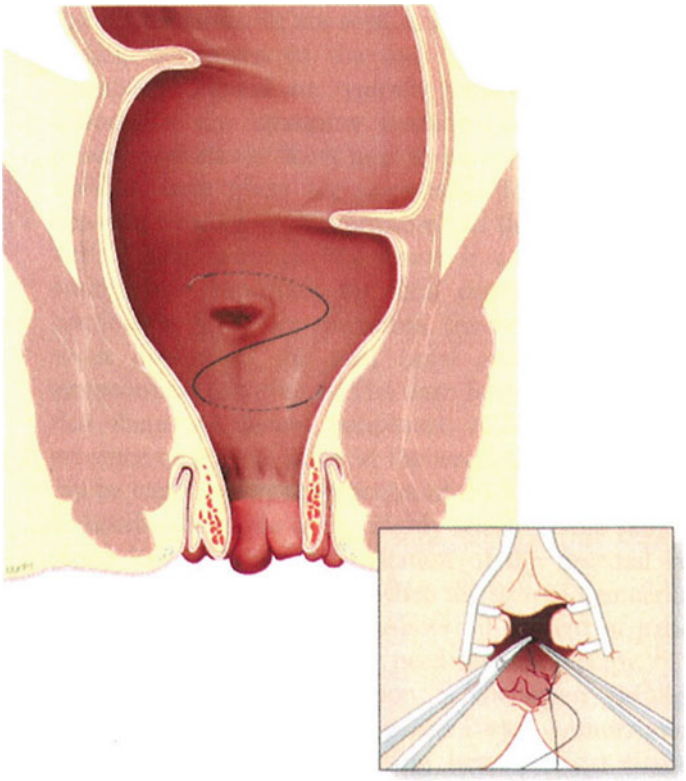


Fig. 4.11 Excision of 1 cm (*square mucosa*)

complaints in one week. Only six patients needed oral analgesics such as diclofenac for postoperative pain. In 6 months follow-up, 34/38 (89%) were asymptomatic, 2 (3%) had a minor segmental prolapse without need for intervention, and 2 (5%) had pruritus ani. The technique of sutured hemorrhoidopexy is depicted in Figs. 4.10, 4.11 and 4.12.

The authors concluded that "Transanal Open Hemorrhoidopexy" is simple, effective, and cost effective in comparison to other tissue sparing procedures [80]. Gemici and colleagues reported a larger series (116) of patients with a one-year follow-up [81]. A "vascular Z-shaped ligation technique" for treatment of hemorrhoids was utilized. Men comprised 65% of the patients and women 35%. The mean operative time was 12 ± 4.8 min. The Visual Analogous Score (VAS) at 3,7,21 days averaged 2.2, 1.8, and 1.2, respectively, during the same intervals. Acute bleeding 4.3%, infection 1.6%, urinary retention 6.9%, and recurrence 3.5% were reported [81]. No stenosis was seen in any patient [81].

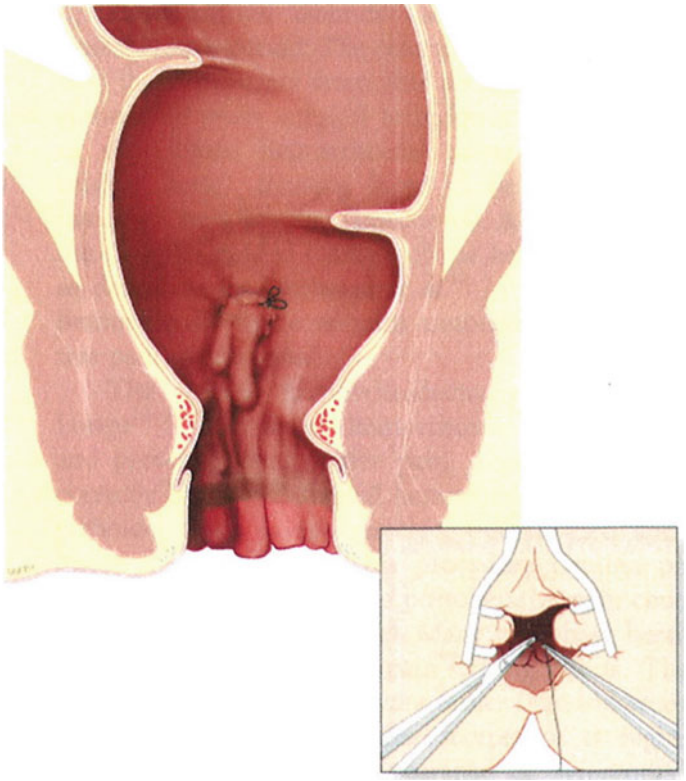


Fig. 4.12 Lifting of the hemorrhoidal tissue by tightening of the Z-shaped suture

Complications of Sutured Hemorrhoidopexy

There are analogous to hemorrhoidal ligation covered in another chapter in this book.

1. *Thrombosed hemorrhoids* occur in 1.9–4.3% of the patients [79–81].
2. *Urinary Retention* occur in 1.4% [79] and 6.9% [81] of the patients.
3. *Hemorrhage* Acute bleeding was seen in 4.3% of patients [81]. Secondary bleeding in 0.6% [79] and 3% [80] was minimal in nature and needing no intervention.
4. *Infection and Anal Stenosis* has not been reported due to minimal tissue necrosis in suture hemorrhiodopexy [79, 81].

The simplicity and cost effectiveness of this procedure mandates its inclusion in the armamentarium of all surgeons operating on treatment hemorrhoids.

Non-excisional Hemorrhoidectomy

Kristine Makiewicz and Marc I. Brand

There is a broad range of surgical options for the management of hemorrhoids. Excisional hemorrhoidectomy remains the most definitive management, but there are always new technologies in development to treat hemorrhoids with the less amount of pain, the lower rates of recurrence, and minimal complications. All hemorrhoid procedures have a similar range of complications and are quite painful since the anoderm is well innervated. Damage to the underlying sphincter complex can cause incontinence, and removing too much tissue causes stenosis. The most feared complication is sepsis and death, fortunately a very rare occurrence.

Introduction

Hemorrhoid symptoms are an extremely common medical condition with a prevalence of 4.4% in the USA [82]. The internal and external hemorrhoidal cushions are a normal part of the continence mechanism but can become pathologically enlarged. Internal hemorrhoids cause bleeding and prolapse, while external hemorrhoids cause intense pain when thrombosed. There are many management choices that are various combinations of removing excess tissue, fixing the prolapsed mucosa in place and managing vascular congestion. Table 4.2 categorizes the management of hemorrhoids into management of thrombosed external hemorrhoids, internal hemorrhoids, and combined internal and external hemorrhoids.

Anatomy and Grading System

Hemorrhoids are typically three vascular plexuses in the anal canal in the right posterior, right anterior, and left lateral positions. The internal component is proximal to the dentate line and the external hemorrhoid is distal. External

Table 4.2 Non-excisional management of hemorrhoids

Condition	Procedure	Grade
Thrombosed external hemorrhoids	Observation Excision	External
Internal hemorrhoids	Rubber band ligation Infrared coagulation Injection sclerotherapy Suture hemorrhoidopexy Transanal dearterialization	II & III I & II I & II II & III II & III
Combined internal and external hemorrhoids	LigaSure™ hemorrhoidectomy Laser hemorrhoidectomy Cryotherapy	III & IV I–IV I–III

hemorrhoids thrombose causes pain. Internal hemorrhoids prolapse and bleed. Grade I hemorrhoids are enlarged but do not prolapse, grade II prolapse and reduce spontaneously, grade III prolapse and reduce manually, and grade IV prolapse and do not reduce.

Excision of Thrombosed External Hemorrhoids

Patients with acute thrombosed external hemorrhoids present with acute anal pain and a bulge thought to be brought on by an intravascular clot triggered by the pressure of constipation [83]. Thrombosed external hemorrhoids are usually managed conservatively with days to weeks of Sitz baths, topical and oral pain medications. As the clot begins to resorb and the swelling improves, the pain resolves. The other option is to excise the thrombosis and clear the clot to speed the healing process. Surgical management can be a formal hemorrhoidectomy or incision and evacuation of clot. Incision and evacuation has been abandoned by colorectal surgeons because of the higher rates of recurrence and bleeding with this procedure [84–86]. Complete excision of the clot under local anesthesia is a common procedure that is not well studied [84, 87, 88].

Complication of Excision of Thrombosed External Hemorrhoid

Early Complications

The primary early complication of thrombosed external hemorrhoid drainage results from inadequate evacuation of the clot from the thrombosed veins. Incomplete clot removal can lead to re-accumulation of clot and bleeding [84, 85]. Under local anesthesia, an ellipse of skin is excised and the underlying clot completely evacuated. Rates of post surgical abscess and fistula development are not well studied. Jongen et al. reported a 2.1% rate in a study of 340 patients but did not evaluate predisposing factors [87].

Late Complications

Late recurrence of thrombosed external hemorrhoids appears to be more common with medical management than with surgical excision [84]. It is unclear if recurrences are repeat episodes in the same hemorrhoids or similar episodes in a new area. Anal tags and hypertrophic papilla can develop from the healing and resorption of clot following either medical or surgical management of thrombosed external hemorrhoids [87]. Excision does not cause anal stenosis or incontinence because these are quite localized one or two column procedures with no involvement of the internal anal canal or sphincter complex. Injudicious extension of the excision into the anal canal may result in an anal fissure as delayed complication (Table 4.3).

Table 4.3 Summary of complications

Complication	Procedures in which they occur	Preventative measures would "none" be appropriate in empty areas?
Early complications		
External hemorrhoid recurrence	External hemorrhoid incision and evacuation	• Early: Excision rather than incision • Late: surgical excision reduces recurrence more than medical management
Anal tags + hypertrophic papilla	External hemorrhoid excision	None
	Suture hemorrhoidopexy	
	Laser hemorrhoidectomy	
	Cryotherapy	
Urinary retention	Rubber band ligation	• Perform only single column banding at any one session
	Infrared coagulation	None
	Suture hemorrhoidopexy and transanal hemorrhoidal dearterialization	
	LigaSure™ hemorrhoidectomy	
	Laser hemorrhoidectomy	
Priapism	Rubber band ligation	• Perform only single column banding at any one session
Thrombosed external hemorrhoids	Rubber band ligation	• Perform only single column banding at any one session
	Injection sclerotherapy	• Avoid injecting intravascularly
Secondary/delayed bleeding	Rubber band ligation	None
	Infrared coagulation	
	Suture hemorrhoidopexy	
	Transanal hemorrhoidal dearterialization	
	LigaSure™ hemorrhoidectomy	• Cut hemorrhoid precisely along line of cautery to remove otherwise cut edge can bleed
	Laser hemorrhoidectomy	• Avoid aiming laser deep toward hemorrhoidal arteries
	Cryotherapy	None
Perianal abscess/sepsis/necrotizing fasciitis	Rubber band ligation	• Removal of bands in the OR at the first sign of increasing pain, fever, urinary retention
	Injection sclerotherapy	None
Hematuria, prostatitis, rectourethral fistula	Injection sclerotherapy	• Do not inject anteriorly

(continued)

Table 4.3 (continued)

Complication	Procedures in which they occur	Preventative measures would "none" be appropriate in empty areas?
Early recurrent hemorrhoidal prolapse	Transanal hemorrhoidal dearterialization	• Adequate stitches to prevent early pull through of mucopexy
Severe post-procedure pain	Rubber band ligation	• Remove band that is in too close proximity to dentate line
	Transanal hemorrhoidal dearterialization	• Remove stitch that is in too close proximity to dentate line
Incontinence	LigaSure™ hemorrhoidectomy	• Often temporary from post operative laxative use and impaired sensation while healing
Discharge from mucosal sloughing	Injection sclerotherapy	• No more than 2–5 mL of injectant • Injection episodes minimum 6–12 weeks apart • Inject into submucosa (not more superficial or deeper)
	LigaSure™ hemorrhoidectomy	• From break down of wound edges since no sutures are placed
	Cryotherapy	None
Late complications		
Fistula	Thrombosed external hemorrhoid excision	None
Anal stenosis	Transanal hemorrhoidal dearterialization	
	Injection sclerotherapy	• Minimize volume injected to prevent radial extravasation
	LigaSure™ hemorrhoidectomy	• Careful elevation of mucosa from sphincter with local anesthesia injection • Short bursts of energy to prevent lateral spread of heat • Sharp excision of anoderm for the external component instead of with LigaSure™ cautery
	Laser hemorrhoidectomy	None
Anal fissure	Rubber band ligation	None

Rubber Band Ligation for Internal Hemorrhoids

Rubber band ligation is an office-based treatment for internal hemorrhoids; it does not address any component of external hemorrhoids. Most practitioners will use this method for grade II and some grade III hemorrhoids. An anoscope is placed and the base of the hemorrhoid elevated with a forceps or suction device. A rubber band is placed just proximal to the base of the hemorrhoid causing ischemia of the banded tissue and resultant scarring and fixation of the hemorrhoid to the underlying tissue. Practitioners vary in the number of hemorrhoids treated in one session. Some will

band all three hemorrhoids in one session while others treat individual hemorrhoids over multiple sessions.

Complications of Rubber Band Ligation Complications

Early Complications

Most patients have mild tenesmus for 24–48 h post procedure [89–93]. If there is immediate pain during placement, the band is too close to the dentate line and should be removed and repositioned. It is helpful to ask the patient to compare the sensation while the anoscope is in place and when the hemorrhoid is drawn up into the banding ring before deployment of the rubber bands. Additionally, a suction ligator tends to mimic the post banding sensation better than a grasping forceps. If a sharp pain is felt, the hemorrhoid should be released and the ligator repositioned more cephalad. If the pain is intense for more than 24 h, patients require operative hemorrhoidectomy and removal of rubber band [92]. A case report exists of referred sciatic pain from rubber band ligation that resolved with removal of the band [94]. Other minor complications occurring at very low rates are urinary retention, priapism, slipped bands, thrombosis of hemorrhoids, and itching [90]. These minor complications occur at higher rates if multiple hemorrhoids are banded in a single treatment [95–97].

Mild bleeding for the first few days is common and should be differentiated from secondary bleeding at 10–14 days. When the banded tissue sloughs off, secondary or delayed bleeding occurs in 1–2% of the patients and can be quite massive. Most large case series have small numbers of hemorrhage requiring admission and transfusion but not operations. Nonoperative hemorrhage is more common in patients on warfarin and aspirin, but the rates are not high enough for these medications to be an absolute contraindication. Cirrhosis and portal hypertension do not appear to be a contraindication to rubber band ligation [97–99]. Topical application of 1/100 Epinephrine on a Q tip causes significant arterial constriction and the bleeding either becomes minimal or stops altogether. The round central dot can then be cauterized with silver nitrate.

The most serious complications are infections. Perianal abscess and resulting fistula can occur at the band site. Bacteremia occurs less than 1% of the time with a single case report of the bacteremia causing endocarditis in a patient with a VSD (Ventricular septal defect) [99–101]. There are case reports of pelvic sepsis requiring ICU admission and IV antibiotics, necrotizing fasciitis causing death or damage to sphincter requiring permanent fecal diversion [97, 102–104]. For this reason, complaints of severe pain or fever, especially when delayed a few days and associated with urinary retention, should be treated aggressively with repeat exam in office or under anesthesia, removal of rubber bands debridement of necrotic tissue, and intravenous broad spectrum antibiotics [103].

Late Complications

Rubber band ligation can lead to problems with delayed healing from mucosal ulceration lasting for months, and anal fissure [88]. Anal stenosis does not appear to be a risk of banding, unlike many other hemorrhoidal procedures. A surprisingly large number (16%) of patients have impaired continence scores when carefully assessed during follow-up [105]. Recurrence rates are higher than with excisional hemorrhoidectomy and increase with greater degrees of initial prolapse. Success and recurrence rates vary widely in the literature, as does the definition of success. Grade II hemorrhoids have approximately a 10% recurrence rate and grade III hemorrhoids about a 25% recurrence rate on long-term follow-up [92, 97]. Educating patients regarding optimal bowel habits and encouraging lifelong maintenance of these habits may help to reduce the likelihood of recurrent symptoms.

Infrared Coagulation

Infrared coagulation (IRC) is an outpatient procedure usually used for grade I and II hemorrhoids. This procedure uses infrared light directed at the mucosa with direct pressure just proximal to the base of the hemorrhoid. Two to five pulses at each hemorrhoid generates heat, promoting coagulation in vessels and hemorrhoidal tissue 3 mm deep by 3 mm wide [106–108]. As the inflamed tissue heals, the resulting scar fixes the hemorrhoid in place preventing prolapse.

Complications of Infrared Coagulation

Early Complications

No major complications from infrared coagulation have been reported. Patients describe mild pain during the procedure and for 24–48 h post procedure. There can be some discharge from the ulceration and mild post procedure bleeding is common for the first week [89, 106, 109]. Two cases of post-procedure hemorrhage requiring admission and observation, resolving without surgical intervention have been reported [110] and low rates of urinary retention and anal fissures have been reported [89, 111].

Late Complications

The primary long-term complication from IRC is inefficient management of hemorrhoidal symptoms. Only about two out of three patients have effective control of symptoms after one treatment for grade I or II hemorrhoids [107, 112]. Over 50% of grade III hemorrhoids will have recurrence of prolapse after 1 year [113].

Injection Sclerotherapy

Injection sclerotherapy is an office procedure designed to cause fixation of excess hemorrhoidal cushions to the underlying tissue and prevent prolapse. It is used for grade I and II hemorrhoids, since it is more effective to prevent bleeding hemorrhoids and less effective against prolapse. This is a long-standing procedure that has

been used for grade I and II hemorrhoids since the 1860s [114]. 2–5 mL of phenol with oil is injected into the submucosa above the dentate line. Repeat injections can be delivered 6–12 weeks apart.

Complications of Injection Sclerotherapy

Early Complications

The early complications of injection sclerotherapy are well documented in numerous case reports. They can be categorized into three different reactions: local, septic, and urologic. Injection of the sclerosant should be into the submucosa. However, if it is injected either too superficially or deep, mucosal sloughing and bleeding can occur. The sloughing can also occur with too large a volume of sclerosant or injection sessions too close together. The sclerosant should not be injected into the vascular space—unlike in lower extremity venous disease—since this can cause hemorrhoid thrombosis [115].

Septic complications can vary from localized abscesses to severe bacteremia, sepsis, necrotizing fasciitis, retroperitoneal abscess, and abdominal compartment syndrome [116–119].

Urinary complications include hematuria, prostatitis, and rectourethral fistula especially if injection sclerotherapy is used for anterior injections [120, 121].

Late Complications

Anal stenosis can occur if too much sclerosant is injected with radial extravasation [122]. Recurrence rates are as high as 30–60% with phenol injections [123]. This high recurrence rate leads to more repeat procedures than other office-based procedures such as rubber band ligation or infrared coagulation [124].

Suture Hemorrhoidopexy

Suture hemorrhoidopexy is a technique that includes a combination of hemorrhoid ligation with fixation. An absorbable suture is placed at the base of the hemorrhoid to ligate all arterial inflow into the hemorrhoid plexus and a running stitch is brought out along the hemorrhoid to ligate all redundant tissue and pexy to the underlying muscle. No tissue is excised and all stitches are placed proximal to the dentate line to minimize pain. The mucopexy is thought to align the hemorrhoidal tissue and recreate a straight venous outflow tract. After the absorbable stitch dissolves, the scar holds the hemorrhoid in place. This is distinct from transanal hemorrhoidal dearterialization (THD) since the stitch is placed blindly and not under Doppler guidance.

Complications of Suture Hemorrhoidopexy

The most common complications are thrombosed external hemorrhoids (1.9%), urinary retention (1.4%), and secondary hemorrhage (0.6%) [122]. There are no reports of anal stenosis or infectious complications because there is minimal necrosis of tissue [125].

Transanal Hemorrhoidal Dearterialization ±Mucopexy

Transanal hemorrhoidal dearterialization (THD) with or without mucopexy is a non-excisional method of managing hemorrhoids. It differs from suture hemorrhoidopexy in that the arterial inflow is identified using a specialized anoscope with attached Doppler probe and then ligated. First described by Morinaga in the 1990s, the Doppler is used to locate the hemorrhoidal arteries which are sutured to interrupt the flow. Six to eight arteries are ligated on average. When mucopexy, also known as recto-anal repair (RAR), is added to the procedure, the hemorrhoid is lifted back to the anatomical position to reduce prolapse. Since no tissue is excised and all stitches are placed proximal to the dentate line, postoperative pain is significantly less than that experienced by patients after excisional hemorrhoidectomy and there is faster return to baseline activities [126, 127]. The majority of studies have evaluated grade II and III internal hemorrhoids but there is evidence that grade IV hemorrhoids can be treated with THD and mucopexy with a recurrence rate of 10% at one-year follow-up [128].

Complications of Transanal Hemorrhoidal Dearterialization ±Mucopexy

Early Complications

Postoperative bleeding occurs 1–5% of the cases and hemorrhage requiring return to the operating room was reported once in several case series [127, 129, 130]. Overall ischemic complications are quite low since there is minimal mucosal necrosis associated with the mucopexy. Some studies reported one to two patients with fissures, indicating perhaps a small component of ischemia [126, 131]. After the procedure, 1% of the patients may develop thrombosed external hemorrhoids and require excision. Urinary retention occurs in 1–2% of patients [127, 128]. Some mild pain is normal for the first few days; if pain is too severe, it is due to close proximity of the sutures to the dentate line. If the suture pulls through, early recurrent prolapse occurs [128].

Late Complications

As expected, there are no reports of incontinence after this procedure, since there is minimal anal dilation and no excision of tissues [131, 132]. The long-term resolution of symptoms—prolapse, pain and bleeding—appears to be 85–90% but most studies have evaluated the procedure in grade II and III hemorrhoids only [126,

127, 131, 133]. Undergoing THD does not preclude patients from subsequent procedures if necessary.

LigaSure™ Hemorrhoidectomy

The LigaSure™ (Medtronic, Minneapolis, MN) technique of hemorrhoidectomy is a modern modification of the classic Ferguson hemorrhoidectomy [134]. The hemorrhoid is grasped with forceps and excised with LigaSure™ cautery. The wound itself is left open to heal or sutured closed. This technique is used to treat grade III and IV hemorrhoids and can be used to remove an external component at the same time. Proponents of LigaSure™ hemorrhoidectomy note the advantages of decreased operative time, less blood loss, and decreased postoperative pain.

Complications of LigaSure™ Hemorrhoidectomy

Early Complications

Multiple meta-analyses and randomized control trials have compared complications of LigaSure™ hemorrhoidectomy with conventional Ferguson hemorrhoidectomy and have failed to demonstrate statistically different rates of postoperative bleeding, urinary retention, or incontinence of gas or stool. Incontinence is likely secondary to impaired sensation during healing process or postoperative laxative use since it resolves within a few weeks after surgery [134–137]. There are only a few case reports of bleeding requiring return to the operating room for control. The LigaSure™ hemorrhoidectomy uses bipolar energy to coagulate the base of the hemorrhoid and scissors to transect the tissue. If the hemorrhoid is not transected precisely along the line of coagulation, the edge may bleed [134]. LigaSure™ hemorrhoidectomy has less immediate postoperative pain and faster return to work for the patient than conventional hemorrhoidectomy. However, since the mucosal edges are not sutured, the wounds can break down and cause some pain from open wounds [138, 139]. These open wounds can cause increased temporary pruritus and mucus discharge [136]. There are no reports of perineal sepsis or abscess from wound break down and only one report of superficial infection requiring IV antibiotics [134].

Late Complications

The most concerning long-term complication of LigaSure™ hemorrhoidectomy is anal stenosis. Most studies report only a few occurrences per study and a few different theories on etiology. If elevation of the submucosa with local anesthetic is not adequate, the underlying sphincter could be damaged by thermal spread and cause stenosis [140] LigaSure™ is often used for combined external and internal hemorrhoids and if the anoderm is not excised sharply with scalpel or scissors, the scarring can cause stenosis [141]. Other authors have advocated short bursts with the energy device to reduce lateral spread of heat [142].

Laser Hemorrhoidectomy

Laser hemorrhoidectomy is a generic term that encompasses two separate techniques and two types of lasers. The most popular technique is similar to Milligan–Morgan or Ferguson conventional hemorrhoidectomy but the dissection is done with laser instead of cautery/scalpel/scissors. The thought was that the laser would provide less tissue destruction than conventional cautery. While most often done with a CO_2 laser, Nd:YAG lasers have also been studied. The second technique is surface laser ablation of the hemorrhoid with CO_2 or Nd:YAG laser causing vaporization and/or coagulation of the hemorrhoidal tissue. The laser can be placed using a direct contact or noncontact technique [143, 144]. This method was developed in the 1980s and has since fallen out of favor. There are few randomized control trials comparing laser hemorrhoidectomy to conventional hemorrhoidectomy and only one comparing cost in the era of outpatient hemorrhoidectomies.

Complications of Laser Hemorrhoidectomy

Early Complications

The early complications of a conventional style hemorrhoidectomy performed with laser are similar to any excisional hemorrhoidectomy. Urinary retention, bleeding, infection and skin tags occur at low rates, similar to that of conventional excisional hemorrhoidectomy [145–147]. Some studies reported lower levels of pain and faster return to work for patients than conventional hemorrhoidectomy but others found no statistical significance [148, 149]. While there were higher rates of wound dehiscence at 10 day follow-up there was no impact on long-term healing or stenosis [148]. There was a case report with evidence of laser damage to underlying sphincter and hemorrhoidal arteries leading to fatal hemorrhage [150]. Proponents of the surface ablation method were enthusiastic that, as a topical method, it would be associated with less pain and minimal scarring compared to conventional hemorrhoidectomy. However, even with the surface coagulation of the mucosa, enough underlying damage can be done to the sphincters promoting stenosis [151].

Late Complications

There were no long-term studies that evaluated recurrence rates after laser hemorrhoidectomy. There were reports of anal stenosis after laser hemorrhoidectomy indicating underlying sphincter damage [144].

Cryotherapy

Cryotherapy for hemorrhoids was an office-based method used in the 1970s to freeze external and internal hemorrhoids. A probe with liquid nitrogen or liquid nitrous oxide was held against the hemorrhoid causing intracellular ice crystallization and cell membrane destruction [146, 152]. Immediately after the procedure, the wound develops intense edema and drainage. As the wound heals, the excess tissue sloughs and is replaced by healthy tissue. With freezing, a white line clearly

marks healthy versus destroyed tissue allowing for precise application. This technique has been abandoned because the open wounds led to prolonged healing, foul smelling drainage and pain [144].

Complications of Cryotherapy

Early Complications

Despite advocates stating that cryotherapy caused no pain during the procedure because the nerve endings were frozen along with the hemorrhoids, most patients reported discomfort lasting for approximately 2 weeks after the procedure [152, 153]. The primary reason this method was abandoned was that patients found the degree of discharge and drainage unacceptable [146, 152, 154]. A small number of patients developed bleeding in the second postoperative week when the greatest degree of slough occurred [153, 154]. Skin tags developed in 25% of patients, although only a few of these were significant enough to require operative removal [152, 154].

Late Complications

Cryotherapy was a procedure in use for only decade with very few long-term follow-up studies. Therefore, the recurrence rates and other late complications are unknown. Oh et al. reported an 11% recurrence rate of the hemorrhoids although it is unclear what the true length of follow-up was for his patients [153].

Traditional Chinese Medicine

Traditional Chinese medicine considers hemorrhoids an imbalance of Yin and Yang with Damp-Heat (traditional concepts) as the underlying pathology. A large variety of herbs are used in combination to counteract these imbalances. Traditionally, the herbs are taken orally. However the Western method of injection sclerotherapy has been modified, with the injection of Xiaozhiling into the hemorrhoid to cause sclerosis. Xiaozhiling is an extract of *Galla chinensis* and *Alumen* herbs [155]. The most common oral compounds are Radix and Sephora species [156]. It is difficult to determine efficacy because recurrence rates were quoted as less than 1% and most studies did not report complications [155]. Injection of Xiaozhiling likely has similar complications to Western injection sclerotherapy of sloughing, infection, and anal stenosis. Many Acupuncture clinics offer treatment for hemorrhoids, however, there were no clinical studies studying its efficacy.

Hemorrhoids and Cancer

Colon, rectal, and anal cancer can present with rectal bleeding, but the most common cause of rectal bleeding is hemorrhoids. Care must be taken by primary care physicians, ER physicians and general surgeons who initially see complaints of rectal bleeding to do a complete history and physical including rectal exam and consider the need for endoscopy to rule out malignant etiologies prior to assuming a

benign diagnosis of hemorrhoids. Anal cancer and melanoma are especially rare diagnoses that must be considered, since there is evidence that a diagnosis of "hemorrhoids" can delay necessary treatment [157]. Anal melanoma, while very rare, is the third most common site of primary melanoma [158].

Hemorrhoidectomy is one of the most commonly performed surgical procedures and pathologic management of the specimens has been debated. Routine pathologic evaluation is expensive but recent studies show rates of 1.4–3.2% of normal appearing hemorrhoid specimens with malignancy on microscopic examination [159, 160]. Adenocarcinoma, squamous cell carcinoma, melanoma, and carcinoid have all been identified [160, 161, 162, 163]. With the increasing prevalence of anal squamous cell cancer, routine histopathological examination should be considered [160, 162].

References

1. Wolff BG, Fleshman JW, Beck DE, Pemberton JH, Wexner SD, editors, The ASCRS Textbook of Colon and Rectal Surgery. New York: Springer; 2007
2. Pavlin DJ, Chen C, Penaloza DA, et al. Pain a factor complicating recovery and discharge after ambulatory surgery. Anesth Analg. 2002;95:627–34.
3. Gramke HF, de Rijke JM, van Kleef M, et al. Predictive factors of postoperative pain after day-case surgery. Clin J Pain. 2009;25:455–60.
4. Li S, Coloma M, White PF, et al. Comparison of the costs and recovery profiles of three anesthetic techniques for ambulatory anorectal surgery. Anesthesiology. 2000;93:1225–30.
5. Gorfine SR, Onel E, Patou G, Krivokapic ZV. Bupivacaine extended-release liposome injection for prolonged postsurgical analgesia in patients undergoing hemorrhoidectomy: a multi-center, randomized, double-blind, placebo-controlled trial. Dis Colon Rectum. 2011;54:1552–9.
6. Haas E, Onel E, Ragupathi M, White PF. A double-blind, randomized, active-controlled study for post-hemorrhoidectomy pain management with liposome bupivacaine, a novel local analgesic formulation. Am Surg. 2012;78:574–81.
7. Richman IM. Use of Toradol in anorectal surgery. Dis Colon Rectum. 1993;36:295–6.
8. O'Donovan S, Ferrara A, Larach S, Williamson P. Intraoperative use of Toradol facilitates outpatient hemorrhoidectomy. Dis Colon Rectum. 1994;37:793–9.
9. White PF, Raeder J, Kehlet H. Ketoralac: its role as part of a multimodal analgesic regimen. Anesth Analg. 2012;114:250–4.
10. Sinatra RS. NSAIDS and COX-2 Inhibitors. In: Sinatra RS, Larach S, Ramamoorthy S, editors. Surgeon's guide to postsurgical pain management: colorectal and abdominal surgery. 1st ed. West Islip, NY: Professional Communications, Inc.; 2012. p. 161.
11. Elia N, Lysakowski C, Tramer MR. Does multimodal analgesia with acetaminophen, nonsteroidal anti-inflammatory drugs, or selective cyclooxygenase-2 inhibitors and patient-controlled analgesia morphine offer advantages over morphine alone? Meta-analyses of randomized trials. Anesthesiology. 2005;103:1296–304.
12. Arici S, Gurbet A, Turker G, et al. Pre-emptive analgesic effects of intravenous paracetamol in total abdominal hysterectomy. Agri. 2009;21:54–6.
13. Carpeti EA, Kamm MA, McDonald PJ, et al. Double-blind randomized controlled trial of effect of metronidazole on pain after day-case haemorrhoidectomy. Lancet. 1998;351:169–72.

14. Balfour L, Stojkovic SG, Botterill ID, et al. A randomized double-blind trial of the effect of metronidazole on pain after closed hemorrhoidectomy. Dis Colon Rectum. 2002;45:1186–90.
15. Ala S, Saeedi M, Eshghi F, et al. Topical metronidazole can reduce pain after surgery and pain on defecation in postoperative hemorrhoidectomy. Dis Colon Rectum. 2008;51:235–8.
16. Fleischer M, Marini CP, Statman R, et al. Local anesthesia is superior to spinal anesthesia for anorectal surgical procedures. Am Surg. 1994;60:812–5.
17. Liu JW, Lin CC, Kiu KT, et al. Effect of glyceryl trinitrate ointment on pain control after hemorrhoidectomy: a meta-analysis of randomized controlled trials. World J Surg. 2016;40:215–24.
18. Joshi GP. Neugebauer EA; PROSPECT collaboration. Evidence-based management of pain after haemorrhoidectomy surgery. Br J Surg. 2010;97:1155–68.
19. Bailey HR, Ferguson JA. Prevention of urinary retention by fluid restriction following anorectal operations. Dis Colon Rectum. 1976;19:250–2.
20. Prasad ML, Abcarian H. Urinary retention following operations for benign anorectal diseases. Dis Colon Rectum. 1978;21:490–2.
21. Toyonaga T, Matsushima M, Sogawa M, et al. Postoperative urinary retention after surgery for benign anorectal disease: potential risk factors and strategy for prevention. Int J Colorectal Dis. 2006;21:676–82.
22. Ternent CA, Fleming F, Welton MA, et al. Clinical practice guideline for ambulatory anorectal surgery. Dis Colon Rectum. 2015;58:915–22.
23. Wolf JS, Munoz JJ, Rosin JD. Survey of hemorrhoidectomy practices: open versus closed techniques. Dis Colon Rectum. 1979;22:536–8.
24. Chen HH, Wang JY, Changchien CR, et al. Effective management of posthemorrhoidectomy secondary hemorrhage using rectal irrigation. Dis Colon Rectum. 2002;45:234–238.
25. Cirocco WC, Golub RW. Local epinephrine injection as treatment for delayed hemorrhage after hemorrhoidectomy. Surgery. 1995;117:235–7.
26. Rosen L, Sipe P, Stasik JJ, et al. Outcome of delayed hemorrhage following surgical hemorrhoidectomy. Dis Colon Rectum. 1993;36:743–6.
27. Basso L, Pescatori M. Outcome of delayed hemorrhage following surgical hemorrhoidectomy. Dis Colon Rectum. 1994;37:288–9.
28. Smellie GD. Control of post-haemorrhoidectomy bleeding with a Foley catheter and a pack. JR Coll Surg Edinb. 1965;10:328.
29. LeFrock JL, Ellis CA, Turchik JB, et al. Transient bacteremia associated with sigmoidoscopy. N Engl J Med. 1973;289:467–9.
30. Bouchard D, Abramowitz L, Castinel A, et al. One-year outcome of haemorrhoidectomy: a prospective multi-center French study. Colorectal Dis. 2012;15:719–26.
31. Chen CW, Lai CW, Chang YJ, et al. Results of 666 consecutive patients treated with LigaSure hemorrhoidectomy for symptomatic prolapsed hemorrhoids with a minimum follow-up of 2 years. Surgery. 2013;153:211–8.
32. Qarabaki MA, Mukhashavaria GA, Mukashavaria GG, et al. J Gastrointest Surg. 2014;18:808–15.
33. Eu KW, Teoh TA, Seow-Chen F, et al. Anal stricture following hemorrhoidectomy: early diagnosis and treatment. Aust N Z J Surg. 1995;65:101–3.
34. Milsom JW, Mazier WP. Classification and management of postsurgical anal stenosis. Surg Gynecol Obstet. 1986;163:60–4.
35. Liberman H, Thorson AG. How I do it. Anal stenosis. Am J Surg. 2000;179:325–9.
36. Blumetti J, Abcarian H. Anal canal resurfacing in anal stenosis. In Zbar AP, Madoff RD, Wexner SD, editors. Reconstructive Surgery of the Rectum, Anus and Perineum. London: Springer; 2013. p. 437-445.
37. Pearl RK, Hooks VH, Abcarian H, et al. Island flap anoplasty for the treatment of anal stricture and mucosal ectropion. Dis Colon Rectum. 1990;33:581–3.

38. Nienhuijs SW, de Hingh IHJT. Conventional versus LigaSure hemorrhoidectomy for patients with symptomatic Hemorrhoids. Cochrane Database of Systematic Reviews 2009, Issue 1. Art. No.: CD006761. doi:10.1002/14651858.CD006761.pub2.
39. Brisinda G, Vanella S, Cadeddu F, et al. Surgical treatment of anal stenosis. World J Gastroenterol. 2009;15:1921–8.
40. Wolff BG, Culp CE. The Whitehead hemorrhoidectomy, an unjustly maligned procedure. Dis Colon Rectum. 1988;31:587–90.
41. Roe AM, Bartolo DC, Vellacott KD, et al. Submucosal versus ligation excision haemorrhoidectomy: a comparison of anal sensation, anal sphincter manometry and postoperative pain and function. Br J Surg. 1987;74:948–51.
42. Madoff RD, Fleshman JW. American gastroenterological association technical review on the diagnosis and treatment of hemorrhoids. Gastroenterology. 2004;126:1463–73.
43. van Tets WF, Kuijpers JH, Tran K, et al. Influence of Parks' anal retractor on anal sphincter pressures. Dis Colon Rectum. 1997;40:1042–5.
44. Felt-Bermsa RJ, van Baren R, Koorevaar M, et al. Unsuspected sphincter defects shown by anal endosonography after anorectal surgery, a prospective study. Dis Colon Rectum. 1995;38:249–53.
45. Johnson CD, Budd J, Ward AJ. Laxatives after hemorrhoidectomy. Dis Colon Rectum. 1987;30:780–1.
46. Longo A. Treatment of hemorrhoid disease by reduction of mucosa and hemorrhoid prolapse with a circular-suturing device: A new procedure. In: Proceedings of the sixth world congress of endoscopic surgery. 1998; Rome, Italy: 777–784.
47. Corman ML, Gravie JF, Hager T. Stapled haemorrhoidopexy: a consensus position paper by an international working party: Indications: Contra-indications and Technique. Colorectal Dis. 2003;5(4):303–10.
48. Singer MA, Cintron JR, Fleshman JW, Abcarian H, et al. Early experience with stapled hemorrhoidectomy in the United States. Dis Colon Rectum. 2002;45(3):360–7.
49. Rowsell M, Bello M, Hemingway DM. Circumferential mucosectomy (stapled haemorrhoidectomy) versus conventional haemorrhoidectomy: randomized controlled trial. Lancet. 2002;355(9206):779–81.
50. Ortiz H, Marzo J, Armendariz P. Randomized clinical trial of staple haemorroidopexy versus diathermy haemorrhoidectomy. Br J Surg. 2002;89(11):1276–81.
51. Mehigan B, Monson JRT, Hartley J. Stapling procedure for haemorrhoids versus Milligan-Morgan haemorrhoidectomy: Randomized controlled trial. Lancet 200:355 (9206);782–785.
52. Khalil KH, O'Bichere A, Sellu D. Randomized clinical trial of sutured versus stapled closed haemorrhoidectomy. Br J Surg. 2008;87(10):1352–5.
53. Correa-Rovelo JM, Telez O, Obregón L, et al. Stapled rectal mucosectomy vs closed hemorrhoidectomy: a randomized, clinical trial. Dis Colon Rectum. 2002;45(10):1367–74.
54. Ceci F, Picchio M, Palimento D, et al. Long-term outcome of stapled hemorrhoidiopexy for grade III and grade IV hemorrhoids. Dis Colon Rectum. 2008;51(7):1107–12.
55. Omner A, Hinrichs J, Möllenberg H, et al. Long-term results after stapled hemorrhoidpexy: a prospective study with a 6-year follow-up. Dis Colon Rectum. 2011;54(5):601–8.
56. Senagore AJ, Singer M, Abcarian H, et al. A prospective, randomized, controlled multicenter trial comparing stapled hemorrhoidopexy and Ferguson hemorrhoidectomy: perioperative and one-year results. Dis Colon Rectum. 2004;47(11):1824–36.
57. Cheetham MJ, Mortensen NJ, Nystrom PO. eta al: Persistent pain and faecal urgency after stapled haemorrhoidectomy. Lancet. 2000;356(9231):730–3.
58. Maw A, Concepcion R, Eu KW, et al. Prospective randomized study of bacteraemia in diathermy and stapled haemorrhoidectomy. Br J Surg. 2003;90(2):222–6.
59. Maw A, Eu KW, Soew-Choen F. Retroperitoneal sepsis complicating stapled hemorrhoidectomy: Report of a case and review of the literature. Dis Colon Rectum. 2002;45 (6):826–8.

60. Guy RJ, Seow-Choen F. Septic complications after treatment of haemorrhoids. Br J Surg. 2003;90(2):147–56.
61. Molloy RG, Kingsmore D. Life threatening pelvic sepsis after stapled haemorrhoidectomy. Lancet. 2000;35(9206):810–2.
62. Mlakar B. Should we avoid stapled hemorrhoidopexy in males and females who practice receptive anal sex? Dis Colon Rectum. 2007;50(10):1727–9.
63. Cipriani S, Pescatori M. Acute rectal obstruction after PPH stapled haemorrhoidectomy. Colorectal Dis. 2002;4(5):367–70.
64. Pescatori M. Management of post-anopexy rectal stricture. Tech Coloproctol. 2002;6 (2):125–6.
65. Dowden JE, Stanely JD, Moore RA. Obstructed defecation after stapled hemorrhoidopexy: a report of four cases. Am Surg. 2010;76(6):622–5.
66. Arroyo A, Pèrez-Legaz J, Miranda E, et al. Long-term clinical results of double-pursestring stapled hemorrhoidopexy in a selected group of patients for the treatment of chronic hemorrhoids. Dis Colon Rectum. 2011;54(5):609–14.
67. Gao XH, Wang HT, Chen JG, et al. Rectal perforation after procedure for prolapsed and hemorrhoids: possible causes. Dis Colon Rectum. 2010;53(10):1439–45.
68. Wong LY, Jiang JK, Chang SC, Lin JK. Rectal perforation: a life-threatening complication of stapled hemorrhoidectomy: report of a case. Dis Colon Rectum. 2003;46(1):116–7.
69. Rippetti V, Caricato M, Arullani A. Rectal perforation, retropneumoperitoneum, and pneumomediastinum after stapling procedure for prolapsed hemorrhoids: report of a case and subsequent considerations. Dis Colon Rectum. 2002;45:268–70.
70. Stelzner F, Staubesand J, Machleidt H. The corpus cavernosum recti—basis of internal hemorrhoids. Langenbecks Arch Klin Chi Ver Dtsch Z Chir. 1962;299:302–12.
71. Thomson WH. The nature of Haemorrhoids. Br J Surg. 1975;62(7):542–52.
72. Parnaud E, Guntz M, Bernard A, et al. Anatomie normale macroscopique et microscopique du réseau vasculaire hémorrïdal. Archives Françaises des Maladies de l'Apparei Digestif. 1976;65:501–14.
73. Davy A, Duval C. Modifications macroscopiques et microscopiqures du reseau vasculaire hemorroïdal dans la maladie hemorroidaire. Arch Fr Appar dig 1976;65:515–512.
74. Haas PA, Fox TA, Haas GP. The pathogenesis of hemorrhoids. Dis Colon Rectum. 1984;27:442–50.
75. Bleday R, Pena JP, Rothenberger DA, et al. Symptomatic hemorrhoids: current incidence and complications of operative therapy. Dis Colon Rectum 1192;35(5):477–481.
76. Morinaga K, Hasuda K, Ikeda T. A novel therapy for internal hemorrhoids: ligation of hemorrhoidal artery with a newly devised instrument (Moricorn) in conjunction with a Doppler flowmeter. Am J Gastroent. 1995;90:610–3.
77. Longo A. A treatment of haemorrhoid disease by reduction of mucosa and haemorrhoid prolapse with a circular-suturing devise: A new procedure. In: Proceedings of the sixth world congress of endoscopic surgery. Rome:1998;777–784.
78. Cheetham MJ, Cohen CR, Kamm MA, et al. A randomized, controlled trial of diathermy hemorrhoidectomy v stapled hemorrhoidectomy in an intended day-care setting with long-term follow-up. Dis Colon Rectum. 2003;46:491–7.
79. Gupta PJ, Kalaskar S. Ligation and mucopexy for prolapsing hemorrhoids—a ten year experience. Ann Surg Innov Res. 2008;2:5–8.
80. Pakravan F, Helmes C, Beaten C. Transanal Open Hemorrhoidopexy. Dis Colon Rectum. 2009;52(3):503–6.
81. Gemici K, Okus A, Serden A. Vascular Z-shaped ligation technique in surgical treatment of haemorrhoid. W J Gastrointest Surg. 2015;27:10–4.
82. Johanson JF, Sonnenberg A. The prevalence of hemorrhoids and chronic constipation. Gastroenterology. 1990;98:380–6.
83. Oh C. Acute thrombosed external hemorrhoids. Mt Sinai J Med New York. 1989;56(1):30–2.

84. Greenspon J, Williams SB, Young HA, Orkin BA. Thrombosed external hemorrhoids: Outcome after conservative or surgical management. Dis Colon Rectum. 2004;47(9):1493–8.
85. Grosz CR. A surgical treatment of thrombosed external hemorrhoids. Dis Colon Rectum. 1990;33(3):249–50.
86. Sakulsky SB, Blumenthal JA, Lynch RH. Treatment of thrombosed hemorrhoids by excision. Am J Surg. 1970;120(October):537–8.
87. Jongen J, Bach S, Stubinger SH, Bock JU. Excision of thrombosed external hemorrhoid under local anesthesia: a retrospective evaluation of 340 patients. Dis Colon Rectum. 2003;46(9):1226–31.
88. Chan KKW, Arthur JDR. External haemorrhoidal thrombosis: evidence for current management. Tech Coloproctol. 2013;17(1):21–5.
89. Poen AC, Felt-Bersma RJF, Cuesta MA, et al. A randomized controlled trial of rubber band ligation versus infra-red coagulation in treatment of internal haemorrhoids. Eur J Gastroenterol Hepatol. 2000;12:535–9.
90. Savoiz D, Roche B, Glauser et al. Rubber band ligation of hemorrhoids: a relapse as a function of time. Int J Colorectal Dis. 1998;13:154–6.
91. Pérez Vicente F, Fernández Frías A, Arroyo Sebastían A, et al. Effectiveness of rubber band ligation in haemorrhoids and factors related to relapse. Rev Esp Enferm Dig. 2003;95 (2):110–4.
92. Forlini A, Manzelli A, Quaresima S, Forlini M. Long-term result after rubber band ligation for haemorrhoids. Int J Colorectal Dis. 2009;24(9):1007–10.
93. Lu LY, Zhu Y, Sun Q. A retrospective analysis of short and long term efficacy of RBL for hemorrhoids. Eur Rev Med Pharmacol Sci. 2013;17(20):2827–30.
94. Levin A, Lysy J. Sciatic pain after rubber band ligation of haemorrhoids. Int J Colorectal Dis. 2011;26(7):955.
95. Lee HH, Spencer RJ, Jr, RWB. Multiple hemorrhoidal bandings in a single session. Dis Colon Rectum, 1994;37(1):37–41.
96. Mattana C, Maria G, Pescatori M. Rubberband ligaton of hemorrhoids and rectal prolapse in constripated pattients. Dis Colon Rectum. 1989;32(5):372–5.
97. El Nakeeb AM, Fikry AA, Omar et al. Rubber band ligation for 750 cases of symptomatic hemorrhoids out of 2200 cases. World J Gastroenterol. 2008;14(42):6525–6530.
98. Bat L, Melzer E, Koler, M, et al. Complications of rubber band ligation of symptomatic internal hemorrhoids. Dis Colon Rectum. 1993;36(3), 287–290.
99. Iyer VS, Shrier I, Gordon PH. Long-term outcome of rubber band ligation for symptomatic primary and recurrent internal hemorrhoids. Dis Colon Rectum. 2004;47(8):1364–70.
100. Bayer I, Myslovaty B, Picovsky BM. Rubber band ligation of hemorrhoids. Convenient and economic treatment. J Clin Gastroenterol. 1996;23(1):50–2.
101. Tejirian T, Abbas MA. Bacterial endocarditis following rubber band ligation in a patient with a ventricular septal defect: report of a case and guideline analysis. Dis Colon Rectum. 2006;49(12):1931–3.
102. Duchateau A, Huyghe M. Perirectal sepsis after rubber band ligation os haemorrhoids: A case report. Acta Chir Belg 2014 114L344–348.
103. Quevedo-Bonilla G, Farkas AM, Abcarian H, et al. Septic complications of hemorrhoidal banding. Arch Surg. 1988;123(5):650–1.
104. Subramaniam D, Hureibi K, Zia K, Uheba M. The development of Fournier's gangrene following rubber band ligation of haemorrhoids. BMJ Case Reports. 2013:1–5.
105. Longman RJ, Thomson WHF. A prospective study of outcome from rubber band ligation of piles. Colorectal Dis. 2006;8(2):145–8.
106. Leicester RJ, Nicholls RJ, Chir M, et al. Infrared coagulation: a new treatment for hemorrhoids. Dis Colon Rectum. 1981;24:602–5.

107. Ahmad A, Kant R, Gupta A. Comparative analysis of doppler guided hemorrhoidal artery ligation (DG-HAL) & infrared coagulation (IRC) in management of hemorrhoids. Ind J Surg. 2013;75(4):274–7.
108. Templeton JL, Spence RA, Kennedy TL, et al. Comparison of infrared coagulation and rubber band ligation for first and second degree haemorrhoids: a randomised prospective clinical trial. Br Med J (Clin Res Ed). 1983;286(6375):1387–9.
109. Marques CFS, Nahas SC, Nahas CSR, et al. Early results of the treatment of internal hemorrhoid disease by infrared coagulation and elastic banding: a prospective randomized cross-over trial. Techniques in Coloproctology. 2006;10(4):312–317.
110. Gupta P. Infra red photocoagulation of early grades of hemorrhoids- 5 year follow-up study. Bratisl Lek Listy. 2007;108:223–6.
111. Dennison A, Whiston RJ, Rooney S, et al. A randomized comparison of infrared photocoagulation with bipolar diathermy for the outpatient treatment of hemorrhoids. Dis Colon Rectum. 1990;33(1):32–4.
112. Ambrose NS, Hares MM, Alexander-Williams J, Keighley MR. Prospective randomised comparison of photocoagulation and rubber band ligation in treatment of haemorrhoids. Br Med J (Clin Res Ed). 1983;286(6375):1389–91.
113. Walker AJ, Leicester RJ, Nicholls RJ, et al. A prospective study of infrared coagulation, injection and rubber band ligation in the treatment of haemorrhoids. Int J Colorectal Dis. 1990;5:113–6.
114. Ray S, Mandal S, Khamrui S. Rectovaginal fistula: an extremely rare complication after injection sclerotherapy for hemorrhoids. Am Surg. 2013;April(79):E143–44.
115. Corman ML (ed.). Colon and rectal surgery: ambulatory treatment. In: Colon and rectal surgery. 3rd ed. lippencott 1993. pp. 61–98).
116. Adami B, Eckardt VF, Suermann RB, et al. Bacteria after proctoscopy and hemorrhoidal injection sclerotherapy. Dis Colon Rectum. 1981;24:373–4.
117. Yang P, Wang YJ, Li F, Sun JB. Hemorrhoid sclerotherapy with the complication of abdominal compartment syndrome: report of a case. Chin Med J (Engl). 2011;124 (12):1919–20.
118. Kaman L, Aggarwal S, Kumar R, et al. Necrotizing fascitis after injection sclerotherapy for hemorrhoids: report of a case. Dis Colon Rectum. 1999;42(3):419–20.
119. Ribbans WJ, Radcliffe AG. Retroperitoneal abscess following sclerotherapy for hemorrhoids. Dis Colon Rectum. 1985;28(3):188–9.
120. Gupta N, Katoch A, Lal P, Hadke NS. Rectourethral fistula after injection sclerotherapy for haemorrhoids, a rare complication. Colorectal Dis. 2011;13(1):105.
121. Al-Ghnaniem R, Leather AJM, Rennie JA. Survey of methods of treatment of haemorrhoids and complications of injection sclerotherapy. Ann R Coll Surg Engl. 2001;83(5):325–8.
122. Gupta PJ, Kalaskar S. Ligation and mucopexy for prolapsing hemorrhoids-a ten year experience. Ann Surg Innovation Res. 2008;1164:2–5.
123. Tomiki Y, Ono S, Aoki J, et al. Treatment of internal hemorrhoids by endoscopic sclerotherapy with aluminum potassium sulfate and tannic acid. Diagn Ther Endosc. 2015:517–690.
124. MacRae HM, McLeod RS. Comparison of hemorrhoidal treatments: a meta-analysis. Can J Surg. 1997;40(1):14–7.
125. Gemici K, Okuş A, Ay S. Vascular Z-shaped ligation technique in surgical treatment of haemorrhoid. World J Gastrointestinal Surgery. 2015;7(1):10–4.
126. Bursics A, Morvay K, Kupcsulik P, Flautner L. Comparison of early and 1-year follow-up results of conventional hemorrhoidectomy and hemorrhoid artery ligation: A randomized study. Int J Colorectal Dis. 2004;19(2):176–80.
127. Infantino A, Bellomo R, Monte Dal, et al. Transanal haemorrhoidal artery echodoppler ligation and anopexy (THD) is effective for II and III degree haemorrhoids: a prospective multicentric study. Colorectal Dis. 2010;12(8):804–9.

128. Elmér SE, Nygren JO, Lenander CE. A randomized trial of transanal hemorrhoidal dearterialization with anopexy compared with open hemorrhoidectomy in the treatment of hemorrhoids. Dis Colon Rectum. 2013;56(4):484–90.
129. Giordano P, Overton J, Madeddu F, et al. Transanal hemorrhoidal dearterialization: a systematic review. Dis Colon Rectum. 2009;52(9):1665–71.
130. Ratto C, Donisi L, Parello A, et al. Evaluation of transanal hemorrhoidal dearterialization as a minimally invasive therapeutic approach to hemorrhoids. Dis Colon Rectum. 2010;53 (5):803–11.
131. Sohn N, Aronoff JS, Cohen FS, Weinstein MA. Transanal hemorrhoidal dearterialization is an alternative to operative hemorrhoidectomy. Am J Surg. 2001;182(5):515–9.
132. De Nardi P, Capretti G, Corsaro A, Staudacher C. A prospective, randomized trial comparing the short- and long-term results of doppler-guided transanal hemorrhoid dearterialization with mucopexy versus excision hemorrhoidectomy for grade III hemorrhoids. Dis Colon Rectum. 2014;57(3):348–53.
133. Avital S, Inbar R, Karin E, Greenberg R. Five-year follow-up of Doppler-guided hemorrhoidal artery ligation. Tech Coloproctol. 2012;16(1):61–5.
134. Chen CW, Lai CW, Chang YJ, et al. Results of 666 consecutive patients treated with LigaSure hemorrhoidectomy for symptomatic prolapsed hemorrhoids with a minimum follow-up of 2 years. Surgery (United States). 2013;153(2):211–8.
135. Arslani N, Patrlj L, Rajkovic Z, et al. A randomized clinical trial comparing ligasure versus stapled hemorrhoidectomy. Surg Laparosc Endosc. 2012;22(1):58–61.
136. Basdanis G, Papadopoulos VN, Michalopoulos A, et al. Randomized clinical trial of stapled hemorrhoidectomy vs open with Ligasure for prolapsed piles. Surg Endosc Other Intervent Tech. 2005;19(2):235–239.
137. Xu L, Chen H, Lin G, Ge Q. Ligasure versus Ferguson hemorrhoidectomy in the treatment of hemorrhoids: a meta-analysis of randomized control trials. Surg Laparosc Endosc Percutaneous Tech. 2015;25(2):106–10.
138. Altomare DF. Tips and tricks: hemorrhoidectomy with LigaSure. Tech Coloproctol. 2009;13 (4):321–2.
139. Khanna R, Khanna S, Bhadani S, et al. Comparison of ligasure hemorrhoidectomy with conventional ferguson's hemorrhoidectomy. Ind J Surg. 2010;72(4):294–7.
140. Tan EK, Cornish J, Darzi AW, et al. Meta-analysis of short-term outcomes of randomized controlled trials of LigaSure vs conventional hemorrhoidectomy. Arch Surg. 2007;142 (12):1209–18.
141. Gravante G, Venditti D. Postoperative anal stenoses with ligasure hemorrhoidectomy. World J Surg. 2007;31(1):245–7.
142. Ramcharan KS, Hunt TM. Anal stenosis after LigaSure hemorrhoidectomy. Dis Colon Rectum. 2005;48(8):1666–7.
143. Brown DC, Smith JS. Surface laser ablation of internal haemorrhoids using the carbon dioxide laser. J R Coll Surg Edinb. 1992;37:51–2.
144. Salvati EP. Nonoperative management of hemorrhoids: evolution of the office management of hemorrhoids. Dis Colon Rectum. 1999;42:989–93.
145. Hodgson WJB, Morgan J. Ambulatory hemorrhoidectomy with CO_2 laser. Dis Colon Rectum. 1995;38:1265–9.
146. Iwagaki H, Higuchi Y, Fuchimoto S, et al. The laser treatment of hemorrhoids: results of a study on 1816 patients. Jpn J Surg. 1989;19(6):658–61.
147. Plapler H, de Faria Netto AJ, da Silva Pedro MS. 350 ambulatory hemorrhoidectomies using a scanner coupled to a CO_2 laser. J Clin Laser Med Surg, 2000;18(5):259–262.
148. Senagore A, Mazier WP, Luchtefeld MA, et al. Treatment of advanced hemorrhoidal disease: a prospective, randomized comparison of cold scalpel vs. contact Nd:YAG laser. Dis Colon Rectum. 1993;36(11):1042–9.
149. Zahir KS, Edwards RE, Vecchia A, et al. Use of the Nd-YAG laser improves quality of life and economic factors in the treatment of hemorrhoids. Conn Med. 2000;64(4):199–203.

150. Gill JR, Morrow JS, West AB. Fatal hemorrhage following laser hemorrhoidectomy. J Clin Gastroenterol. 1994;19(4):344–6.
151. Wang JY, Chang-Chien CR, Chen JS, et al. The role of lasers in hemorrhoidectomy. Dis Colon Rectum. 1991;34(1):78–82.
152. Lewis MI, de la Cruz T, Gazzaniga DA, Ball TL. Cryosurgical hemorrhoidectomy—preliminary report. Dis Colon Rectum. 1969;12(5):371–8.
153. Detrano SJ. The role of cyrosurgery in management of anorectal disease: three hundred and fifty cases. Dis Colon Rectum. 1975;18(4):284–8.
154. Oh C. Problems of cryohemorrhoidectomy. Cryobiology. 1982;19:283–6.
155. Oh C, Dreiling DA. Cryohemorrhoidectomy. The Mount Sinai Journal of Medicine New York. 1974;41(5):658–64.
156. Savin S. Hemorrhoidectomy-how i do it: results of 444 cryorectal surgical operations. Dis Colon Rectum. 1977;20(3):189–96.
157. An A, Feng D, Wang C, Shi Y, et al. Comparing the effect of An's Shaobei Injection (安氏芍倍注射液) with Xiaozhiling Injection (消痔灵注射液) in patients with internal hemorrhoids of grade I-III: a prospective cohort study. Chin J Integr Med. 2014;20(7):555–60.
158. Gan T, Liu Y-D, Wang Y, Yang J. Traditional Chinese medicine herbs for stopping bleeding from haemorrhoids. Cochrane Database Syst Rev. 2010;(10):CD00679.
159. Chiu S, Joseph K, Ghosh S, Schiller RCD. Reasons for delays in diagnosis of anal cancer and the efect on patient satisfaction. Can Famly Physicians. 2015;61:509–16.
160. Turner G, Abbott S, Eglinton T, Wakeman C, Frizelle F. Anorectal melanoma: not a haemorrhoid. N Z Med J. 2014;127(1395):73–81.
161. Bauer P, Flejou JF, Etienney I. Prospective single-center observational study of routine histopathologic evaluation of macroscopically normal hemorrhoidectomy and fissurectomy specimens in search of anal intraepithelial neoplasia. Dis Colon Rectum. 2015;58(7):692–7.
162. Timaran CH, Sangwan YP, Solla JA. Adenocarcinoma in a hemorrhoidectomy specimen: case report and review of the literature. Am Surg. 2000;66(August):789–92.
163. Lohsiriwat V, Vongjirad A, Lohsiriwat D. Value of routine histopathologic examination of three common surgical specimens: appendix, gallbladder, and hemorrhoid. World J Surg. 2009;33(10):2189–93.

Anal Fissure

5

Richard Nelson

Anal fissure is a small ulcer located at the opening of the anal canal just distal to the mucocutaneous junction. It causes pain upon defecation, a pain so severe that it has been likened to sliding down the razor blade of life or defecating broken glass; Pain way out of proportion to its size. It occurs typically in the posterior midline though it may also be seen in the anterior midline (Fig. 5.1).

It has findings of chronicity which include a white base of deluded internal sphincter, heaped up edges, possibly a sentinel polyp at its proximal margin and a sentinel pile at the distal margin. The polyp is small and fibrotic, not a neoplasm. Chronicity is also established by duration of symptoms. More on this is given below. Patients will have constipation, since they fear the pain of defecation, and a stenotic anal canal. Bleeding, if any at all, is usually minimal. Though many complications are described related to surgery for anal fissure, certainly the most discussed and the one that is of greatest concern is anal incontinence. This is also, for reasons discussed below, perhaps the most puzzling of all complications of anorectal surgery.

However, it is important before undertaking a detailed discussion of incontinence related to fissure surgery to consider in some detail first who it is that should be subjected to an operation. There are a number of lesions in the anal canal which might be considered to be fissure except for an atypical appearance that differentiates them from the findings of the typical fissure described above. Several examples of these atypical lesions are shown (Fig. 5.2).

These would include large or irregular fissures, possibly located off the midline, multiple fissures, and fissures with edematous piles at the anal opening and fissures not associated with anal stenosis or constipation. Diseases that should come to mind

R. Nelson (✉)
Epidemiology/Biometry Division, School of Public Health,
University of Illinois at Chicago, 1603 West Ashland, Room 956,
Chicago, IL 60612, USA
e-mail: altohorn@uic.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_5

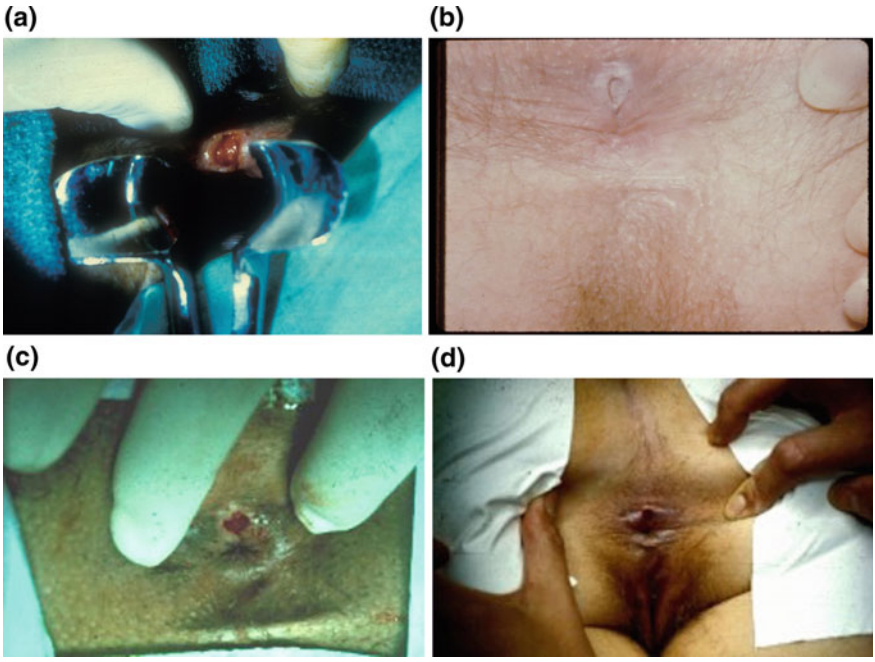


Fig. 5.1 **a**, **b**, and **c** are all typical chronic posterior midline anal fissures with a pale base and heaped up edges, **d** is also a typical pair of fissures located in both the anterior and posterior midlines in a woman

in such a situation would be Crohn's disease or anal neoplasm. To proceed with a partial lateral internal sphincterotomy in any of these situations could be catastrophic. If any operation is to be done in such a situation it is an examination under anesthesia and biopsy.

Surgery is almost never recommended for patients with an acute anal fissure. It is thought that acute anal fissure will often resolve spontaneously or with minimal medical intervention and that only a small proportion would evolve into a chronic fissure. Diagnostic criteria for acute anal fissure are the absence of the findings described above, that is a flat lesion with a red base, friability, and a short symptom history (Fig. 5.3).

This in fact is another puzzling problem in anal rectal surgery in that to diagnose chronic anal fissure requires only one of any of the findings described above, including simply a longer symptom history. So a fissure that looks in every way acute but that has been causing symptoms for six months is a chronic anal fissure. But what is the threshold? In fact it is different in almost every publication on this topic ranging from two weeks to three months. Symptoms of anal fissure often wax and wane so that the short symptom history may have been preceded by similar symptoms many months earlier, which would categorize this as a chronic fissure [1]. This topic will be revisited at the end of the chapter.

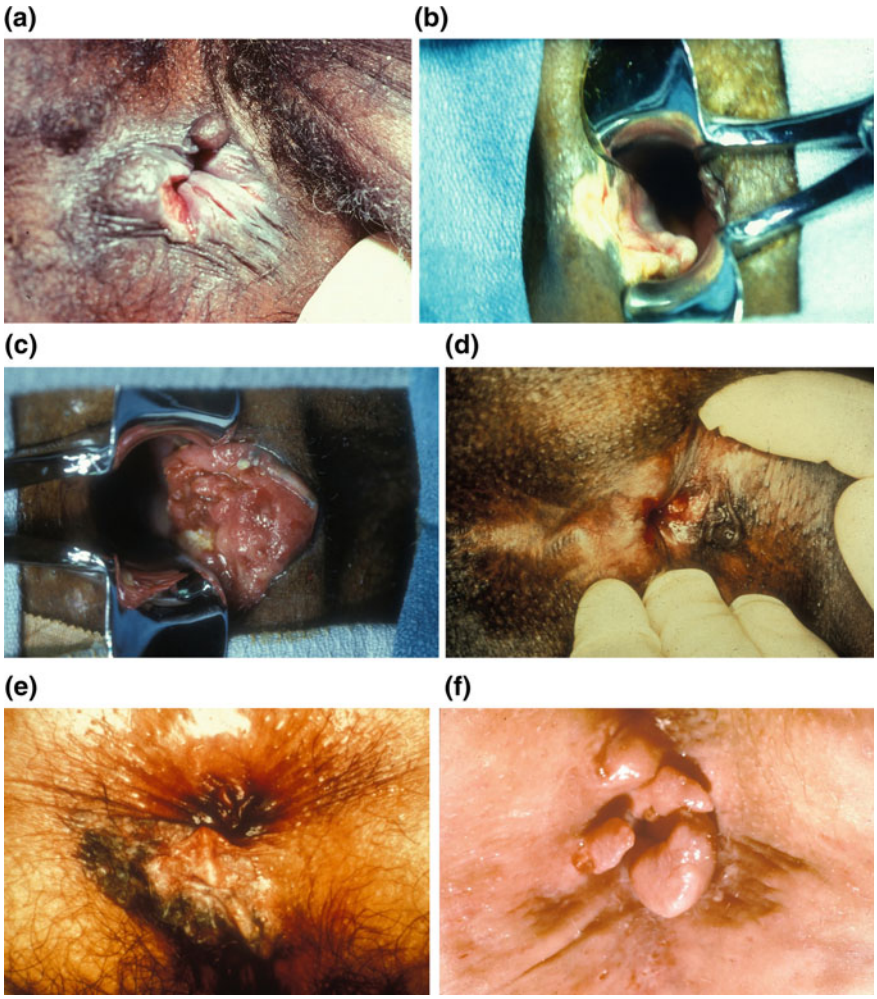


Fig. 5.2 **a** An irregular fissure which might be of some concern. **b** Epidermoid cancer of the anus. **c** Adenocarcinoma of the anus. **d** Lymphoma. **e** Melanoma of the anus. **f** Crohn's disease of the anus

Incontinence, a History

Anal fissure is not really mentioned by any ancient author. Sushruta, Hippocrates, and Galen all write extensively about hemorrhoids and fistula including surgical treatment. This was usually cauterization, and ulcers are mentioned but never separated from hemorrhoids or fistula. Abū Bakr Muḥammad ibn Zakariyyā al-Rāzī does specifically mention fissure but felt it was due to constipation and was treated

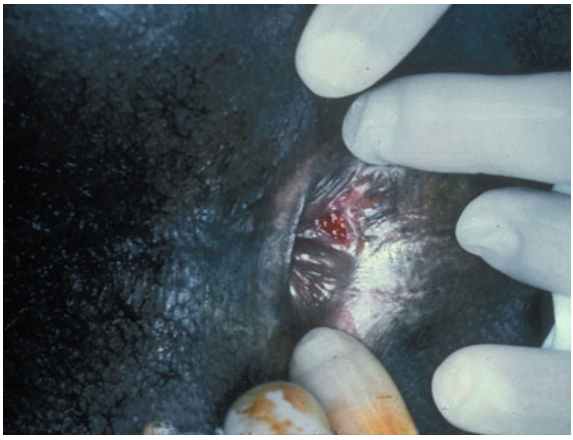


Fig. 5.3 This fissure can either be an acute fissure, or a chronic one if present for a longer time. The findings in Fig. 5.1 do not develop in all chronic fissures

with laxatives [2]. John of Arderne does mention fissuring associated with tenesmus but felt that it was secondary to piles rather than a specific diagnosis [3]. The treatment for fissure alone was nonsurgical.

It was not until the nineteenth century that fissure seems to be recognized as a specific diagnosis and that surgical intervention was needed for its cure. The birth of sphincterotomy was attributed to Alexis Boyer's eleven volume *Traitē des Maladies Chirurgicales* published between 1818 and 1826 [4]. The operation described is very little different from that which is performed today. The extent of sphincterotomy is not delineated. It is also not in most publications today. In a very thorough and fascinating book by Bodenhamer about anal fissure published in 1868, he writes that Ambrose Pare may have described the same operation and Albucasis described a more conservative incisional approach at the end of the tenth century [4]. The number of surgeons actively involved in investigating the surgical treatment of fissure by the time of Bodenhamer and communicating their results was outstanding. Not only efficacy in pain relief and healing were described but also harms of the procedures including incontinence. The procedure of Boyer was regarded as too extensive and a lesser incision recommended, especially for continence preservation.

Another approach described in this book above is forced dilation as a method for relieving the outlet obstruction associated with fissure. One operation of some note was that of Maisonneuve who inserted his whole hand and then closed his fist upon withdrawal. The risk of incontinence was obviously recognized with this procedure and so lesser dilatations were investigated, including two thumbs stretch to the ischial tuberosities, also found to have a high risk of incontinence and finally just two index fingers inserted until a release was palpated. All that these surgeons seem to have been lacking in their investigations were the statisticians.

In the 1920s an old approach got a new name: pectenosis [5]. The previously described internal sphincter fibers were thought to have undergone fibrosis, the so-called pectin band, through chronic congestion. The preferred method of treatment was to incise the fibers, apparently away from the fissure in most descriptions,

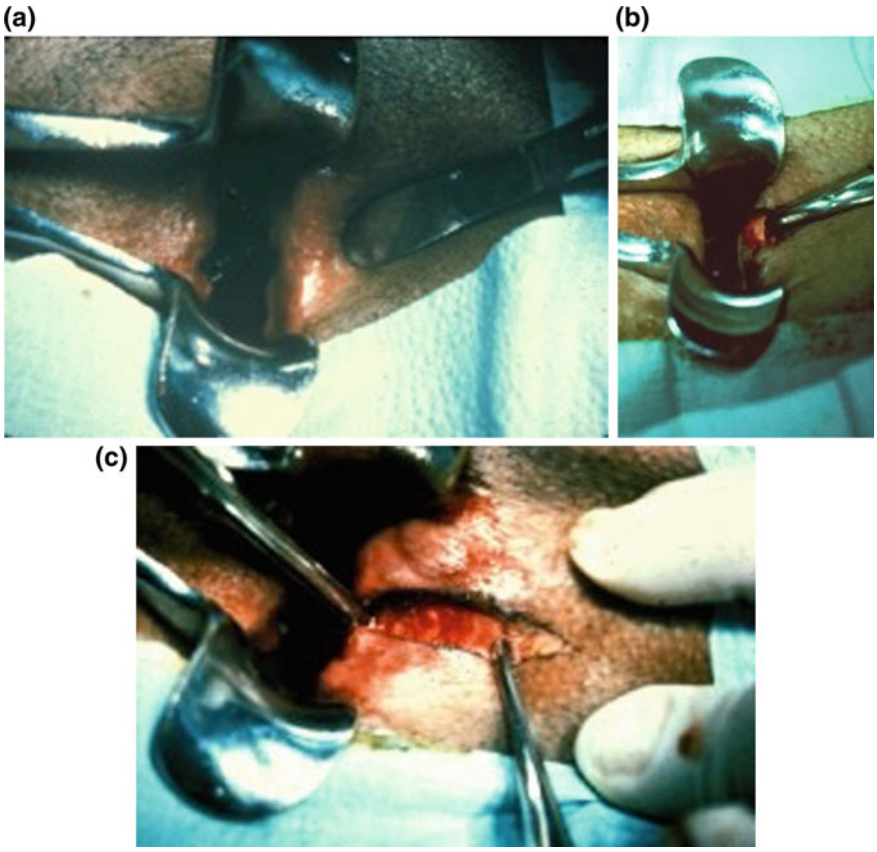


Fig. 5.4 **a** The ridge is an hypertrophied internal sphincter in a patient with fissure. **b** Isolated internal sphincter about to be divided in an LIS. **c** Completed LIS with an intact external sphincter at the base. Does it support the anal canal better here than in the poster mid line?

until the anus could accommodate a two finger insertion, a much more conservative procedure than Maisonneuve's or Boyer's procedures. It was subsequently found that the presumed fibrosis was intact spastic internal sphincter fibers. By the late 1930s, attention had shifted to the external sphincter with injections or actual division of external fibers by Gabriel [6]. Kilbourne also raised the possibility that fissures could be caused by tuberculosis or syphilis at that time [7].

Then in 1951, Eisenhammer [8] described the partial lateral internal sphincterotomy (LIS), though he combined this with a rather liberal dilation of the anal canal after the sphincterotomy. He is, I believe the first to list the number of patients treated by his method (181) and states that none had any defecation difficulties afterwards [8] Fig. 5.4.

This procedure was enthusiastically adopted by surgeons around the world. It was also thought by others that incontinence was not an issue [9]. The first publication to quantify continence disturbance was in 1985. It stated that, out of 306 patients who had had an LIS at least one year earlier, only 15 suffered from any degree of incontinence. This was principally to flatus. In no patient was it severe enough for the patient to wear a pad [10].

However in 1989 everything changed. Khubchandani published a large case series of follow-up after LIS, in which 36% of the patients were incontinent to flatus and 5% to solid stool [11]. In 1996, from the University of Minnesota, which had reported such low incontinence rate in 1985 [10] in a retrospective comparison of open versus closed LIS now found that 30.3% of their patients were incontinent to flatus and 11.8% to solid stool [12]. The age of GTN (glyceryl trinitrate), Botox, and calcium channel blockers was born. In many countries it appears that LIS had been abandoned in favor of medical therapy [13]. In a systematic review of anal incontinence following LIS, 22 studies, mostly nonrandomized case series or cohorts found an overall incontinence rate of 14% with less than 1% having incontinence to solid stool [14]. Yet patient satisfaction with LIS has been reported to be high [1]. The often crippling pain of fissure is almost immediately relieved. And the rest of us colorectal surgeons wondered: "Where are all these incontinent patients?"

The most recent update of the Cochrane review of medical therapy for anal fissure, 28 different medical therapies are investigated in randomized clinical trials [15]. If it was assumed that the only available treatments were GTN, Botox, and calcium channel blockers, all of which appeared in the late 1990s, then it could be surmised by this explosion of new therapies that the older ones were not doing so well. Again, in the Cochrane review that has not been found not to be the case. Whereas LIS has an efficacy of between 90 and 95% in systematic reviews (7.9% in the aforementioned review [1]), no medical therapy has achieved a long term cure rate of 50% [15]. And in the mean time, patients are in pain.

So how big is the anal incontinence problem after LIS really? With the first Cochrane review, looking at all randomized trials of LIS, it was 10%, which was almost, as with Garg, incontinence to flatus [1]. These numbers do not match the findings of Khubchandani [11] and Garcia-Aguilar [12]. These two reports were both from retrospective surveys comparing open LIS and closed LIS from single practices including 715 and 549 patients, respectively. The Cochrane reviews include 143 randomized trials of both medical and surgical treatment of anal fissure published from 1976 to 2016 of which 2523 had LIS and postoperative continence assessment [1, 15]. These data were collected according to an established protocol prospectively. The main difference between the retrospective cohorts and the studies included in the Cochrane reviews is selection bias, e.g., the responders to the retrospective surveys could well have been those with the worst outcomes.

What is more interesting is that with each subsequent update of the of the two Cochrane reviews (There is one only comparing surgical procedures and another Cochrane review comparing any medical therapy to any other therapy, which in 29 of the included studies the comparitor was LIS) the risk of incontinence had declined. From the risk in the original review published in 2000 of 10%, it has

declined to 3.4% for those studies published since 2000. Though this may seem significant if nonsurgical therapies had no risk of incontinence, in fact incontinence developed after therapy (for whatever reason) in 1.1% of those using GTN, 2.2% of those using botox and 1.4% of those using calcium channel blockers [15].

Why did the risk of incontinence decline? There are several possibilities to consider. More haphazard ascertainment is unlikely. The protocols of all randomized trials are carefully scrutinized by ethics committees. The operation might have changed? I think this is likely only insofar as all surgeons are extremely aware of the risk of incontinence related to LIS. If anything, the extent of sphincterotomy (or concomitant dilation) has diminished without apparently diminishing efficacy. This is exactly what was described by Bodenhamer in the USA in the 1860s, wherein the LIS, which was quite extensive in the description of Boyer, was barely more than a mucosal incision with, as he described good results [4]. The length of the sphincterotomy has been studied: either extending proximally to the dentate line, or just to the level of the proximal margin of the fissure. The longer incision showed an insignificantly better efficacy but slightly worse continence [1].

Or possibly different patients are getting the surgery. I also think this is also likely. There may be more careful selection of those patients with an obviously hypertrophied internal sphincter and stenotic anal orifice, and not just anybody with an anal fissure for surgery. Reasonable medical therapies were now available for most fissure patients. This also suggests that for instance patients with fissure but no hypertrophy or stenosis may have been more prone to postoperative incontinence and are now being treated medically. But these are both guesses.

There is one more facet of anal fissure surgery in which history of the early to mid-nineteenth century has repeated itself: forced anal dilation. From Maisonneuve, we can fast forward to Lord and in recent years Bodenhamer to Renzi or Gaj, and others who have investigated a measured anal dilation, in place of the older forced 8 finger wide stretch. So far no incontinence has been reported in the modern dilation groups, which include just six studies, and efficacy is superior to GTN [14].

Myths Concerning Fissure and Incontinence

Posterior midline partial internal sphincterotomy, unlike LIS, is thought to leave a **keyhole defect** in the distal anal canal that results in incontinence of flatus (Fig. 5.5).

All internal sphincterotomies, open or closed, posterior or lateral, leave a keyhole defect and there have been no data from randomized trials to suggest that either efficacy or incontinence are greater with any of these procedures [1].

Acute anal fissure should never be operated upon. It is generally thought that chronic fissures arise from preexisting acute fissures (of course) but that the hypertrophy and stenosis of the sphincter take time to develop, and this will not happen in most acute fissure patients, and that surgery not be considered until this has happened. Fine. In order to defend the above statement, step one is for there to

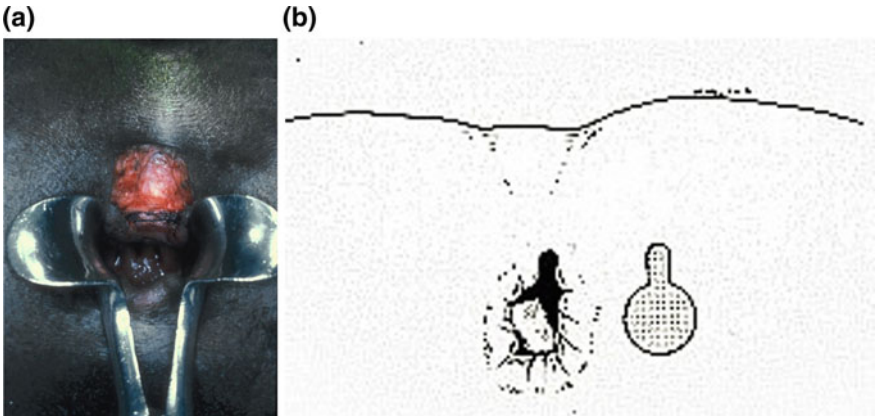


Fig. 5.5 **a** Completed posterior midline internal sphincterotomy **b** The resulting keyhole defect

be general agreement as to what constitutes an acute anal fissure. Physical findings can separate them. With many fissures there is no doubt that they are chronic. But there is no general agreement as to duration. In today's climate most patients with an acute looking fissure will be started on GTN and by the time it has failed, they have usually crossed the chronic fissure dateline. LIS probably should still not be considered in these patients unless sphincter hypertrophy and anal stenosis are present, and its appearance is otherwise not atypical.

Virtually every paper published in the past 20 years has a sentence prominently placed in its first paragraph that LIS causes permanent incontinence. Anybody who runs an incontinence clinic knows there is no such thing as **permanent incontinence**. A broad range of excellent therapies exist for anal incontinence, and most are nonsurgical. Internal sphincter repair has been performed, and should work well, since the muscles are otherwise normal, unlike childbirth-related incontinence. But only a very small percentage of colorectal surgeons have undertaken this operation [16]. What makes these rather alarming statements irresponsible is not just that they are not true, but also that none of these authors have undertaken studies that rigorously characterized the presumed incontinence or investigated various therapies for incontinence in randomized trials. One would have thought that the patients would have demanded it. Presumably they have not.

What Else?

Abscess: I have seen abscess in the sphincterotomy incision in open LIS that has not happened since I switched to closed LIS 20 years ago. That does not seem logical. The randomized studies showed no difference in abscess between the two forms of LIS [1].

Other procedures for which there are insufficient data to establish efficacy or risk of complications, or even to characterize complications include:

fissurectomy
dermal flap coverage of the fissure
bilateral LIS
levatorplasty
removal of anal papillae
Ayurvedic sutures.

References

1. Nelson RL, Chattopadhyay A, Brooks W, Platt I, Paavana T, Earl S. Operative procedures for fissure in ano. Cochrane Database Syst Rev. 2011;(11): update in press.
2. AL-Humadi AH, Al-Samarrai S. Treatment of Anorectal Diseases by al-Rāzī. JIMA. 2009;41:122–34.
3. Power D. Treatises of Fistula m Ano Haemorrhoids, and Clysters of John Arderne. Kegan Paul: Oxford University Press; 1910.
4. Bodenhamer W. Practical observations on the aetiology, pathology, diagnosis and treatment of anal fissure. New York: Wm Wood & Co.; 1868.
5. Abel AL. The pecten, pecten band, pectenosis and pectenotomy. Lancet. 2 April, 1932v 1;5666:714–8.
6. Gabriel WB. ANAL FISSURE. Br Med J. 1939;1(4079):519–21.
7. Kilbourne NJ. The injection treatment of anal fissure. Cal West Med. 1931;35(5):384.
8. Eisenhammer S. The surgical correction of chronic internal anal (sphincteric) contracture. S African Med J 1951;25:486–9.
9. Abcarian H. Surgical correction of chronic anal fissure: results of lateral internal sphincterotomy vs. fissurectomy–midline sphincterotomy. Dis Colon Rectum. 1980 Jan-Feb;23(1):31–6.
10. Walker WA, Rothenberger DA, Goldberg SM. Morbidity of internal sphincterotomy for anal fissure and stenosis. Dis Colon Rectum. 1985;28(11):832–5.
11. Khubchandani IT, Reed JF. Sequelae of internal sphincterotomy for chronic fissure in ano. Br J Surg. 1989;76(5):431–4.
12. Garcia-Aguilar J, Belmonte C, Wong WD, Lowry AC, Madoff RD. Open versus closed sphincterotomy for chronic anal fissure: long-term results. Dis Colon Rectum. 1996;39 (4):440–3.
13. Ommer A. Management of complications of fissure and fistula surgery. Chirurg. 2015;86 (8):734–40.
14. Garg P, Garg M, Menon GR. Long-term continence disturbance after lateral internal sphincterotomy for chronic anal fissure: a systematic review andmeta-analysis. Colorectal Dis. 2013;15(3):e104–17.
15. Nelson RL, Thomas K, Morgan J, Jones A. Non surgical therapy for anal fissure. Cochrane Database Syst Rev. 2012;15:2.
16. Najarian M. Surgeons' beliefs and experiences with the surgical treatment of anal fissure. Sem Colon Rectal Surg. 2006;17:116–119.

Pilonidal Cyst

6

Sany Thomas and Johan Nordenstam

Overview

Pilonidal disease is an acquired benign condition of the skin and subcutaneous tissue of the sacrococcygeal region [1–3]. The term pilonidal is derived from the Latin word pilus meaning hair and nidus meaning nest, describing the disease of the hair follicle in the 'nest' formed in the natal cleft [4–6]. The disease was first described in 1833 by British pathologist, anatomist, and surgeon Harold Mayo, who described the finding of a hair follicle containing sinus in the sacrococygeal region in a female patient [4]. Hair in the natal cleft is thought to be the culprit as per the causative theory [7]. Bascom stated in his original paper that pilonidal disease was caused by enlargement of the midline pits in the natal cleft that contained distorted hair follicles. He further described that the enlarged pits had keratin accumulation and the distorted hair pushes inferiorly causing coalescence of adjacent follicles. This results in inflammation in the subcutaneous tissue and progresses to abscess formation [8, 9].

Pilonidal disease is more common in obese individuals, people with thick hair in the natal cleft, patients with sedentary lifestyles and following trauma to the sacrococcygeal region [7, 10–12]. Buie referred to it as 'Jeep disease' in 1944 and Hardaway called it 'Jeep rider disease' in 1958. This was because young male soldiers, who were predominately affected, had the highest risk of disease. It was suggested that the soldiers' frequent sitting on the uncomfortable jeep seats, in combination with uneven driving condition caused coccygeal trauma resulting in the disease [5]. The etiology of disease is not completely understood, but it is

S. Thomas · J. Nordenstam (✉)
Division of Colon and Rectal Surgery, Department of Surgery,
University of Illinois at Chicago, 840 South Wood Street, 518 E CSB,
Chicago, IL 60612, USA
e-mail: drjohan@uic.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_6

thought that the anatomy of the natal cleft is the culprit. The natal cleft has minimal subcutaneous tissue with the skin being closely adherent to the sacrum and coccyx. The gluteal region adds downward strain to this tightly adherent skin, due to the weight of the musculature. In addition to the anatomy, the natal cleft, like the axilla, is more likely to be an area of moisture and bacterial accumulation. The mechanics of walking allows the skin of the gluteal folds and natal cleft to rub against each other, causing the migration of debris as well as skin trauma. These factors contribute to pilonidal disease formation [5, 12].

The disease often has a chronic course, initially presenting with pilonidal abscess formation, with most patients experiencing disease recurrence [5, 7]. The disease affects males more often than females (3:1 prevalence), affecting males between the ages of 15–30 [2, 3, 5, 11, 12]. The most common presentation is pain, swelling, and/or drainage from the natal cleft [2]. The disease can also be asymptomatic in 3.7% of affected individuals, with one or more blind sinus openings in the natal cleft [3].

Management

The aim of treatment is to cure disease in the simplest way possible, while causing little pain and minimal effect on patients' lifestyles, while achieving low recurrence rates [1, 4, 6, 13]. Treatment can be divided into conservative and surgical approaches to management of both disease and recurrence (Figs. 6.1, 6.2 and 6.3).

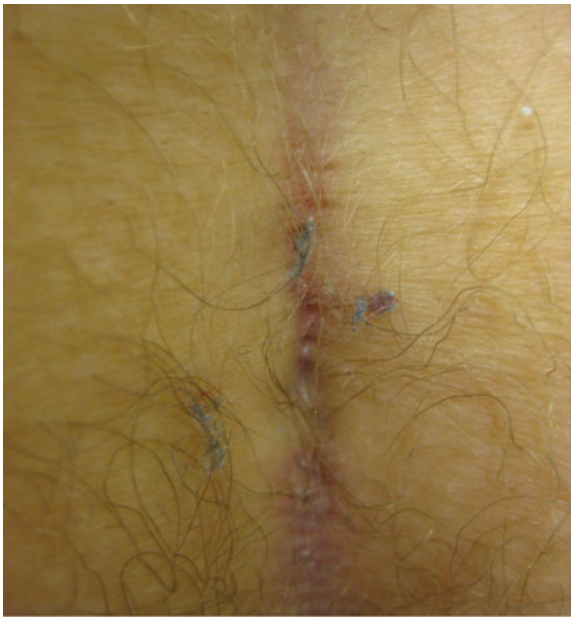


Fig. 6.1 Midline pits (Photograph courtesy of Charles O. Finne MD, Minneapolis)

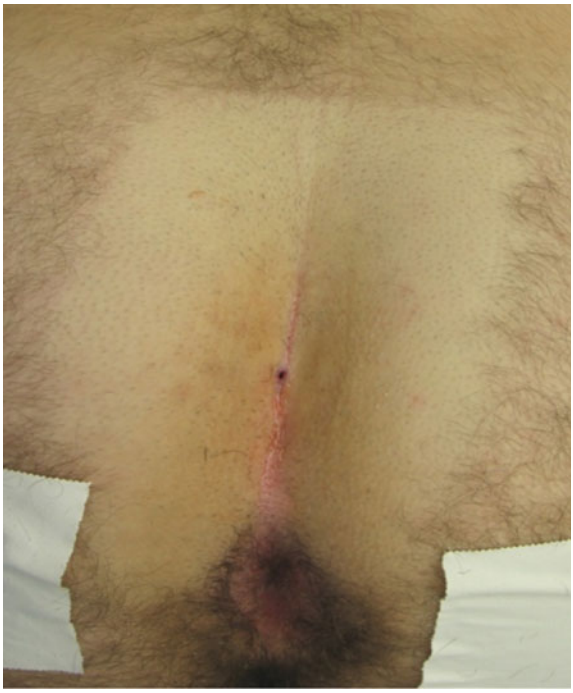


Fig. 6.2 Pilonidal sinus (Photograph courtesy of Charles O. Finne MD, Minneapolis)

Conservative Approaches

Controlling hair growth in the sinuses is important in preventing disease progression in early pilonidal disease and preventing recurrences as hair growth in the natal cleft has been linked to pilonidal disease [7]. Hair growth can be controlled by shaving, waxing, electrolysis, and use of depilatory creams [7]. Another technique for hair removal is laser depilation. Khan et al. had good results in preventing disease recurrence using this technique. Photoelectrolysis has the advantage of being able to reach deep areas not easily accessed by other techniques of hair removal [7]. The complications of laser depilation include skin erythema and irritation, hyperpigmentation or hypopigmentation, and skin crusting [7]. The paper emphasized the adherence to hair removal techniques and suggested that lapse in adherence as the cause in disease recurrence.

Phenol injection into pits has been suggested. The mechanism of action is thought to be due to destruction of the epithelium in the pit, leading to inflammation and scar formation [14]. The procedure is performed under local anesthesia on an outpatient basis. Weekly phenol instillation in addition to local hair removal has a success rate of approximately 60% [6] with recurrence rate of approximately 11% [15]. High recurrence rate is a disadvantage of phenol injection, and is thought to be due to inadequate phenol penetration of extensive sinus tracts [3]. The

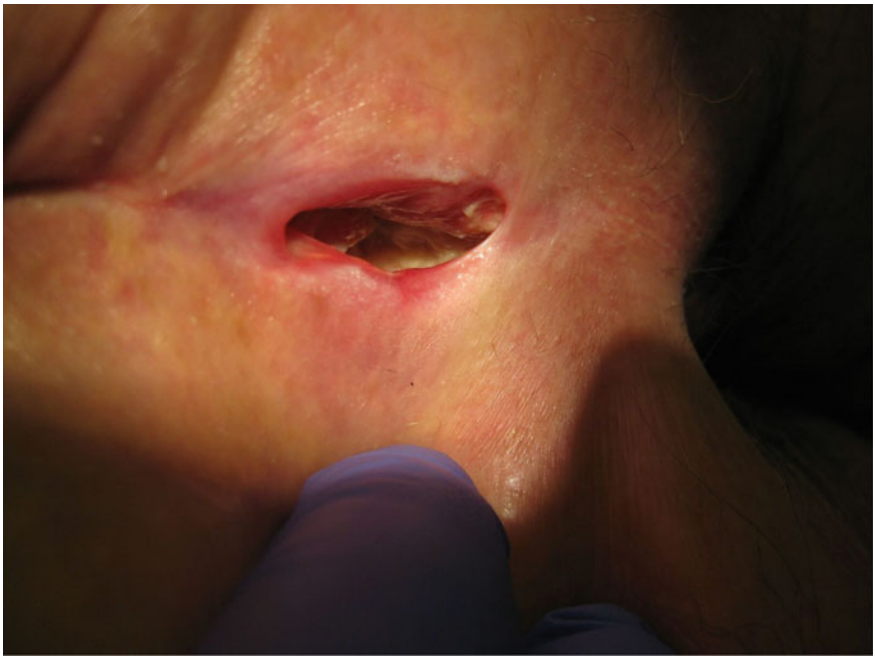


Fig. 6.3 Chronic pilonidal wound (Photograph courtesy of Charles O. Finne MD, Minneapolis)

complications of this therapy are local toxicity, resulting in skin irritation, burns, cellulitis, and abscess formation [6]. This is avoided by protecting the surrounding skin and with the application of ointment containing nitrofurantoin prior to phenol application which can reduce the risk of skin burns [6, 16]. Analgesia, topical anesthetics use, and wound care can aid in skin healing if phenol toxicity occurs (Figs. 6.4, 6.5, 6.6, and 6.7).

Antibiotic use has a limited role in conservative management of pilonidal disease. The use of preoperative antibiotics has not shown benefit in wound healing, preventing complications, or disease recurrence [16]. Equivocal data exists for the use of antibiotics postoperatively [2, 16]. In chronic pilonidal disease, antibiotic use is only recommended in cases of associated cellulitis, immunosuppression, and systemic illness [14].

Surgical Approach

Surgical approaches are offered when there is failure of conservative management and in cases of chronic pilonidal disease [1, 15]. There are several approaches ranging from minimally invasive procedures, such as pit picking and more extensive procedures such as wide local excision.

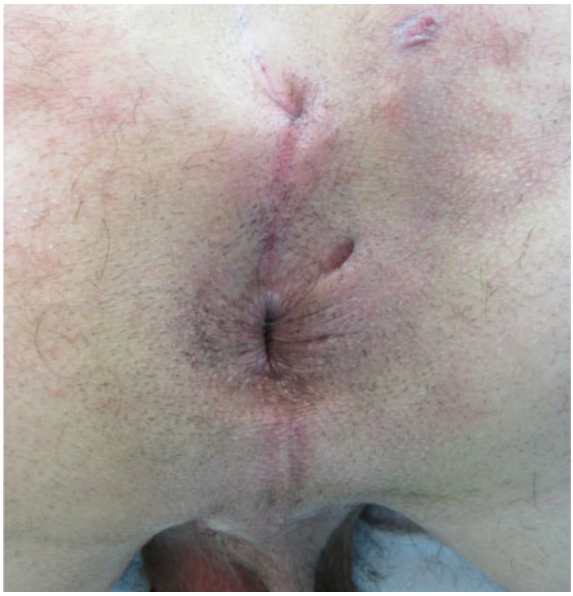


Fig. 6.4 Pilonidal sinuses

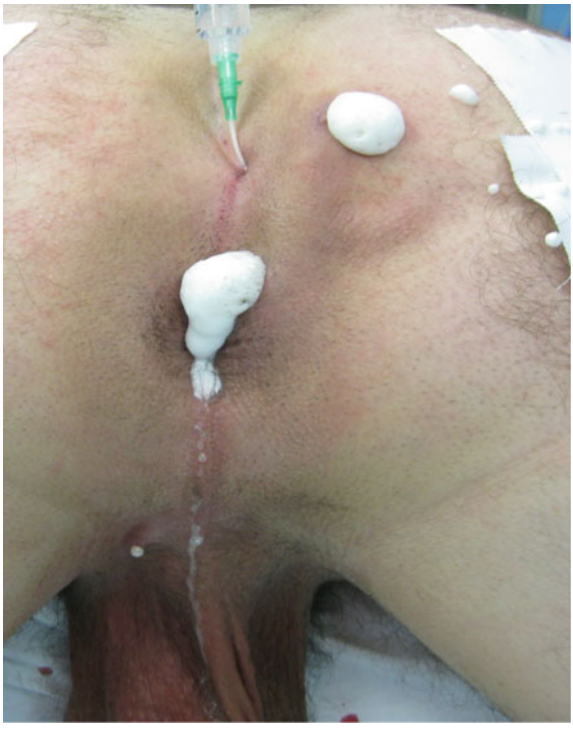


Fig. 6.5 Connection between pilonidal sinus tracts delineated following peroxide injection

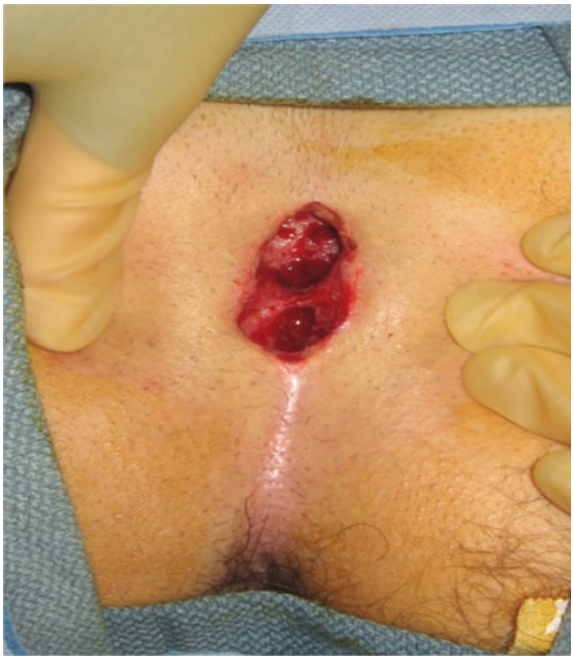


Fig. 6.6 Unroofing of pilonidal disease (Photograph courtesy of Charles O. Finne MD, Minneapolis)

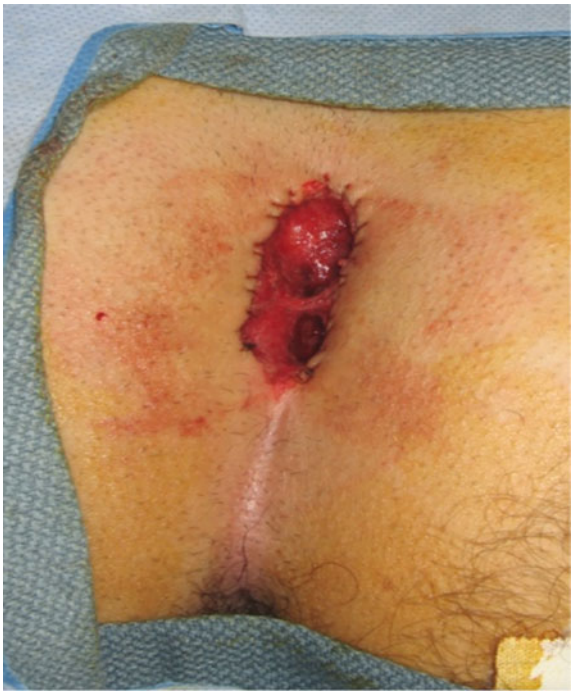


Fig. 6.7 Marsulpialzation following unroofing (Photograph courtesy of Charles O. Finne MD, Minneapolis)

In the acute stage incision and drainage vs needle aspiration followed by antibiotic course is recommended for acute pilonidal abscesses [14]. Incision and drainage results in complete wound healing in 60% of cases [15]. Definitive surgical excision is recommended after inflammation subsides, to address the resultant wound. Disease recurrence occurs in 10–15% of cases despite complete wound healing, as drainage of a pilonidal abscess does not address the underlying cause of its pathology [15].

Pit picking is one of the minimally invasive procedures. There are various methods of performing this type of surgery. A common feature in all these methods is the excision and removal of midline pits followed by drainage or curetting of the subcutaneous tissues. The aim of these techniques is to remove minimal amount of tissues. It is important to note that the sinus tract is not excised with these techniques. The advantage of this method is that it is performed on an outpatient basis, has short wound healing time and short recovery time. The disadvantage is a recurrence rate of approximately 20–25% in 5 year follow-up [14].

Sinusectomy first described by Soll et al. is another minimally invasive technique. The sinus tracts are probed and injected with methylene blue. The sinus tracts are then excised following the methylene blue delineation. The wounds are left open to close by secondary intent [17]. A recurrence rate of 5% was reported in the study [17]. This technique is recommended for patients with less than three pilonidal pits [14].

Unroofing and marsupialization (UM) of the sinus tracts is another surgical option [1]. In this procedure no healthy, normal tissue is removed and only affected tissue is incised [1]. This technique still results in a 1–2 cm open wound, but the wound is much smaller than the wound caused by wide local excision (WLE) [14]. Rouch et al. described a low recurrence rate with UM when compared to WLE in their retrospective review [1].

The most common procedure offered is wide local excision with or without closure [1, 2, 12, 14, 17]. In this procedure all of the involved tissue is excised and the resultant wound is either closed or left to close by secondary intent [1]. The technical approach of WLE is similar to sinusectomy and UM, in that the sinus tracts are probed and sometimes injected with methylene blue prior to being excised; however, the extent of excision is larger [14]. The disadvantage of allowing the wound to close by secondary intention is prolonged wound healing time, increased recurrence rate, patient effort in wound care and time off work [2, 14].

Midline and off midline closure is used in primary closure following WLE. Shorter time of wound healing is noted with primary closure. Off midline closure is shown to have faster healing rates, lower infection as well as lower recurrence rates compared to midline closure [2]. Three off midline procedures commonly used are the Karydakis flap, the Limberg flap and the cleft lift procedure (Bascom II). The advantage of off midline closure is that it first removes the chronically diseased tissue and second it flattens the natal cleft, thereby minimizing recurrence due to anatomic and mechanical stress [5]. Disadvantage of the off midline closure is tension on the suture line, resulting in wound dehiscence, and esthetic of ultimate

Table 6.1 Flap closure techniques following WLE and their complications

Flap procedure	Technique	Complication
Karydakis flap	Asymmetrical excision of pilonidal sinus and lateral closure of flap secured to sacrococcygeal fascia	Wound separation and delayed wound healing
Limberg flap	Rhomboid excision of pilonidal tissue using closure with a rotational fasciocutaneous flap	Surgical site infections and wound separation
Cleft lift procedure	Excision of midline pits with mobilization of healthy skin adjacent to the midline. Skin and subcutaneous tissue is apposed for off midline closure	Seroma, hematoma and wound separation

scar [18]. The most common complications following off midline flap closure is hematoma, seroma and wound separation [5, 11]. The use of drains intra-operatively may prevent the formation of seromas and hematomas. If wound hematoma or seroma develop, fluid aspiration with large bore needle is suggested. Wound separation is treated with wet to dry dressing applied to the region (Table 6.1).

Pilonidal disease can recur up to 20 years after surgery, but 60% will recur within 5 years [12]. Early recurrence in midline closures is thought to be secondary to the surgical site infection and occur in up to 24% of case that undergo WLE with primary closure [2, 3]. The administration of systemic antibiotics has been reviewed in several randomized controlled trials, showing no significant benefit [2, 14, 15]. Postoperative antibiotics can be used as an adjunct following surgical excision; however studies have shown mixed results in term of wound healing and recurrence rate [15]. Nyugen et al. suggested the use of gentamycin collagen sponge to reduce the local infection rates; however, the study did not reach statistical significance [2]. Other studies failed to show that the use of gentamycin improved wound healing and prevented disease recurrence [2, 15].

Complications

Regardless of whether conservative or surgical treatment strategies are utilized, pilonidal disease often leads to post-therapeutic complications, including poor wound healing and disease recurrence. Male gender, obesity, hirsutism, smoking, family history, poor hygiene, sinus size, and the surgical procedures are risk factors for complications and recurrence [7, 10–12, 15, 19]. One study, done by Lesalnieks, showed that smokers had increased postoperative wound complications following both minor surgical procedures as well as larger procedures with off midline closures [19]. Pilonidal disease recurrence was also reported to be increased in smokers when compared to nonsmokers [19]. Surgeon experience was also considered in disease recurrence. Pilonidal disease recurred in 44% of patients when the Karydakis flap was performed by an inexperienced surgeon, while the recurrence rate was 9% when performed by an experienced surgeon [19]. A correlation

also exists between sinus pit size and number of pits and disease recurrence [20]. Incomplete sinus tract excision results in disease recurrence [20]. Method of anesthesia also affected disease recurrence. Smaller and inadequate surgical excision with local anesthesia use had higher recurrence rates compared to either spinal or general anesthesia [20].

Wide local excision with primary closure minimizes wound healing time and has shorter recovery time prior to patients returning to work [2]. Off midline flap closures are preferred as these procedures have lower recurrence rate compared to midline closures [20]. Onder et al. suggested that primary midline closures had higher recurrence rates while flap closure had higher postoperative complications [20].

Minor postoperative complications, such as seroma, hematoma, local wound infections, and wound dehiscence is reported to between 16 and 17% following WLE and primary closure [21]. Should a seroma or hematoma develop, fluid aspiration is recommended. Intra-operative wound drain placement is used to prevent fluid accumulation. Antibiotics, be it systemic vs local, is used to address the complication of local wound infection. Wound separation is treated with local dressing (Figs. 6.8, 6.9, 6.10 and 6.11).

Irrespective of surgical technique chosen, hair removal and maintaining strict hygiene have been shown to prevent disease recurrence [16].

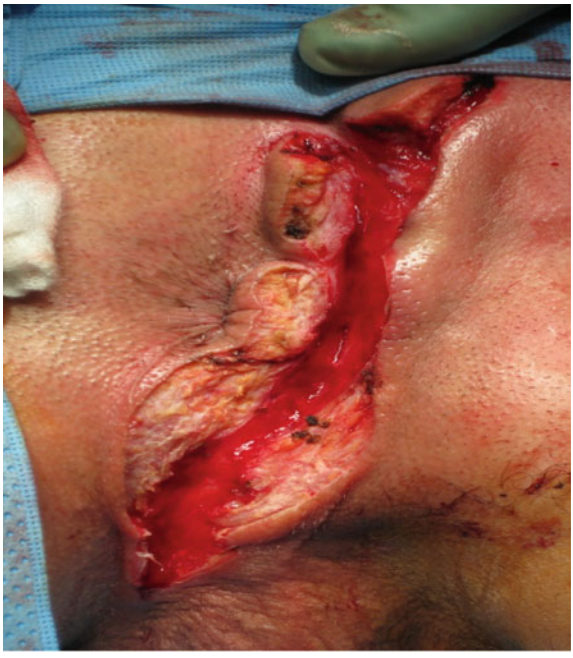


Fig. 6.8 Unroofing of extensive pilonidal disease

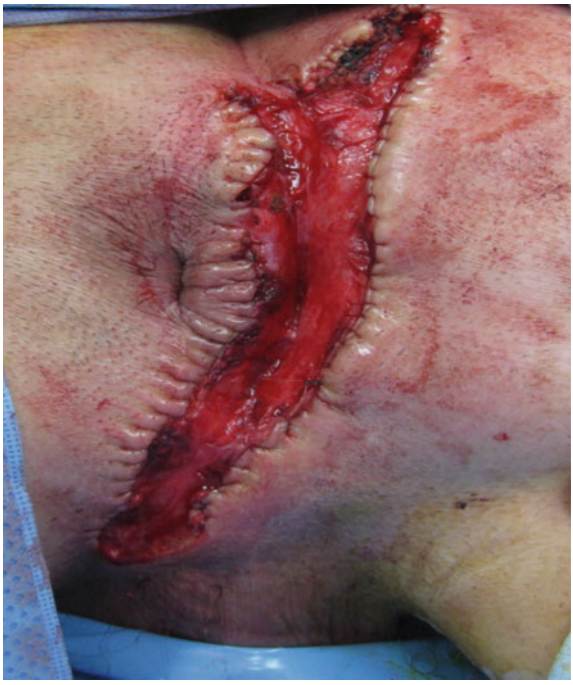


Fig. 6.9 Marsupialization

Malignant transformation is a rare complication of chronic recurrent pilonidal disease. Carcinoma developing in the pilonidal sinus tract is rare and occurs in less than 0.1% of cases of chronic, untreated, recurrent pilonidal disease [16, 22]. Chronic pilonidal disease is present for approximately 20 years prior to malignant degeneration [22, 23]. Squamous cell carcinoma is the most common carcinoma, occurring in 90% of cases. The remaining 10% is made up of basal cell, mixed squamous and basal cell, and adenocarcinoma [22]. The disease presents as an aggressive, rapidly progressing fungating ulcer [16]. The carcinoma is locally invasive but rarely has distant metastasis. Treatment of choice is en-bloc surgical excision with closure of the resultant defect with skin grafting or flaps [22]. The disease has a poor prognosis and high recurrence rate of 50% despite intervention [16]. Adjuvant chemotherapy and radiotherapy is used to reduce disease recurrence [23].

Misdiagnosis

Differential diagnosis for pilonidal disease includes hidradenitis suppurativa, congenital dermal tract, myelomeningocele, meningocele, dermoid cyst, tailgut cyst, teratoma, or lipoma to mention a few misdiagnoses.

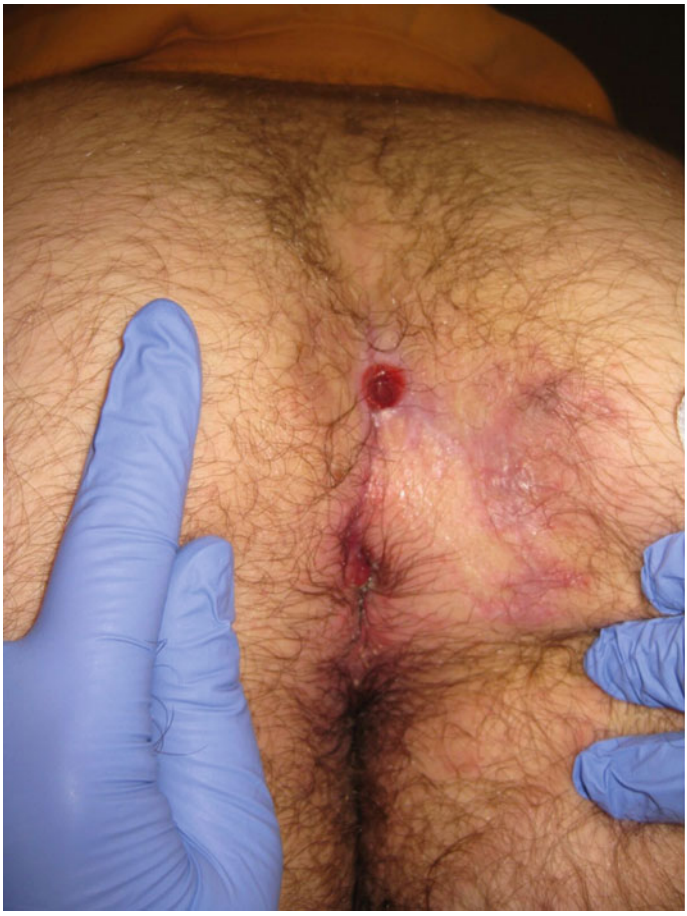


Fig. 6.10 Recurrent pilonidal cyst initially treated with Limberg flap (Photograph courtesy of Charles O. Finne MD, Minneapolis)

Hidradenitis suppurativa (HS) is a disease that affects skin with high concentration of apocrine glands especially the axilla, inframammary, inguinal, perineal, and perianal regions. The etiology of HS is thought to be secondary to occlusion of hair follicles, with resultant dilation, follicle rupture, and coalescing tract formation [24]. This is similar to the pathogenesis of pilonidal disease. If disease is confined to the perianal and perineal tissue, patients present with pain and malodorous drainage similar to that of pilonidal disease. On physical examination subcutaneous abscesses with multiple draining tracts are seen. Treatment ranges from conservative management to surgical management with wide local excision and wound closure by secondary intent [24].

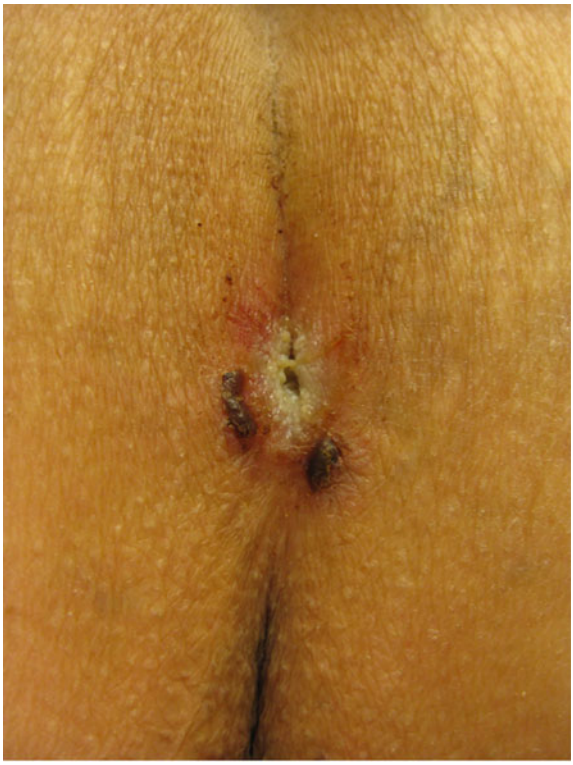


Fig. 6.11 Lichen Sclerosis minimking pilonidal disease (Photograph courtesy of Charles O. Finne MD, Minneapolis)

Congenital sinus tracts may be seen anywhere from the nose to the coccyx, occurring at the midline or adjacent to the midline [25]. The sinus tracts are lined with stratified squamous epithelium, like skin, and contain dermal appendages [25]. The tracts can extend as far as the spinal cord and may be complicated by meningitis or be linked to tracts ending in the subcutaneous tissue.

Tailgut cysts are congenital lesion in the retrorectal space, considered to be embryological remnants of postnatal intestinal tract [26]. As tailgut cysts are found in the retrorectal space they present with signs of mechanical obstruction to the rectal/anal canal or urinary system as the cysts increase in size. Tail gut cysts may be misdiagnosed as pilonidal disease and they can also incidentally found as a sacrococcygeal dimpling in the natal cleft [26]. Tailgut cysts may be surgically excised; however, this is associated with a high morbidity and complication rate [26].

Myelomeningocele, meningocele, and ependymoma are defects of the central nervous system that can occur along the central nervous tract in the sacrococcygeal region [27]. As these lesions present as a fluctuant mass in the sacrococcygeal region they may be misdiagnosed as pilonidal disease. The initial management would be to aspirate or incise and drain the lesion, which will not result purulent fluid. Surgical excision and pathological evaluation confirms diagnosis [27].

Understanding the epidemiology and disease presentation is important in effective diagnosis of pilonidal disease. Sending tissue sample for pathological evaluation will also aid in confirming diagnosis.

References

1. Rouch JD, Keeley JA, Scott A, et al. Short- and long-term results of unroofing and marsupialization for adolescent pilonidal disease. JAMA Surg. 2016. doi:10.1001/jamasurg.2016.0850
2. Nguyen AL, Pronk AA, Furnee EJ, et al. Local administration of gentamicin collagen sponge in surgical excision of sacrococcygeal pilonidal sinus disease: a systematic review and meta-analysis of the literature. Tech. Coloproctol. 2016;20(2):91–100. doi:10.1007/s10151-015-1381-7.
3. Furnee EJ, Davids PH, Pronk A, et al. Pit excision with phenolisation of the sinus tract versus radical excision in sacrococcygeal pilonidal sinus disease: study protocol for a single centre randomized controlled trial. Trials. 2015;16:92. doi:10.1186/s13063-015-0613-5.
4. Kanat BH, Sozen S. Disease that should be remembered: Sacrococcygeal pilonidal sinus disease and short history. World J. Clin. 2015;Cases 3(10):876–9. doi:10.12998/wjcc.v3.i10.876.
5. Favuzza J, Brand M, Francescatti A, Orkin B. Cleft lift procedure for pilonidal disease: technique and perioperative management. Tech. Coloproctol. 2015;19(8):477–82. doi:10.1007/s10151-015-1333-2.
6. Girgin M, Kanat BH. The results of a one-time crystallized phenol application for pilonidal sinus disease. Indian J. Surg. 2014;76(1):17–20. doi:10.1007/s12262-012-0548-y.
7. Khan MA, Javed AA, Govindan KS, et al. Control of hair growth using long-pulsed alexandrite laser is an efficient and cost effective therapy for patients suffering from recurrent pilonidal disease. Lasers Med. Sci. 2016;31(5):857–62. doi:10.1007/s10103-016-1920-0.
8. Bascom J. Pilonidal disease: origin from follicles of hairs and results of follicle removal as treatment. Surgery. 1980;87(5):567–72.
9. Bascom J. Pilonidal disease: long-term results of follicle removal. Dis. Colon. Rectum. 1983;26(12):800–7.
10. Demircan F, Akbulut S, Yavuz R, et al. The effect of laser epilation on recurrence and satisfaction in patients with sacrococcygeal pilonidal disease: a prospective randomized controlled trial. Int. J. Clin. Exp. Med. 2015;8(2):2929–33.
11. Bali I, Aziret M, Sozen S et al. Effectiveness of Limberg and Karydakis flap in recurrent pilonidal sinus disease. Clinics (Sao Paulo) 2015;70(5):350–5. doi:10.6061/clinics/2015(05)08.
12. Ortega PM, Baixauli J, Arredondo J, et al. Is the cleft lift procedure for non-acute sacrococcygeal pilonidal disease a definitive treatment? Long-term outcomes in 74 patients. Surg. Today. 2014;44(12):2318–23. doi:10.1007/s00595-014-0923-3.
13. Wang C, Yao Y, Cao Y. The integrative method “suture dragging and simplified vacuum assisted therapy” for complex pilonidal sinus disease. Case Rep. Surg. 2014;425–97. doi:10.1155/2014/425497.
14. Iesalnieks I, Ommer A, Petersen S, et al. German national guideline on the management of pilonidal disease. Langenbecks Arch. Surg. 2016;401(5):599–609. doi:10.1007/s00423-016-1463-7.
15. Steele SR, Perry WB, Mills S, et al. Standards practice task force of the American Society of C, Rectal S Practice parameters for the management of pilonidal disease. Dis. Colon. Rectum. 2013;56(9):1021–7. doi:10.1097/DCR.0b013e31829d2616.
16. Humphries AE, Duncan JE. Evaluation and management of pilonidal disease. Surg. Clin. North Am. 2010;90(1):113–24, Table of Contents. doi:10.1016/j.suc.2009.09.006.

17. Soll C, Hahnloser D, Dindo D, et al. A novel approach for treatment of sacrococcygeal pilonidal sinus: less is more. Int. J. Colorectal Dis. 2008;23(2):177–80. doi:10.1007/s00384-007-0377-9.
18. Saydam M, Ozturk B, Sinan H, et al. Comparison of modified Limberg flap transposition and lateral advancement flap transposition with Burow's triangle in the treatment of pilonidal sinus disease. Am. J. Surg. 2015;210(4):772–7. doi:10.1016/j.amjsurg.2015.03.031.
19. Iesalnieks I, Deimel S, Zulke C, Schlitt HJ. Smoking increases the risk of pre- and postoperative complications in patients with pilonidal disease. J. Dtsch. Dermatol. Ges. 2013;11(10):1001–5. doi:10.1111/ddg.12140.
20. Onder A, Girgin S, Kapan M, et al. Pilonidal sinus disease: risk factors for postoperative complications and recurrence. Int. Surg. 2012;97(3):224–9. doi:10.9738/CC86.1.
21. Karaca AS, Ali R, Capar M, Karaca S. Comparison of Limberg flap and excision and primary closure of pilonidal sinus disease, in terms of quality of life and complications. J. Korean Surg. Soc. 2013;85(5):236–9. doi:10.4174/jkss.2013.85.5.236.
22. Eryilmaz R, Bilecik T, Okan I, et al. Recurrent squamous cell carcinoma arising in a neglected pilonidal sinus: report of a case and literature review. Int. J. Clin. Exp. Med. 2014;7(2):446–50.
23. Matsushita S, Ohtake N, Mochitomi Y, et al. A case of squamous cell carcinoma arising in a pilonidal sinus. J. Dermatol. 2002;29(11):757–8.
24. Velasco AL, Dunlap WW. Pilonidal disease and hidradenitis. Surg. Clin. North Am. 2009;89(3):689–701. doi:10.1016/j.suc.2009.02.003.
25. Ikwueke I, Bandara S, Fishman SJ, et al. Congenital dermal sinus tract in the lateral buttock: unusual presentation of a typically midline lesion. J. Pediatr. Surg. 2008;43(6):1200–2. doi:10.1016/j.jpedsurg.2008.01.021.
26. Satyadas T, Davies M, Nasir N, Halligan S, et al. Tailgut cyst associated with a pilonidal sinus: an unusual case and a review. Colorectal. Dis. 2002;4(3):201–4.
27. McEachron KR, Gaertner WB. Extradural sacrococcygeal subcutaneous ependymoma misdiagnosed as pilonidal disease: case report and review of the literature. J. Surg. Case Rep. 7. 2016;doi:10.1093/jscr/rjw121.

Hidradenitis Suppurativa

7

Jacqueline Harrison and Francois Dagbert

Hidradenitis suppurativa is a cutaneous disorder involving apocrine gland bearing skin regions. Rich in apocrine glands, the perianal region is frequently involved, as well the gluteal, inguinal, and axillary regions. Women are more frequently affected than men (3:1), and obesity and cigarette smoking are known risk factors [1]. The course of the disease is variable, but frequently progresses to a chronic condition with subcutaneous abscesses, draining sinuses and extensive skin fibrosis. Even though medical therapy, as well as simple incision and drainage, may be adequate for the management of early, limited disease and acute infection, their role in the management of chronic, extensive disease is limited [2]. Recurrence rate of 100% after simple incision and drainage is common [3].

The treatment of chronic, severe hidradenitis suppurativa is primarily surgical. For patients with extensive disease, a staged procedure may be required. On average, patients suffer 10 years of active disease before undergoing radical excision [4]. The resulting wounds can take many weeks, even months, to heal completely and can be associated with significant morbidity and disability. Quality of life is adversely affected, by the disease and its treatment, and depression and anxiety are more frequent in patients with hidradenitis suppurativa [5]. There is extensive debate in the literature regarding the extent of excision of perianal hidradenitis suppurativa and options for closure of these often massive wounds. Frequent coexistence of inflammatory bowel disease and hidradenitis suppurativa can make diagnosis and treatment challenging. Practitioners need to be aware of the risk of malignancy associated with long-standing hidradenitis suppurativa, especially in the perianal and perineal regions, and appropriate treatment of this devastating complication.

J. Harrison (✉) · F. Dagbert
Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County,
1900 W. Polk Street, Chicago, IL 60612, USA
e-mail: Sbhayden@hotmail.com

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_7

Delayed Healing and Its Management

Surgical treatment of perianal hidradenitis suppurativa can result in wounds that may be difficult to manage. Healing by secondary intention is the most widely used method after wide local excision down to healthy tissue. These wounds require aggressive local wound care to achieve complete healing. Sitz baths or showers three to four times daily with frequent dressing changes are usually required to keep the area clean. Using this regimen, Thornton and Abcarian were able to achieve complete healing by secondary intention in 104 patients after wide local excision of perianal and perineal hidradenitis in a mean time of 3.5–7 weeks (3.5 weeks for small wounds, 7 weeks for wounds over 5 cm) [4]. Bocchini et al. presented similar results with a mean time to complete healing of 10 weeks after wide local excision and healing by secondary intention [6] while Balik et al. reported a mean time to healing of 12 weeks [7]. The time to complete healing correlates with the extent of excision. An early, aggressive surgical excision could therefore potentially shorten recovery time in these patients by reducing the surface of skin excision.

Management of extensive open wounds in the perineal or perianal area is often difficult. Multiple techniques have been described to reduce the time to complete healing of these large defects. Negative-pressure dressings have been used to help accelerate healing in chronic wounds by increasing oxygen tension and granulation tissue formation, decreasing bacterial counts, and preventing shearing forces on the wound [8]. They have also been used to bolster split-thickness skin grafts [9–11]. However, the application of such dressings in the perianal region is difficult because these devices require an air-tight seal at all times. Vacuum dressing application is mainly limited to buttock, natal cleft, or inguinal wounds with an adequate margin of normal skin from the anal verge.

Split-thickness skin grafting is commonly used for coverage of large skin defects [12]. Its use in perianal and perineal hidradenitis suppurativa is well described [2, 7, 13–17]. The use of skin grafts has been associated with a lower mean time to complete healing after wide local excision than healing by secondary intention (12.2 weeks for secondary intention versus 8 weeks for delayed skin grafting) [7]. Skin grafts are typically harvested on the thigh and morbidity from the donor site (aside from pain) is usually low. In many instances, a delayed skin graft will be performed two to three weeks after the excisional surgery. The results of split-thickness skin grafts are generally good. Partial graft loss is fairly frequent but can generally be managed with local wound care and healing by secondary intention. Bocchini et al. reported a 37.5% partial loss of skin graft in their series with 8% of patients requiring a new grafting procedure [6] while Harrison et al. reported a 45% incidence of partial skin graft loss without need for further grafting [18]. The main disadvantage of skin grafts is the need for immobilization, prolonged hospital stay, potential graft failure, and the necessity of a donor site.

Maeda et al. have described a reused skin graft technique with immediate grafting on the wound where the graft is harvested from the surface of the lesions before radical excision to eliminate the need for a donor site. They did not find any

clinical recurrence in the skin graft, even if histological evaluation revealed that buried epidermal cysts could cause recurrence [19]. More research is needed to validate this novel technique.

Another approach for reconstruction after wide local excision is the use of local flaps. Their utility is greatest for areas with the highest risk of contracture, especially in the groin crease [20]. Closure with local cutaneous flaps cannot be performed if the resection is too extensive and not enough skin is available for coverage. Good results have been reported with local rotation flaps or V-Y advancement flaps with shorter times to complete wound closure [6, 21, 22]. Local flap closure has also been advocated for non-healing chronic perianal wounds [23]. Minor wound dehiscence is frequent but generally can be managed with local wound care only [24]. Major dehiscence and infection is rare but can result in larger wounds even more difficult to manage than the original wide excision defect.

The use of diverting colostomy does not seem to be indicated in the vast majority of patients because the anoderm can usually be preserved, even with extensive resection. Diversion of the fecal stream may be indicated for debilitated patients or in some instances after a split-thickness skin graft for extensive perianal disease. Indwelling soft rectal tubes can be used to divert the fecal stream during the initial phase of healing [25].

Fistulas

The coexistence of perianal hidradenitis suppurativa and perianal fistulas is not uncommon, ranging from 2 to 14% according to different series [6, 15]. For this reason, an anoscopic examination should always be performed during surgical treatment of perianal hidradenitis, and some advocate colonoscopy in those with a diagnosis of perianal hidradenitis suppurativa to rule out coexisting Crohn's disease. Missed fistulas can lead to recurrent abscess and persistent chronic drainage. Hidradenitis itself can cause superficial fistula to the distal anal canal [26], while more proximal fistulas should raise the possibility of concomitant cryptoglandular disease or the diagnosis of Crohn's disease as there are no apocrine glands in the proximal anal canal [27]. Drainage of associated abscess and seton placement is the treatment of choice for associated perianal fistula to allow healing of the wound before considering definitive surgery for the fistula.

Perianal fistula associated with hidradenitis suppurativa should prompt evaluation for possible Crohn's disease. Crohn's disease is associated with hidradenitis suppurativa in 2–38% of patients and the perianal area is the most frequently involved site [15, 28–30]. In these patients, Crohn's disease is usually affecting the colon and rectum and severe rectal disease leads to a rate of proctectomy as high as 70%. Skin graft after wide local excision of perianal disease has been reported in a patient with well-controlled Crohn's disease with medical therapy [29].

Anal Stricture

Extensive excision of anoderm and perianal skin can lead to anal stricture. This is often avoidable in the treatment of perianal hidradenitis as the anoderm is usually spared but can complicate circumferential excision of severe perianal disease [6]. It has also been described following skin grafting of the perianal area. If resection near the anal verge is necessary, a staged excision may limit the risk of developing an anal stricture. Mild stricture usually responds to simple dilation but severe stricture may require anoplasty and local skin advancement flaps or circumferential full thickness skin grafting [31–33].

Recurrence of Perianal Hidradenitis Suppurativa After Surgical Treatment

Surgical treatment remains the mainstay of definitive therapy for patients with severe hidradenitis suppurativa. Surgical therapies for perianal hidradenitis include simple incision and drainage of acute abscesses, deroofing, and wide radical excision. Lack of prospective randomized controlled trials and heterogeneity of the existing retrospective data make comparison of results among different surgical treatments difficult. A 2015 meta-analysis by Mehdizadeh et al. looking specifically at recurrence of hidradenitis suppurativa after surgical management concluded that recurrence rates are lowest with wide radical excision (estimated average recurrence 13.0%), and moderate for deroofing procedures (27.0%) and limited excision (22.0%) [13]. Deroofing is a compromise between simple incision and drainage and radical excision. It consists of probe directed excision of all overlying skin with exposure of the floors of sinuses and fistulae, with curettage of granulation tissue of the fistulae, leaving islands of skin amidst the deroofed lesions so that the resulting wound is less extensive and healing more rapid [34–36]. It can be carried out with a knife, cautery, or laser.

Differences in recurrence rates among wound closure methods are to some degree a reflection of the underlying extent of tissue excision. The high rate of recurrence reported in some series with primary closure may reflect compromise of the extent of excision in order to make primary wound closure possible. The aforementioned meta-analysis by Mehdizadeh excluded patients who had wide excision and were allowed to heal by secondary intention without a flap or graft. The definition of radical excision is also somewhat variable, with some articles reporting the excision must reach underlying fascia, and some describing a deep excision of subcutaneous tissue down to normal appearing fat, with the lateral extent of excision to include all subcutaneous tracts [13]. One method of determining extent of excision was reported by Morgan and Hughes, which consists of administering intravenous atropine to block eccrine secretion. Oxytocin is then given to stimulate sweat secretion. The skin is painted with iodine, then starch powder in castor oil is applied. This process highlights the apocrine sweat gland

distribution as black dots where sweat contacts the iodine-starch concoction, guiding wide excision of all apocrine sweat glands [37]. Another described adjunct to defining extent of excision is the injection of methylviolet (Gentian violet), or methylene blue, 3–5 ml, into the sinus tracts to color the sinus tracts and fistulae and guide excision [38]. Although these methods have been described, the frequency of their use, especially the atropine/iodine starch method, is unknown. More commonly, excision is guided by tactile and visual clues to extent of diseased tissue, i.e., induration caused by inflammation, along with granulation tissue encountered if one should divide a sinus/fistula tract during excision, as well as the use of metal probes to probe fistula tracts and sinuses. While the usual means of spread of the inflammatory process of hidradenitis suppurativa is in a lateral fashion, mandating adequate width of excision of affected tissue, the process can also extend quite deeply into the subcutaneous tissue. Involvement of underlying bone leading to osteomyelitis, and fistulization to the anus or rectum has been described, but should also lead one to rule out other diagnoses, and to consider malignant degeneration.

Method of closure is less important a factor in recurrence when compared to the extent of excision, but may influence recurrence rates to some degree. Mehdizadeh's meta-analysis found that in the patients who had wide excision, recurrence was 15% for primary closure, 8% for flap closure, and 6% for grafting. Patients who were allowed to heal solely by secondary intention were excluded from this meta-analysis [13].

Unlike the axilla and groin, where contracture of the area may occur with healing by secondary intention, contracture is rare after wide excision of perianal hidradenitis [4].

Coexistence of Crohn's disease with hidradenitis suppurativa has been reported in the literature to be as high as 38% [28]. Yadav et al. reported that patients with inflammatory bowel disease were found to be 9 times more likely than the general population to develop hidradenitis suppurativa [30]. Certainly distinguishing between the two entities can be difficult, sometimes necessitating examination under anesthesia to elucidate the correct diagnosis. On examination of the anal canal, sparing of the anal canal from the dentate line and proximally points to a diagnosis of hidradenitis suppurativa, whereas involvement of the anal canal at the dentate line may indicate Crohn's disease or cryptoglandular fistula in ano [26]. This rule of thumb arises from the histologic absence lack of apocrine sweat glands proximal to the distal two-thirds of the anal canal [27]. However, Crohn's disease and hidradenitis suppurativa frequently coexist. Convincing evidence of this is the fact that the hidradenitis suppurativa in patients with both Crohn's disease and hidradenitis suppurativa often involves typical apocrine gland bearing areas far removed from the perianal region, such as the axillae. Recurrence or persistence of perianal lesions in those patients may warrant careful examination to rule out fistula in ano associated with Crohn's disease. Even if the diagnosis seems clear, before undertaking a wide local excision of perianal hidradenitis suppurativa, performing preoperative colonoscopy to rule out concomitant inflammatory bowel disease is

prudent, as unwittingly creating a large perianal wound in a patient with Crohn's proctitis can be disastrous.

Complex fistula in ano may also be confused with perianal hidradenitis suppurativa, especially if neglected, with development of multiple branches, and in horseshoe fistulas, with bilateral involvement of the ischiorectal spaces. Again, examination of the anal canal with identification of the primary fistula opening and its relationship to the dentate line, may clarify the diagnosis.

Squamous Cell Carcinoma Arising in Hidradenitis Suppurativa

At this point, squamous cell carcinoma arising in the setting of long-standing hidradenitis suppurativa is a well-known entity, in many ways analogous to Marjolin's ulcer arising in chronic burn wounds. Jackman found an incidence of squamous cell carcinoma arising in hidradenitis suppurativa of 3.2% [39] and one large series of 217 hidradenitis suppurativa patients reported an incidence of 4.6% [40]. Location of disease in the perineum appears to be a separate risk factor for development, as squamous cell carcinoma arising in axillary hidradenitis suppurativa has not been described in the literature [41]. All of the described cases occurred in the perianal, perineal, gluteal, thigh, groin, or vulvar regions. Although hidradenitis suppurativa is more common in women, extra-axillary hidradenitis suppurativa is more common in men, as is malignant degeneration to squamous cell carcinoma, with a 4:1 ratio. Typically, the duration of symptomatic hidradenitis is lengthy, with a mean duration of 25 years (range 3–50 years) [41]. It has been suggested that human papilloma virus infection may have a role in causation [42, 43], which would help to explain the lack of cases of squamous cell carcinoma in axillary or inframammary hidradenitis suppurativa. Prognosis is variable, but the diagnosis is often delayed, with a poor outcome. Two year mortality in Maclean's series from 2007 was 48% [41], and in Pena's review of 21 patients reported in the literature, the mortality rate was at least 43%, although time from diagnosis was lacking in many of those patients [44]. Of those whose time from diagnosis was noted, one died just 2 days after diagnosis, another, one month after diagnosis. Presence of regional lymph nodes virtually assures that cure is not achievable [41]. Wide radical excision is the treatment of choice in those patients without lymph node or distant metastases. Palliative radiation and chemotherapy as appropriate for anal squamous cell carcinoma should be considered for those patients whose disease is metastatic. Radical "palliative" surgery should be discouraged, especially if there is little hope of R0 resection. MacLean and Coleman present the case of a 50 year old man who 32 years after gluteal suppurative hidradenitis, was diagnosed with a squamous cell carcinoma within the hidradenitis. He was given chemotherapy and radiation, without surgery, with some relief of symptoms. The patient developed a second area of squamous cell cancer on the contralateral buttock 8 months later, and ultimately underwent radical excision of his disease from

both buttocks and perineum, abdominoperineal resection with pedicled rectus abdominis musculocutaneous flap and split-thickness skin grafts to close the resulting tissue defect, bilateral groin dissections, and right hip disarticulation. After hip disarticulation he developed a local recurrence on the buttock after 4 months and thereafter rapidly died, 26 months after his initial cancer diagnosis [41]. This case demonstrates the aggressiveness of this entity and serves as a warning to temper enthusiasm for attempts at surgical eradication in the face of relentless local disease. Altunay et al's series describes a 54 year old man with bilateral nodal metastases, 7 cm × 4 cm on one side, with contralateral lymphadenopathy. "The amputation of the lower half of the trunk from the upper level of the perlvis and the sacral region was planned, but the patient refused. He died 3 months later" [45]. Figures 7.1, 7.2 and 7.3 are from a 54 year old woman who suffered from

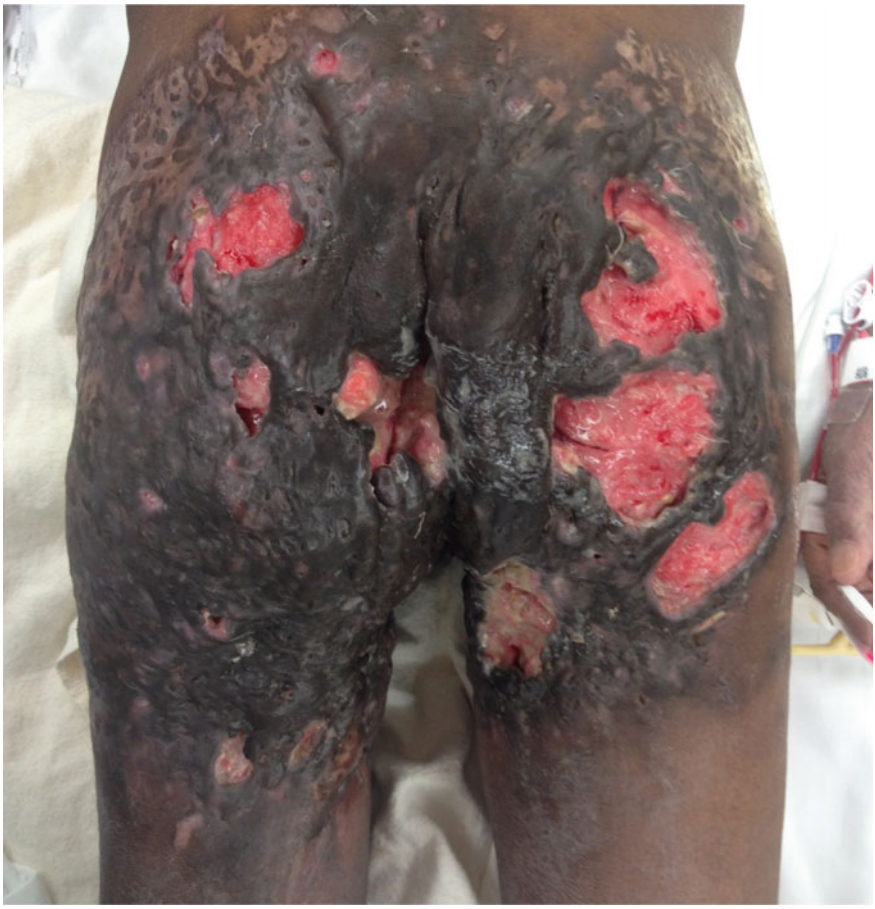


Fig. 7.1 Preoperative severe perianal, gluteal, and thigh hidradenitis suppurativa, of 27-year duration. She was found to have multifocal squamous cell carcinoma

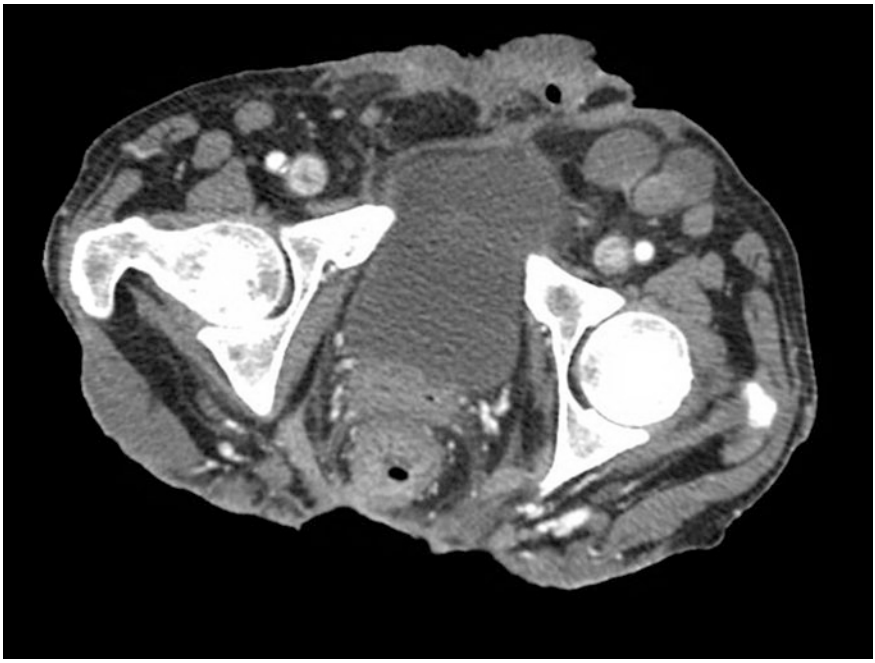


Fig. 7.2 Preoperative CT scan of the pelvis in a 54-year old woman with multifocal squamous cell carcinoma of the perineum arising in a background of hidradenitis suppurativa. Note the left groin lymphadenopathy

hidradenitis suppurativa of the axillae, pubis, groins, perineum, and thighs for over 25 years before presenting with unrelenting gluteal and perineal pain, associated with leg pain and swelling. Malignancy was not recognized preoperatively and she underwent wide resection of her hidradenitis. Multifocal squamous cell carcinoma was identified in the surgical specimen. Her last known follow up was less than 6 months postoperatively, when chemotherapy was suspended due to lack of efficacy and progression of disease, and she was referred to palliative care. At the time, she was bedridden secondary to debilitating pain and non-healing surgical wounds.

Certainly, squamous cell carcinoma arising in a background of hidradenitis suppurativa is a much more aggressive entity than squamous cell carcinoma in "normal" skin. Early diagnosis, aided by a high index of suspicion, and prompt surgical excision with adequate margins when an R0 resection can be achieved provides the only hope of cure. Multiple rounds of biopsies may be required to establish a diagnosis of squamous cell carcinoma in this setting. This malignancy has a propensity to spread along the subcutaneous tracts of the hidradenitis suppurativa so that the underlying malignancy in these tracts may be missed if biopsies are not adequately deep. Biopsies that are too superficial may reveal only atypical pseudoepitheliomatous hyperplasia. Repeat biopsy with deeper sampling is in order if clinical suspicion is high [40].

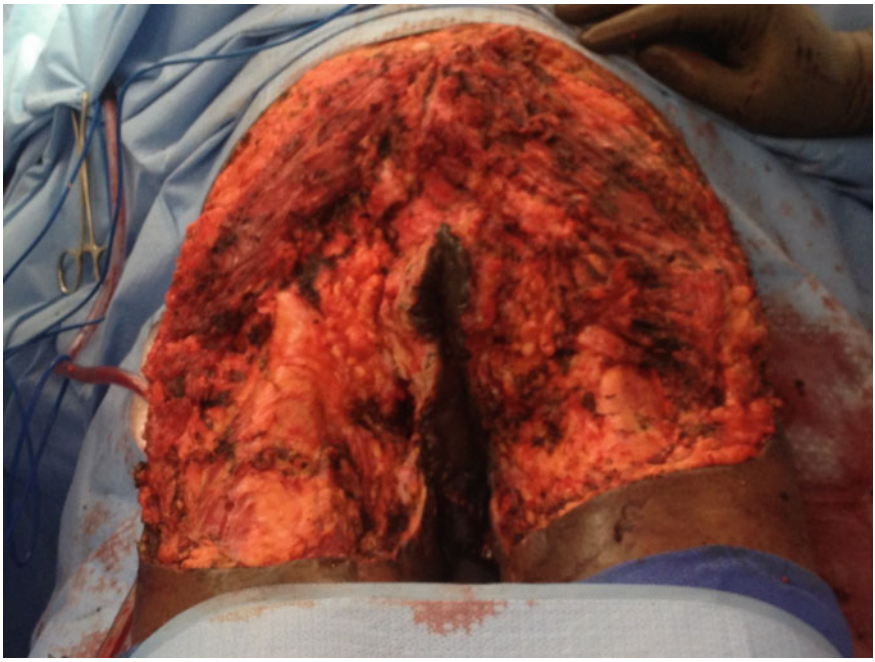


Fig. 7.3 Immediately postoperative after massive excision of severe gluteal and perineal hidradenitis suppurativa. The surgical specimen revealed multifocal squamous cell carcinoma. There are multiple areas of residual carcinoma

New case reports in the literature of this entity associated with the use of biologic treatments such as infliximab [40, 46] demonstrate the aggressiveness of squamous cell carcinoma in this setting, especially in combination with the immunosuppressive effects of monoclonal antibodies, and underscore the advantages of early surgical treatment of suppurative hidradenitis.

The Microbiology of Hidradenitis Suppurativa

One of the mainstays of therapy for mild hidradenitis suppurativa is antibiotic therapy. The etiology of hidradenitis suppurativa has long been debated. The most widely accepted theory posits that keratin plugging of the hair follicle leads to follicular dilation, rupture, and surrounding inflammation. What part bacteria play in the process is still under debate. Does bacterial invasion trigger inflammatory pathways? Or is bacterial invasion secondary? The fact that clindamycin, rifampicin, and tetracycline, which are three of the most effective antibiotics used in hidradenitis suppurativa, have known anti-inflammatory properties also clouds the issue. Ring et al. published a review of 9 studies from between 1988 and 2014 on

the bacteriology of hidradenitis suppurativa. Most of these bacteriologic studies did not confine themselves to one area, i.e., they contained patients suffering from axillary hidradenitis, inguinal lesions, vulvar and scrotal lesions, as well as perianal disease, or a combination of these. They also did not break down the microbiologic findings by site of collection [47]. A 1988 study by Highet et al., confined itself to microbiology of perineal hidradenitis suppurativa. This included perianal, upper thigh, inguinal, buttock, and genital lesions. The authors implicated *Streptococcus milleri* most significantly in causing disease exacerbation in their patients, and antibiotics clearing that bacterium were often successful in improving suppuration. *Staphylococcus aureus* was implicated as well, but to a lesser degree. Interestingly, coagulase negative staphylococcus (CoNS) was frequently cultured from patients, but was essentially disregarded as being a ubiquitous commensal and considered nonpathogenic [48]. Subsequent studies have lent CoNS much more of a role in propagation of the disease process because of its production of a biofilm ("slime"), a matrix of extracellular polymeric substance comprised of proteins, polysaccharides, and extracellular DNA. The bacteria which produce this biofilm are embedded in the slime, which gives them some protection from antibiotics as well as natural host defenses. Both *Staphylococcus epidermidis* and *Staphylococcus lugdunensis* are coagulase negative staph species that produce biofilm. Both have been implicated in hidradenitis suppurativa lesions [49–51].

Anaerobic actinomycetes (*Actinomyces turicensis, Actinomyces radingae, Actinomyces neuii,* and *Actinobaculum schaalii*) have been recovered from a majority of hidradenitis suppurativa lesions in later studies. These species are slow growing and difficult to identify, which may account for them not being cultured in earlier studies. They typically are difficult to eradicate and require prolonged antibiosis. They usually coexist with strict anaerobes [50, 52].

Finally, there exists a case report of a patient with gluteal hidradenitis suppurativa who underwent ileostomy and local incisions and drainages, fistulectomies, and unroofings, only to have his sepsis recur on ileostomy reversal. A large resection of one buttock was performed, with a finding of a 9 mm larva of *Ancyclostoma braziliense* (hookworm) in one of the abscess cavities. This larva is responsible for cutaneous larva migrans when it migrates through the skin causing serpiginous raised tunnels. In this case, it was felt that the larva may have incited further inflammation. It was not implicated as causing the hidradenitis suppurativa [53].

In summary, many bacterial species have been implicated in pathogenesis or superinfection of hidradenitis lesions, including skin commensals, such as coagulase negative staph species *S epidermidis and lugdunensis*, *Streptococcus milleri,* pathogens, such as *Staph aureus* and actinomycetes, as well as rarer bacteria such as *Bilophila wadsworthia* that colonize the GI tract, but have been implicated in other disease processes such as appendicitis [54]. Antibiotic regimens active against these may result in improvement in suppurative lesions, but recurrence after their cessation is the norm. In addition, many of the more common antibiotic regimens used in hidradenitis suppurativa employ clindamycin, tetracycline, and rifampin, that

have anti-inflammatory properties which may account for some of their beneficial effects in hidradenitis.

Summary

Perianal and gluteal hidradenitis suppurativa is a chronic, relapsing disease capable of causing patients great disability and decreased quality of life. There is still often a significant delay in diagnosis. While a myriad of medical treatments can afford temporary relief, surgical excision is the most effective means of cure. Prolonged disease can be associated with the development of squamous cell carcinoma. These neoplasms tend to be more aggressive than denovo squamous cell carcinoma. Early diagnosis and appropriate R0 resection, if possible, is the only means of cure. Patients with long-standing hidradenitis in the perianal and gluteal areas need to be adequately informed of the risk of development of cancer.

References

1. Jemec G. Hidradenitis suppurativa. N Engl J Med. 2012;366:158–64. doi:10.1056/NEJMcp1014163.
2. Kagan RJ, Yakuboff KP, Warner P, Warden GD. Surgical treatment of hidradenitis suppurativa: A 10-year experience. Surgery. 2005 Oct;138(4):734–40;discussion740–41.
3. Ritz JP, Runkel N, Haier J, Buhr HJ. Extent of surgery and recurrence rate of hidradenitis suppurativa. Int J Colorectal Dis. 1998;13(4):164–8.
4. Thornton JP, Abcarian H. Surgical treatment of perianal and perineal hidradenitis suppurativa. Dis Colon Rectum. 1978;21(8):573–7.
5. Shavit E, Dreiher J, Freud T, Halevy S, Vinker S, Cohen AD. Psychiatric comorbidities in 3207 patients with hidradenitis suppurativa. J Eur Acad Dermatol Venereol. 2015;29:371–6. doi:10.1111/jdv.12567.
6. Bocchini SF, Habr-Gama A, Kiss DR, Imperiale AR, Araujo SEA. Gluteal and perianal hidradenitis suppurativa: surgical treatment by wide excision. Dis Colon Rectum. 2003;46(7):944–9.
7. Balik E, Eren T, Bulut, Büyükuncu Y, Bugra D, Yamaner S. Surgical approach to extensive hidradenitis suppurativa in the perineal/perianal and gluteal regions. World J Surg. 2009;33(3):481–7.doi:10.1007/s00268-008-9845-9.
8. Elwood ET, Bolitho DG. Negative-pressure dressings in the treatment of hidradenitis suppurativa. Ann Plast Surg. 2001;46(1):49–51.
9. Ye J, Xie T, Wu M, Ni P, Lu S. Negative pressure wound therapy applied before and after split-thickness skin graft helps healing of Fournier gangrene: a case report (CARE-Compliant).Medicine (Baltimore). 2015;94(5):e426. doi.10.1097/MD.0000000000000426.
10. Petkar KS, Dhanraj P, Kingsly PM, Sreekar H, Lakshmanarao A, Lamba S, et al. A prospective randomized controlled trial comparing negative pressure dressing and conventional dressing methods on split-thickness skin grafts in burned patients. Burns. 2011p;37(6):925–9. doi:10.1016/j.burns.2011.05.013. Epub.
11. Chen E, Friedman HI. Management of regional hidradenitis suppurativa with vacuum-assisted closure and split thickness skin grafts. Ann Plast Surg. 2011;67(4):397–401. doi:10.1097/SAP.0b013e3181f77bd6.

12. Karian LS, Chung SY, Lee ES. Reconstruction of defects after Fournier gangrene: a systematic review. Eplasty. 2015;15:e18.
13. Mehdizadeh A, Hazen PG, Bechara FG, Zwingerman N, Moazenzadeh M, Bashash M, et al. Recurrence of hidradenitis suppurativa after surgical management: a systematic review and meta-analysis. J Am Acad Dermatol. 2015;73(5 Suppl 1):S70–7. doi:10.1016/j.jaad.2015.07.044.
14. Alharbi Z, Kauczok J, Pallua N. A review of wide surgical excision of hidradenitis suppurativa. BMC Dermatol. 2012;12(1):9. doi:10.1186/1471-5945-12-9.
15. Wiltz O, Schoetz DJ, Murray JJ, Roberts PL, Coller JA, Veidenheimer MC. Perianal hidradenitis suppurativa. The lahey clinic experience. Dis Colon Rectum. 1990;33(9):731–4.
16. Banerjee AK. Surgical treatment of hidradenitis suppurativa. Br J Surg. 1992;79(9):863–6.
17. Bohn J, Svensson H. Surgical treatment of hidradenitis suppurativa. Scand J Plast Reconstr Surg Hand Surg. 2001;35(3):305–9.
18. Harrison BJ, Mudge M, Hughes LE. Recurrence after surgical treatment of hidradenitis suppurativa. BMJ. 1987;294(6570):487–9.
19. Maeda T, Kimura C, Murao N, Takahashi K. Promising long-term outcomes of the reused skin-graft technique for chronic gluteal hidradenitis suppurativa. J Plast Reconstr Aesthet Surg. 2015;68(9):1268–75. doi:10.1016/j.bjps.2015.05.025. Epub. 2015 May 29.
20. Reiger UM, Erba P, Pierer G, Kalbermatten DF. Hidradenitis suppurativa of the groin treated by radical excision and defect closure by medical thigh lift: aesthetic surgery meets reconstructive surgery. J Plast Reconstr Aesthet Surg. 2009;62(10):1355–60. doi:10.1016/j.bjps.2008.04.035. Epub. 2008 Aug 8.
21. Orkin BA. Perineal reconstruction with local flaps: technique and results. Tech Coloproctol. 2013;17(6):663–70. doi:10.1007/s10151-013-0978-y. Epub. 2013 Feb 21.
22. Kishi K, Nakajima H, Imanishi N. Reconstruction of skin defects after resection of severe gluteal hidradenitis suppurativa with fasciocutaneous flaps. J Plast Reconstr Aesthet Surg. 2009;62(6):8005. doi:10.1016/j.bjps.2007.09.063. Epub. 2008 Apr 21.
23. Bernardi C, Pescatori M. Reconstructive perineoplasty in the management of non-healing wounds after anorectal surgery. Tech Coloproctol. 2001;5(1):27–32.
24. Chen ML, Odom B, Santucci RA. Surgical management of genitoperineal hidradenitis suppurativa in men. Urology. 2014;83(6):1412–7. doi:10.1016/j.urology.2014.01.011. Epub. 2014 Mar 28.
25. Menderes A, Sunay O, Vayvada H, Yilmaz M. Surgical management of hidradenitis suppurativa. Int J Med Sci. 2010;7(4):240–7.
26. Culp CE. Chronic hidradenitis suppurativa of the anal canal. A surgical skin disease. Dis Colon Rectum. 1983;26(10):669–76.
27. McColl I. The comparative anatomy and pathology of anal glands. Ann R Coll Surg Engl. 1967;40:36–7.
28. Church JM, Fazio VW, Lavery IC, Oakley JR, Milsom JW. The differential diagnosis and comorbidity of hidradenitis suppurativa and perianal Crohn's disease. Int J Colorectal Dis. 1993;8(3):117–9.
29. Kamal N, Cohen BL, Buche S, Delaporte E, Colombel J-F. Features of patients with Crohn's disease and hidradenitis suppurativa. Clin Gastroenterol Hepatol. 2016;14(1):71–9. doi:10.1016/j.cgh.2015.04.180. Epub. 2015 May 5.
30. Yadav S, Singh S, Edakkanambeth Varayil J, Harmsen WS, Zinsmeister AR, Tremaine WJ, et al. Hidradenitis suppurativa in patients with inflammatory bowel disease: A population-based cohort study in Olmsted County, Minnesota. clin gastroenterol hepatol. 2016;14(1):65–70. doi:10.1016/j.cgh.2015.04.173. Epub. 2015 May 5.
31. Maria G, Brisinda G, Civello IM. Anoplasty for the treatment of anal stenosis. Am J Surg. 1998;175(2):158–60.
32. Katdare MV, Ricciardi R. Anal stenosis. Surg Clin North Am. 2010;90(1):137–45. Table of contents. doi:10.1016/j.suc.2009.10.002.

33. Szeto P, Ambe R, Tehrani A, Cagir B. Full-thickness skin graft anoplasty: novel procedure. Dis Colon Rectum. 2012;55(1):109–12. doi:10.1097/DCR.0b013e318236b513.
34. van der Zee HH, Prens EP, Boer J. Deroofing: a tissue-saving surgical technique for the treatment of mild to moderate hidradenitis suppurativa leisons. J Am Acad Dermatol. 2010;63:475–80. doi:10.1016/j.jaad.2009.12.018.
35. van Hattem S, Spoo JR, Horvath B, Jonkman MF, Leeman FW. Surgical treatment of sinuses by deroofing in hidradenitis suppurativa. Dermatol Surg. 2012;38:494–7. doi:10.1111/j.1524-4725.2011.02255.x. Epub. 2011 Dec 30.
36. Lin CH, Chang KP, Huang SH. Deroofing: An effective method for treating chronic diffuse hidradenitis suppurativa. Dermatol Surg. 2016;42(2):273–5. doi:10.1097/DSS.0000000000000609.
37. Morgan WP, Hughes LE. The distribution, size and density of the apocrine glands in hidradenitis suppurativa. Br J Surg. 1979;66:853–6.
38. Rompel R, Petres J. Long-term results of wide surgical excision in 106 patients with hidradenitis suppurativa. Dermatol Surg. 2000;26:638–43.
39. Jackman RJ. Hidradenitis suppurativa: diagnosis and management of perianal manifestations. Proc R Soc Med. 1959;52:110–2.
40. Lavogiez C, Delaporte E, Darras-Vercambre S, Martin De Lassalle E, Castillo C, Mirabel X, et al. Clinicopathological study of 13 cases of squamous cell carcinoma complicating hidradenitis suppurativa. Dermatology. 2010;220(2):147–53. doi:10.1159/000269836. Epub. 2009 Dec 23.
41. Maclean GM, Coleman DJ. Three fatal cases of squamous cell carcinoma arising in chronic perineal hidradenitis suppurativa. Ann R Coll Surg Engl. 2007;89:709–12.
42. Brown MD, Zachary CB, Grekin RC, Swanson NA. Genital tumors: their management by micrographic surgery. J Am Acad Dermatol. 1988;18:115–22.
43. Cosman BC, O'Grady TC, Pekarske S. Verrucous carcinoma arising in hidradenitis suppurativa. Int J Colorectal Dis. 2000;15:342–6.
44. Pena ZG, Sivamani RK, Konia TH, Eisen DB. Squamous cell carcinoma in the setting of chronic hidradenitis suppurativa; report of a patient and update of the literature. Dermatol Online J. 2015;21(4). pii: 13030/qt9q9707dp.
45. Altunay IK, Godkemir G, Kurt A, Kayaoglu S. Hidradenitis suppurativa and squamous cell carcinoma. Dermatol Surg. 2002;28(1):88–90.
46. Verdelli A, Antiga E, Bonciani D, Bonciolini V, Volpi W, Caproni M. A fatal case of hidradenitis suppurativa associated with sepsis and squamous cell carcinoma. Int J Dermatol. 2016;55(1):e52–3. doi:10.1111/ijd.13006. Epub. 2015 Oct 16.
47. Ring HC, Mikkelson PR, Miller IM, Jenssen H, Fuursted K, Saunte DM, et al. The bacteriology of hidradenitis suppurativa: A systematic review. Exp Dermatol. 2015;24(10):727–31. 10.1111/exd.12793. Epub. 2015 Aug 21.
48. Highet AS, Warren RE, Weekes AI. Bacteriology and antibiotic treatment of perineal suppurative hidradenitis. Arch Dermatol. 1988;124(7):1047–51.
49. Lapins J, Jarstrand C, Emtestam L. Coagulase-negative staphylococci are the most common bacteria found in cultures from the deep portions of hidradenitis suppurativa lesions, as obtained by carbon dioxide laser surgery. Br J Dermatol. 1999;140(1):90–5.
50. Guet-Revillet H, Coignard-Biehler H, Jais JP, Quesne G, Frapy E, Poiree S, et al. Bacterial pathogens associated with hidradenitis suppurativa, France. Emerg Infect Dis. 2014 Dec;20(12):1990–8. doi:10.3201/eid2012.140064.
51. Ring HC, Emtestam L. The microbiology of hidradenitis suppurativa. Dermatol Clin. 2016;34(1):29–35. doi:10.1016/j.det.2015.08.010.
52. Nikolakis G, Join-Lambert O, Karagiannidis I, Guet-Revillet H, Zouboulis CC, Nassif A. Bacteriology of hidradenitis suppurativa/acne inversa: a review. J Am Acad Dermatol. 2015;73(5 Suppl 1):S12–8. doi:10.1016/j.jaad.2015.07.041.

53. Sciaudone G, Limongelli P, Selvaggi F. The image of surgical infection:Hidradenitis suppurativa and *Ancylostoma braziliense*. Surg Infect (Larchmt). 2008;9(2):217–8. doi:10.1089/sur.2007.077.
54. Baron EJ, Curren M, Henderson G, Jousimies-Somer H, Lee K, Lechowitz K, et al. *Bilophila wadsworthia* isolates from clinical specimens. J Clin Microbiol. 1992;30(7):1882–4.

Perineal Repair of Rectal Prolapse

8

Richard Nelson

What Are the Options?

A number that has floated around medical literature for several generations is 130. One hundred thirty operations have been described for the treatment of rectal prolapse. That has been interpreted to mean that none of them work very well. Or that it is still uncertain what the pathophysiology is of rectal prolapse: a hernia? an intussusception? a motility disorder? inevitable result of a defecation disorder? not enough lumbar lordosis? and so on.

Are there really 130? I do not know anybody who in fact has made the count. The most available methods for the most part are fairly simple in their conception, which is to ignore the pathophysiology and either suspend the rectum from above to prevent it dropping, or reduce the prolapse from below to prevent it from finding its way out. And in some cases remove a length of colon in the belief that what attaches the colon uphill will prevent further dropping. In this chapter, the focus will be on procedures accomplished from the perineum to prevent future prolapse. Without much data (i.e., randomized controlled trials—RCTs) to support this, there is also the hope that these might in some way ameliorate the defecation disorder that is always part of prolapse.

R. Nelson (✉)
Epidemiology/Biometry Division, School of Public Health,
University of Illinois at Chicago, 1603 West Taylor, Room 956,
Chicago, IL 60612, USA
e-mail: altohorn@uic.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_8

The procedures discussed will be:

Perineal Proctectomy, aka the Altemeier procedure
Anorectal mucosectomy, aka Delorme procedure
Anal circlage, the Theirsch, using wire, silastic, fabric, or biologic materials
Fully stapled perineal proctectomies (TRANSTAR and PSP)

All of these are often combined with a striated muscular tightening of the pelvic outlet/anal canal. Or anal circlage with a foreign body. My experience with the former I will deal with quickly and not again. A "levatorplasty" or otherwise plication of uninjured external sphincter feels like an anal canal with restored muscle tone immediately afterwards, and that tightness is totally absent 4 months later. One has to wonder about how possible it is to tighten striated muscle that has not been previously injured, as in childbirth, in which case the muscle is reinforced with scar tissue.

The chapter will not compare efficacy. That is way too difficult to sort out and is better described, as far as it can be, in the Cochrane review [1].

It is important first to deal with the elephant in the room. Are there any reports I found of a previously continent patient made incontinent by any of these procedures? Not that I could find.

Incontinence persisted in a very variable percentage of patients post surgery, as one would expect when the pelvic nerves and muscles were inured for very variable lengths of time by an obturation which is not much different that a vaginal delivery from the perspective of these pelvic nerves and muscles. But the case series available show pretty routinely some degree of improvement after prolapse repair.

So, on to complications. First the disclaimers. This is not a systematic review. The casual reports (i.e., case series, case reports, etc.) are so numerous and so haphazard in their trip to publication that an accurate assessment of the statistical risk of each complication would be at best very inaccurate and, even more, tedious. The most serious ones will be discussed, as in "How could this have happened? And is it likely to happen again?" Beneath that here is a scoring system for complications that is pretty broadly used: numbered from 1 to 4: 1 being minor complication requiring no interventions, four being death, three being loss of an organ, or lasting disability and two has lots of subdivisions depending on the invasiveness of the correcting intervention [2] (Fig. 8.1).

Altemeier Procedure

Trans-Anal Evisceration After Perineal Proctectomy

First of all, there is an urban myth that this could happen without previous surgery if one is corpulent and flushes an airplane toilet without first standing up, because of

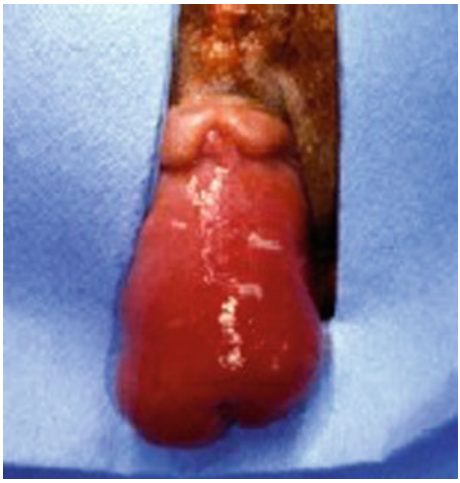


Fig. 8.1 A rectal prolapse

the negative pressure flushing mechanism. This has never been reported. There is one case report in JAMA in 1987 without photographs or an eye witness in which the individual involved said. "It all came out" [3]. Whether this was a small bowel evisceration through a ruptured rectum, or simply a rectal prolapse is not clear in that article. And in any case it was not in an airplane, but a cruise ship. There are documented cases with photographs and detailed clinical histories of children sitting on swimming pool drains with massive trans-anal small bowel eviscerations and massive small bowel loss [4]. There are also case reports of rectal rupture with small bowel evisceration in patients with known rectal prolapse, and no particular triggering event [5, 6] (Fig. 8.2). This seems also not to be rare. Morris cites 53 case reports in 2003 going back to the original report by Brodie in the Lancet in 1827 [7]. Screening PubMed since 2003, 15 more cases would be added for a total of 68 case reports. Not all of these were in patients with rectal prolapse, but at least 70% were.

To these I add one published case report of the same event shortly after a perineal proctectomy with presumed rupture of the anastomosis ([8], Fig. 8.3), and add an additional case of my own, never published. She was a patient from a mental hospital and prone to rather wild behavior. The photograph of my patient was taken the evening of her surgery, (Fig. 8.4), and immediately repaired by reduction of the small bowel via laparotomy and reinforcement of the anastomotic line, through which the small bowel had come. She had thereafter an uneventful recovery. In the published case report the prolapse did not occur until four days after surgery while straining to defecate in a 42-year-old male. The small bowel was necrotic by the time he made it back to the hospital and to surgery, so the anastomosis was taken down, the ileum resected and an end colostomy formed. A grade 4 surgical complication [2].

So did these two cases occur due to can anastomotic leak, a poorly constructed anastomosis, or a sudden vast increase in intra-abdominal pressure stressing an

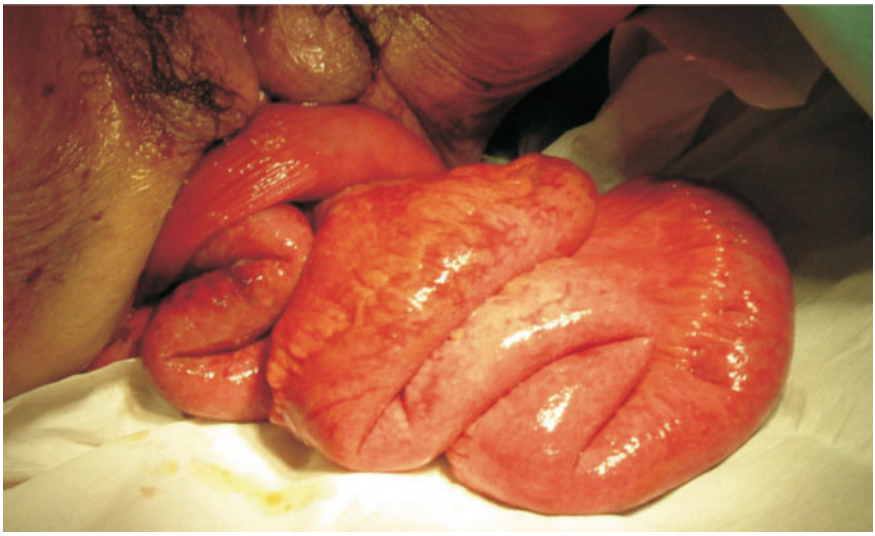


Fig. 8.2 Eviscerated small intestine in a patient with known rectal prolapse

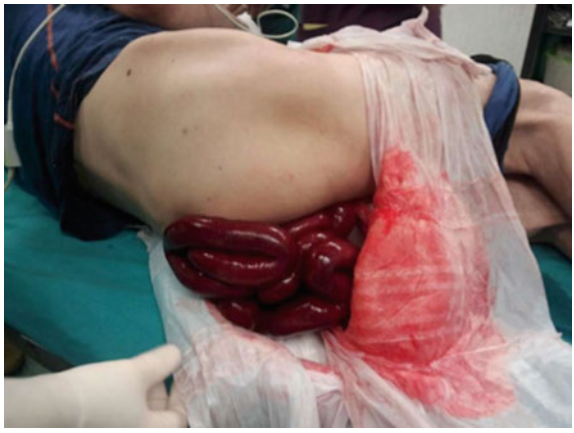


Fig. 8.3 Eviscerated small intestine in a patient who had had an Altemeier 4 days earlier

anastomosis? The other eviscerations that have been reported have resulted in the absence of an anastomosis. In the case of swimming pools and toilets, there was a large pressure gradient across the rectal wall. In the case of preexisting rectal prolapse, might there have been a solitary rectal ulcer not previously diagnosed? In any case it is not a rare event.

Anastomotic leaks have been often reported after perineal proctectomy, including four in the original paper by Altemeier in 1971 [9]. (Digression. Perineal proctectomies had been done for many years before Altemeier's 1971 series was published [10]. To the original operation he added a levatorplasty. But with the passage of time virtually all (non-stapled) perineal proctectomies have become

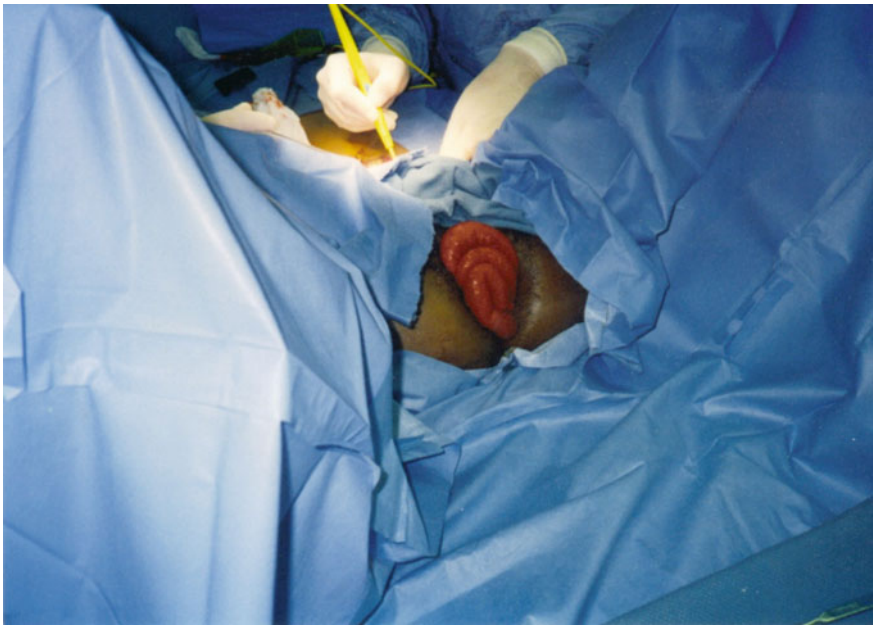


Fig. 8.4 Eviscerated small intestine in a patient who had had an Altemeier 8 hours earlier

known as Altemeiers.) There were eight leaks in a rather massive series of 518 patients reported from the University of Minnesota [11], a leak rate that compares very favorably with the published risk of leak of colonic anastomoses of between 7 and 8% in Holland [12]. Though some are described as asymptomatic, it is hard to imagine how or why they could have been found in the absence of symptoms. Many were treated by prolonged courses of intravenous antibiotics and bowel rest. A grade 2 complication. If a pelvic abscess results from the leak, trans-anastomosis drainage has been effective. In some cases stomas were done as well, which implies that the patient must have been pretty sick. No additional details were given. One case of massive surgical emphysema was reported extending through the retroperitoneum all the way to the patients face early in the post operative period [13] (Fig. 8.5a and b).

Ischaemia

Rectal ischemia or infarctions are not an unusual presentation of rectal prolapse that becomes incarcerated [14]. Indeed, perineal proctectomy is well suited to that presentation and as long as both ends after resection are healthy. However, acute ischaemic infarction of the segment of colon above the anastomosis in an Altemeier is also worth mentioning. There are no published case reports of such an event, but there is a case with which I am familiar. A patient had had a perineal proctectomy.

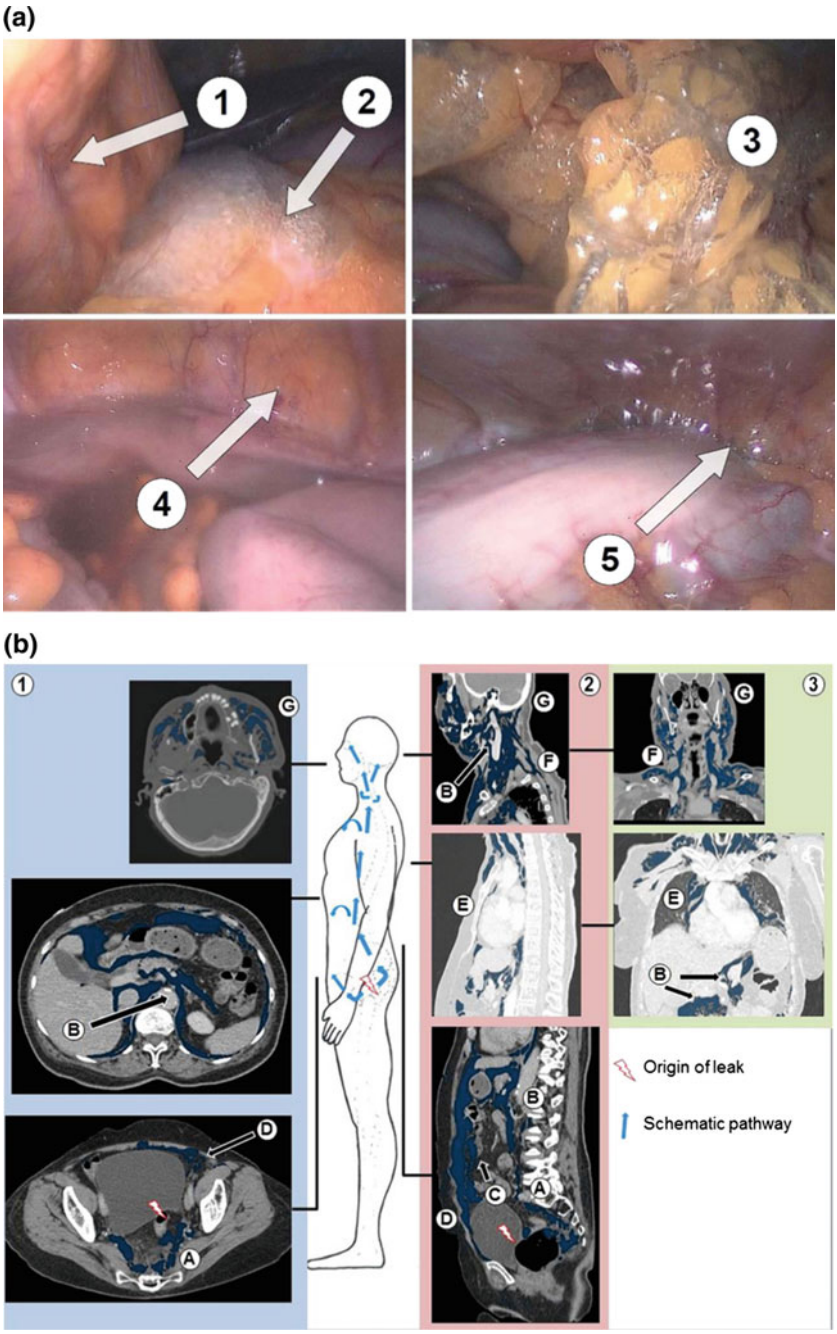


Fig. 8.5 **a** Emphysema seen at surgery in a patient with development of subcutaneous emphysema early in the postoperative period. **b** Mapping on CT of the extent of the patient's emphysema

She had a lower midline scar on her abdomen that was many years old and she was unable to tell where it came from. It was found too late to be from a previous sigmoid resection for an unknown disease. There was a segment of sigmoid colon that lost its blood supply from both above and below, and nothing available in between. The result of that event, because it was not suspected was not good.

However before abandoning perineal proctectomy because of the risk of ischemia, be aware that there is also a case of rectosigmoid ischaemia after a Delorme procedure, which is only a very limited mucosectomy [15]. How could such a thing happen? The published case report offers no suggestions beyond a proximal impaction (Fig. 8.6).

Bleeding

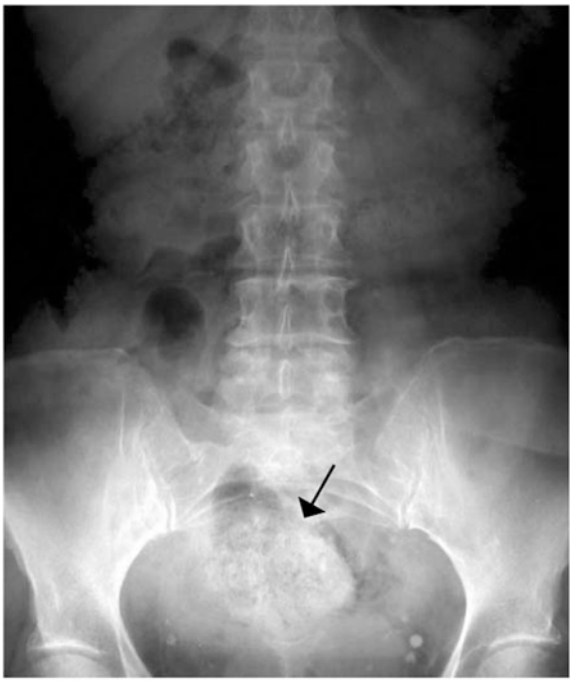
Bleeding is a prominent risk in all of perineal surgery from hemorrhoids to fistulas, and certainly for all perineal procedures for prolapse, with the possible exception of injection sclerotherpy [16, 17] (Injection sclerotherapy is also a procedure for rectal prolapse, especially in children, but with no reported complications). There are individual case reports of the need to return to surgery to suture the bleeding anastomosis but that is not unique to the Altemeier. But there is a special risk related to perineal proctectomy. Once the rectal mucosa and muscularis are divided proximal to the dentate line, mesenteric vessels are serially divided and ligated. As that progresses upwards, every surgeon who does this procedure thinks more and more about how well controlled the mesenteric division is. If a vessel slips away it will retract into the upper pelvis, completely out of reach. Laparotomy is the only option regardless of the fragility of the patient. There are no case reports of this occurring. None of the larger series specifically mention it. It has not happened to me. But I have spent plenty of time worrying about it.

Stricture, etc

Anastomotic stricture has been reported but is rare, in the one to 2% range. Of course it is easily accessed for repair. Urinary difficulties are frequently mentioned in reports, common in all disabled or hospitalized patients [18, 19].

Delorme Procedure

This operation interestingly, in cruising PubMed, is discussed in by far the most publications of all perineal operations for rectal prolapse. Yet it is one with which I have relatively little exposure. It is quite simple: a sleeve mucosectomy and corrugation of the submucosal muscle with anastomosis of the mucosa over the corrugated muscle. So it is sort of an autologous Thiersch. This and all treatments of



Plain radiograph shows fecal impaction in the rectum (*arrow*) without colonic distension

Histological analysis shows infarction involving the mucosa with abscesses inside the rectal wall. Hematoxylin-eosin stain original magnification X 125

Fig. 8.6 Fecal impaction in a previous Delorme who developed rectal ischaemia one month post op

rectal prolapse, as well as history, anatomy and physiology are discussed at length in the excellent review by Wu et al. [20].

As written above, what could go wrong with the Delorme? Ischemia did develop 4 weeks after surgery in a patient who had a fecal impaction just above the peritoneal

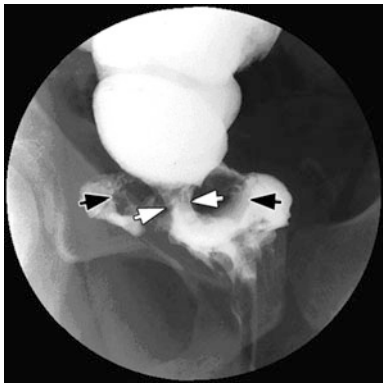


Fig. 8.7 Presumably what a barium enema should show in a patient after a Delorme

reflection requiring major surgery [14] (Fig. 8.6). Complications were not mentioned in many publications but as frequent as 45% in others. Suture line dehiscence was perhaps the most common serious complication and again its frequency and severity varied greatly. Several are described as asymptomatic but again, why would one look if there were no symptoms? Still, with an intact muscularis, the danger should have been small. Reoperation was mentioned in several publications, usually to control bleeding but even for fecal diversion after a mucosal suture line separation [21]. How could that happen? Stricture requiring dilation was reported in many publications [22]. The corrugation would in fact function as stated above as a muscular circlage (Fig. 8.7). Delorme in many publications is combined with either levatorplasty or circlage, a sort of belt and suspenders, which, among other things, added subcutaneous wound infection to its possible complications [23].

Reports Comparing Two or More Procedures

Several case series have been published in which there are comparisons of more than one operation in a prolapse population. These are not randomized trials so they are clearly subject to selection bias in allocation of patients to one procedure or another, particularly in those publications comparing an abdominal approach to a perineal one. Efficacy is not really assessable. But it was hoped that these comparative studies could provide information about relative risk of complications from a group of surgeons doing both in the same institution. Of the many, there are two comparing abdominal surgery to a Delorme. Interestingly, in one there were four deaths in the Delorme group, three in the early postoperative period including one small bowel perforation (??), and one six months later after a dilation of a rectal stricture [24]. The second study only reported a post op bleed requiring intervention in the Delorme group [25]. Altemeier was compared to Delorme in two studies. In one there were four leaks in 22 Altemeiers, three requiring stomas (!!) and one small bowel obstruction and in the Delorme group, one anal stenosis, a congestive heart failure, and two urinary tract infections [26]. Complications arose in 22% of Altemeier

patients and 7% of those having Delorme. In the second there were no complications in the Delorme group but two leaks in 32 patients, one death early and a hematoma requiring a stoma in the Altemeier meter group. In addition, there were four late strictures [27].

Thiersch

This much maligned procedure [28] is still the frequent subject of surgical publication. The silver wire has in most cases been replaced with more malleable materials such a silk sutures or elastic meshes [29]. Recurrences for the most part occurred when the circlage became infected, or ruptured or eroded into the rectum or peri-anal skin, and were removed. Chronic sepsis has also been reported after Thiersch, and can present in an area away from the anus [30] (Fig. 8.8) as in this scrotal abscess in a child. All perineal procedures are often described as preferred in the most fragile patients (more about that below) and the Thiersch even for the most

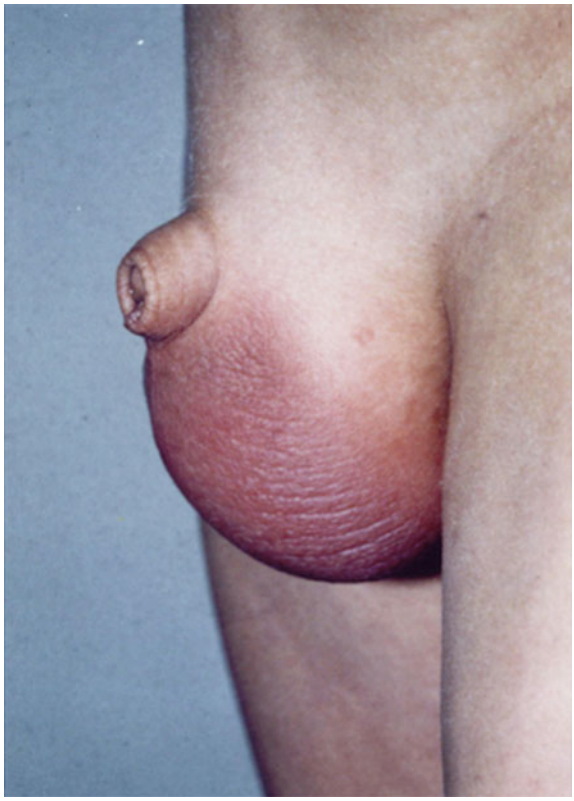


Fig. 8.8 Scrotal abscess in a child who had had a previous Thiersch procedure

fragile among the perineal procedures [31]. Post operative deaths have been reported but attributed to underlying disease. The goal of the procedure was simply to get the prolapse out of sight and with no real intention of improving bowel function.

Staples

Also know as Transtar (I think is stands for stapled trans-anal rectal resection), and PSP (perineal stapled prolapse), these are approximations of the Altemeier perineal proctectomy. Some of these are covered in other chapters. Being new technologies, the complications are reported in a bit more detail [32–34]. One publication described a leak treated medically, two bleeds requiring operation and two urinary retentions, one requiring suprapubic cystotomy. The second described a leak in one of 27 patients requiring a stoma and two bleeds, one requiring surgery, and two retroperitoneal hematomas. Leaving staples in the distal rectum may cause some of the difficulties seen in stapled hemorrhoidectomy [35].

Randomized Controlled Trials

Besides the advantages the RCTs provide in selection bias, they also would ideally provide more information about complications. Ethics committee approval, which is required for all RCTs, require the collection of all complications. These studies include:

Boccassanta [36]: compared two different instruments used for dissection in Altemeier resections in 58 patients. No complications were reported.

Deen [37]: Compared resection rectopexy to Altemeier plus post anal repair in 20 patients. There was one post op ileus in the abdominal group and one stricture in the perineal group.

Emile [38]: Compared ventral mesh rectopexy with Delorme in 50 patients. Very minor wound complications were seen in five patients in the first group and three in the second.

Senapati [39]: The PROSPER trial is a large multi center trial comparing two abdominal and two perineal procedures. 293 patients were randomized, 213 of them to perineal procedures comparing 106 Altemeier to 107 Delorme patients. But there was considerable attrition by the time of assessment of almost 50%. Four early deaths were reported in the perineal group: a ruptured aortic aneurysm in the Delorme group and in the Altemeier group a leak with sepsis, chest infection, and myocardial infarction. There were an additional four leaks in the Altemeier group.

Youseff [40]: Compared Delorme plus or minus a post anal repair with levatorplasty in 82 patients. There was one stricture and another suture line disruption in the sphincter repair group.

Rothenhoefer [41]: The DeloRes trial. This is published as only a protocol comparing Delorme to abdominal resection rectopexy so no outcome data are yet available.

So not a great deal of insight is added to this summary by these six RCTs.

References

1. Tou S, Brown SR, Nelson RL. Surgery for complete (full-thickness) rectal prolapse in adults. Cochrane Database Syst Rev. 2015 Nov 24;(11):CD001758. doi:10.1002/14651858.CD001758.pub3. Review. PubMed PMID: 26599079.
2. Dindo D, Desatines N, Clavien PA. Classification of surgical complications a new proposal with evaluation in a cohort of 6336 patients and results of a survey. Ann Surg. 2004;240(2):205–13.
3. Wynne JB. Vacuum toilet evisceration. JAMA. 1987;257(9):1177.
4. Neil R Price, S V Soundappan, Anthony L Sparnon, Danny T Cass. Swimming pool filter-induced transrectal evisceration in children: Australian experience. Med J Australia. 2010;192(9).
5. Kumar S, Mishra A, Gautam S, Tiwari S. Small bowel evisceration through the anus in rectal prolapse in an Indian male patient. BMJ Case Rep. 2013;6:2013.
6. Shoab SS, Saravanan B, Neminathan S, Garsaa T. Thiersch repair of a spontaneous rupture of rectal prolapse with evisceration of small bowel through anus—a case report. Ann R Coll Surg Engl. 2007;89(1):W6–8.
7. Morris AM, Setty SP, Standage BA, Hansen PD. Acute transanal evisceration of the small bowel: report of a case and review of the literature. Dis Colon Rectum. 2003;46(9):1280–3.
8. Di Lena Maria, Angarano Emanuele, Giannini Ivana, Guglielmi Altomarino. Donato Francesco Altomare. Strangulated ileal trans-coloanal-anastomotic hernia: A complication of Altemeier's procedure previously never reported. World J Gastroenterol. 2013;19(5):776–7.
9. Altemeier WA, Culbertson WR, Schowengerdt C, Hunt J. Nineteen years experience with the one-stage perineal repair of rectal prolapse. Ann Surg. 1971;173(6):99301001.
10. Cirocco WC. The Altemeier procedure for rectal prolapse: an operation for all ages. Dis Colon Rectum. 2010;53(12):1618–23.
11. Tiengtianthum R, Goldberg SM, Mellgren A. Clinical outcomes of perineal proctectomy among patients of advanced age. Dis Colon Rectum. 2014;57:1298–303.
12. Daams F, Luyer M, Lange JF. Colorectal anastomotic leakage: Aspects of prevention, detection and treatment. World J Gastroenterol. 2013;19(15):2293–7.
13. Valente F, Angehrn EJ, Däster S, Antonescu M. Massive surgical emphysema after perineal proctosigmoidectomy. BMJ Case Rep. 2014;. doi:10.1136/bcr-2014-206257.
14. Abdelhedi C, Frikha F, Bardaa S, Kchaw A, Mzali R. Altemeier operation for gangrenous rectal prolapse. S Afr J Surg. 2014;52(3):86–7. doi:10.7196/sajs.2157.
15. De Nardi P, Osman N, Viola M, Staudacher C. Ischemic proctitis following Delorme procedure for external rectal prolapse. Tech Coloproctol. 2006;10:253–5.
16. Chan WK, Kay SM, Laberge JM, Gallucci JG, Bensoussan AL, Yazbeck S. Injection sclerotherapy in the treatment of rectal prolapse in infants and children. J Pediatr Surg. 1998;33(2):255–8.
17. Zganjer M, Cizmic A, Cigit I, Zupancic B, Bumci I, Popovic L, Kljenak A. Treatment of rectal prolapse in children with cow milk injection sclerotherapy: 30-year experience. World J Gastroenterol. 2008;14(5):737–40.
18. Ris F, Colin JF, Chilcott M, Remue C, Jamart J, Kartheuser A. Altemeier's procedure for rectal prolapse: analysis of long-term outcome in 60 patients. Colorectal Dis. 2012;14(9):1106–11.

19. Pinheiro LV, Leal RF, Coy CS, Fagundes JJ, Martinez CA, Ayrizono ML. Long-term outcome of perineal rectosigmoidectomy for rectal prolapse. Int. J. Surg. 2016;32:78–82.
20. Wu JS. Rectal Prolapse: a historical perspective. Curr Probl Surg. 2009;46:602–716.
21. Lieberth M, Kondylis LA, Reilly JC, Kondylis PD. The Delorme repair for full-thickness rectal prolapse 2009 a retrospective review. Am J Surg. 2009;197(3):418–23.
22. Patel S, Levine MS, Rombeau JL. Appearance of the rectum on barium enema examination after the Delorme procedure. AJR Am J Roentgenol. 2007;188(4):W396.
23. Warwick AM, Zimmermann E, Boorman PA, Smart NJ, Gee AS. Recurrence rate after Delorme's procedure with simultaneous placement of a Thiersch suture. Ann R Coll Surg Engl. 2016;98(6):419–21.
24. Marchal F, Bresler L, Ayav A, Zarnegar R, Brunaud L, Duchamp C, Boise P. Long-term results of delorme's procedure and orr-loygue rectopexy to treat complete rectal prolapse. Dis Colon Rectum. 2005;48(9):1783–8.
25. Emile SH, Elbanna H, Youssef M, Thabet W, Omar W, Elshobaky A, Abd El-Hamed TM, Farid M. Laparoscopic ventral mesh rectopexy versus Delorme's operation in management of complete rectal prolapse: a prospective randomized study. Colorectal Dis. 2016 May 26.
26. Elagili F, Gurland B, Liu X, Church J, Ozuner G. Comparing perineal repairs for rectal prolapse: Delorme versus Altemeier. Tech Coloproctol. 2015;19(9):521–5.
27. Agachan F, Pfeiffer J, Joo JS, Nogueras JG, Weiss EG, Wexner SD. Resuts of perineal procedures for rectal prolapse. Am Surg. 1997;63:9–12.
28. Berkowitz J. Correction of rectal procidentia; the Thiersch operation as a simple palliative procedure. N Eng J Med. 1953;248(17):720–2.
29. Amar A, Jougon J, Hillion G, Leroux F, Chapel N, Egarnes M, Valyi L, Marry JP. Treatment of rectal prolapse with elastic circling of the anus: Perspectives of utilization. J Chir (Paris). 1996;133(4):183–5.
30. Saleem MM, Al-Momani H. Acute scrotum as a complication of Thiersch operation for rectal prolapse in a child. BMC Surg. 2006;28(6):19.
31. Naalla R, Prabhu R, Shenoy R, Hendriks IG. Thiersch wiring as a temporary procedure in a haemodynamically unstable patient with an incarcerated rectal procidentia. BMJ Case Rep. 2014;23:2014.
32. Boccasanta P, Venturi M, Calabro G, Maciocco M, Roviaro GC. Stapled transanal rectal resection in solitary rectal ulcer associated with prolapse of the rectum: a prospective study. Dis Colon Rectum. 2008;51(3):348–54.
33. Mistrangelo M, Tonello P, Brachet Contul R, Arnone G, Passera R, Grasso L, Rapetti L, Borroni R, Pozzo M, Roveroni M, Morino M, Perinotti R. Perineal stapled prolapse resection for full thickness external rectal prolapse: a multicentre prospective study. Colorectal Dis. 2016 Mar 11.
34. Sehmer D, Marti L, Wolff K, Hetzer FH. Midterm results after perineal stapled prolapse resection for external rectal prolapse. Dis Colon Rectum. 2013;56(1):91–6.
35. De Nardi P, Bottini C, Faticanti Scucchi L, Palazzi A, Pescatori M. Proctalgia in a patient with staples retained in the puborectalis muscle after STARR operation. Tech Coloproctol. 2007;11 (4):353–6.
36. Boccasanta P, Rosati R, Venturi M, Cioffi U, De Simone M, Montorsi M, Peracchia A. Surgical treatment of complete rectal prolapse: results of abdominal and perineal approaches. J Laparoendosc Adv Surg Tech A. 1999;9(3):235–8.
37. Deen KI, Grant E, Billingham C, Keighley MR. Abdominal resection rectopexy with pelvic floor repair versus perineal rectosigmoidectomy and pelvic floor repair for full-thickness rectal prolapse. Br J Surg. 1994;81(2):302–4.
38. Emile SH, Elbanna H, Youssef M, Thabet W, Omar W, Elshobaky A, Abd El-Hamed TM, Farid M. Laparoscopic ventral mesh rectopexy versus Delorme's operation in management of complete rectal prolapse: a prospective randomized study. Colorectal Dis. 2016 May 26. doi:10.1111/codi.13399. [Epub ahead of print] PubMed PMID: 27225971.

39. Senapati A, Gray RG, Middleton LJ, Harding J, Hills RK, Armitage NC, Buckley L, Northover JM; PROSPER Collaborative Group. PROSPER: a randomised comparison of surgical treatments for rectal prolapse. Colorectal Dis. 2013;15(7):858–68.
40. Youssef M, Thabet W, El Nakeeb A, Magdy A, Alla EA, El Nabeey MA, el Fouda Y, Omar W, Farid M. Comparative study between Delorme operation with or without postanal repair and levateroplasty in treatment of complete rectal prolapse. Int J Surg. 2013;11(1):52–8.
41. Rothenhoefer S, Herrle F, Herold A, Joos A, Bussen D, Kieser M, Schiller P, Klose C, Seiler CM, Kienle P, Post S. DeloRes trial: study protocol for a randomized trial comparing two standardized surgical approaches in rectal prolapse—Delorme's procedure versus resection rectopexy. Trials. 2012;29(13):155.

Rectocele Repair (ODS)

9

Steven Brown, Salvador G. Guevara and Linda M. Farkas

Complications After STARR and How to Deal with Them

The STARR procedure or stapled transanal resection of rectum was developed from the stapled haemorrhoidopexy or PPH (procedure for prolapsed haemorrhoids) operation. This operation was introduced the late twentieth century as a novel method of treating haemorrhoids [1]. The concept was based on the underlying theory of Thompson that the pathology of haemorrhoidal disease was related to damage to the supporting connective tissue of the anal cushions resulting in prolapse and subsequent kinking and engorgement of the contained vasculature [2]. By resuspending the anal cushions, a more physiological result was obtained. Theoretically at least, the restoration of normal anatomy would maintain the function of the haemorrhoidal tissue in, regards to continence and aiding evacuation. An additional advantage is a more rapid recovery after surgery. Unlike traditional haemorrhoidectomy there is no excision of the sensitive anoderm and pain, which can be a major issue for both Milligan-Morgan and Ferguson excisional procedures.

From the initial introduction of this PPH operation it became clear, by all of those who practiced the technique, that there were a group of patients who had more than just haemorrhoidal prolapse and that there was an element of internal full thickness prolapse or internal intussusception. These patients often described evacuation dysfunction or obstructed defaecation. A simple excision of the mucosal

S. Brown (✉)
Thornbury Hospital, 312 Fulwood Road, Sheffield
South Yorkshire S10 3BR, UK
e-mail: Steve.brown@sth.nhs.uk

S.G. Guevara · L.M. Farkas
Division of Colon and Rectal Surgery, Department of Surgery,
University of California, 2221 Stockton Blvd, Davis, Sacramento,
CA 95817, USA

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_9

element of this prolapse was not sufficient to resolve the dysfunction. More needed to be excised. Hence the STARR procedure was borne.

Utilizing the same instrumentation, the PPH operation was modified to allow this larger and deeper rectal wall excision [3]. Resolution of the obstructed defaecation was the aim and several studies subsequently suggested that this was the case in a significant proportion of patients. Modifications of the technique and the instrumentation (including the use of the Trans STARR mini-contour stapler) were made in order to achieve an even larger resection in the hope of better results.

Both the STARR and the PPH operations gained popularity in Europe throughout the beginning of the twenty first century. However, more recently, in the UK in particular, both procedures have become less popular. This is due to a variety of reasons including the suspicions about its effectiveness and alternative options for treating obstructed defaecation (including laparoscopic ventral rectopexy). However, another major influence on its falling popularity is the variety of complications that may occur, including some novel and challenging problems. These are the focus of this chapter.

Common Complications

Failure to Resolve ODS

Although the literature (including higher quality randomized controlled trials) would suggest the STARR procedure is very successful in resolving obstructed defaecation [4, 5], this is not the case in all patients. Even in the best hands and the most optimistic case series the STARR procedure can be estimated to resolve symptoms in about 60–70% of patients [4]. When carried out correctly, imaging studies postoperatively confirm that normal anatomy is invariably restored. It is likely therefore that restoration of normal anatomy is not the only cause to the evacuation dysfunction. Pescatori put this very succinctly when he described his surgical iceberg [6]. Obstructed defaecation can have a complex etiology including both anatomical (e.g., intussusception, rectocoele) and functional (e.g., anxiety/depression, anismus, neuropathy) elements. Correction of the anatomy may not be enough and merely uncover other underlying pathology resulting in ‘sinking of the surgical ship’.

Faecal Urgency and Incontinence

A large survey of practice in Europe identified common complications arising from the STARR procedure [7]. Top of the list and occurring in over 20% of patients was urgency. Almost all other case series describe the same phenomenon in some patients. This urgency can be severe, result in incontinence and have a significant effect on quality of life.

There are various explanations for the urgency. Damage to the sphincter complex is one obvious potential cause. The accessories that allow access to the rectal wall in order to carry out the STARR procedure have a diameter of 33 mm. Stretching of the sphincter complex may result in internal sphincter disruption [8]. However, this is unlikely to be the major cause of the urgency; the degree of dilatation is rarely sufficient to cause sphincter disruption [9, 10] and there is little evidence of sphincter disruption in those trials that have assessed the sphincters after surgery [11]. In addition, disruption of the internal sphincter does not usually lead to urgency; external sphincter injury is also necessary. An alternative explanation is that the urgency and any incontinence pre-exists surgery, and correction of the obstructive defaecation leads to 'unmasking' of these underlying symptoms [7]. It is the authors opinion that the most likely explanation of the urgency is the inflammation and edema that inevitably results around the staple line after surgery. This would explain why, with time, many patients have resolution of symptoms [12].

There may also be a contribution from the unavoidable reduction in rectal capacity and compliance that occurs if a section of rectal wall has been excised. One study suggested that patients with a preoperative rectal lumen diameter of <40 mm were more prone to urgency after STARR than those with a more capacious rectum [13]. Finally a more controversial explanation to the urgency is that it is part of a neuroendocrine enteric motility disorder [14]. This would seem unlikely but protagonists claim a response to H2 antagonists and baclofen.

In terms of other treatment options, reassurance that symptoms often improve with time combined with biofeedback and even neuromodulation for those with persistent symptoms is recommended [15]. However, avoidance is better than cure. Identification of a pre-existing weak pelvic floor due to age or previous childbirth is essential with caution in offering surgery to these patients and those with a small volume rectum.

Persistent Pain

Pain is another relatively common occurrence after both PPH and STARR. Like urgency the pain can also be severe and unrelenting. Such discomfort was first reported very early on in the use of the PPH stapling device [16, 17]. Again various explanations to the discomfort have been put forward. Of course one potential cause is incorrect technique. If the staple line is too low it may abut on the sensitive anoderm and result in discomfort [18, 19]. Fissuring and haematoma development and even infection may occur and should be excluded or treated appropriately. However, there remains a group of patients where no such explanation exists. The etiology of pain in these patients has been explained via various theories. Possibly the most credible is sphincter spasm. Hypertensive internal sphincter pressures have been demonstrated in several studies examining these patients. This allows a basis for treatment which is aimed at reducing spasm. Smooth muscle relaxants such as Nifedipine and GTN have been described and are worth trying [20, 21].

Other theories regarding the pain include staple line that impinge on pelvic floor musculature or even nervous tissue [22]. Excision of the scar has benefitted some individuals and is again worth considering. As with urgency there is a tendency for improvement with time, most resolving within 3 months [17].

Bleeding and Haematoma Formation

The incidence of significant bleeding may be as high as 5% of cases. Postoperative bleeding at the suture line was more frequently reported in early series [23] resulting in recommendations regarding technique. As much compression as possible should be used and a period of waiting before and after firing the staple gun and before releasing this compression is essential before firing the staple gun. Even then, meticulous haemostasis with under-running of any bleeding vessels should be carried out [23]. Use of the PPH03 gun, which provides greater compression of the staples, certainly reduces the bleeding potential, but does not allow such a large resection as the PPH01 [24].

Urinary Retention

With any pelvic floor procedure urinary retention can be an issue, particularly if there is a pre-existing urinary outlet flow compromise. Careful fluid management during surgery and adequate initial pain relief is essential. If occurs temporary catheterization may be necessary.

Stricturing

Mild stricturing after both the stapled haemorrhoidopexy and STARR procedures is a relatively common occurrence in the authors experience and is of no consequence or responds to mild dilation on per rectal examination if the patient is examined in the office a few weeks after surgery [25]. More severe stricturing probably relates to an element of sepsis or ischemia. However, technical errors including an oblique staple line can be avoided by keeping the instrument in perfect line with the longitudinal axis of the rectum during anvil closure [23]. In cases of severe stenosis a worsening of evacuation dysfunction may result. Intervention in the form of gentle dilatation under anesthetic often suffices with more severe cases requiring excision of the scar and re-anastomosis.

Rarer Complications

There are some rare and rather serious complications that have been described. These require significant surgical intervention to remedy, some are even life threatening. The potential for such events does create hesitation for the surgeon who is contemplating treating what is essentially a benign condition, with surgery only indicated in order to improve quality of life. Justification comes from the belief that these complications are rare and undoubtedly unlikely if the procedure is carried out carefully and correctly. However, it should be said that these serious complications may not be as rare as one thinks. A survey of 23 centers revealed that one third admitted to at least one serious complication occurring after a stapling procedure [26].

Rectovaginal fistula [7, 27, 28], rectal necrosis, perforation [29], lumen obliteration [30], small bowel injury [25, 27, 31] and rectal pocket syndrome [32] are examples of these rare but potentially serious complications. They usually relate to technical faults by the surgeon.

Regarding rectovaginal fistula, the vaginal wall should be assessed repeatedly during the procedure and it is essential that the wall can be moved independently from the rectum when the staple gun is closed. However, even when the vagina is assessed in this way, fistulae have been described, presumably related to relative ischemia or haematoma formation and subsequent infection.

Rectal necrosis is very rare. In some cases where it has been described it has related to poor patient selection. For instance, a STARR carried out in a patient who had undergone a previous resection rectopexy was the cause in at least one case [29]. Rectal necrosis may also relate to technique; spiraling of the staple line during a trans STARR procedure may result in an ischemic section of rectal wall.

Rectal lumen obliteration has been described during a PPH procedure presumably when the purse-string is tied beside the stapler anvil rather than around the anvil [30].

Small bowel injury may theoretically occur if there is an enterocoele sufficiently large and low enough to be incorporated in the stapling device. Certainly such an anatomical abnormality should be checked for both during the preoperative workup and intraoperatively via bimanual palpation. Any suspicion was initially considered by many as a contraindication to STARR [25, 27, 31] but subsequent consensus opinion is that STARR can be carried out safely if combined with tilting the table to allow the enterocoele to fall away [33] or even laparoscopy to lift the bowel out of the pelvis [34, 35].

Rectal pocket syndrome [32] is a term that has been used to describe a cul-de-sac near the staple line causing entrapment of fecal material which may result in severe proctalgia and soiling. This has also been reported following PPH, and it probably represents a failure in complete placement of the purse-string favoring fecalith deposition with formation of a chronic abscess and intramural fistula. It can be treated by suture line revision, staple removal, and curettage.

Retroperitoneal sepsis deserves a specific mention. This is a well-recognized phenomenon and has been described after numerous procedures for haemorrhoid treatment [36]. The common presenting symptoms are unexpected abdominal or perineal pain, urinary retention or difficulty micturating and fever. Findings at surgery may be minimal, consisting of oedema and purulent peritoneal fluid or just general inflammation. However, in many cases there may be a predisposing cause indicating a suboptimal operation. For instance a deficient staple line or inadvertent rectal perforation may lead to the sepsis [37, 38].

Conclusion

With an efficacy of 60–70% even in the best hands and an overall complication rate of about 36%, some complications being severe and possibly even life threatening, it would appear reasonable to dismiss the STARR procedure as a bad idea. However, in its favor is the fact that it does resolve symptoms in a significant proportion of patients and significantly more patients than respond to conservative therapy according to the small quantity of good quality literature available [4, 5]. Many of the complications relate to poor technique and adequate training and experience is essential. Poor results also inevitably relate to poor patient selection. Apart from the above factors that have been mentioned, concomitant psychoneurosis may play a very prominent role in poor outcome with one group suggesting a success rate for STARR dropping from 74 to 26% in those with associated psychological pathology [39].

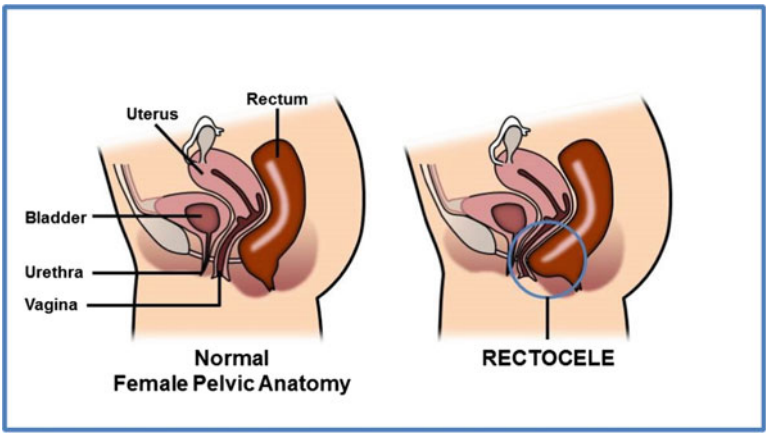
It should be remembered that the STARR procedure has had a beneficial effect on the development of colorectal pelvic floor surgery. Interest and research in this area has increased exponentially and we now have a better understanding of the pathophysiology of obstructed defaecation. The industry backing in promoting this procedure has enabled networks of interested pelvic floor surgeons to be formed and in turn newer operations have been developed. Further understanding should result in better patient (and operation) selection, better training and experience and an overall improvement in outcome.

Complications of Rectocele Repair

As rectoceles are prevalent in women, rectocele repair is a commonly performed procedure, executed by gynecologists and colon and rectal surgeons. A common difficulty in treating rectoceles is that they are not typically found in isolation, but frequently in association with other structural abnormalities noted on physician examination and imaging, e.g.: enterocele, sigmoidocele, colpocele, rectal intussusception, and solitary rectal ulcer, to name a few. In addition there are associated functional disorders: anismus, pudendal neuropathy, irritable bowel syndrome,

rectal hypo sensation, and anxiety/depression. Therefore it is no surprise that the array of treatment modalities ranges from dietary fiber, laxatives, enemas, pelvic floor pelvic training, psychotherapy, to surgery by transanal, trans perineal or transvaginal or abdominal approach. This chapter will address the complications of transanal, trans perineal and transvaginal approaches. The stapled transanal rectal resection (STARR) is also discussed.

The most common complications are nonsuccess and recurrences. Complications such as dyspareunia and incontinence are also commonly reported. To optimize outcome, due to the complexity of the presentation of the rectoceles with concomitant structural and functional disorders, careful patient selection extensive workup with multi-disciplinary input, optimization of bowel function, clear outcome expectations and frequently psychological evaluation prior to intervention is necessary.



Used by permission from the American Society of Colon and Rectal Surgery.

Introduction

Rectoceles are described as an outpouching and a bulge into the posterior wall of the vagina. There is a loss of the intervening layer and the rectum is in direct contact with the vaginal wall. The bulge can reach to the level of the hymen or in most severe may descend below the hymen. Rectoceles are prevalent and noted in 12.9–18.6% women with an average annual incidence of 5.7 cases per 100 women years [40, 41].

While the anatomic presence of a rectocele is quite common, most women are not symptomatic. Therefore careful assessment and treatment strategies are necessary prior to embarking on the correction of an anatomical abnormality, which

can lead to potential complications and/or failure to resolve symptoms. While frequently there are other associated disorders with bulging of other organs into this area due to the weakening of the pelvic floor, such as cystocele, this chapter will focus only on the isolated rectocele treatment strategies and their complications.

This weakening of the pelvic floor can occur from an amalgamation of various processes. These can include excessive straining from multiple etiologies such as: anxiety, constipation, and discordant bowel evacuation. Trauma (birthing, chronic coughing, obesity or gynecological/anorectal surgeries) can damage to and weaken of the pelvic floor. Straining due to any primary root cause, can not only create damage to the pelvic floor muscles, but also lead to pudendal nerve stretch, which may impair further sensation, and aggravating constipation.

Presentation and Workup

While most rectoceles are asymptomatic, others can have a plethora of symptoms, and an initial assessment, carefully teasing out these complaints, needs to be performed to discern how much of the symptoms can be attributed to the presence of the rectocele. This is beyond the scope of this chapter and includes incomplete evacuation, fragmentation of bowel movements, fecal incontinence, fecal leakage, or aided man evers for bowel evacuation such as pressing fingers on the perineum or against the posterior wall of the vagina, or significant repositioning techniques on the toilet, or frank digitation and disimpaction. The mechanical bulge can lead to complaints of vaginal fullness or dyspareunia.

Workup includes a minimum of physical exam and endoscopy. Further workup strategies including: transvaginal ultrasound (TVUS), defecography, anorectal manometry (ARM) and balloon expulsion, entero-defecography, dynamic perineal ultrasound, magnetic resonance imaging (MRI) defecography, pudendal nerve terminal motor latency (PNTML) and psychologic evaluation have all been described [42–48]. Workup is dependent on physician preferences, the available facilities, and other concomitant patient complaints.

Surgical Approaches and Their Complications

The three main non-abdominal approaches for an isolated rectocele repair are: transvaginal, transperineal and transrectal. The aim of this chapter is to discuss the common complications that occur with each of these.

Transvaginal Approach

The transvaginal approach consists of an incision on the posterior vaginal wall and eventual plication of the rectovaginal fascia. Incorporating the underlying levator ani muscles with interrupted sutures from the levator plate to the perineal body, while reducing the anterior rectal wall, is the classic "posterior colporrhaphy". The excess vaginal tissue is excised and repair is completed. This technique tends to be favored by the gynecologists given their natural comfort with a transvaginal operations. It also allows an avenue to address other gynecological concerns such as concomitant vaginal hysterectomy or cervical amputation. Since most of the reported studies describing transvaginal approaches are performed by gynecologists, the great majority of patients do not have the functional preoperative workup to assess the type of constipation (outlet obstruction vs. slow transit) or the imaging (defecography) that colon and rectal surgeons tend to employ. Much of this variability is due to differences in training, but this may also be by natural selection, as the gynecologists' patients will have presenting symptoms typically more gynecologically focused: i.e., vaginal bulge and dyspareunia. Therefore, anal manometry, colonic transit studies and defecography are less utilized in gynecology preoperative workup and also the reported postoperative complications is also gynecologically focused. For example, in the gynecology literature frequently defines recurrence as the relapse of the vaginal bulge with a generalization of the postoperative defecatory complaints. Karram and Maher [49] as part of the Fifth International Collaboration on Incontinence summarized an extensive review of studies and outcomes (Table 9.1). The patients were followed a minimum of 12 months in most studies. Anatomic cure ranged from 76 to 96%, vaginal bulge persisted 4–31%, vaginal digitation continued 0–33%, defecatory dysfunction was seen in 8–36%, and dyspareunia in 8–45%. Many of the trials did not include rates of dyspareunia or defecatory disorders preoperatively to allow postoperative comparison. The postoperative dyspareunia incidence can be assumed to be underestimated as many of the subjects are elderly women and sexually inactive. While the Maher study [50] noted 37/38 women with dyspareunia preoperatively and only 2 postoperatively, the Abramov [51] study noted an increase from 8/183 to 31/183 respectively. Kahn [52] also reported worsening postoperative dyspareunia. Weber illustrate in her study that the resultant vaginal dimension did not correlate with sexual function [53].

Comparisons amongst these studies are difficult as there is a variable selection process that led to surgery. Other complications noted in these repairs as reported by Mellgren include 12% post-operative hematoma, 4% urinary retention and 4% urinary tract infection [54]. Arnold et al. noted: 10% urinary retention, 4% wound breakdown, 3% infection (not abscess) and 7% impaction in the initial post-operative period in their 29 patients [55]. Long-term follow-up noted 54% with constipation, 36% incontinence, 32% pain, 41% bleeding, 23% sexual dysfunction. Despite these findings, patients reported 77% rate of improvement and 77% satisfaction.

Table 9.1 Review of posterior colporrhaphy/midline plication

	No.	Review (months)	Anatomic cure (%)	Vaginal bulge (recurrence) (%)	Vaginal digitalization (%)	Defecatory dysfunction (%)	Dyspareunia (%)
Arnold	24		80			36	23
Mellgren	25	12	96	4	0	8	8
Kahn	171	42	76	31	33	11	16
Weber	53	12					26
Sand	67	12	90				
Maher	38	12	87	5	16	16	5
Abramov	183	>12	82	4		18	17
Paraiso	28	17.5	86		26	32	45
Total			83	9.2	26	17	18

While still a trans vaginal approach, others favor a discrete identification of fascial defects and doing a localized repair with nonabsorbable suture. This is described as a "site-specific repair" in which only the area where the levator defect is seen is plicated. The theory is that minor levator ani plication will decrease incidence of dyspareunia [52, 56]. However, collection of many series (Table 9.2) by Karam [49] in comparison with posterior colporrhaphy to site-specific repair, showed no difference in regards to postoperative complications and success. The wide range of results may be attributed to the observation of Nichols that the anterior compartment repair is: "…the most misunderstood and poorly performed" gynecological surgery [57].

Other techniques described may or may not incorporate mesh (biologic: autologous/allograft/xenograft and synthetic) into the repair. Sand [58] reported on 132 women with polyglactin mesh to reinforce the repair and found no difference in comparison to those without mesh. Sung [59] compared tissue porcine sub intestinal submucosal tissue graft repair with native tissue repair in a double-blind multicenter randomized trial with 137 total women for grade 2 symptomatic rectoceles. At one year no difference was seen in objective and subjective success rates for defecatory symptoms. Dyspareunia rates were also not statistically different with 7% and 12.5% respectively. Paraiso [60] evaluated 3 techniques in a prospective randomized trial of posterior colporrhaphy, versus site-specific repair and site-specific repair augmented with porcine small intestine submucosa. While fairly small numbers in each (n = 37, 37, 32 respectively) the anatomic failure rate was statistically highest in the graft augmented group and no significant difference was seen in subjective symptoms or dyspareunia. These results do not support the use of mesh.

Transrectal Approach

Transrectal approach, also described as transanal or endorectal repair, has long been reported by colon and rectal surgeons as their procedure of choice, presumably because the approach is within these surgeons' technical area of expertise. This technique is characterized by the plication of rectovaginal septum after raising rectal mucosal flaps, removing excess tissue and obliterating the rectocele defect. Anterior levatorplasty is frequently incorporated if incontinence is an issue [61]. While one can also address other anorectal pathology, the transrectal approach is limited in that it can only access the rectocele defect without any opportunity for repairing any other concomitant pelvic pathology such as enterocele and/or cystocele, two known contraindications [62, 63].

While the transanal technique attempts to obliterate the anatomic defect, some studies suggest this specific approach may yield better functional outcomes than other techniques [64]. Hammond studied 88 women who underwent transanal rectocele repair, specifically focusing on bowel and urinary symptoms pre and postoperatively. When compared to a control group without rectocele, women had

Table 9.2 Review of Site-Specific posterior vaginal repair

	No.	Review (months)	Anatomic cure (%)	Vaginal bulge (recurrence) (%)	Vaginal digitalization (%)	Defecatory dysfunction (%)	Dyspareunia (%)
Cundiff	61	12	82	18	18	8	19
Porter	72	6	82	14	21	21	46
Kenton	46	12	90	9	15		8
Glavind	67	3	100				3
Singh	33	18	92	7		5	125
Abramov	124	>12	56	11	21	19	16
Paraiso	27	17.5	78				28
Sung	70	12	90	7	15.5	21	7
Total			83	11.4	18	17	18

significant improvement in multiple aspects of defecation, including straining, sensation of incomplete emptying, and need for digital support or laxatives [61].

Despite good symptomatic results with transanal repairs, a retrospective study with long term outcomes (mean 74 months) in 71 patients who underwent transanal rectocele repair showed an overall 50% recurrence rate, with 41% rate of isolated rectocele recurrence and 8% rectocele recurrence with an associated enterocele [65]. Nieminen's randomized control trial comparing transanal to transvaginal repair in 30 patients suggested that while both techniques offered reliable repairs with associated symptomatic relief, the transanal repair led to more frequent recurrence. Rectocele recurrence was statistically significantly higher in the transanal repair group (40%) versus transvaginal group (7%), after 12-month follow-up [66]. Furthermore, transanal repair also caused weakened anal sphincter tone postoperatively more so than transvaginal repair, a findings supported by other groups [67]. Despite Nieminen noting worse anatomic repair and weakened sphincter tone in the transanal repair group, patients in both groups had significantly decreased need to digitate themselves during defecation and decreased rectocele symptoms. The number of patients followed was too small to detect a superiority between the two approaches [66].

Careful selection may optimize success. A prospective review of transanal repair in 59 women with obstructed defecation over 19 months found especially superior evacuation (93%) if the patients were free of anismus [67]. Another study of 45 woman who underwent transanal repair only if they demonstrated greater than 15% contrast retention on defecography, noted improvement in complete emptying, reduction in manual maneuvers, reduction in dyspareunia (11–3%) and no new reports of sexual dysfunction [69]. A third study noted 80% improvement in pre-operative symptoms when surgery was offered only to those that had admitted to defecatory support and retention of barium on defecography. In this retrospective review of selective criteria for primarily transanal repair, 88% of 33 women reported complete resolution of vaginal bulge, with 92% reporting symptomatic improvement and operative satisfaction after a mean follow-up period of 31 months [68].

The transanal approach shares many of the same complications as the transvaginal approach. Nieminen's small randomized trial of these two techniques failed to show significant differences with respect to complications, with only 1 out of 15 transanal repairs having a postoperative infection [66]. Commonly reported complications include: fecal impaction, urinary retention, bleeding, wound breakdown, sinus formation, and short longevity of the repair. Thornton [71] in his Posterior colporrhaphy: its effects on bowel and sexual function observed a 13% decline in anal continence and 36% dyspareunia in the transanal arm. Complications unique to the transanal approaches include rectovaginal fistula and stenosis [62, 68]. These unusual complications were reported rarely as a single incidence in most reported studies.

Transperineal Approach

The transperineal approach for rectocele repair stems from the interest to avoid the complications from the other two techniques. By focusing on the failed rectovaginal septum itself, this approach attempts to avoid vaginal tightness and dyspareunia arising from the transvaginal approach or sphincter impairment and incontinence from the transanal approach [73]. The perineal entry allows direct access to the typically widened levator ani muscles, facilitating suture plication of the perineal tissue and rectal submucosa. A transperineal (anterior) levatorplasty, can also be added performed to buttress the rectovaginal septum simultaneously symptoms of dysfunctional defecation. On review of Medline Search, there was a scarcity of papers note in comparison to the other two techniques, One can only assume it is not as commonly performed as the other two approaches already discussed.

A 1998 prospective study of 35 women with rectocele with symptoms of outlet obstruction who underwent transperineal levatorplasty reported a 74% improvement in postoperative defecation, without need for digital maneuvers. Of the 20 women who reported pre-operative incontinence, 75% experienced an improvement in continence [73]. Lamah's mean 3.2 year follow-up by patient questionnaire after transperineal levatorplasty for rectocele noted 88% had improvement in defecation without the need for digitation, with 74% reporting excellent/good satisfaction [74].

A small study of 15 women who had transperineal rectocele repair at St Mark's Hospital with 27 months mean follow-up noted that both transanal and transperineal rectocele repair demonstrated a reduction in rectocele size, improvement in sensation of emptying, and less need for digitation. The transperineal repair, however, showed greater overall improvement overall in symptoms. Interestingly, the size of remaining rectocele, if any, did not correlate with symptom improvement in either group [75].

Because of concern for dyspareunia with the use of levatorplasty, a randomized control trial comparing transanal repair to transperineal repair with and without levatorplasty was conducted [76]. On 6-month follow-up, while a significant improvement in constipation was reported in both transperineal groups, it was greater within the transanal group. The transperineal levatorplasty group, however, had the best improvement with less need for digitation, better sensation of complete evacuation, without reporting major complications like hemorrhage, infection or perforation/fistulization. No significant improvement in sexual function was reported in any of the three groups, but the addition of levatorplasty to the transperineal approach was associated with 13% worsening dyspareunia, leading to their recommendation to avoid levatorplasty in sexually active women with rectocele [76].

Because repair of a chronically weakened rectovaginal septum may not be lasting, the use of mesh was introduced in transperineal approaches to augment the rectovaginal septal repair, without placing the levator muscles on tension. Polyglycolic acid, porcine dermal collagen implant and prolene mesh have all been described. Good to excellent results were reported with no mesh erosion. This is

more than likely a result of typical bias in reporting. Mesh erosion within the rectovaginal septum remains problematic for the surgeons and of potential economic benefit for the lawyers. Overall, reported results are no more superior to studies without mesh. Also, most cases are small in numbers [72, 77, 78] while larger series have short follow-up [79]. Therefore there is no high level of evidence to justify this technique with its increased expense and the known mesh risks well documented in other procedures.

Similar complications such as dyspareunia, wound infections, hemorrhage have all been reported. A unique issue is the contraindication to place mesh if rectal perforation occurs during dissection in the rectovaginal septum [72].

Medical Management

Since the most common complication of surgical management of a rectocele by any means is recurrence, careful evaluation and medical management should proceed any of the above surgical interventions. This includes careful assessment for the cause(s) of symptoms other than the rectocele. When found, the treatment of the true root cause of the rectocele or defecatory complaints needs to be addressed prior to embarking on surgery. During this time dietary and frequently psychological/physical therapy can be pursued. While most gynecological literature stress the connective tissue strength, the colon and rectal literature frequently addresses the underlying psychological factors and presence of colonic inertia or anismas. Without identification of the primary cause of the rectocele, any surgical procedure with longer follow-up will be fraught with increasing recurrence rates and patient dissatisfaction.

Adequate fiber intake is an essential first step as smaller and hard stool is more difficult to evacuate and less likely to stimulate the rectal wall for triggering the rectoanal inhibitory reflex (RAIR) to facilitate stool evacuation. Some have suggested avoidance of certain foods which increases food viscosity such as chocolate [80]. Diet manipulations obviously have the most minimal risks and therefore should be tried initially in all patients and can be embarked upon while workup ensues. Other low risk interventions include: enemas, yoga, biofeedback, and home electrostimulators, etc. [81–85]. An obvious but frequently forgotten part of the evaluation is a careful history of anxiety, depression and sexual trauma. Thirty-three percent of women with complaints of obstructive disorders and proctalgia have had sexual trauma as a minor [86].

Summary

Rectoceles are a common anatomical finding which does not always require repair. This is replicated in many studies that an anatomical correction does not translate into functional success and correction of all symptomatic dysfunctions. The presence of multiple surgical strategies, exemplifies that there is not one best technique. While rectoceles can be seen and felt, the true underlying disorder is frequently a functional defecatory disorder or a hidden psychological affliction. When symptomatic, the underlying disorders should be corrected first, maximal medical management should be exhausted, and physical therapy and frequently psychotherapy should be employed to optimize success, as surgical repair is not without its complications. Medical treatments have very little if any side effects and may offer good results. Since rectoceles, are the resultant sign of the primary disease, surgery will not be successful without treatment of the underlying root cause. When nonsurgical modalities fail, a thorough assessment of the cause of the symptoms must be undertaken. A frank discussion with the patient of whether repairing an anatomic condition will correct their symptoms is essential. Restoring anatomy does not equate to restoring function. The trade-off of dyspareunia needs a careful discussion with the women who are still sexually active. With these precautions in mind, the numbers of rectoceles truly deserving surgical repair are probably much less than the incidence of repairs reported in the literature.

References

1. Mehigan BJ, Monson JR, Hartley JE. Stapling procedure for haemorrhoids versus Milligan-Morgan haemorrhoidectomy: randomised controlled trial. Lancet. 2000;355 (9206):782–5.
2. Thompson H. The anal cushions- a fresh concept in diagnosis. Postgrad Med J. 1979;55:403–5.
3. Boccasanta P, Venturi M, Salamina G, et al. New trends in the surgical treatment of outlet obstruction: clinical and functional results of two novel transanal stapled techniques from a randomised controlled trial. Int J Colorectal Dis. 2004;19:359–69.
4. Van Geluwe B, Stuto A, Da Pozzo F, et al. Relief of obstructed defaecation syndrome after stapled transanal rectal resection (STARR): a meta-analysis. Acta Chir Belg. 2014;114:189–97.
5. Lehur PA, Stuto A, Fantoli M, et al. Outcomes of Stapled Transanal Rectal Resection versus Biofeedback for the Treatment of Outlet Obstruction Associated with Rectal Intussusception and Rectocele: A Multicenter, Randomized, Controlled Trial.
6. Pescatori M, Spyrou M, Pulvirenti-D'Urso A. A prospective evaluation of occult disorders in obstructed defecation using the 'iceberg diagram'. Colorect Dis. 2006;8:785–78.
7. Jayne DG, Schwandner O, Stuto A. Stapled transanal rectal resection for obstructed defecation syndrome: One-year results of the European STARR Registry. Dis Colon Rectum. 2009;52:1205.
8. Ho YH, Tsang SM, Tang CLM, Nyam DM, Eu KW, Seow-Choen F. Ana sphincter injuries from stapling instruments introduced transanally: randomised, controlled study with endoanal ultrasound and anorectal manometry. Dis Colon Rectum. 2000;43:169–73.

9. Creve U, Hubens A. The effect of Lord's procedure on anal pressure. Dis Colon Rectum. 1979;22:483–5.
10. Kreis ME, Jehle EC, Haug V, et al. Functional results after transanal endoscopic microsurgery. Dis Colon Rectum. 1996;39:1116–21.
11. Naldini G, Cerullo G, Claudia Menconi C, et al. Resected specimen evaluation, anorectal manometry, endoanal ultrasonography and clinical follow-up after STARR procedures. World J Gastroenterol. 2011;17:2411–6.
12. Stuto A, Adolfo Renzi A, Carriero A, et al Stapled Trans-anal rectal resection (STARR) in the surgical treatment of the obstructed defecation syndrome: results of Starr italian registry. Surg Innovation 18;248–253.
13. Boenicke L, Reibetanz J, Kim M, et al. Predictive factors for postoperative constipation and continence after stapled transanal rectal resection. Brit J Surg. 2012;99:416–22.
14. Goede AC, Glancy D, Carter H, et al. Medium-tern results of stapled transanal rectal resection (STARR) for obstructed defaecation and symptomatic rectal-anal intussusception. Colorectal Dis. 2011;13:1052–7.
15. Boenicke L, Kim M, Reibetanz J, et al. Stapled transanal rectal resection and sacral nerve stimulation–impact on faecal incontinence and quality of life. Colorectal Dis. 2011;14:480–9.
16. Cheetham MJ, Mortensen NJ, Nystrom PO, Kamm MA, Phillips RK. Persistent pain and faecal urgency after stapled haemorrhoidectomy. Lancet. 2000;356:730–3.
17. Dodi G, Pietroletti R, Milito G, Binda G, Pescatori M. Bleeding, incontinence, pain and constipation after STARR transanal double stapling rectotomy for obstructed defecation. Tech Colproctol. 2003;7:148–53.
18. Arroyo A, Perez-Vicente F, Serrano P, et al. Proctitis complicating stapled hemorrhoidectomy: report of a case. Int J Colorectal Dis. 2006;21:197–8.
19. P´erez-Vicente F, Arroyo A, Serrano P, et al. Prospective randomized clinical trial of single versus double purse-string stapled mucosectomy in the treatment of prolapsed haemorrhoids. Int J Colorectal Dis. 2006;21:38–43.
20. Thaha MA, Irvine LA, Steele RJ. Campbell KL (2005) Post defaecation pain syndrome after circular stapled anopexy is abolished by oral nifedipine. Br J Surg. 2005;92:208–10.
21. Izzo D, Brillantino A, Iacobellis F, et al. Role of 0.4% glyceryl trinitrate ointment after stapled trans-anal rectal resection for obstructed defecation syndrome: a prospective, randomized trial. Int J Colorectal Dis. 2014;29:105–10.
22. De Nardi P, Bottini C, Scucchi L, Palazzi A, Pescatori M. Proctalgia in a patient with staples retained in the puborectalis muscle after STARR operation. Tech Coloproctol. 2007;11:353–6.
23. Frascio M, Stabilini C, Ricci B, et al. Stapled transanal rectal resection for outlet obstruction syndrome: results and follow-up. World J Surg. 2008;32:1110–5.
24. Madbouly KM, Abbas KS, Hussein AM. Disappointing long-term outcomes after stapled transanal rectal resection for obstructed defecation. World J Surg. 2010;34:2191–6.
25. Boccasanta P, Venturi M, Stuto A, et al. Stapled transanal rectal resection for outlet obstruction: a prospective, multicenter trial. Dis Colon Rectum. 2004;47:1285–97.
26. Naldini G. Serious unconventional complications of surgery with stapler for haemorrhoids prolapse and obstructed defection because of rectocele and rectal intussusception. Colorectal Dis. 2011;13:323–7.
27. Pescatori M, Gagliardi G. Postoperative complications after procedure for prolapsed hemorrhoids (PPH) and stapled transanal rectal resection (STARR) procedures. Tech Coloproctol. 2008;12:7.
28. Bassi R, Rademacher J, Savoia A. Rectovaginal fistula after STARR procedure complicated by hematoma of the posterior vaginal wall: Report of a case. Tech Coloproctol. 2006;10:361.
29. Zehler O, Vashist YK, Bogoevski D, et al. Quo Vadis STARR? A prospective long-term follow-up of stapled transanal rectal resection for obstructed defecation syndrome. J Gastrointest Surg. 2010;14:1349–54.

30. Baraza W, Shorthouse A, Brown S. Obliteration of the rectal lumen after stapled haemorrhoidopexy: report of a case. Dis Colon Rectum. 2009;52:1524–5.
31. Corman ML, Carriero A, Hager T, et al. Consensus conference on stapled transanal rectal resection (STARR) for disordered defaecation. Colorectal Dis. 2006;8:98–101.
32. PescatoriM, Spyrou M, Cobellis L, Bottini C, Tessera G. Rectal pocket syndrome after stapled mucosectomy. Colorect Dis. 2006;8:808–811.
33. Reibetanz J, Boenicke L, Kim M, Germer CT, Isbert C. Enterocele is not a contraindication to stapled transanal surgery for outlet obstruction: an analysis of 170 patients. Colorectal Dis. 2011;13,:e131–e136.
34. PetersenS HellmichG, Schuster A, et al. Stapled transanal rectal resection under laparoscopic surveillance for rectocele and concomitant enterocele. Dis Colon Rectum. 2006;49:685.
35. Carriero A, Picchio M, Martellucci J, Talento P, Palimento S, Spaziani E. Laparoscopic correction of enterocele associated to stapled transanal rectal resection for obstructed defecation syndrome. Int J Colorectal Dis. 2010;25:381–7.
36. Maw A, Eu KW, Seow-Choen F. Retroperitoneal sepsis complicating stapled hemorrhoidectomy: report of a case and review of the literature. Dis Colon Rectum. 2002;45:826–8.
37. Gagliardi G, Pescatori M, Altomare DF, et al. Results, outcome predictors, and complications after stapled transanal rectal resection for obstructed defecation. Dis Colon Rectum. 2008;51:186.
38. Molloy RG, Kingsmore D. Life threatening pelvic sepsis after stapled haemorrhoidectomy. Lancet. 2000;355(9206):810.
39. Pescatori M, Boffi F, Russo A. Zbar AP (2006) Complications and recurrence after excision of rectal internal mucosal prolapse for obstructed defaecation. Int J Colorect Dis. 2006;21:160–5.
40. Handa V, Garrett E, Hendrix S, et al. Progression and remission of pelvic organ prolapse: a longitudinal study of menopausal women. Am J Obstet Gynecol. 2004;190(1):27–32.
41. Hendrix S, Clark A, Nygaard I, et al. Pelvic organ prolapse in the women's health initiative: Gravity and gravidity. Am J Obstet Gynecol. 2002;186(6):1160–6.
42. Brusciano L, Limongelli P, Pescatori M, et al. Ultrasonographic patterns in patients with obstructed defaecation. Int J Colorectal Dis. 2007;22(8):969–77.
43. Taylor SA. Defecographic study of rectal evacuation in constipated patients. In: Santoro GA, Di Falco G, editors. Benign anorectal diseases: diagnosis with endoanal and endorectal ultrasound and new treatment options. Milan, Italy: Springer; 2006. p. 231–41.
44. Pucciani F. Anorectal manometry. In: Santoro GDi Falco G. Benign anorectal diseases. Milan: Springer; 2006.
45. Piloni V, Tosi P, Vernelli M. MR-defecography in obstructed defecation syndrome (ODS): technique, diagnostic criteria and grading. Tech Coloproctol. 2013;17(5):501–10.
46. Beer-Gabel MCarter D. Comparison of dynamic transperineal ultrasound and defecography for the evaluation of pelvic floor disorders. Int. J. Colorectal Dis. 2015;30(6):835–41.
47. Hill J, Hosker G, Kiff E. Pudendal nerve terminal motor latency measurements: what they do and do not tell us. Br J Surg. 2002;89(10):1268–9.
48. Russo A, Pescatori M. Psychological assessment of patients with proctological disorders. In: Wexner S, Zbar A, Pescatori M. Complex anorectal disorders London: Springer; 2005.
49. Karram Maher C. Surgery for posterior vaginal wall prolapse. Int Urogynecol J. 2013;24 (11):1835–41.
50. Maher C, Qatawneh A, Baessler K, Schluter P. Midline rectovaginal fascial plication for repair of rectocele and obstructed defecation. Obstet Gynecol. 2004;104(4):685–9.
51. Abramov Y, Kwon C, Gandhi et al. Long –term anatomic outcome of discrete site-specific defect repair versus standard posterior colporrhaphy for the correction of advanced rectocele: a 1 year follow-up analysis. Neurourol Urodyn 22(5):520–521.
52. Kahn MA, Stanton SL. Posterior colporrhaphy: its effects on bowel and sexual function. Int J Gynecol Obstet. 1997;57(2):243.

53. Weber A. Sexual function in women with uterovaginal prolapse and urinary incontinence. Int J Gynecol Obstet. 1995;51(3):296.
54. Mellgren A, Anzén B, Nilsson B, et al. Results of rectocele repair. Dis Colon Rectum. 1995;38(1):7–13.
55. Arnold M, Stewart W, Aguilar P. Rectocele repair. Dis Colon Rectum. 1990;33(8):684–7.
56. Cundiff G, Fenner D. Evaluation and treatment of women with rectocele: focus on associated defecatory and sexual dysfunction. Obstet Gynecol. 2004;104(6):1403–21.
57. Nichols D. Posterior colporrhaphy and perineorrhaphy: Separate and distinct operations. Am J Obstet Gynecol. 1991;164(3):714–21.
58. Sand P, Koduri S, Lobel R, et al. Prospective randomized trial of polyglactin 910 mesh to prevent recurrence of cystoceles and rectoceles. Am J Obstet Gynecol. 2001;184(7):1357–64.
59. Sung V, Rardin C, Raker C, et al. Porcine sub intestinal submucosal graft augmentation for rectocele repair. Obstet Gynecol. 2012;119(1):125–33.
60. Paraiso M, Barber M, Muir T, Walters M. Rectocele repair: a randomized trial of three surgical techniques including graft augmentation. Am J Obstet Gynecol. 2006;195(6):1762–71.
61. Hammond KEllis C. Outcomes after transanal repair of rectoceles. Dis Colon Rectum. 2010;53(1):83–87.
62. Sullivan E, Leaverton G, Hardwick C. Trans rectal perineal repair. Dis Colon Rectum. 1968;11(2):106–14.
63. Khubchandani IT, Sheets JA, Stasik JJ, Hakki AR. Endorectal repair of rectocele. Dis Colon Rectum. 1983;26(12):792–6.
64. Khubchandani IT, Clancy J, Rosen L, et al. Endorectal repair of rectocele revisited. Br J Surg. 1997;84(1):89–91.
65. Roman H, Michot F. Long-term outcomes of transanal rectocele repair. Dis Colon Rectum. 2005;48(3):510–7.
66. Nieminen K, Hiltunen K, Laitinen J, et al. Transanal or vaginal approach to rectocele repair: a prospective, randomized pilot study. Dis Colon Rectum. 2004;47(10):1636–42.
67. Ho Y, Ang M, Nyam D, et al. Transanal approach to rectocele repair may compromise anal sphincter pressures. Dis Colon Rectum. 1998;41(3):354–8.
68. Murthy V, Orkin B, Smith L, Glassman L. Excellent outcome using selective criteria for rectocele repair. Dis Colon Rectum. 1996;39(4):374–8.
69. Tjandra J, Ooi B, Tang C, et al. Transanal repair of rectocele corrects obstructed defecation if it is not associated with anismus. Dis Colon Rectum. 1999;42(12):1544–50.
70. Heriot A, Maxwell P, Kumar D. Functional and physiological outcome following transanal repair of rectocele. Gastroenterology. 2000;118(4):A126.
71. Thornton MJ, Lam A, King DW. Laparoscopic or transanal repair of rectocele? A retrospective matched cohort study. Dis Colon Rectum. 2015;48:792–8.
72. Mercer-Jones MA, Sprowson A, Varma JS. Outcome after transperineal mesh repair of rectocele: a case series. Dis Colon Rectum. 2004;47(6):864–8.
73. Ommer A, Köhler A, Athanasiadis S. Results of transperineal levator-plasty in treatment of symptomatic rectocele. Der Chirurg; Zeitschrift fur alle Gebiete der operativen Medizen. 1998;69(9):966–72.
74. Lamah M, Ho J, Leicester RJ. Results of anterior levatorplasty for rectocele. Colorectal Dis. 2001;3(6):412–6.
75. Van Laarhoven CJ, Kamm MA, Bartram CI, et al. Relationship between anatomic and symptomatic long-term results after rectocele repair for impaired defecation. Dis Colon Rectum. 1999;42(2):204–10.
76. Farid M, Madbouly KM, Hussein A, et al. Randomized controlled trial between perineal and anal repairs of rectocele in obstructed defecation. World J Surg. 2010;34(4):822–9.
77. Leventoğlu S, Menteş B, Akn M, et al. Transperineal rectocele repair with polyglycolic acid mesh: a case series. Dis Colon Rectum. 2007;50(12):2085–95.

78. Smart N, Mercer-Jones M. Functional outcome after transperineal rectocele repair with porcine dermal collagen implant. Dis Colon Rectum. 2007;50(9):1422–7.
79. Lechaux JP, Lechaux D, Bataille P, Bars I. [Transperineal repair of rectocele with prosthetic mesh. A prospective study]. In Annales de chirurgie. 2004;129(4):211–217.
80. Dietz H. Rectocele or stool quality: what matters more for symptoms of obstructed defecation? Tech Coloproctol. 2009;13(4):265–8.
81. Dolk A, Holmström B, Johansson C, et al. The effect of yoga on puborectalis paradox. Int J Colorectal Dis. 1991;6:139–42.
82. Pucciani F, Reggioli M, Ringressi M. Obstructed defaecation: what is the role of rehabilitation? Colorectal Dis. 2012;14(4):474–9.
83. Bove A. Consensus statement AIGO/SICCR diagnosis and treatment of chronic constipation and obstructed defecation (Part II: Treatment). World J Gastroenterol. 2012;18(36):4994.
84. Peticca L, Pescatori M. Outlet obstruction due to anismus and rectal hypo sensation: effect of biofeedback training. Colorect Disease. 2002;4(1):67.
85. Matzel KE. Invited comment. In: Santoro GA, Di Falco G, editors. Benign anorectal disease: diagnosis with endoanal and endorectal ultrasound and new treatment options. Milan, Italy: Springer; 2006. p. 367–8.
86. Devroede G. Early life abuses in the past history of patients with gastrointestinal tract and pelvic floor dysfunctions. Prog Brain Res. 1999;122:131–55.

Complications of Rectovaginal Fistula Repair

10

Slawomir Marecik, Ariane M. Abcarian and Leela M. Prasad

Introduction

Despite advances in surgical techniques, rectovaginal fistulas (RVF) remain a challenging surgical problem. Women with RVF experience severe physical, social, and emotional trauma [1]. Additionally, when complications arise during treatment, their suffering is greatly magnified.

The medical literature on RVF generally focuses on reports of successful surgical procedures. It is rare to find the "negative studies" that focus on reports of failures. There is even less educational material on RVF, which could serve to analyze the reasons for failure. This chapter will discuss specific complications of RVF repair procedures.

It has been said that the quality of a surgeon is measured not only by how well he or she can correct the problem with surgery, but also how well complications can be managed if and when they arise. Complications arising from any surgical

Leela M. Prasad (Deceased)

S. Marecik (✉) · L.M. Prasad
Division of Colon and Rectal Surgery, Advocate Lutheran General Hospital,
1775 W. Dempster Ave, Park Ridge, IL 60068, USA
e-mail: Smarecik@hotmail.com

S. Marecik · L.M. Prasad
Division of Colon and Rectal Surgery, University of Illinois at Chicago,
840 S. Wood St., Suite 518E, Chicago, IL 60612, USA

A.M. Abcarian
Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County,
1600 W. Polk Street, Chicago, IL 60612, USA

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_10

treatment can lead to litigation [2]. Oftentimes, this is due, in part, to the lack of adequate explanation with the patient during preoperative discussion (informed consent) as to the potential complications. The information presented herein is intended to shed more light on this important topic.

Anatomy

Knowledge of surgical anatomy and proper identification of anatomical structures during dissection cannot be overemphasized when undertaking RVF repair. It is one of the main factors leading to success of the procedure and avoidance of complications.

The ultimate goal of the surgical technique is clean, precise, bloodless, and meticulous dissection based on anatomical landmarks and tissue planes. This should be followed by reconstruction using well-vascularized and healthy tissues, while also avoiding creation of dead space.

In colorectal surgical practice, RVFs are encountered infrequently [3]. Unfortunately, this results in lack of exposure to RVFs by trainees who often only have opportunity to participate in two or three repairs during their year of specialty training. In fact, more often than not, trainees witness no more than one or two surgical approaches during that time period.

Due to the nature of the surgical field, the exposure and anatomy of RVFs can easily be obscured by blood, which can lead to disorientation and errors. Bleeding can originate from an extremely well-vascularized vaginal wall, perivaginal plexuses, or hemorrhoidal vessels in the lower rectum and anal canal. Surgical dissection can also be secondarily obscured due to loss of the anatomic landmarks, either by the primary offending process or as a result of previous repairs. For this reason, finding the available anatomical reference points is very important.

The surgeon should be equally familiar with the anatomy of the perineum in both the lithotomy and prone positions. The following is a review of the important anatomic structures which are useful as reference points during RVF repair.

Perineal Body

The perineal body, also known as the perineal raphe, is an anatomic location in the center of the perineum, just in front of the anal sphincter complex. It has tendinous characteristics because it includes crisscrossing of the bilateral contributing muscles, which include the superficial and deep transverse perineal muscles, the bulbospongiosus muscle, and the external sphincter complex. The perineal body separates the anus from the vaginal introitus.

Sphincter Complex

The sphincter muscle complex is comprised of two muscular tubes. The external tube (external sphincter) is longer and thicker and is derived from striated (skeletal) muscle. The internal tube (internal sphincter) is significantly thinner, slighter shorter, and comprised of smooth muscle. The difference in length between the two sphincter tubes creates the intersphincteric groove, which is an important landmark located just proximal to the anal verge. The internal sphincter is made of concentric smooth muscle lamellae. Overall, the thickness of the internal sphincter tube is approximately 2–4 mm. At the anorectal junction, the internal sphincter transitions into the inner circular muscle layer of the rectal wall.

The anal canal is a functional unit created by the sphincter muscle complex. It is posterior to the lower part of the vagina and the introitus, the origin of the bulbospongiosus (often described as bulbocavernosus) muscles, and Bartholin's glands. The average length of the anal canal in women is 2.5–3.5 cm, however, this is dependent on the muscle tension and is significantly shorter under regional or general anesthesia or deep sedation. Prominent internal hemorrhoids often cover the proximal half of the anal canal, while the lower half has a smooth surface lined by shiny anoderm.

Puborectalis Muscle

The puborectalis muscle is a band-like structure that forms the middle portion of the levator ani. It originates from the posterior pubis, runs dorsally, and slings around the anorectal junction. This muscle is responsible for creating the anorectal angle. It exerts a compression effect on the anorectal junction and largely contributes to fecal continence.

Pubococcygeus Muscle

The pubococcygeus muscle is a sheet-like structure. It originates from the anterior half of the internal obturator fascia along the tendinous arch, then runs dorsally, medially, and caudally toward the lower sacrum. In the midline, the fibers intertwine with the fibers from the opposite site to create anterior part of the anococcygeal raphe, located just posterior to sphincter complex. More anteriorly, some of the fibers do not come together and it is here, joining the puborectalis muscle, that they create an open space in the anterior pelvic floor, called the levator hiatus. The posterior aspect of the levator hiatus accommodates the anorectal junction (rectal hiatus) and the anterior aspect is reserved for the urogenital structures (urogenital hiatus). A thickening of the endopelvic fascia creates the urogenital membrane (hiatal ligament), thereby fixing the urogenital structures to the levator muscle complex.

Levators

During RVF repair, the term levators is frequently used to describe the fused puborectalis and pubococcygeus muscles (see above) directly opposed to the lateral aspect of the lower vagina and lower rectum. Separation of these organs from the levators can be performed along the avascular plane.

Deep Transverse Perineal Muscle

The deep transverse perineal muscle is a sheet-like muscular structure that spreads between the inferior pubic rami in the transverse fashion. It is located anteriorly to the sphincter muscle complex, below the anterior portion of the levators, and directly underneath the hiatal ligament, covering the urogenital hiatus of the levator ani. This muscle contains an anterior opening surrounded by the circular urethral sphincter and the vaginal opening.

Superficial Transverse Perineal Muscle

The superficial transverse perineal muscle is a narrow band-like, paired muscular structure that spreads between the ischial tuberosities across the center of the perineum. Both muscles join in the center, in front of the sphincter muscle complex, and contribute to creation of the perineal body. The muscles are in direct contact with the deep transverse perineal muscle located directly above them.

Bulbospongiosus Muscle

The bulbospongiosus (also known as bulbocavernosus) muscle is a paired, thin, muscular cover of the inferior-lateral base of vestibular (clitoral) bulbs. It originates from the center of the perineum. A fat pad of the labium majora, which can be harvested for reconstructive purposes, covers the bulbospongiosus muscle. The blood supply to the labial fat pad is provided by the posterior labial branches of the perineal artery, and is itself a branch of the internal pudendal artery. This blood supply reaches the fat pad from the posterolateral direction.

Rectovaginal Septum

The rectovaginal septum is a conventional structure separating the lumen of the rectum from the vaginal lumen. It is created by adherence of the anterior rectal and posterior vaginal walls, which are fused together by the areolar tissue of vaginal wall adventitia. It is important to note that in obese individuals, some anterior mesorectal fat can be present between the muscular layer of the rectum and the

vaginal wall. Occasionally, a well-defined mesorectal fascia (fascia propria recti) can also be identified. Another important detail is the presence of vaginal venous plexus in the form of sinuses surrounding the vaginal wall. This is mainly in the lateral aspects but also extends onto the posterior vaginal wall. During dissection within the rectovaginal septum, any persistent venous bleeding is likely a result of violation of the above-mentioned venous plexus. In the lower part of the vagina, the rectovaginal septum extends between the almost vertically oriented levator muscles comprised of the puborectalis and pubococcygeus muscles. In addition, both the rectum and vagina can be dissected off the levators, allowing for their exposure and aid in levatorplasty.

General Complications Related to the Repair of RVF

General complications of RVF repair include recurrence, bleeding, infection, and injury to the surrounding structures. These can result in bowel related dysfunction and genitourinary complications, which can then adversely affect the quality of life. In addition, complex repairs will often require fecal diversion, bringing with it inherent potential complications. Radiation therapy adds still another layer of complexity to RVFs.

Recurrence

Failure to heal (recurrence/persistence) is probably the most dreaded and upsetting complication following a properly executed RVF repair. Recurrence is a complication in itself and can often be a result of other complications such as bleeding and infection. The reported rate of recurrence if often measured as a negative to the success rate.

Success and recurrence of RVF repair depends on a number of factors. It is well known that RVFs constitute a large non-uniform group of different types of communication between the vagina and all possible levels of rectum or anus. These openings vary in size and reflect the amount of tissue loss of the rectovaginal septum, sphincter complex, perineal body, and pelvic floor muscles. The remaining tissue may have a different quality, reflecting the amount of residual inflammation and potential deleterious effects of previous radiation. Larger size fistulas with poorer quality of tissues are classified as complex and are generally associated with poorer outcomes [4]. It has also been well documented that repeat RVF repairs are associated with poorer outcomes [4–6]. For this reason, analysis of operative reports from the previous repairs, as well as a good understanding of the most common corrective procedures, is crucial.

Despite significant suffering by patients with RVFs, and regardless of their understandable impatience to have the problem corrected, the treatment policy should always be based on the conservative approach. In other words, the least

invasive procedures should be utilized first, before the more radical approaches are considered. The timing of surgery is also important and, in line with the conservative approach, preference is for a longer interval in order to allow the tissues to obtain their optimal healing ability. Finally, it is important to understand that it is not an easy task to identify the right corrective procedure to minimize the chance of recurrence. It is something that comes with experience, which comes with time. In fact, because the prevalence of RVF is so low, many surgeons gain their experience the hard way—by learning from their own mistakes.

Most fistulas initially present with some degree of inflammation associated with primary infection. Rarely are the conditions optimal for the corrective procedure to take place right away. Because of this, it is always wise to consider undertaking all possible measures to resolve the local sepsis before attempting corrective surgery [7]. On occasion, a short course of antibiotics can be helpful, although patients often benefit more from examination under anesthesia (EUA), surgical debridement removal of any and all non-absorbable sutures, and placement of a silastic draining seton (Fig. 10.1). In this instance, the corrective procedure should be deferred for 8–10 weeks [8]. Examination under anesthesia also adds a benefit, one that is rarely mentioned in surgical textbooks, and allows for thorough planning of the definitive corrective procedure (simulation of possible final procedure in the future). Based on the findings from the EUA, the surgeons can also decide whether to utilize a

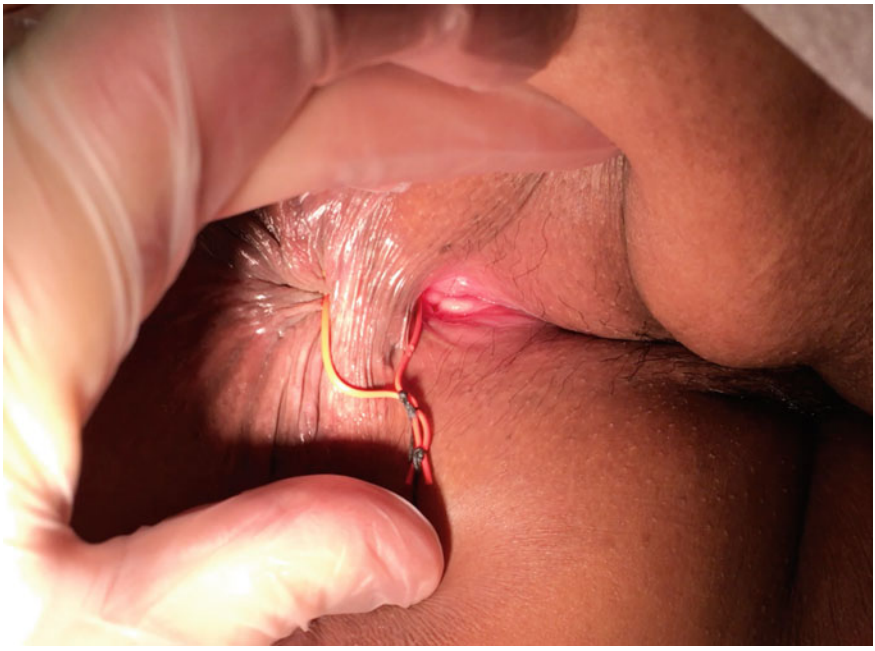


Fig. 10.1 A thin and soft silastic seton to allow the inflammation to subside before the final repair

multidisciplinary approach during the final procedure, such as involving plastic surgery consultants for more complex reconstruction.

The principles of proper wound healing should always be followed in order to decrease any chances of failure. Diabetic patients require optimization of their blood sugar. Because most repairs are scheduled well in advance, a window of opportunity exists during the preoperative period that allows for close monitoring of blood sugars and glycated hemoglobin (A1C). Euglycemia is crucial during the perioperative period, although achieving it might be difficult in patients with advanced diabetes. In these instances, glucose levels of 140–200 mg/dL are set as a preoperative goal, with fasting levels at <140 mg/dL and random checks of <180 mg/dL [9].

Smoking is also associated with impaired wound healing, through a multifactorial mechanism that includes vasoconstriction, leading to decreased perfusion, relative ischemia of dissected tissues, reduction of inflammatory response, impaired bactericidal mechanisms, and alterations of collagen metabolism [10]. This is especially important when tissue flaps are considered during reconstructive procedures [11, 12]. In certain cases, in order to provide an optimal environment for healing, complete abstinence from smoking can be monitored by measuring cotinine levels in body fluids. Non-compliant patients should have their cases postponed.

On occasion, the patient will present with a more complex fistula associated with a side branch or a secondary primary opening on the rectal side. The surgeon should always maintain a high index of suspicion for that possibility, particularly during re-operative approaches and recurrent fistulas. Consideration should also be given to performing a real-time endorectal ultrasound during EUA or a preoperative MRI [13, 14].

The recurrence rate might also be due to other causes, including hematoma, infection, poor tissue quality, inflammation, foreign body including staples (Fig. 10.2), mesh and non-absorbable sutures, plugs, inflammatory bowel disease (IBD), malignancy, radiation vasculitis, obliterative endarteritis, ischemia, dead space, obstructed defecation syndrome, or steroids. Thus, in the surgical treatment of RVF, many things can go wrong. It also helps to explain why it is often difficult for RVFs to heal after multiple operations, especially considering that the rate of success diminishes after each subsequent procedure [4].

Because of its transmural nature, patients with Crohn's disease often develop fistulas. The anorectum is the most common anatomic area for fistula formation in these patients and the close proximity of the posterior vaginal wall also enables the formation of RVF. These fistulas often involve severe inflammation and stricture and are especially prone to recurrence following repair [7]. It is not uncommon for patients to undergo multiple procedures such as incision and drainage, placement of seton, plug, and sealant and flap procedures, and for all of them to fail. In fact, because of the frustrating results of treatment, many patients choose to forego further treatment and opt to live with the fistula. Likewise, surgeons who are concerned with inflamed rectal mucosa, prefer to approach Crohn's related RVFs transanally and under the protection of diverting stoma [15].

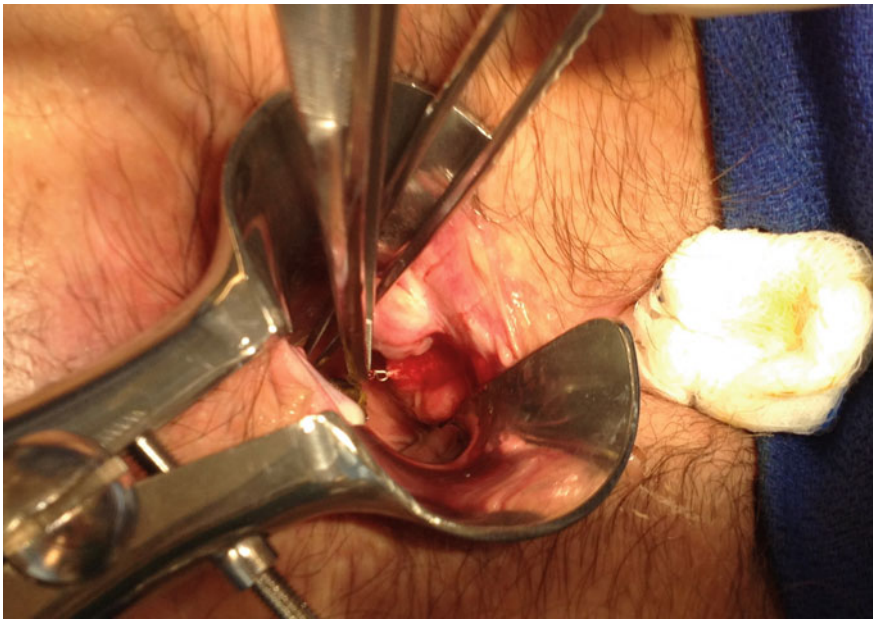


Fig. 10.2 A staple (foreign body) found to be a reason for failure after second rectal advancement flap repair for fistula from PPH (procedure for prolapsed hemorrhoids)

Treatment with biologics, such as adalimumab (Humira, AbbVie) and infliximab (Remicade, Janssen Biotech), can often reduce inflammation and contribute to the closure of some fistulas. However, the long-term closure of fistulas, documented by MRI, is less certain. In the ACCENT II study of Crohn's disease, Sands and colleagues studied 25 patients (18%) with RVFs [16]. Of these, 60% of fistulas closed in 10 weeks and 44.8% closed in 14 weeks using Infliximab infusion. Additionally, fistula closure lasted longer (46 weeks) with 5 mg/kg Infliximab infusion for maintenance vs. the placebo group (33 weeks). None of the patients received surgical intervention.

Fecal diversion, although helpful in reducing inflammation and infection, has not been shown to be effective in closing RVFs [17]. One of the benefits of fecal diversion, however, is the improvement in quality of life which might encourage patients to ultimately consider a proctocolectomy. Scott and colleagues reported that 13 of 38 patients with perianal Crohn's disease, without RVF, needed stoma or proctectomy; while 18 of 29 patients with Crohn's RVF ultimately needed stoma or proctectomy, and the differences were statistically significant [18].

In patients with cancer, RVFs may develop. Brachytherapy can lead to RVFs in patients with cervical cancer, while external beam radiation can lead to RVFs in patients with rectal cancer. In an anteriorly located rectal cancer invading the vagina, radiation-induced necrosis can sometimes result in RVF. Similarly, low RVF can be seen following chemoradiation in the treatment of advanced anal

cancer. If the cause of RVF is cervical cancer therapy, the patient must have EUA and biopsies of both the vaginal and rectal sides of the fistula in order to exclude residual malignancy. If no cancer is diagnosed, a very low anterior resection (VLAR) and low colorectal or coloanal anastomosis can be attempted. The omentum can then be mobilized and interposed between the vagina and the colorectal anastomosis [19].

Bleeding

Bleeding is a possible complication of almost any surgical procedure. Operations involving the anorectum, rectovaginal septum, and vaginal wall are inherently associated with a higher risk of bleeding. The main reason for this is hypervascularity of the vaginal wall, perivaginal venous plexus, mainly in the lateral aspects and extending onto the posterior wall, and the prominent vasculature of the rectal wall, particularly the lower region where the hemorrhoidal plexus is located. Of importance is the possibility of direct communication between the rectal and vaginal venous plexus [20].

Bleeding can occur intraoperatively, which can lead to significant blood loss but is rarely life threatening. The main consequence of bleeding during RVF repair is the loss of helpful anatomical landmarks and excessive thermal tissue trauma from bleeding control using cautery. The risk for intraoperative bleeding is increased if the dissection veers off the proper, avascular (or the least vascular) tissue plane. The risk of this happening is higher when the anatomy is distorted either by previous surgeries or acquired conditions (e.g., large rectocele with thinned-out rectovaginal septum). In these cases, the use of fine suture ligatures is recommended, instead of high wattage cautery, to control the bleeding from the persistent vaginal venous plexus. Metallic clips should also be avoided to control bleeding in this area, due to the risk of foreign body retention in a potentially contaminated field. Persistent intraoperative bleeding also has a psychological effect on the surgical team by bringing morale down, creating an atmosphere of impatience, and limiting control over the operating field, all of which predispose to errors. The best way to avoid or stop any bleeding is by providing an adequate and stable exposure of the operating field, using self-retaining retractors, and following the avascular anatomical planes.

It is the practice of many surgical teams to inject the local anesthetic with epinephrine solution into the dissected tissues preemptively. This is done primarily to prevent intraoperative bleeding but also to decrease postoperative pain. This technique has been used successfully for many decades, although it is important to note that it can lead to distortion of the tissue planes as well as creation of hematoma, if the blood vessel is injured by the needle. In such cases, not only are the tissues changed by edema but they are also stained by hematoma. It is the authors' experience that this infiltration technique be used only in cases of unexpected hypervascularity of the operating field. Conversely, local anesthetic with or without epinephrine is commonly used for anesthetic purposes once the tissues have been dissected.

Hematomas often form postoperatively and are frequently related to a bloody operative field. They can be of arterial nature (vaginal or rectal wall, levators) or have venous character (paravaginal venous plexus). They can also have a mixed character when originating from the hemorrhoidal plexus. Hematomas will likely occur more often in the potential dead space such as the dissected rectovaginal septum. Physical exercise and straining during constipation or diarrhea can lead to increased abdominal and pelvic pressure, which can then cause the hematoma. During sphincteroplasty it is possible to close the sphincter too tight, which can lead to obstructed defecation and abnormally high pelvic pressure during evacuation. The same scenario can happen in patients with obstructed defecation symptoms from other causes or hypertonic sphincters. Fiber bulking therapy has been found to be helpful in evacuation and should be recommended for most patients following repair.

Bleeding can also occur early in the postoperative period and should be considered a technical error. Passage of fresh blood or clots through the anal canal or vagina soon after surgery (within the first 24 h), mandates a return to the operating room, EUA, and control of the bleeding. Delayed bleeding, occurring many days postoperatively, is usually caused by a low-grade infection or disruption of the suture line and is associated with early failure of RVF repair. The patient must be taken back to the operating room for EUA, however, re-repair at this time typically fails and it is best to debride and clean the wound, leaving open to heal by secondary intention. Plans for re-repair should be postponed for 3–4 months.

Sepsis

Sepsis should be considered the primary reason for failure of RVF repair. If a small amount of drainage is noted in the perineum, the patient can be placed on more frequent wound cleansing, low residue diet, and a short course of antibiotics, in the hopes of preventing a full-blown infection or abscess [21]. However, if frank purulent discharge or evidence of abscess with pain, swelling, erythema, and fever is encountered, the only option is to return to the operating room for a EUA and lay the abscess open for debridement and drainage of the infection, without attempting a simultaneous re-repair.

Continuous fecal contamination, particularly in patients with diarrhea-predominate IBS, might also be at fault. Some surgeons advocate bowel confinement for 3–5 days following RVF repair. One has to consider the deleterious effect of passage of rock hard stool a few days later. It is unclear why repair of RVF undertaken after proximal diversion gets infected but it is safe to say that fecal diversion is not the final answer in the prevention of wound infection [22].

Dead space in RVF repairs is often the primary site of infection or abscess and can obscure sepsis for days (Fig. 10.3). It is therefore imperative to obliterate all dead spaces during the primary operation. If this is not possible, the vaginal side of the repair should be left open slightly in order to allow drainage and prevent fluid collection between the repaired rectal and vaginal walls. Similarly, if postoperative

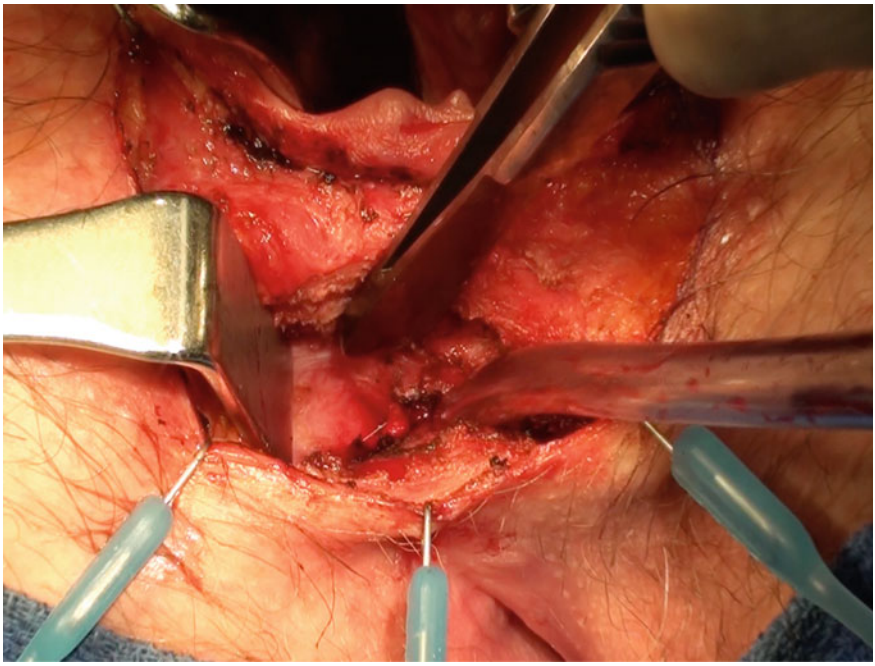


Fig. 10.3 Large amount of dead space after transperineal approach

infection or abscess needs surgical drainage, the vaginal side of the repair should be opened sufficiently to allow for proper dependent drainage.

Liquid stool found in the colon during RVF repair should not be taken lightly due to higher risk of repair failure. It is most often the result of incomplete bowel preparation in patients who have tendency for severe constipation or severe diverticulosis. This results in a large load of liquid fecal matter. In this instance, it is recommended that a colonoscopy be attempted before the formal repair in order to suction out all the liquid material. If this cannot be done, the procedure should be rescheduled. On occasion, diarrhea in the postoperative period can also be a harbinger of repair failure, due to inflammatory reaction in the lower rectum resulting from regional sepsis.

Recently, Alverdy at el. studied the influence of colonic microbiome on the failure of lower rectal repairs [23]. Their investigation focused primarily on the low and ultralow anastomosis performed in rectal cancer resections, which can likely be transposed to RVF repair. The early result of their research indicates that certain species of intestinal flora demonstrate higher tissue destructive potential. For this reason, oral antibiotics meant to sterilize the gut flora, in addition to mechanical bowel preparation, can often help in preventing fistula repair failure [24].

GI Complications

Constipation early in the postoperative period is frequently related to low oral intake of food and water, use of narcotic analgesics, and apprehension in having a bowel movement. This must be dealt with by counseling the patient, increasing oral fluid intake, and providing stool softeners and non-narcotic analgesics. If the patient is unable to defecate after a few days, particularly if there are obvious signs of fecal impaction, increased pelvic pressure, dysuria, or frequent return to the bathroom with only a small passage of liquids, a EUA is indicated in order to disimpact the stool and irrigate the rectum. If the repair becomes partially or completely disrupted during disimpaction, it must be left open and managed conservatively rather than attempting to perform a re-repair. If the wound needs to lay open, all foreign bodies must be removed, including sutures, plug, mesh, and staples.

Incontinence following RVF repair is strictly related to the preoperative status of the patient's continence. Preoperative endoanal ultrasound, anorectal manometry, and pudendal nerve terminal motor latency studies will all provide information to enable the surgeon to have a detailed and meaningful discussion with the patient and the family during the informed consent process. This is critical because, in the United States, postoperative fecal incontinence is the most common reason for litigation in anorectal surgery [25].

If the patient's RVF is the result of obstetric injury, a preoperative workup might reveal a source neurologic injury (prolonged pudendal nerve latency) which can result in persistent incontinence, despite an excellent anatomic repair [26]. Alternatively, if the endoanal ultrasound shows significant concomitant injuries to the internal and external sphincter, a simultaneous or delayed sphincter repair might be indicated and this possibility should also be included in the informed consent discussion.

Diarrhea in the early postoperative period may be related to preoperative bowel preparation (incomplete prep), irritable bowel syndrome (D predominant), rising prevalence of *Clostridium difficile* colitis, as well as early signs of fistula repair failure. Underlying Crohn's disease and radiation-induced diarrhea should also be ruled out, preferably before the repair.

Genitourinary Complication

Urinary retention after any anorectal operation, especially hemorrhoidectomy and complex restorative procedures under spinal or epidural anesthesia, is common and can occur in up to 10% of patients [27]. Following a complex RVF repair, a Foley catheter can be inserted for 24–48 h if urinary retention occurs. If the patient is unable to void after removal of the catheter, it is best to reinsert the Foley catheter and send the patient home with the catheter in place for 4–5 additional days. Daily administration of oral Flomax® (0.4 mg) prior to removal of the catheter will obviate the need for further catheterization. This policy is preferred over repeated catheterization in emergency rooms, which can increase the risk of urinary tract

infection and, even worse, can result in disruption of the repair. Judicious use of narcotic analgesics, which can contribute to urinary retention, is also important [28]. If urinary tract infection does develop, urine cultures and sensitivities and antibiotics are indicated according to standard practice protocols.

Following RVF repair, women should be instructed to refrain from having sexual intercourse for 6–12 weeks. Dyspareunia is not uncommon and usually subsides with time. It can, however, be particularly bothersome in young, sexually active women who have had extensive repair such as levatorplasty, which can contribute to narrowing of the introitus [29]. Physical therapy and use of vaginal dilators are rarely needed.

Surgeons should counsel the patient that subsequent vaginal delivery can put the repair at risk and lead to higher failure rates of repeated repairs. This allows the patient to make an informed decision as to whether to defer the RVF repair until after subsequent vaginal deliveries, particularly if she is experiencing minimal symptoms. Conversely, she may choose to have the RVF repair and undergo elective Caesarian section for subsequent deliveries. This information and recommendations should be provided in writing to the patient and her obstetrician to prevent subsequent claims of negligence.

Complications Related to Particular Repairs

Transanal Approaches

Rectal Advancement Flap

Rectal advancement flap (RAF) is a relatively simple technique with a high success rate. While rates vary, reports have them at 80% in most cases [7, 30–33]. Despite its popularity, however, the technique has many variations and modifications, as can be seen in the major surgical textbooks. In the authors' experience, the procedure involves mobilization of a well-vascularized full thickness segment of the rectal wall, which is then used to cover the repaired fistula defect. Although the technique can be used in the mid and lower rectum, it is most often used for anovaginal fistulas. In such cases, the creation of a flap is begun just distal to the fistula. Initially, the flap involves only the anoderm, but at the level of the anorectal junction the dissection is moved deeper to involve the full thickness of the rectal wall. Of note, the internal and external sphincter muscles are not incorporated into the flap and can be used for the fistula repair. Since the repair frequently involves a two-layer closure (muscle layer and the anorectal wall layer), the vaginal opening can be left open for drainage. This approach allows for more muscle bulk to be used for the repair. The anoderm should be detached from the internal sphincter in a relatively narrow (around 1–1.5 cm) segment, as compared to the width of the flap mobilized in the rectum. A resulting thick flap is then used to cover the repair in the exposed muscle.

One of the major complications of RAF is necrosis of the flap due to ischemia, which inevitably leads to fistula recurrence. This can often be avoided by following the principle that the base of the flap should be at least twice the size of its apex. In reality, the base of the flap (in the rectum) stretches widely between the levators (antero-lateral aspects of the lower rectum adjacent to the levators). Additionally, since the dissection is performed in the anterior aspect of the rectum, the prone position is often the most preferred position. Some surgeons claim that the submucosal flap or partial thickness flaps are sufficient for reconstruction, however, the full thickness flap naturally has the best perfusion, and therefore should be the preferred method. It is possible, although rare, to create vasoconstriction of the flap, which can then cause hypoperfusion by injection of epinephrine solution given preemptively for anesthetic and hemostatic purposes. Smoking has also been found to impair blood flow of the rectal mucosa [12, 34].

Another complication of RAF is retraction of the flap. In general, when dissected from the surroundings, most tissues have tendency to retract and shrink. For this reason, the mobilized RAF always shrinks and retracts if not secured properly to the donor site. Therefore, tension on the distal suture line should be avoided when the flap is sutured in place. In order to achieve this, the most proximal sutures securing the flap are placed more distally on the recipient rectal wall than on the flap side. This will result in flap advancement relative to the donor site and eliminate tension on the distal suture line. Flap retraction can also result from breaks of sutures. In order to avoid this, it is recommend that absorbable sutures (e.g., Vicryl) be used, at least 3-0 size along the 2-0 sutures. If possible, plication of the vaginal wall can serve to shorten the distance between the base of the flap and the sphincter and can also increase the final reach of the flap while taking any unnecessary tension from the apex after suturing [35]. Anal stenosis is rare and only seen in some cases of failed repair.

Incontinence following the RAF procedure is usually mild, if at all present, and is related to the procedure itself, particularly if a large portion of the internal sphincter muscle is excised and incorporated into the flap (this is not recommended, as stated earlier). Some seepage might also be observed if a large portion of the flap creates ectropion, which is rectal mucosa protrusion at the anal verge. For symptomatic ectropion, it is best to allow the RVF repair to heal completely, wait 4–6 months, and then excise the ectropion, leaving the wound open or closing it with dermal advancement [36–38]. Fortunately, incontinence is a rare complication following a successful RAF procedure [39]. Preservation of the internal sphincter is crucial in maintenance of continence in these patients with already compromised sphincter mechanism.

Intraoperative bleeding is rare during flap procedures, although mild oozing can be disturbing to the patient if not controlled adequately. Repositioning the Lone Star ® retractor prongs (if used) to achieve optimal exposure can cause bleeding from the anoderm or hemorrhoidal plexus, thereby obscuring the deeper operative field. A simple suture ligature can control this bleeding more efficiently than cautery. Additionally, dissection of the flap in the submucosal plane along the internal sphincter muscle can cause bleeding from the internal hemorrhoidal plexus. Here

again, excessive cautery should be avoided to prevent damage to the internal sphincter. Oftentimes, injection of the anoderm with a lidocaine/epinephrine solution can be helpful. During further dissection, some bleeding from the posterior vaginal wall can also be encountered if dissection veers off the avascular rectovaginal septum toward the perivaginal venous plexus. It is important to recognize this mistake. Again, small suture ligatures are better that cautery and decreases injury to the vaginal wall.

Hematoma can form in any dead space following the surgical dissection of well-vascularized tissues. This can include RAF repair. The amount of dissection should always be balanced to allow for appropriate tissue advancement, yet still minimize the amount of dead space. In the authors' experience, redundant vaginal wall can be often plicated/imbricated during closure of the defect, reinforcing the repair just as the anterior rectum is plicated during transvaginal rectocele repair [35]. By doing this, flap is advanced distally while the amount of dead space is minimized.

Bleeding due to an increase in pelvic pressure from diarrhea or constipation can also cause hematomas. For this reason, postoperative constipation should be carefully addressed with stool softening but not forcing agents. Diarrhea can be controlled with bulking (fiber) or hypomotility (e.g., Imodium) agents.

Rectal Sleeve Advancement

Rectal sleeve advancement is a rarely used procedure reserved for RVFs that are associated with significant disease or scarring of the distal rectum or anorectal ring; and is seen mainly in patients with Crohn's disease [40]. The dissection starts at the level of the dentate line, with preservation of as much sphincter as possible, and is then carried out in the perimesorectal plane in order to achieve adequate mobilization of the distal rectum. The vaginal opening is then closed and the rectum is advanced in a pull-through fashion [41]. The diseased anorectal segment is removed and a coloanal hand-sewn anastomosis is created.

Rectal sleeve advancements are typically performed under the protection of diverting stoma. The main complications are similar to those seen in the pull-though repairs that are done for rectal cancer. They include dehiscence of anastomosis, retraction of the advanced segment, pelvic sepsis, and anastomotic stricture. The recurrence rate of the fistula is low, as reported by a few studies [40, 42]. In some cases, closure of the fistula is seen, despite initial dehiscence of the rectal repair, and as long as the vaginal defect remains closed. In those cases, conservative management allows for secondary healing of the rectal defect. Certain modifications of this technique have also been described [43, 44]. As with most coloanal pull-through procedures, a certain degree of incontinence can be expected.

Vaginal Advancement Flap

Vaginal advancement flap (VAF) is a relatively simple technique with similar, basic principles associated with the rectal advancement flap [15, 45, 46]. Of note, it is rarely used in colorectal practice because most rectal surgeons agree that creation of the primary flap on the high-pressure side of the fistula (anorectum) should be the

priority. It does, however, make sense if the underlying disease, such as Crohn's disease, involves the anorectum and/or the patient is diverted. Similar to RAF, the other side of the repair (in this case the anorectum) can be left open for drainage and secondary healing, as long as the patient is diverted.

The VAF can also be used with the Martius flap, which involves transposition of bulbospongiosus (frequently named bulbocavernosus) muscle and labial fat pad (described later in this chapter) [45, 47–49].

Complications associated with the vaginal advancement flap are of similar nature to RAF, since the technique is the mirror image of the latter. It is important to note that the perivaginal venous plexus is concentrated mainly in the lateral aspect of the vagina with some extension on the posterior wall. Because of this, more venous bleeding is to be expected during lateral vaginal flap dissection.

Due to frequent disease in the anorectum, the recurrence rate of VAF is expected to be higher than the recurrence rate for RAF. If the fistula persists after VAF, there is the possibility of more than one vaginal opening (fistula branching), due to violation of the vaginal wall during flap creation.

Dermal Advancement Flap

The dermal advancement flap (DAF) technique for treatment of fistula-in-ano (including anovaginal fistulas) was introduced by Del Pino and Nelson in 1996 [50, 51]. The technique can be used selectively to treat low anovaginal fistulas in patients with sufficient amount of skin and subcutaneous tissue of the perineal body [52]. The technique involves excision of the internal opening within the anus, creation of a proximal anodermal mini-flap (lip), closure of the sphincter defect, and advancement of the mobilized flap which is comprised of skin, distal anoderm, and subcutaneous tissue into the anal canal. Subsequently, the flap is secured into its target site (lip) with the interrupted sutures.

Complications of the DAF procedure are similar to the above-mentioned flap techniques and, like those, the procedure follows the principles of vascular blood preservation. Complications are also similar to those associated with anoplasty. Candidates for DAF should be evaluated for true feasibility by taking into account previous episiotomy scars and the risk of vascular compromise if the perineum has previously been repaired. Patients with a short perineal body (the distance between the anus and introitus) and thin body habitus are usually not good candidates for this type of reconstruction, unless more soft tissue can be found just lateral to the perineal body (obesity is a favorable factor here). Similar to the advancement flap techniques used for anal stenosis, the risk for infection leading to flap failure is not low. In order to decrease the infection and suture line dehiscence, the harvest defect is often left open for healing by secondary intention.

Fistulectomy with Layer Closure

In selected patients, fistulectomy with layer closure can be considered. Because anovaginal fistulas have more available tissue for reconstruction than RVFs, the chances for success are higher for anovaginal fistulas. In these cases, the fistula tract

epithelium is excised and the sphincter defect and anoderm are closed separately. If recurrence occurs, it often leads to creation of a wider fistula than before.

In patients with more proximal RVFs, there is a higher risk of recurrence due to two directly opposing suture lines from the rectal and vaginal sides. For this reason, some authors advocate interpositioning of the repairs with a biologic sheath of mesh [53]. The potential complication for this approach is mainly infection of the mesh, its liquefaction, foreign body reaction, and mesh extrusion.

Plug Repair

The fistula plug repair using biologic material was designed primarily for cryptoglandular fistulas. With time, however, it has also been used with some success for anovaginal fistulas [54]. The plug was commonly used for anovaginal fistulas but was found to be impractical to repair more proximal RVFs due to lack of supporting tissues, as well as shortness of the fistula tract. A modified plug was introduced to circumnavigate this problem, and consisted of a flat anchoring portion (button/disk) and a tail [55]. In recent years, the plug repairs have fallen out of favor due to their poor success rate [56].

A commonly mentioned statement about the safety of the plugs, despite their poor success is only partially true. It should be remembered that insertion of the plug into a tight fistula channel is inevitably associated with widening of the internal opening. In cases of failure, the plugs are commonly extruded, but significant infection (abscess) can form, leading to more tissue destruction and creation of a larger fistula than before.

Fibrin Glue

The use of fibrin glue was introduced by Hjortrup and colleagues for the treatment of fistula-in-ano and has had very minimal success in the treatment of RVF [57–59]. Logistically, it is difficult to use this technique for anovaginal fistulas and impossible to use for true RVFs. If an attempt is made to use it for anovaginal fistulas, it is recommended that the internal opening be closed to prevent the escape of glue into the anal canal. In theory, proximal diversion could increase the chances of success by eliminating the high-pressure zone in the anorectum, although this hasn't been studied.

Complications associated with fibrin glue, although rare, include retention of fibrin glue material in the fistula tract, thereby risking the infection and redevelopment of abscess, which in turn can lead to further destruction of the rectovaginal septum.

Transperineal Approaches

Ligation of Intersphincteric Fistula Tract

Ligation of the intersphincteric fistula tract, or LIFT procedure, was invented by Rojanasakul in Thailand in 2007, as an option for treating anal fistulas, although it can also be incorporated into treatment of anovaginal and selected low RVF [60].

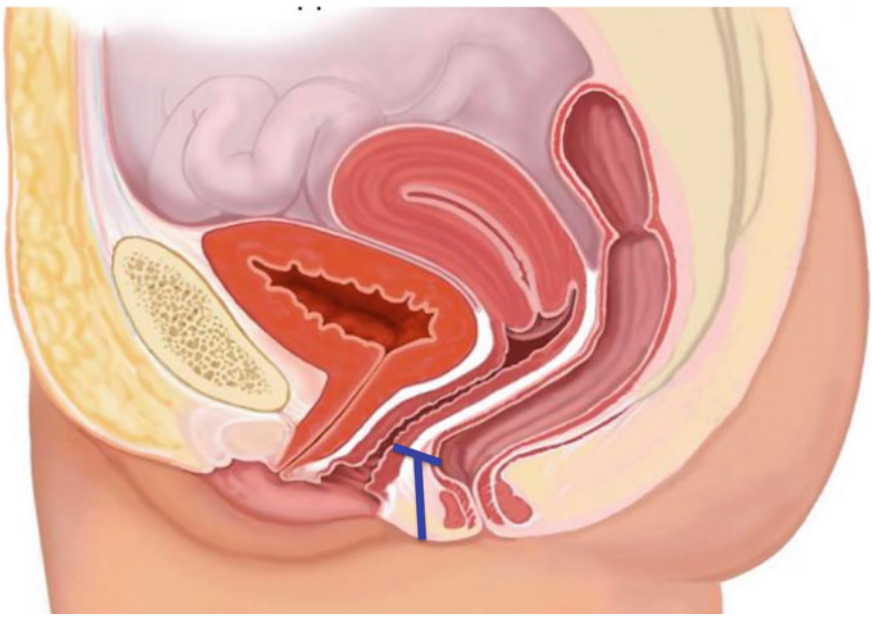


Fig. 10.4 T-type perineal type fistula complicating RVF after failed transperineal repair

The procedure is relatively simple and is associated with a limited amount of dissection along the anatomical planes. It involves the transverse incision over the perineal body, at or close to the intersphincteric groove. Subsequently, the dissection is continued in the avascular intersphincteric plane to the level of the fistula tract. The tract is then ligated, divided, and the incision closed.

Because there is a limited amount of dissection and the dissection is performed in the avascular plane, complications of the LIFT procedure are rare. The main complication is recurrence (overall success rate 74% for all anorectal fistulas) which typically presents in the form of minor perineal infection [61]. In this case, the perianal body creates a T-type fistula (Fig. 10.4) or, in more favorable situations, an intersphincteric perineal fistula.

Sphincteroplasty (with and Without Levatorplasty) with Repair of Fistula

Sphincteroplasty is best suited for patients with anovaginal fistulas and underlying fecal incontinence due to sphincter defect. The intent is to reconstruct the anal sphincter ring and to provide a locally harvested muscle tissue for a solid foundation under the fistula repair. In addition, an important and frequently underappreciated advantage is perineal body reconstruction, which can translate into improved sexual function and decreased frequency of urinary tract infections.

The sphincteroplasty is preferably done using an overlapping technique [62]. This can be performed with transection or preservation of anterior sphincteric scar.

Repair without division of the scar has a theoretical advantage for preservation of the sphincteric ring, in case the repair fails. On the other hand, it is often more practical and easier to execute the sphincteroplasty and repair the fistula when an attenuated scar is transected and direct access is provided to the perineal body and the deeper rectovaginal septum. Occasionally, the sphincteroplasty can be created in such a way that one of the free ends of the muscle is opposing the anorectal repair, thus providing a solid foundation. Additionally, in selected patients, levatorplasty can be added to augment the sphincter repair [63].

The complications of sphincteroplasty and concomitant fistula repair are rare. They can be related to the fistula repair itself or the sphincteroplasty. These repairs have rather high success rates (65–100%), although failures do occur [4, 5, 64]. In order to increase the success rate, some reports suggest adding rectal advancement flaps to the sphincteroplasty [65]. In some cases, the anorectal opening is closed by creating mini-flaps or simple approximation of the debrided fistula edges. In cases with a larger anorectal opening, it might be difficult to approximate the devascularized edges together. Then, the only good way to secure that part of the repair is to tuck them to the healthy underlying muscle using absorbable sutures. Similar to the LIFT procedure, recurrence can show itself as a T-type fistula to the perineal body, or convert the fistula into an anterior perineal fistula-in-ano.

The golden principle of any perineal repair is to perform an absolute minimal amount of necessary dissection to prevent denervation of the sphincter or creation of dead space, which can lead to infection and subsequent recurrence. For this reason, many surgeons opt to either leave the vaginal defect open or leave the perineal wound partially open while the vaginal opening is closed. Leaving the perineal wound open when the repair fails can result in the RVF converting into a perineal fistula-in-ano. In order to prevent devascularization of the anoderm, the internal sphincter should not be isolated (dissected off the anoderm). Instead, isolation of the external sphincter should be performed along the intersphincteric groove, thereby keeping the internal sphincter attached to the anoderm.

Bleeding during sphincteroplasty occurs most commonly from the lateral and posterior vaginal sinus and can be easily controlled with fine hemostatic absorbable sutures. This is preferred over excessive cauterization, which can inevitably lead to more tissue damage. This type of bleeding usually happens when the external sphincter muscle is being dissected from the lateral aspects of vagina.

Infections during sphincteroplasty are rare. They can be prevented by preoperative bowel preparation, standard preoperative antibiotics, hemostatic technique, avoidance of dead space, frequent water rinsing to wash off debris and prevent tissue desiccation, and adequate drainage of the operative site, as mentioned above. If infection occurs it is likely to respond to oral antibiotics as long as no foreign bodies persist. This can include thick sutures with an extended degradation period, which might need to be removed to allow for quicker recovery.

In some instances, sphincteroplasty can result in stricture formation if the muscle is tightened excessively. This can lead to evacuation problems and create unusually high intrarectal pressure, which can in turn be the reason for fistula repair failure.

Superficial dehiscence of the perineal wound is common, which is why the wound is often left partially open. The likelihood of partial skin necrosis depends on the thickness and vascularity of skin flaps, as well as any previous scars in the area. Conversely, muscle repair dehiscence is very rare, as long as the overlap repair is created using viable muscle fragments. In most of cases, it is partially the scarred muscle, through which the sutures are placed. Still, when the sutures are placed through the viable muscle, care should be taken to avoid close bites that would compromise the muscle blood supply.

Levatorplasty is frequently incorporated into sphincteroplasty to augment the repair [64, 66, 67]. Two or three strong sutures used to bring the puborectalis and pubococcygeus muscles together can also be associated with temporary deeper pelvic pain and occasional urinary retention. It should be noted that not all patients are candidates for levatorplasty. For instance, it can be difficult in patients with long anal canal and strong levator muscles to approximate the levators, as compared to patients with thin, lax and pliable levators. The decision to perform levatorplasty should be individualized. Additionally, any excessive tension on the approximated muscle can lead to significant pain. Attempt to perform levatorplasty can also lead to muscle tears and bleeding into the perineum and levatorplasty can lead to temporary dyspareunia [29, 68].

Episioproctotomy

Episioproctotomy involves complete transection of all perineal tissues distal to the fistula tract and was popularized by Hull and colleagues [32, 69]. The episioproctotomy involves both the internal and external components of an intact sphincter complex, the anoderm, perineal body, and part of the vaginal and rectal wall. In essence, a controlled fourth degree laceration through the center of the perineum is created to the level of fistula. The fistula is then excised and layered anatomical closure of all corresponding structures takes place [70].

The success rate for episioproctotomy is high, as reported in several small series from tertiary institutions [32, 71, 72]. A word of caution should be given, however, that despite the initial success rate, the rate of late incontinence has not yet been studied. The likelihood of this is based on the increased incidence of fecal incontinence that is frequently observed several decades later in females who underwent the fourth degree laceration repair during delivery [73]. Partial dehiscence of the repair, mainly due to infection, can lead to re-creation of a much larger RVF. Likewise, complete dehiscence will lead to cloaca deformity. After transection of the perineum to the level of the fistula (essentially a perineal fistulotomy), special attention should be given to not overstretch the vaginal or rectal lumen with a self-retaining retractor. This can help to avoid inadvertent extension of the tear to more proximal rectovaginal septum.

Transperineal Anatomical Deconstruction with Layered Anatomical Closure

Transperineal anatomical deconstruction of the perineal body to address the rectovaginal or anovaginal fistulas is usually performed following one or two failed

transanal approaches [74]. The main implication of this is a higher level of procedural complexity. Scarring can also be expected not only from the vaginal side (e.g., episiotomy) but also from the rectal side (previous advancement flap). The risk of partial devascularization is also higher (anoderm, sphincter), as it the chance of muscle weakening due to denervation. Fecal incontinence is usually a late occurrence.

Transperineal anatomical deconstruction can create large dead space (Fig. 10.4), mainly due to the shrinking effect of surrounding scars and muscles, and also due to the fact that the perineal body is a central anchoring point of all the surrounding structures. The dead space is often deeper in obese patients or in patients with well-developed pelvic floor musculature. This is sometimes associated with fluid accumulation and subsequent infection, and can lead to recurrence and creation of a perineal T-type fistula. Frequently, dead space formation can be decreased by approximation of the levators (levatorplasty), which can also augment the repair and help with continence. Perineal sepsis, when developed, often persists until the postoperative cavity decreases and becomes shallow. The use of biologic mesh has been found to augment the transperineal repairs with some success, however, when infection occurs, the infected mesh can be the source of suppuration until it is removed or completely liquefied [53].

Transperineal Repair with Gracilis Muscle Interposition

Tissue flap interposition utilizing the gracilis muscle for transperineal repair is usually considered after other, less complex repairs fail [75]. The procedure involves transperineal anatomical deconstruction of the rectovaginal septum, closure of anorectal and vaginal defects, and gracilis muscle harvest based on the proximal neurovascular bundle supply. Following this, the mobilized muscle is then delivered through the subcutaneous tunnel and secured in the rectovaginal septum, thereby separating the rectal and vaginal defects with well-vascularized tissue. The perineal incision is closed over the repair while drainage of the rectovaginal septum is frequently performed.

The complications of tissue flap interposition can be related to the transperineal deconstruction of the rectovaginal septum (mentioned earlier), and the muscle harvest. In addition, many of these complex repairs are performed under protection of stoma, therefore stoma-inherent complications should also be considered. The success rate for gracilis muscle interposition has been reported to be between 53 and 92% [22, 75–77].

Harvesting of the gracilis muscle is performed in lithotomy position through a full-length incision along the muscle, or with two separate smaller incisions at the proximal and distal end of the muscle. Viability of the muscle depends on the constant proximal neurovascular pedicle, which cannot be kinked when the mobilized muscle is deflected and brought through the subcutaneous tunnel. The perforating vessels along the muscle course are then divided. In order to decrease the chance of leg wound infection, this part of procedure should be performed using a separate instrument tray and draping.

The complication rate for this procedure has been reported by Carr et al. as less than 10% for in-hospital donor site complications [78]. These included local wound problems (i.e., pain, infection, bleeding) and a single case of temporary sciatic nerve palsy. Long-term donor site issues related to scar (e.g., pruritus, discoloration, width, sensitivity) were reported in approximately half of the cases. Other complications included tingling and hypesthesia. Functional difficulties were reported by 26% of patients, with 15% reporting temporary weakness that lasted an average of 6 months. Six percent of participants reported persistent weakness that interfered with running, walking, or participation in sports. Another possible complication is the muscle retracting from its rectovaginal recipient site, often resulting in fistula recurrence.

Perineal wound infection can occur, as can hypesthesia of the perineum. If ischemia of the muscle occurs, the muscle will turn dark during surgery, which should alter the surgeon to abort the procedure and examine the muscle. More insidious postoperative ischemia will manifest itself as pain, swelling, erythema, and wound drainage in the ensuing days, which will ultimately result in muscle necrosis and wound disruption. The muscle will then need to be excised. Recovery from a failed gracilis muscle interposition can take weeks to months. Dyspareunia was reported in 57% of patients and was partly related to perineal scarring [79].

Martius Flap

The Martius flap technique involves harvesting the labial fat pad and frequently the bulbospongiosus (bulbocavernosus) muscle, based on the blood supply from the posterior labial branches of the perineal artery, which is itself a branch of the internal pudendal artery [47–49, 80–82]. This blood supply reaches the fat pad from the posterolateral direction. The flap can be performed during transperineal or transvaginal approaches. Complications of the Martius flap are similar to RAF or VAF [80]. Ischemia of the flap is directly related to its solitary blood supply and can be caused by sharp angulation (kinking) of the vascular pedicle. Ischemia of the fat (necrosis) underneath the closed repair can result in local sepsis. Other complications include dehiscence of the donor site, labial scaring, and dyspareunia. At 35-month follow up, Pitel et al. reported a 50% success rate using the Martius flap technique in Crohn's disease patients with RVF [83].

Indocyanine Green

The use of indocyanine green (ICG) to evaluate real-time adequacy of the blood supply is increasing in popularity in colorectal, vascular, and reconstruction procedures [84]. Although its use in ascertaining adequacy of the flap vascularity might seem unnecessary in rectovaginal repair procedures, this emerging technology should not be forgotten in more complex cases when an adequate blood supply is uncertain.

Mesh Interposition

This concept of mesh interposition involves the insertion of biologic mesh (e.g., porcine intestinal submucosa) in the rectovaginal septum following separation of

the two organs [85, 86]. The interposed mesh must sit flat without pleating or bulking and be cultured with absorbable sutures. An excellent blood supply and dry operating field are essential in order to incorporate the mesh into the healing wound and avoid any complications. The true rate of success for this procedure is unknown due to a small number of patients in the reported series [87]. A short-term success rate of 71–80% has been reported at 1 year but no long-term data is available [53]. The complications of mesh insertion consist mostly of extrusion of the material and infection. The loss of mesh results in recurrence (persistence) of RVF but has no documented effect on anal continence. It might, however, create recurrent infection, which in turn can lead to further sphincter damage.

Transabdominal Operations

Bricker Procedure

The Bricker procedure is one of the earliest innovative procedures for high RV fistulas resulting from radiotherapy and was proposed by Bicker and Johnston in 1979 [88]. Today, it is very rarely performed due to advancement in the ultralow and coloanal pull-through techniques. The Bricker technique involves disconnecting the sigmoid and using its proximal end to seal the rectal side of RVF as an onlay patch, then re-anastomosing the colon to the healthy folded segment of the sigmoid while frequently leaving the vaginal side alone [89]. Complications of this procedure have been associated with difficulty in performing hand-sewn suture lines of the onlay patch as well as the colosigmoid anastomosis.

Pull-Through Procedures

Due to advancements in the technique of total mesorectal excision, pull-through operations have replaced the Bricker procedure [90]. Today, the preferred procedure is very low anterior resection with ultralow colorectal or coloanal anastomosis and diverting ileostomy. These cases can be performed in a minimally invasive fashion. The vaginal opening is debrided, and left open or closed with an omental graft (if possible) inserted in between the vagina and the colorectal anastomosis [91]. A side-to-end or pouch coloanal or colorectal anastomosis is preferred to an end-to-end anastomosis due to a better blood supply [92, 93]. The complications of very low colorectal or coloanal anastomosis are discussed in a separate chapter.

Omental Interposition

The excellent blood supply of the omentum makes it an ideal tissue flap in many operations. A well-mobilized omental graft, placed either on the right or left gastroepiploic artery, can be brought into the pelvis for interposition [91]. After completion of dissection of the rectovaginal septum (or proctectomy), the omentum is placed in the pelvis to cover the fistula repair. Failure of this operation is mainly due to omental reach, tension, and ischemia of the graft, in addition to deep pelvic infection [91].

Diversion

Temporary colostomy or ileostomy is often constructed after multiple rectovaginal repair failures. It is unclear as to whether fecal diversion helps heal the RVF but it certainly can divert stool in diarrheal predominant IBS patients [22]. Endoscopic and radiologic studies should be performed 3 months after RVF to confirm complete healing prior to stoma closure. Permanent diversion is often the final option in the treatment of RVF and fecal incontinence, and in some cases of severe Crohn's disease. This should not be construed as failure but instead as the best option for selected patients. The inherent complications of ileostomy or colostomy should also be considered.

Conclusion

RVF is frequently a frustrating problem for both the patient and surgeon. It encompasses a large spectrum of clinical scenarios, caused by a variety of etiologies and requiring different treatment modalities [6, 94]. Each of the treatment modalities has its own potential complications. Because of this, it is not unusual for an experienced surgeon to refer a patient with RVF to a center known for its large experience and good results. This should be viewed as admirable and responsible, and not as a sign of weakness, but rather good judgment. Surgeons who treat patients with RVF need to be familiar with many different surgical procedures in order to offer each patient a carefully selected, tailored operation with a low rate of morbidity.

References

1. Ng'ang'a N. Women of the courtyard. A nurse's journey to treat obstetric fistulae in Niger. AWHONN Lifelines. 2006;10(5):410–7.
2. Hoexter B, Labow SB, Moseson MD. Transanal rectovaginal fistula repair. Dis Colon Rectum. 1985;28(8):572–5.
3. Adler AJ, et al. Estimating the prevalence of obstetric fistula: a systematic review and meta-analysis. BMC Pregnancy Childbirth. 2013;13:246.
4. Halverson AL, et al. Repair of recurrent rectovaginal fistulas. Surgery 2001;130(4):753–7; discussion 757–8.
5. Lowry AC, et al. Repair of simple rectovaginal fistulas. Influence of previous repairs. Dis Colon Rectum. 1988;31(9):676–8.
6. MacRae HM, et al. Treatment of rectovaginal fistulas that has failed previous repair attempts. Dis Colon Rectum. 1995;38(9):921–5.
7. Sonoda T, et al. Outcomes of primary repair of anorectal and rectovaginal fistulas using the endorectal advancement flap. Dis Colon Rectum. 2002;45(12):1622–8.
8. Oakley SH, et al. Practice patterns regarding management of rectovaginal fistulae: a multicenter review from the Fellows' Pelvic Research Network. Female Pelvic Med Reconstr Surg. 2015;21(3):123–8.
9. Buchleitner AM, et al. Perioperative glycaemic control for diabetic patients undergoing surgery. Cochrane Database Syst Rev. 2012;(9):CD007315.

10. Sorensen LT. Wound healing and infection in surgery: the pathophysiological impact of smoking, smoking cessation, and nicotine replacement therapy: a systematic review. Ann Surg. 2012;255(6):1069–79.
11. Moller AM, et al. Effect of preoperative smoking intervention on postoperative complications: a randomised clinical trial. Lancet. 2002;359(9301):114–7.
12. Zimmerman DD, et al. Smoking affects the outcome of transanal mucosal advancement flap repair of trans-sphincteric fistulas. Br J Surg. 2003;90(3):351–4.
13. Yin HQ, et al. Clinical value of endoluminal ultrasonography in the diagnosis of rectovaginal fistula. BMC Med Imaging. 2016;16:29.
14. Sofic A, et al. MRI in evaluation of perianal fistulae. Radiol Oncol. 2010;44(4):220–7.
15. Bauer JJ, et al. Transvaginal approach for repair of rectovaginal fistulae complicating Crohn's disease. Ann Surg. 1991;213(2):151–8.
16. Sands BE, 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(10):912–20.
17. Hannaway CD, Hull TL. Current considerations in the management of rectovaginal fistula from Crohn's disease. Colorectal Dis. 2008;10(8):747–55; discussion 755–6.
18. Scott NA, Nair A, Hughes LE. Anovaginal and rectovaginal fistula in patients with Crohn's disease. Br J Surg. 1992;79(12):1379–80.
19. Schloericke E, et al. Transperineal omentum flap for the anatomic reconstruction of the rectovaginal space in the therapy of rectovaginal fistulas. Colorectal Dis. 2012;14(5):604–10.
20. McHugh PP, et al. Vaginal varices with massive hemorrhage in a patient with nonalcoholic steatohepatitis and portal hypertension: successful treatment with liver transplantation. Liver Transpl. 2008;14(10):1538–40.
21. Ruminjo JK, et al. Clinical procedures and practices used in the perioperative treatment of female genital fistula during a prospective cohort study. BMC Pregnancy Childbirth. 2014;14:220.
22. Pinto RA, et al. Are there predictors of outcome following rectovaginal fistula repair? Dis Colon Rectum. 2010;53(9):1240–7.
23. Shogan BD, et al. Intestinal anastomotic injury alters spatially defined microbiome composition and function. Microbiome. 2014;2:35.
24. Morowitz MJ, et al. The human microbiome and surgical disease. Ann Surg. 2011;253 (6):1094–101.
25. Kern KA. Medical malpractice involving colon and rectal disease: a 20-year review of United States civil court litigation. Dis Colon Rectum. 1993;36(6):531–9.
26. Lawson J. Rectovaginal fistulae following difficult labour. Proc R Soc Med. 1972;65(3):283–6.
27. Prasad ML, Abcarian H. Urinary retention following operations for benign anorectal diseases. Dis Colon Rectum. 1978;21(7):490–2.
28. Verhamme KM, et al. Drug-induced urinary retention: incidence, management and prevention. Drug Saf. 2008;31(5):373–88.
29. Miklos JR, Kohli N, Moore R. Levatorplasty release and reconstruction of rectovaginal septum using allogenic dermal graft. Int Urogynecol J Pelvic Floor Dysfunct. 2002;13(1):44–6.
30. Rothenberger DA, et al. Endorectal advancement flap for treatment of simple rectovaginal fistula. Dis Colon Rectum. 1982;25(4):297–300.
31. Watson SJ, Phillips RK. Non-inflammatory rectovaginal fistula. Br J Surg. 1995;82 (12):1641–3.
32. Hull TL, et al. Surgeons should not hesitate to perform episioproctotomy for rectovaginal fistula secondary to cryptoglandular or obstetrical origin. Dis Colon Rectum. 2011;54(1):54–9.
33. de Parades V, et al. Endorectal advancement flap with muscular plication: a modified technique for rectovaginal fistula repair. Colorectal Dis. 2011;13(8):921–5.

34. Zimmerman DD, et al. Smoking impairs rectal mucosal bloodflow–a pilot study: possible implications for transanal advancement flap repair. Dis Colon Rectum. 2005;48(6):1228–32.
35. Marecik S, et al. Repair of a Recurrent Traumatic Rectovaginal Fistula. ACS Video Library Cine-Med, 2015(ACS-3956).
36. Pearl RK, et al. Island flap anoplasty for the treatment of anal stricture and mucosal ectropion. Dis Colon Rectum. 1990;33(7):581–3.
37. Pidala MJ, Slezak FA, Porter JA. Island flap anoplasty for anal canal stenosis and mucosal ectropion. Am Surg. 1994;60(3):194–6.
38. Angelchik PD, Harms BA, Starling JR. Repair of anal stricture and mucosal ectropion with Y-V or pedicle flap anoplasty. Am J Surg. 1993;166(1):55–9.
39. Ortiz H, Marzo J. Endorectal flap advancement repair and fistulectomy for high trans-sphincteric and suprasphincteric fistulas. Br J Surg. 2000;87(12):1680–3.
40. Simmang CL, Lacey SW, Huber PJ Jr. Rectal sleeve advancement: repair of rectovaginal fistula associated with anorectal stricture in Crohn's disease. Dis Colon Rectum. 1998;41(6):787–9.
41. Berman IR. Sleeve advancement anorectoplasty for complicated anorectal/vaginal fistula. Dis Colon Rectum. 1991;34(11):1032–7.
42. Hull TL, Fazio VW. Surgical approaches to low anovaginal fistula in Crohn's disease. Am J Surg. 1997;173(2):95–8.
43. Veronikis DK, Nichols DH, Spino C. The Noble-Mengert-Fish operation-revisited: a composite approach for persistent rectovaginal fistulas and complex perineal defects. Am J Obstet Gynecol. 1998;179(6 Pt 1):1411–6; discussion 1416–7.
44. Mengert WF, Fish SA. Anterior rectal wall advancement; technic for repair of complete perineal laceration and recto-vaginal fistula. Obstet Gynecol. 1955;5(3):262–7.
45. Nosti PA, Stahl TJ, Sokol AI. Surgical repair of rectovaginal fistulas in patients with Crohn's disease. Eur J Obstet Gynecol Reprod Biol. 2013;171(1):166–70.
46. Sher ME, Bauer JJ, Gelernt I. Surgical repair of rectovaginal fistulas in patients with Crohn's disease: transvaginal approach. Dis Colon Rectum. 1991;34(8):641–8.
47. Kin C, et al. Martius flap repair for complex rectovaginal fistula. Pol Przegl Chir. 2012;84(11):601–4.
48. Kniery K, Johnson EK, Steele SR. How I do it: martius flap for rectovaginal fistulas. J Gastrointest Surg. 2015;19(3):570–4.
49. Kniery KR, Johnson EK, Steele SR. Martius flap for repair of recurrent rectovaginal fistulas. Dis Colon Rectum. 2015;58(12):1210.
50. Del Pino A, et al. Island flap anoplasty for treatment of transsphincteric fistula-in-ano. Dis Colon Rectum. 1996;39(2):224–6.
51. Nelson RL, Cintron J, Abcarian H. Dermal island-flap anoplasty for transsphincteric fistula-in-ano: assessment of treatment failures. Dis Colon Rectum. 2000;43(5):681–4.
52. Draganic B, Solomon MJ. Island flap perineoplasty for coverage of perineal skin defects after repair of cloacal deformity. ANZ J Surg. 2001;71(8):487–90.
53. Ellis CN. Outcomes after repair of rectovaginal fistulas using bioprosthetics. Dis Colon Rectum. 2008;51(7):1084–8.
54. O'Connor L, et al. Efficacy of anal fistula plug in closure of Crohn's anorectal fistulas. Dis Colon Rectum. 2006;49(10):1569–73.
55. Ratto C, et al. Gore Bio-A(R) Fistula Plug: a new sphincter-sparing procedure for complex anal fistula. Colorectal Dis. 2012;14(5):e264–9.
56. El-Gazzaz G, Zutshi M, Hull T. A retrospective review of chronic anal fistulae treated by anal fistulae plug. Colorectal Dis. 2010;12(5):442–7.
57. Hjortrup A, Moesgaard F, Kjaergard J. Fibrin adhesive in the treatment of perineal fistulas. Dis Colon Rectum. 1991;34(9):752–4.
58. Venkatesh KS, Ramanujam P. Fibrin glue application in the treatment of recurrent anorectal fistulas. Dis Colon Rectum. 1999;42(9):1136–9.

59. Abel ME, et al. Autologous fibrin glue in the treatment of rectovaginal and complex fistulas. Dis Colon Rectum. 1993;36(5):447–9.
60. Rojanasakul A. LIFT procedure: a simplified technique for fistula-in-ano. Tech Coloproctol. 2009;13(3):237–40.
61. Bastawrous A, et al. Results from a novel modification to the ligation intersphincteric fistula tract. Am J Surg. 2015;209(5):793–8; discussion 798.
62. Fang DT, et al. Overlapping sphincteroplasty for acquired anal incontinence. Dis Colon Rectum. 1984;27(11):720–2.
63. Miller R, et al. Anterior sphincter plication and levatorplasty in the treatment of faecal incontinence. Br J Surg. 1989;76(10):1058–60.
64. Tsang CB, et al. Anal sphincter integrity and function influences outcome in rectovaginal fistula repair. Dis Colon Rectum. 1998;41(9):1141–6.
65. Khanduja KS, et al. Reconstruction of rectovaginal fistula with sphincter disruption by combining rectal mucosal advancement flap and anal sphincteroplasty. Dis Colon Rectum. 1999;42(11):1432–7.
66. Corman ML. Anal incontinence following obstetrical injury. Dis Colon Rectum. 1985;28 (2):86–9.
67. Russell TR, Gallagher DM. Low rectovaginal fistulas Approach and treatment. Am J Surg. 1977;134(1):13–8.
68. Melich G, et al. Transverse incision transvaginal rectocele repair combined with levatorplasty and biological graft insertion: technical details and case series outcomes. Tech Coloproctol. 2016;20(1):51–7.
69. Hull TL, et al. Multimedia article. Success of episioproctotomy for cloaca and rectovaginal fistula. Dis Colon Rectum. 2007;50(1):97–101.
70. Tancer ML, Lasser D, Rosenblum N. Rectovaginal fistula or perineal and anal sphincter disruption, or both, after vaginal delivery. Surg Gynecol Obstet. 1990;171(1):43–6.
71. Mazier WP, Senagore AJ, Schiesel EC. Operative repair of anovaginal and rectovaginal fistulas. Dis Colon Rectum. 1995;38(1):4–6.
72. Pepe F, et al. Low rectovaginal fistulas. Aust N Z J Obstet Gynaecol. 1987;27(1):61–3.
73. Guise JM, et al. Incidence of fecal incontinence after childbirth. Obstet Gynecol. 2007;109(2 Pt 1):281–8.
74. Gottgens KW, et al. Rectovaginal fistula: a new technique and preliminary results using collagen matrix biomesh. Tech Coloproctol. 2014;18(9):817–23.
75. Wexner SD, et al. Gracilis muscle interposition for the treatment of rectourethral, rectovaginal, and pouch-vaginal fistulas: results in 53 patients. Ann Surg. 2008;248(1):39–43.
76. Furst A, et al. Gracilis transposition for repair of recurrent anovaginal and rectovaginal fistulas in Crohn's disease. Int J Colorectal Dis. 2008;23(4):349–53.
77. Lefevre JH, et al. Operative results and quality of life after gracilis muscle transposition for recurrent rectovaginal fistula. Dis Colon Rectum. 2009;52(7):1290–5.
78. Carr MM, Manktelow RT, Zuker RM. Gracilis donor site morbidity. Microsurgery. 1995;16 (9):598–600.
79. Papadopoulos O, et al. Gracilis myocutaneous flap: evaluation of potential risk factors and long-term donor-site morbidity. Microsurgery. 2011;31(6):448–53.
80. Boronow RC. Repair of the radiation-induced vaginal fistula utilizing the Martius technique. World J Surg. 1986;10(2):237–48.
81. White AJ, et al. Use of the bulbocavernosus muscle (Martius procedure) for repair of radiation-induced rectovaginal fistulas. Obstet Gynecol. 1982;60(1):114–8.
82. McNevin MS, Lee PY, Bax TW. Martius flap: an adjunct for repair of complex, low rectovaginal fistula. Am J Surg. 2007;193(5):597–9; discussion 599.
83. Pitel S, et al. Martius advancement flap for low rectovaginal fistula: short- and long-term results. Colorectal Dis. 2011;13(6):e112–5.

84. Atallah SB, et al. Application of laser-assisted indocyanine green fluorescent angiography for the assessment of tissue perfusion of anodermal advancement flaps. Dis Colon Rectum. 2013;56(6):797.
85. Pye PK, et al. Surgisistrade mark mesh: a novel approach to repair of a recurrent rectovaginal fistula. Dis Colon Rectum. 2004;47(9):1554–6.
86. Moore RD, Miklos JR, Kohli N. Rectovaginal fistula repair using a porcine dermal graft. Obstet Gynecol. 2004;104(5 Pt 2):1165–7.
87. Schwandner O, et al. Innovative technique for the closure of rectovaginal fistula using Surgisis mesh. Tech Coloproctol. 2009;13(2):135–40.
88. Bricker EM, Johnston WD. Repair of postirradiation rectovaginal fistula and stricture. Surg Gynecol Obstet. 1979;148(4):499–506.
89. Steichen FM, et al. Bricker-Johnston sigmoid colon graft for repair of postradiation rectovaginal fistula and stricture performed with mechanical sutures. Dis Colon Rectum. 1992;35(6):599–603.
90. Thomford NR, Smith DE, Wilson WH. Pull-through operation for radiation-induced rectovaginal fistula: report of a case. Dis Colon Rectum. 1970;13(6):451–3.
91. van der Hagen SJ, et al. Laparoscopic fistula excision and omentoplasty for high rectovaginal fistulas: a prospective study of 40 patients. Int J Colorectal Dis. 2011;26(11):1463–7.
92. Rubin F, Douard R, Wind P. The functional outcomes of coloanal and low colorectal anastomoses with reservoirs after low rectal cancer resections. Am Surg. 2014;80(12):1222–9.
93. Fazio VW, et al. A randomized multicenter trial to compare long-term functional outcome, quality of life, and complications of surgical procedures for low rectal cancers. Ann Surg. 2007;246(3):481–8; discussion 488–90.
94. Tozer PJ, et al. Surgical management of rectovaginal fistula in a tertiary referral centre: many techniques are needed. Colorectal Dis. 2013;15(7):871–7.

Incontinence

11

Christina Warner and Anders Mellgren

Introduction

Fecal continence is a complex process mediated by sensation of anorectal contents, anorectal reflexes, reservoir ability, voluntary control of stool, cognitive awareness, and stool volume and consistency [1]. Any disruption in the above factors may result in fecal incontinence (FI). By estimation, FI affects between 7 and 15% of the United States population with an increased prevalence in women and the elderly [2]. FI appears to effect women more than men secondary to obstetric injury. Risk factors for FI include advanced age, gender (women), diabetes mellitus, urinary incontinence, loose stools, poor health status, and chronic illness [3]. Overall, the true prevalence of FI may be underreported because of embarrassment caused by the condition and suboptimal screening efforts amongst health care providers. FI carries significant psychological and socioeconomic burdens. The necessity for diet modification, the constant need for proximity to a bathroom, fear of embarrassment and odor, and shame often results in social restriction amongst patients affected by FI.

Numerous scales have been developed to discriminate the severity and impact of FI [4]. The Cleveland Clinic Fecal Incontinence Score (CCFIS; Table 1), often referred to as the Wexner score, is the most commonly used severity scale to assess pre- and postoperative results. It calculates a score of 0–20 based on the patient's frequency of symptoms, where 0 is no incontinence and 20 is complete involuntary

C. Warner
Department of General Surgery, University of Illinois at Chicago,
840 South Wood Street #578, Chicago, IL 60612, USA

A. Mellgren (✉)
Division of Colon and Rectal Surgery, Department of General Surgery,
University of Illinois at Chicago, 840 South Wood Street #578, Chicago,
IL 60612, USA
e-mail: AFMellgren@uic.edu; Anders.mellgren@icloud.com

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_11

Table 1 The Cleveland clinic incontinence score

Type of incontinence	Never	Rarely	Sometimes	Usually	Always
Solid	0	1	2	3	4
Liquid	0	1	2	3	4
Gas	0	1	2	3	4
Wears pad	0	1	2	3	4
Lifestyle alteration	0	1	2	3	4

Never 0. *Rarely* <1/month. *Sometimes* <1/week, ≥1/month. *Usually* <1/day, ≥1/week. *Always* ≥1/day
Score Range 0 (perfect)–20 (complete incontinence)
Adapted from Jorge and Wexner [5]

loss of feces [5]. The Fecal Incontinence Quality of Life (FIQoL) questionnaire is a validated scoring of the impact of FI according to four categories including lifestyle, coping, self-perception and depression, and embarrassment [6].

Etiology and Evaluation of Fecal Incontinence

The pathogenesis and etiology of fecal incontinence may be multifactorial. It may be due to anal sphincter or pelvic floor muscle weakness (e.g., childbirth, operative, trauma), neuropathy (e.g., stretch injury of pudendal nerve, diabetes mellitus), abnormalities of the pelvic floor (e.g., fistula, rectal prolapse), anorectal inflammation (e.g., inflammatory bowel disease, radiation proctitis), central nervous system disease (e.g., cerebellar vascular accident, spinal cord lesion, multiple sclerosis), or bowel disturbances (e.g., irritable bowel syndrome, overflow diarrhea) [2]. Treatment should be directed by a detailed history and physical examination. It is important to ascertain the frequency of FI episodes, clarify symptoms of partial (involuntary leakage or flatus) or complete incontinence, determine stool characteristics (liquid, solid, mucous), and determine if there are symptoms of urgency. Dietary habits, history of congenital abnormalities, childbirth history, previous anorectal procedures, or low colon anastomosis should also be discussed.

Physical examination of the anorectum should include inspection, digital examination, and endoscopic assessment. Normally, the presence of soiled undergarments, anal fissures, prolapsing hemorrhoids, scars from previous surgeries, perineal length (decreased length is often associated with external anal sphincter defects), sphincter tone, a palpable defect, sensation to pinprick and the presence of an anocutaneous reflex should be noted [7].

There are several investigative tools available for assessment of fecal incontinence [7]. Endoanal ultrasound can define the presence and extent of an anatomical sphincter (internal or external) injury. Pelvic floor ultrasound can delineate other injuries and abnormalities of the pelvic floor and the pelvis. Anorectal manometry can objectively document anal resting and squeeze pressures and assess anorectal

compliance and the anorectal inhibitory reflex. Pudendal nerve terminal motor latency (PNTML), although its utility is controversial, may be used to assess possible nerve damage such as pudendal neuropathy. Defecography may be used to visualize the function of the rectum and document functional abnormalities, such as rectal intussusception, rectocele, enterocele, etc. It can be performed using fluoroscopic or MRI techniques.

Treatment of Fecal Incontinence

After a thorough assessment of FI symptoms, treatment usually starts with conservative measures: patient education, normalization of stool consistency, behavioral techniques, and pelvic floor exercises [8]. Dietary modifications with avoidance of food triggers (e.g., caffeine, spicy foods, and alcohol), supplemental fiber and antidiarrheal agents (e.g., loperamide) are usually recommended for diarrhea-associated FI. On the other hand, laxatives are recommended in individuals with FI-associated fecal impaction. Topical treatments that increase smooth muscle tone (e.g., phenylephrine, valproate) have also been trialed in patients with FI and have shown mild, statistically significant, improvement in bowel control [8]. Behavioral training includes scheduled toileting attempts and preventive techniques such as squeezing prior to increased intraabdominal pressure activity such as bending, coughing, or lifting.

Pelvic floor exercises to strengthen the pelvic floor musculature are frequently recommended. Although there is no consensus on how to perform pelvic floor exercises, patients are usually taught methods to increase self-awareness of contraction and relaxation of the pelvic musculature. Biofeedback therapy, an instrument-assisted training strategy, allows patients to visually assess the character and quality of their pelvic strengthening exercises [7]. Biofeedback implementation for FI has success rates between 40 and 100% in different studies [9]. Heymen and coworkers [9] sought to determine the effectiveness of biofeedback training compared to pelvic floor exercises for FI in a clinical, randomized-controlled trial. In their study, prior to treatment, patients received educational training on anatomy and physiology of the pelvic floor muscles, a review of their anorectal manometry results, and instructions on fiber supplements and antidiarrheal medications. After four weeks of conservative measures, 21% of patients experienced adequate relief from FI, and there was an overall decrease of 41% in FI days. Three months after initiation of either biofeedback training or pelvic floor exercises, 67% of patients treated with biofeedback reported adequate relief from FI compared to 41% of patients treated with pelvic floor exercises. Moreover, biofeedback patients had fewer days with FI (not statistically significant), greater reduction in their Fecal Incontinence Severity Index Scores anal canal squeeze pressures, as well as less abdominal tension during squeeze [10]. These results were sustained at 12-month follow-up.

Conservative and nonsurgical treatment options are usually risk free and significant complications are usually not reported.

Surgical Anal Sphincter Repair

If conservative and nonsurgical treatment do not yield satisfactory improvement, surgical anal sphincter repair can be pursued if there is a localized sphincter injury. The repair usually involves repair of defects of the anal sphincters and the most common repair is performed in the anterior aspect (after obstetric injuries). The sphincteroplasty is usually performed with an overlapping technique. A curvilinear incision (200°–240° arc) is made parallel to the outer edge of the external sphincter and the anoderm is mobilized from the underlying sphincter and scar tissue. Dissection is continued cephalad to the sphincter injury. Two rows of interrupted absorbable sutures are used to overlap the ends of the severed sphincter and the anal aperture should permit a snugly fit index finger.

Fecal diversion after sphincter repair was previously common, with the assumption it would improve primary wound healing and functional outcome. Over time, the use of diversion has decreased, and today the repair is routinely performed without diversion. A randomized-controlled study of 27 patients by Hasegawa et al. [11] concluded fecal diversion in sphincter repair is not necessary. The authors assert diversion adds no benefit to wound healing or functional outcomes, but rather contributes to morbidity from stoma-related complications. Currently, fecal diversion is reserved for technically difficult sphincter repair operations, repeat sphincter repair, or if patients develops a postoperative infection.

Outcomes

Most reported literature on outcomes after sphincteroplasty for FI is based upon patient-reported satisfaction, grading scales such (i.e., modified-Parks Continence Score [12]), severity scales (i.e., CCFIS), and/or quality of life scores (i.e., FIQoL). Failure is usually defined as lack of improvement in FI symptoms, the need for reoperation for FI, and/or patient dissatisfaction. Overall, the short-term (<5 years) outcomes of sphincteroplasty have been considered favorable with excellent (no incontinence) or good (continence to flatus, some stain or urgency) in the range of 50–80%) [13–20]. Likewise, functional outcomes graded by the CCFIS have shown improvement in the short term [21, 22]. Unfortunately, long-term studies demonstrate that success rates deteriorate over time (>5 years) [13, 18–20, 23].

There are several studies reporting data after sphincteroplasty. In a retrospective study, Lamblin and coworkers [24] demonstrated in 23 patients that the CCFIS decreased from 12.7 preoperatively to 7.5 postoperatively. Overall, 17 patients reported being satisfied with their results, three (13%) expressed unsatisfactory or

dissatisfactory outcomes, and three (13%) patients demonstrated early recurrence of FI symptoms. Both Maslekar et al. [14] and Lehto et al. [23] conducted prospective, observational studies in >50 individuals and they found patients experiencing deterioration in efficacy with time at term follow-up. For example, Maslekar et al. evaluated a group of patients with a CCFIS of 16 preoperatively. Postoperatively, the CCFIS score improved to five at 12-months of follow-up and subsequently seven at 84 months of follow-up. The authors reported an overall 80% surgical success rate. On the other hand, Lehto et al. [23] reported FI improved in 67% of their patients at short-term follow-up but after a longer follow-up the severity of FI was about the same as preoperatively. Barisic et al. [13] prospectively studied 56 patients with a preoperative CCFIS of 17.8. They found a significant improvement in CCFIS scores, four at 3-months of follow-up and six at 80-months of follow-up. Using the modified Parks grading system, 48% of patients had successful results and 42% experienced recurrent or continuing FI symptoms at the latest follow-up. Finally, Oom et al. and Bravo Gutierrez et al. conducted retrospective reviews of >120 patients with 10-year follow-up. In both studies, approximately 40% of patients reported satisfactory fecal continence (excellent or good results) and about 60% had continued FI symptoms or needed additional surgery for their symptoms. Interestingly, about 60% of patients were satisfied with their results due to patient-perceived improvement in FI episodes compared to preoperative baseline.

Anal manometry has not routinely been included in assessments of the efficacy of anterior sphincteroplasty. In most studies including manometric evaluations, no significant change in resting pressures has been reported [17, 18, 25]. Some studies also fail to demonstrate a significant change in squeeze pressures or sphincter length [18, 25], while another study found a postoperative increase in squeeze pressures and anal sphincter length [17]. A separate study demonstrated that preoperative anal manometry and PNTML measurements are not predictive of postoperative success [26].

Complications

The incidence of postoperative complications after sphincteroplasty is difficult to ascertain, as this has rarely been the primary study outcome when reported. Based on available data, it appears that the overall immediate postoperative complication rate ranges between 5 and 30%. The most common complication is postoperative wound infection, reported between 1 and 20% in different studies. In clinical practice, however, superficial infection that leads to opening up of the wound is rather common and these infections typically heal without further intervention [24, 27, 28]. The patient should be informed about this risk, however, since this will extend the healing time. Despite a fairly high rate of superficial infections that leads to the wounds to open up, the incidence of breakdown of the sphincteroplasty is rather uncommon. Some authors use a perineal drain, because of the infection risk, but there is not a proven benefit in the literature [29].

Perioperative events, such as deep venous thrombosis, pulmonary embolism and ileus, are rare [30]. Other reported complications include postoperative hematoma, prolonged perineal pain, UTI, and fecal impaction. Adverse functional events such as temporary urinary retention [21, 26, 31] and impairment in fecal evacuation requiring long-term use of laxatives and suppositories have been occasionally reported [21]. Some women report problems with dyspareunia [32–35].

Sacral Nerve Stimulation

In 1981, Tanago and Schmidt [36] reported on symptom improvement in urinary urge incontinence and non-obstructive urinary retention using an implanted stimulator for stimulation of the sacral nerve roots. A simultaneous improvement was observed in bowel symptoms in some patients and studies were soon undertaken to assess this effect.

Sacral nerve stimulation (SNS) is usually performed in two stages. The first stage confirms that there is a proper neuromuscular response (contraction of the pelvic floor and plantar flexion of the big toe) with stimulation of the third sacral nerve [37]. Thereafter, a lead is placed and connected to an external stimulator. The patient thereafter undergoes a test-stimulation phase for 2 weeks. Greater than 50% reduction in incontinence episodes is considered a significant response and the patient then becomes eligible for permanent electrode placement.

Outcomes

SNS works by electrical stimulation of sacral nerve roots. Its exact mechanism of action is not clear. Studies indicate that FI may be improved by improved colorectal motility, improved rectoanal sensitivity, and spinal or supraspinal afferent inputs [38]. SNS therapy has demonstrated improved outcomes in a wide array of patient types. FI symptoms are improved in patient with or without sphincter injuries, patients with neuropathic fecal incontinence, cauda equina syndrome, and patients with low anterior resection syndrome [39–41].

According to a meta-analysis conducted by Tan et al. [42], SNS therapy has shown a statistically significant change in CCFIS and a significant increase in FIQoL scores subcategories. Success rates range between 55 and 80% with patients reporting improved decreased FI symptoms. About 30–40% report complete resolution of FI symptoms, while about 20% may have diminished efficacy of the treatment within 5 years [43–51].

Complications

SNS is associated with overall adverse event rate of 5–33%, but serious complications are rare [39, 48, 52–58]. Pain is the most common postoperative complication. Pain usually occurs at the site of implantation, but can also be experienced as extremity pain [46, 47]. To distinguish pain from sacral nerve stimulation versus pain from the device location under the skin, the device can be switched off. Resolution of symptoms indicates sacral nerve stimulation as the culprit of the patient's discomfort. This pain can usually be alleviated by reducing the pulse width, changing the electrode configuration, or reducing the stimulation amplitude. Rarely, has the device been explanted for pain, although resitting the device may be necessary [47].

Wound infection may occur in up to 8–10% of patients [46, 47, 58]. An infection of the device or the lead, usually necessitates explant of the lead and stimulator and treatment with antibiotics. Patients can then usually be reimplanted about 3 months later when the infection has healed. Another local colication is hematoma, but this is quite rare.

There are some technical problems that may occur with SNS. Dislodgement of the unipolar test lead can happen, but this is less common with the tined lead electrode [59]. The neurostimulator generator is battery-operated and has a lifespan of 3–6 years if used for chronic stimulation [48].

Detailed instructions for troubleshooting have been well described by Dudding and coworkers [60]. A measurement of impedence provides valuable insight into a malfunctioning neurostimulator. An impedence of >4000 Ω indicates a technical failure of the implant or lead fracture, where as <15 Ω suggests a short circuit. In either situation, it is best to pursue surgical exploration to replace faulty components as a sacral radiograph is often nondiagnostic. At times, patients may complain of decreased or absent sensation of stimulation or require high-amplitude stimulation for sensation. If impedence measurements are normal, these findings suggest suboptimal lead placement or migration and significant lead migration will require replacement.

Maeda and colleagues [45] reported on 101 patients undergoing SNS in an observational study. They reported a total of 521 reportable events in 94 patients. The most commonly reported events were loss of efficacy (193 events, 37%) and lack of efficacy (141 events, 27%). A total of 422 events required reprogramming, such as changing electrode pole combinations, and/or stimulation amplitudes and switching the device on/off. Eight patients were lost to follow-up, 20 patients had their device removed (eight for loss of efficacy, six for lack of efficacy in which two also had pain, two for pain and discomfort, two for infection, one for a required MRI scan for an unrelated condition, and one was removed per patient request after a new colostomy formation for an unrelated condition. Four patients had their device permanently switched off because of loss of efficacy (two), lack of efficacy (one) and spontaneous improvement in FI symptoms (one).

Magnetic Anal Sphincter

There have been several interventions to improve the function of the anal sphincter, including the artificial bowel sphincter and dynamic graciloplasty. The artificial bowel sphincter has been discontinued and dynamic graciloplasty is not approved in the United States and is rarely performed. These procedures will therefore not be discussed.

Magnetic anal sphincter augmentation has been recently approved by the United States Food and Drug Administration (FDA), through the humanitarian device exemption process, for patients with FI who failed other surgical interventions or are poor surgical candidates [61]. For any institution in the United States to be able to offer magnetic anal sphincter augmentation, approval is required by an institutional review board [62]. The FENIX™ Magnetic Sphincter Augmentation Continence Restoration System (MSA) is a dynamic, annular band of 14–20 interlinked, titanium beads with a magnetic core that is surgically placed around the anal sphincter complex. At rest, the band maintains the anus closed, simulating anal sphincter tone. To defecate, increased rectal pressure from normal Valsalva maneuver overcomes the attractive force of the beads. They then separate, opening the anal canal for fecal egress, and self-retract afterwards.

To implant the device, an anterior incision is made in the perineal body. Careful dissection along the rectovaginal septum is performed to 3–5 cm depth. A tunnel is circumferentially created around the anal canal to implant the device. The correct size is estimated and the device is implanted. The perineal incision is subsequently closed.

Outcomes

To date, there are four clinical studies on magnetic anal sphincter. Two are single center, prospective observational studies [63, 64], one is a prospective comparison study with SNS [65] and one is a multicenter feasibility study [61]. Study results demonstrate an encouraging short-term benefit of ≥50% improvement in incontinence in approximately 70% of patients and there were improvements in both CCFIS and FIQoL.

Complications

Between the four studies, a total of 67 patients were selected for implantation of the MSA device, and there were 26 device-related adverse events reported. Two patients experienced intraoperative rectal perforation and had no device placed and one device was accidently cut during a separate procedure for rectal prolapse repair [63, 64]. The most common complication was wound infection (seven events; 15%) [61, 63, 64]. Four of the infections were accompanied with wound dehiscence and

resolved with antibiotics. Two patients had the device explanted due to abscess or chronic infection. In one patient, although the infection resolved with antibiotics, the device was explanted per patient request for lack of efficacy. Perianal and gluteal swelling with erythema were reported in five patients in one study and resolved with conservative treatment [63]. Straining during defecation seems to risk cracking and self-expulsion of the MSA device and was observed in three patients [64, 65]. Three patients experienced fecal impaction which resolved with enemas and three patients experienced rectal bleeding which resolved spontaneously [61, 65]. Pain related to the device has been reported in two patients and resolved with medications [61].

Ventral Rectopexy for Fecal Incontinence

Both internal rectal intussusception and external rectal prolapse are associated with FI [66–68]. These conditions are, in addition, frequently associated with difficulties in rectal emptying. Internal and external prolapse may be treated with ventral rectopexy, which involves an anterior mobilization of the rectum after which a mesh is placed between the rectum and the posterior vaginal wall and secured to the sacrum.

Outcomes

In a systematic review of ventral rectopexy procedure for overt rectal prolapse, the authors report preoperative incontinence ranging between 23 and 93% in 191 patients [69]. Postoperatively, there was a statistically significant decrease in symptoms (FI ranging between 0 and 29%). This improvement corresponded to a significant decrease in CCFIS in two of the studies (the other four studies did not report pre-and postoperative CCFIS) [68, 70].

Complications

According to a systematic review by Gouvas et al. [69], the recurrent rate of rectal prolapse after ventral rectopexy is about 2–4% with an overall complication rate of 8.9%. In the studies reviewed, pelvic sepsis, pelvic hematoma, visceral erosion, mesh dislocation and infection occurred in up to 3% of patients. Port site infection or hematoma (1–6%) and port site hernias (0–7%) were observed. Other complications included urinary dysfunction and or infection, chronic abdominal pain, ileus, and cardiopulmonary complications.

The authors of a separate, retrospective review [71] of the safety of ventral rectopexy performed at five institutions reported their results on 2203 patients who underwent ventral rectopexy for rectal prolapse. Their most serious adverse event

related to ventral rectopexy was mesh-related. Two percent developed mesh erosion and a majority of these patients presented within 36 months of the operation. Treatment of mesh complications varied from minor (51% had local excision of stitch/exposed mesh) to major (40% had either laparoscopic removal, removal with colostomy, or anterior resection) procedures. Eleven percent of patients developed non-mesh complications (e.g., port site hernias, urinary retention, or urinary tract infection, postoperative pain) with postoperative pain as the most common complaint. Conservative treatment was appropriate for most non-mesh complications. Four patients required pudendal nerve blocks for perianal pain and four patients were treated for anal fissures. Six patents required a diagnostic laparoscopy for pain symptoms.

Vaginal Bowel-Control System for Fecal Incontinence

The vaginal bowel-control system (Eclipse System) was approved by the FDA in 2015 as a nonsurgical treatment option for fecal incontinence. The system consists of a silicone-coated device with a stainless steel base and a posteriorly directed balloon, both of which are available in different sizes. It is inserted into the vagina and the air-filled balloon compresses the rectum to improve continence [72]. The device can be placed in the clinic setting and five preset pressure settings are available for a comfortable fitting. The device can be deflated to enable/facilitate defecation. The device is cleaned daily with menstruation or otherwise once weekly.

Outcomes

To date, there is only one clinical study reporting results of for the vaginal bowel-control system [72]. All patients had ≥4 FI episodes over two weeks. Of the 100 patients fitted with the device, 56 patients entered treatment and completed follow-up after one month. 86% of patients experienced treatment success (≥50% reduction in FI episodes) at 1 month. At 3 months, 86% of 44 patients had >50% reduction in FI episodes and 72.7% had ≥75% reduction in FI episodes according to the last 2 weeks of their bowel diaries.

Complications

In the published study [72], there were no major adverse events associated with the vaginal bowel-control system. Minor complications amongst the 110 patients fitted for the device included pelvic cramping (15%), urinary incontinence/urgency (10%), vaginal symptoms (9%), pelvic pain (8%) and spotting (7%). At one month, the two most common symptoms were pelvic cramping (10%) and vaginal findings

(5%). At 3 months, the only complaints were pelvic cramping (9%) and pelvic pain (3%).

Rectal Sling for the Treatment of Fecal Incontinence

The TOPAS pelvic floor repair system (American Medical Systems, INC [AMS], Minnetonka, MI) was first described in 2014 by Rosenblatt et al. [73]. The device includes a monofilament mesh that is placed about the anal canal using a tension-free, transobturator approach. Using a curved insertion needle, the mesh is tunneled between two small buttock incisions posterior to the rectum. The sling is then passed lateral to the rectum and vagina, around the ischiopubic ramus, and out through the obturator foramen on each side. The mesh and sheath arms are then gently pulled upward for tension adjustment of the mesh [73, 74]. Currently, the TOPAS system is not commercially available.

Outcomes

Only two studies exist on rectal sling treatment for FI. In a study of 29 patients with FI symptoms for >6 months and who failed at least once conservative treatment, Rosenblatt et al. reported a significant decrease in mean FI episodes (6.9 FI episodes in a 14-day period to 3.5 FI episodes) [73]. Likewise, Wexner scores significantly improved from a mean of 13.2–9.9 at 24 months. Overall, patients had a 56% success rate, defined as ≥50 reduction in FI episodes. FIQoL scores also significantly improved. There was no difference in PNTML nor anal manometry measurements between baseline and follow-up. This was also true in the 11 patients with preoperative imaging (endoanal ultrasound or MRI) confirming an anal sphincter defect.

In a separate multicenter, prospective, observational study on the TOPAS system, stricter inclusion criteria were followed [74]. Namely, enrolled patients failed at least two conservative therapy modalities and had ≥4 incontinence episodes within a 14 day period. Sixty-nine percent of patients achieved at least a 50% reduction in FI episodes, and FIQoL measurements significantly improved from baseline to 12 months. Overall, mean Wexner scores significantly decreased from 13.9 at baseline to 9.6 at 12 months.

Complications

In the Rosenblatt study [73], there were 68 adverse events in 22 patients. The most common were new-onset urinary incontinence/urgency (9%), followed by worsening FI (3%) and constipation (3%). Five patients experienced serious adverse events; four were unrelated to the procedure, and one (disk herniation) was related

to patient-positioning during the procedure. There were no device-related erosions or extrusions.

In the second FDA controlled study by Mellgren et al. [74], there were 104 adverse events in 66 patients. The most common adverse events included pain, in either the buttock, pelvic or groin areas and infection. There were six serious adverse events; de novo or worsening pelvic organ prolapse with need for surgical repair (six patients), chronic obstructive pulmonary disease exacerbation (one patient), post-traumatic stress disorder exacerbation (one patient), deep vein thrombosis (one patient), and methicillin-resistent *Staphylococcus aureus* infection outside of the surgical site (one patient).

Injection Therapy

The use of bulking agents as an injectable material to augment the tissue volume of the anal sphincter complex was described in 1993 [75]. Since then, there have been several different injectable biomaterials studied for the treatment of FI. The paucity of long-term efficacy and safety data has limited their clinical application [76].

In 2011, FDA approved the first bulking agent for fecal incontinence, nonanimal stabilized hyaluronic acid with dextranomer (NASHA™ Dx; Solesta®, Salix Pharmaceuticals, Inc. Raleigh, NC, USA). Hyaluronan is a biodegradable substance and it is believed that the dextranomer skeleton provides a framework for collagen growth. The added bulk to the sphincter complex may improve anal canal sealing and improve continence. Normally, one ml of sterile, viscous gel is injected in each quadrant within the deep submucosa of the anal canal, just above the dentate line (total 4 mm).

Outcomes

There are several studies on the effectiveness and safety of NASHA™ Dx for FI treatment: one single center, prospective observational study over 24 months [77, 78]; one multicenter, observational study over 24 months [79, 80], and one international, multicenter, randomized, double-blind sham-controlled trial (RCT) over 6 months, and one 36 month follow-up study of treated patients in the multicenter study [81–83]. Most studies had similar inclusion and exclusion criteria, except there were stricter enrollment criteria for the RCT. Primary success was defined as ≥50% reduction of FI episodes.

The number of FI episodes decreased significantly in all studies. In the single-site observational study, in which 33 out of 34 enrolled patients had 24 month follow-up, the median Miller fecal incontinence severity score decreased from a baseline of 14–10.5 [84]. Twenty patients (59%) achieved successful results, however there was no significant change in SF-36 scores at 24 months.

Dodi et al. [79] and La Torre and De La Portilla [80] reported data on 115 enrolled patients in a multicenter, observational study. Eighty-four patients completed the 24-month follow-up and 63% of these patients achieved success at the 24-month follow-up. In this group, there was an improvement in CCFIS (13.5–8.9) and FIQoL scores showed significant improvement in all four subcategories.

In the RCT comparing NASH™ DX to sham injections, the patients in the NASHA™ Dx treatment arm had a higher success rate that the sham arm at 6 months. Improvement in FI episodes was demonstrated in treated patients at 36-month follow-up.

Complications

Few treatment-related, serious adverse events occurred amongst the combined 283 patients from the three studies. The most common serious adverse event included infections (two rectal abscesses, one perineal abscess, one rectovaginal septum abscess, and one prostatic abscess [79, 81]. Other significant adverse events included one case of concurrent rectal prolapse, proctalgia, and rectal hemorrhage [79] and three cases of proctitis (associated with urgency, diarrhea, and tenderness) [77]. The most common mild to moderate side effects after NASHA™ Dx therapy was proctalgia (13%).

Gatekeeper™ Sphincter Augmentation

The Gatekeeper™ (THD SpA, Correggio, Italy) is a novel inert prosthesis composed of HYEXPAN™ (polyacrylonitrile), a hydrophilic material that is inserted under local anesthesia in the intersphincteric space. Upon contact with human tissue, the thin, solid cylinder (length 21 mm, diameter 1.2 mm) absorbs water and adapts into a shorter (length 17 mm), thicker (diameter 7 mm) and softer prosthesis. The insertion is made with help of ultrasound and a special introducer [85, 86].

Initially 4–6 implants were utilized. In 2016, a new prosthesis was introduced; SphinKeeper™ (THD SpA, Correggio, Italy). The SphinKeeper™ is larger and ten insertions, instead of six, are applied with the SphinKeeper™ [87].

Outcomes

There are two reported clinical studies; one single center observational study [85] with up to 3 year of follow-up, and one multicenter, observational study [86] with up to 1 year of follow-up. Improvements in number of FI episodes and CCFIS were seen in both studies. There was a trend towards slightly increased mean functional anal canal length and rectal sensation but this was not statistically significant [85].

Complications

In the multicenter, observational study on the Gatekeeper™ prostheses, three patients had migration of a prosthesis. This did not affect the efficacy. Another single case study of the Gatekeeper™, however, reported prosthesis migration one year after implantation, and this was associated with the return of FI symptoms.

The only other complication reported is perianal pain, which was observed in 13% of patients in the multicenter study. The pain resolved with temporary nonsteroidal anti-inflammatory drug therapy [86]. One patient in the SphinKeeper™ study developed perianal pain associated with prosthesis migration. This likewise resolved with local lidocaine application and systemic acetaminophen.

Summary

The treatment of FI has made significant progress over the past decade. Several new treatment options have been introduced and other options are under evaluation. We have reviewed the most common options that are approved today, and we have also included a few options that are yet not used in normal clinical practice (yet). We expect more options will be introduced in the future, and we are looking forward to learning more about the role of stem cell therapy in the treatment of FI.

Treatment of FI usually starts with nonsurgical options including diet modifications, medication (loperamide) and other measures to normalize stool consistency. Most patients benefit from stools that are firm. Pelvic floor exercises with biofeedback are frequently effective in patients with FI, especially if the patients experience urgency. Biofeedback is less effective in patients with passive FI.

Previously, patients with a localized sphincter defect were automatically considered candidates for surgical sphincter repair (sphincteroplasty). However, long-term follow-up has demonstrated disappointing results in some patients, and, with the introduction of SNS, there is a debate which treatment should be preferred. Young patients with recent sphincter injury are frequently recommended to undergo sphincteroplasty and likewise patients with symptoms from anatomic deficiencies. SNS has otherwise become increasingly more popular for patients that fail nonsurgical alternatives, because of the technique's efficacy and benign side effect profile.

Injection therapy has become an alternative for patients with milder symptoms, including patients with soiling. The therapy has a benign side effect profile and the therapy is easily administered without anesthesia in the outpatient clinic.

The role of the vaginal bowel-control system is yet to be defined. Long-term assessments are needed. Patients who fail the above-mentioned treatment options will be candidates for the Magnetic Anal Sphincter augmentation device. If this device proves to be safe and effective in a larger patient population over a longer time, this therapy may have an increased role in the future.

References

1. Madoff RD, Williams JG, Caushaj PF. Fecal incontinence. N Engl J Med. 1992;326 (15):1002–7.
2. Bharucha AE, Dunivan G, Goode PS, et al. Epidemiology, pathophysiology, and classification of fecal incontinence: state of the science summary for the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) workshop. Am J Gastroenterol. 2015;110(1):127–36.
3. Ditah I, Devaki P, Luma HN, et al. *Prevalence, trends, and risk factors for fecal incontinence in United States adults, 2005–2010.* Clin Gastroenterol Hepatol, 2014;12(4):636–43 e1–2.
4. Pfeifer J. Quality of life after sphincteroplasty. Acta Chir Iugosl. 2004;51(2):73–5.
5. Jorge JM, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum. 1993;36(1):77–97.
6. Rockwood TH, Church JM, Fleshman JW, et al. Fecal incontinence quality of life scale: quality of life instrument for patients with fecal incontinence. Dis Colon Rectum, 2000;43 (1):9–16; discussion 16–7.
7. Gordon PH, Nivatvongs S. Principles and practice of surgery for the colon, rectum, and anus. 3rd ed. New York: Informa Healthcare; 2007, xxii, 1330 p.
8. Whitehead WE, Rao SS, Lowry A, et al. Treatment of fecal incontinence: state of the science summary for the National Institute of Diabetes and Digestive and Kidney Diseases workshop. Am J Gastroenterol, 2015;110(1):138–46; quiz 147.
9. Heymen S, Scarlett Y, Jones K, et al. Randomized controlled trial shows biofeedback to be superior to pelvic floor exercises for fecal incontinence. Dis Colon Rectum. 2009;52 (10):1730–7.
10. Rockwood TH, Church JM, Fleshman JW, et al. Patient and surgeon ranking of the severity of symptoms associated with fecal incontinence–the fecal incontinence severity index. Dis Colon Rectum. 1999;42(12):1525–31.
11. Hasegawa H, Yoshioka K, Keighley MR. Randomized trial of fecal diversion for sphincter repair. Dis Colon Rectum. 2000;43(7):961–4; discussion 964–5.
12. Browning GG, Parks AG. Postanal repair for neuropathic faecal incontinence: correlation of clinical result and anal canal pressures. Br J Surg. 1983;70(2):101–4.
13. Barisic GI, Krivokapic ZV, Markovic VA, et al. Outcome of overlapping anal sphincter repair after 3 months and after a mean of 80 months. Int J Colorectal Dis. 2006;21(1):52–6.
14. Maslekar S, Gardiner AB, Duthie GS. Anterior anal sphincter repair for fecal incontinence: good longterm results are possible. J Am Coll Surg. 2007;204(1):40–6.
15. McManus BP, Allison S, Hernanchez-Sanchez J. Anterior sphincteroplasty for fecal incontinence: predicting incontinence relapse. Int J Colorectal Dis. 2015;30(4):513–20.
16. Fleshman JW, Dreznik Z, Fry RD, et al. Anal sphincter repair for obstetric injury: manometric evaluation of functional results. Dis Colon Rectum. 1991;34(12):1061–7.
17. Oliveira L, Pfeifer J, Wexner SD. Physiological and clinical outcome of anterior sphincteroplasty. Br J Surg. 1996;83(4):502–5.
18. Malouf AJ, Norton CS, Engel AF, et al. Long-term results of overlapping anterior anal-sphincter repair for obstetric trauma. Lancet. 2000;355(9200):260–5.
19. Halverson AL, Hull TL. Long-term outcome of overlapping anal sphincter repair. Dis Colon Rectum. 2002;45(3):345–8.
20. Bravo Gutierrez A, Madoff RD, Lowry AC, et al. Long-term results of anterior sphincteroplasty. Dis Colon Rectum. 2004;47(5):727–31; discussion 731–2.
21. Tjandra JJ, Han WR, Goh J, et al. Direct repair versus overlapping sphincter repair: a randomized, controlled trial. Dis Colon Rectum. 2003;46(7):937–42; discussion 942–3.
22. Barisic G, Krivokapic Z, Markovic V, et al. The role of overlapping sphincteroplasty in traumatic fecal incontinence. Acta Chir Iugosl. 2000;47(4 Suppl 1):37–41.
23. Lehto K, Hyoty M, Collin P, et al. Seven-year follow-up after anterior sphincter reconstruction for faecal incontinence. Int J Colorectal Dis. 2013;28(5):653–8.

24. Lamblin G, Bouvier P, Damon H, et al. Long-term outcome after overlapping anterior anal sphincter repair for fecal incontinence. Int J Colorectal Dis. 2014;29(11):1377–83.
25. Elton C, Stoodley BJ. Anterior anal sphincter repair: results in a district general hospital. Ann R Coll Surg Engl. 2002;84(5):321–4.
26. Buie WD, Lowry AC, Rothenberger DA, et al. Clinical rather than laboratory assessment predicts continence after anterior sphincteroplasty. Dis Colon Rectum. 2001;44(9):1255–60.
27. Arnaud A, Sarles JC, Sielezneff I, et al. Sphincter repair without overlapping for fecal incontinence. Dis Colon Rectum. 1991;34(9):744–7.
28. Morren GL, Hallbook O, Nystrom PO, et al. Audit of anal-sphincter repair. Colorectal Dis. 2001;3(1):17–22.
29. Fleshman JW, Peters WR, Shemesh EI, et al. Anal sphincter reconstruction: anterior overlapping muscle repair. Dis Colon Rectum. 1991;34(9):739–43.
30. Oom DM, Gosselink MP, Schouten WR. Anterior sphincteroplasty for fecal incontinence: a single center experience in the era of sacral neuromodulation. Dis Colon Rectum. 2009;52 (10):1681–7.
31. Fang DT, Nivatvongs S, Vermeulen FD, et al. Overlapping sphincteroplasty for acquired anal incontinence. Dis Colon Rectum. 1984;27(11):720–2.
32. Cichowski SB, Komesu YM, Dunivan GC, et al. The association between fecal incontinence and sexual activity and function in women attending a tertiary referral center. Int Urogynecol J. 2013;24(9):1489–94.
33. Palm A, Israelsson L, Bolin M, et al. Symptoms after obstetric sphincter injuries have little effect on quality of life. Acta Obstet Gynecol Scand. 2013;92(1):109–15.
34. Salim R, Peretz H, Molnar R, et al. Long-term outcome of obstetric anal sphincter injury repaired by experienced obstetricians. Int J Gynaecol Obstet. 2014;126(2):130–5.
35. Marsh F, Lynne R, Christine L, et al. Obstetric anal sphincter injury in the UK and its effect on bowel, bladder and sexual function. Eur J Obstet Gynecol Reprod Biol. 2011;154(2):223–7.
36. Tanagho EA, Schmidt RA. Bladder pacemaker: scientific basis and clinical future. Urology. 1982;20(6):614–9.
37. Gordon PH, Nivatvongs S. Principles and practice of surgery for the colon, rectum, and anus. 2nd ed. St. Louis, Mo.: Quality Medical Pub. 1999, xxxi, 1455 p.
38. Gourcerol G, Vitton V, Leroi AM, et al. How sacral nerve stimulation works in patients with faecal incontinence. Colorectal Dis. 2011;13(8):e203–11.
39. Gstaltner K, Rosen H, Hufgard J, et al. Sacral nerve stimulation as an option for the treatment of faecal incontinence in patients suffering from cauda equina syndrome. Spinal Cord. 2008;46(9):644–7.
40. Jarrett ME, Matzel KE, Christiansen J, et al. Sacral nerve stimulation for faecal incontinence in patients with previous partial spinal injury including disc prolapse. Br J Surg. 2005;92 (6):734–9.
41. Ramage L, Qiu S, Kontovounisios C, et al. A systematic review of sacral nerve stimulation for low anterior resection syndrome. Colorectal Dis. 2015;17(9):762–71.
42. Tan E, Ngo NT, Darzi A, et al. Meta-analysis: sacral nerve stimulation versus conservative therapy in the treatment of faecal incontinence. Int J Colorectal Dis. 2011;26(3):275–94.
43. Altomare DF, Ratto C, Ganio E, et al. Long-term outcome of sacral nerve stimulation for fecal incontinence. Dis Colon Rectum. 2009;52(1):11–7.
44. Altomare DF, Giuratrabocchetta S, Knowles CH, et al. Long-term outcomes of sacral nerve stimulation for faecal incontinence. Br J Surg. 2015;102(4):407–15.
45. Maeda Y, Lundby L, Buntzen S, et al. Outcome of sacral nerve stimulation for fecal incontinence at 5 years. Ann Surg. 2014;259(6):1126–31.
46. Hull T, Giese C, Wexner SD, et al. Long-term durability of sacral nerve stimulation therapy for chronic fecal incontinence. Dis Colon Rectum. 2013;56(2):234–45.
47. Uludag O, Melenhorst J, Koch SM, et al. Sacral neuromodulation: long-term outcome and quality of life in patients with faecal incontinence. Colorectal Dis. 2011;13(10):1162–6.

48. Tjandra JJ, Lim JF, Matzel K. Sacral nerve stimulation: an emerging treatment for faecal incontinence. ANZ J Surg. 2004;74(12):1098–106.
49. Altomare DF, Binda GA, Dodi G, et al. Disappointing long-term results of the artificial anal sphincter for faecal incontinence. Br J Surg. 2004;91(10):1352–3.
50. Melenhorst J, Koch SM, Uludag O, et al. Sacral neuromodulation in patients with faecal incontinence: results of the first 100 permanent implantations. Colorectal Dis. 2007;9(8):725–30.
51. Hollingshead JR, Dudding TC, Vaizey CJ. Sacral nerve stimulation for faecal incontinence: results from a single centre over a 10-year period. Colorectal Dis. 2011;13(9):1030–4.
52. Kenefick NJ, Vaizey CJ, Cohen RC, et al. Medium-term results of permanent sacral nerve stimulation for faecal incontinence. Br J Surg. 2002;89(7):896–901.
53. Jarrett ME, Varma JS, Duthie GS, et al. Sacral nerve stimulation for faecal incontinence in the UK. Br J Surg. 2004;91(6):755–61.
54. Holzer B, Rosen HR, Novi G, et al. Sacral nerve stimulation for neurogenic faecal incontinence. Br J Surg. 2007;94(6):749–53.
55. O'Riordan JM, Healy CF, McLoughlin D, et al. Sacral nerve stimulation for faecal incontinence. Ir J Med Sci. 2008;177(2):117–9.
56. Munoz-Duyos A, Navarro-Luna A, Brosa M, et al. Clinical and cost effectiveness of sacral nerve stimulation for faecal incontinence. Br J Surg. 2008;95(8):1037–43.
57. Altomare DF, Binda G, Ganio E, et al. Long-term outcome of Altemeier's procedure for rectal prolapse. Dis Colon Rectum. 2009;52(4):698–703.
58. 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(9):1065–75.
59. Altomare DF, Giannini I, Giuratrabocchetta S, et al. The effects of sacral nerve stimulation on continence are temporarily maintained after turning the stimulator off. Colorectal Dis. 2013;15(12):e741–8.
60. Dudding TC, Hollingshead JR, Nicholls RJ, et al. Sacral nerve stimulation for faecal incontinence: optimizing outcome and managing complications. Colorectal Dis. 2011;13(8):e196–202.
61. Lehur PA, McNevin S, Buntzen S, et al. 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.
62. Eydelman MB, Chen EA. The FDA's humanitarian device exemption program. Health Aff (Millwood). 2011;30(6):1210–2; author reply 1212.
63. Pakravan F, Helmes C. Magnetic anal sphincter augmentation in patients with severe fecal incontinence. Dis Colon Rectum. 2015;58(1):109–14.
64. Barussaud ML, Mantoo S, Wyart V, et al. The magnetic anal sphincter in faecal incontinence: is initial success sustained over time? Colorectal Dis. 2013;15(12):1499–503.
65. Wong MT, Meurette G, Wyart V, et al. Does the magnetic anal sphincter device compare favourably with sacral nerve stimulation in the management of faecal incontinence? Colorectal Dis. 2012;14(6):e323–9.
66. Collinson R, Wijffels N, Cunningham C, et al. Laparoscopic ventral rectopexy for internal rectal prolapse: short-term functional results. Colorectal Dis. 2010;12(2):97–104.
67. Formijne Jonkers HA, Poierrie N, Draaisma WA, et al. Laparoscopic ventral rectopexy for rectal prolapse and symptomatic rectocele: an analysis of 245 consecutive patients. Colorectal Dis. 2013;15(6):695–9.
68. Boons P, Collinson R, Cunningham C, et al. Laparoscopic ventral rectopexy for external rectal prolapse improves constipation and avoids de novo constipation. Colorectal Dis. 2010;12(6):526–32.
69. Gouvas N, Georgiou PA, Agalianos C, et al. Ventral colporectopexy for overt rectal prolapse and obstructed defaecation syndrome: a systematic review. Colorectal Dis. 2015;17(2):O34–46.

70. Lauretta A, Bellomo RE, Galanti F, et al. Laparoscopic low ventral rectocolpopexy (LLVR) for rectal and rectogenital prolapse: surgical technique and functional results. Tech Coloproctol. 2012;16(6):477–83.
71. Evans C, Stevenson AR, Sileri P, et al. A multicenter collaboration to assess the safety of laparoscopic ventral rectopexy. Dis Colon Rectum. 2015;58(8):799–807.
72. Richter HE, Matthews CA, Muir T, et al. A vaginal bowel-control system for the treatment of fecal incontinence. Obstet Gynecol. 2015;125(3):540–7.
73. Rosenblatt P, Schumacher J, Lucente V, et al. A preliminary evaluation of the TOPAS system for the treatment of fecal incontinence in women. Female Pelvic Med Reconstr Surg. 2014;20 (3):155–62.
74. Mellgren A, Zutshi M, Lucente VR, et al. A posterior anal sling for fecal incontinence: results of a 152-patient prospective multicenter study. Am J Obstet Gynecol. 2016;214(3):349 e1–8.
75. Shafik A. Polytetrafluoroethylene injection for the treatment of partial fecal incontinence. Int Surg. 1993;78(2):159–61.
76. Luo C, Samaranayake CB, Plank LD, et al. Systematic review on the efficacy and safety of injectable bulking agents for passive faecal incontinence. Colorectal Dis. 2010;12(4):296–303.
77. Danielson J, Karlbom U, Sonesson AC, et al. Submucosal injection of stabilized nonanimal hyaluronic acid with dextranomer: a new treatment option for fecal incontinence. Dis Colon Rectum. 2009;52(6):1101–6.
78. Danielson J, Karlbom U, Wester T, et al. Efficacy and quality of life 2 years after treatment for faecal incontinence with injectable bulking agents. Tech Coloproctol. 2013;17(4):389–95.
79. Dodi G, Jongen J, de la Portilla F, et al. An open-label, noncomparative, multicenter study to evaluate efficacy and safety of NASHA/Dx gel as a bulking agent for the treatment of fecal incontinence. Gastroenterol Res Pract. 2010;2010:467136.
80. La Torre F, de la Portilla F. Long-term efficacy of dextranomer in stabilized hyaluronic acid (NASHA/Dx) for treatment of faecal incontinence. Colorectal Dis. 2013;15(5):569–74.
81. 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.
82. Mellgren A, Matzel KE, Pollack J, et al. Long-term efficacy of NASHA Dx injection therapy for treatment of fecal incontinence. Neurogastroenterol Motil. 2014;26(8):1087–94.
83. Franklin H, Barrett AC, Wolf R. Identifying factors associated with clinical success in patients treated with NASHA((R))/Dx injection for fecal incontinence. Clin Exp Gastroenterol. 2016;9:41–7.
84. Miller R, Bartolo DC, Locke-Edmunds JC, et al. Prospective study of conservative and operative treatment for faecal incontinence. Br J Surg. 1988;75(2):101–5.
85. Ratto C, Parello A, Donisi L, et al. Novel bulking agent for faecal incontinence. Br J Surg. 2011;98(11):1644–52.
86. Ratto C, Buntzen S, Aigner F, et al. Multicentre observational study of the Gatekeeper for faecal incontinence. Br J Surg. 2016;103(3):290–9.
87. Ratto C, Donisi L, Litta F, et al. Implantation of sphinkeeper(TM): a new artificial anal sphincter. Tech Coloproctol. 2016;20(1):59–66.

Transanal Excision of Rectal Tumor (TEM or TAMIS)

12

Kunal Kochar and Vivek Chaudhry

Transanal surgery encompasses a wide spectrum of surgical techniques ranging from conventional Transanal Excision (TAE), Transanal Endoscopic Microsurgery (TEM), Transanal Minimally Invasive Surgery (TAMIS) to a more recent development of Transanal Total Mesorectal Excision (TATME). TAE was first described by Lisfranc in 1826, and then popularized by Parks [1] in 1960s. Though conventional TAE remains a viable option for benign rectal lesions within 10 cm from anal verge, the use of this technique has been questioned for malignant lesions of the rectum. Transanal excision is widely considered low risk, but complications of bleeding, urinary retention, perforation/fragmentation/recurrence of tumors, anal stenosis, sepsis, and fistulas have been reported.

Bleeding

Bleeding following TAE can happen in the immediate post-operative period or later. It has been reported in 10–15% of patients following TAE. Nivatvongs et al. [2] reported an incidence of 3% in their series of 72 patients. In a large case series of 100 patients treated with TAE at St Marks hospital, 3 patients (3%) developed hemorrhage [3]. Similarly, of the 117 patients who underwent TAE for rectal villous adenoma at Ferguson clinic, 8.5% developed hemorrhage following the procedure. Early hemorrhages were treated with return to the operating room and control of bleeding with either suture ligation or cautery. Late hemorrhages did not require any operative intervention [4]. With the improved optics of TEM and better

K. Kochar · V. Chaudhry (✉)
Division of Colon and Rectal Surgery, John H Stroger Hospital of Cook County, 1600 W. Polk Street, Chicago, IL 60612, USA
e-mail: Vchaudhr@gmail.com

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_12

instrumentation, it is not surprising that the incidence of hemorrhage is lower as compared to TAE. In a prospective comparison of TAE and TEM, Winde et al. noted a higher rate of post-operative bleeding in the TAE group, 6% versus 2% [5]. In 89 patients who underwent TAE, Moore et al. reported hemorrhage in 1 patient in the TAE group, whereas no significant bleeding was reported in 82 patients undergoing TEM [6]. In contrast, Langer et al. had a higher rate of blood transfusion in the TEM group as compared to TAE group, 8.9% versus 5.3% respectively, though it did not reach statistical significance [7]. In 260 patients undergoing TEM over a 10 year period, Said et al. reported hemorrhage in 4 (1.4%) patients [8]. Of the 590 patients enrolled in a multi-center TEM Italian study, 8 (1.3%) patients had rectal hemorrhage that required blood transfusion and there were 3 (0.5%) patients with post-operative rectal bleeding that required a repeat TEM procedure for suture control of the bleeding. One patient had intra-operative bleeding that required conversion to open surgery for control of hemorrhage [9]. Transanal Minimally Invasive Surgery (TAMIS) was introduced as an alternative to TEM in 2009 [10]. The main purported advantages of TAMIS over TEM includes lower cost, shorter learning curve, better visualization affording a 360-degree visibility as compared to 220 degrees with TEM, wider operative angle and range of motion of standard laparoscopic instruments [11]. In their initial experience in 50 patients who underwent TAMIS for local excision of benign neoplasms and early rectal cancer, Albert et al. reported delayed hemorrhage in only one patient [11]. Transanal Total Mesorectal excision (TATME) is a new evolving technique for low rectal cancers and utilizes a "bottoms up" approach. Surgeons are still in their learning curves and bleeding can occur if the wrong planes are entered. The pre-sacral veins as well as the lateral pelvic walls are potential places of injury and bleeding from these areas can be difficult to control via the transanal route. In the largest published series of 140 TATME procedures, intra-abdominal bleeding was reported in 2 patients (1.4%) and anastomotic bleeding in 3 patients (2.4%) [46]. In 50 cases of TATME reported by Burke et al. [47], there were no cases of bleeding related to the transanal part of the procedure.

Incomplete Excision, Fragmentation, and Local Recurrence

Though there is controversy regarding local excision of early rectal cancer as it does not address the regional mesorectal lymph nodes, local excision of early rectal cancer has significant advantages with decreased morbidity and avoidance of a stoma [12]. Transanal excision is limited to tumors of less than 4 cm in diameter, within 6–8 cm of the anal verge [13, 14]. It has the advantage of no significant learning curve or associated equipment cost [6, 13]. However, multiple studies have shown that TAE is associated with higher probability of positive margins as well as

fragmentation of specimen. Christoforidis et al. [15] compared TAE versus TEM in 129 patients with pT1 and pT2 rectal cancers. 16% of TAE specimens had a positive margin versus 2% in TEM, and the rate of specimen fragmentation was also higher, 6% versus 0%. Moore et al. also compared TEM versus TAE in 171 patients [6]. TEM was more likely to yield clear margins (90% versus 71%) and a non fragmented specimen (94% versus 65%) compared with TAE. With a mean follow up of 39 months, recurrence was less frequent with TEM, 5% versus 27%. In a national cancer database study, TAE was compared with radical resection, the 5-year local recurrence after local excision was significantly higher than standard excision, 12.5% versus 6.9%, $P = 0.003$ for T1 tumors, and 22.1% versus 15.1% for T2 tumors [16]. Using TAE for rectal cancer, Garcia-Aguilar et al. reported a recurrence rate of 18% for T1 tumors and 37% for T2 tumor at 54 months of follow up [17]. With a median follow up of 55 months, Madbouly et al. had a recurrence rate of 28.8% after TAE of T1 rectal cancers [18]. Mellgren et al. reported an estimated 5-year local recurrence rate of 18% for T1 tumors and 47% for T2 tumors after TAE as compared to 0% for T1 tumors and 6% for T2 tumors after radical resection [19]. Similarly, Chorost et al. reported 31% local recurrence rate after local excision of T1 tumors [20]. In a meta-analysis of TEM versus TAE, TEM had a statistically significant lower rate of negative microscopic margins, reduced rate of specimen fragmentation and recurrence compared with transanal excision [21]. In a retrospective study comparing TEM versus anterior resection versus TAE for rectal lesions (adenoma and carcinoma), there was a higher incidence of incomplete resection (R1 resection) in TAE group (37%) as compared to TEM group (19%). The overall recurrence rate was also higher in the TAE group as compared to TEM and anterior resection (26.3% versus 8.9% versus 3.7%) [5]. The advantages gained due to decreased morbidity and avoidance of a stoma with a TAE is offset by to the high rate of incomplete resection and local recurrence rate. TEM/TAMIS on the other the hand may offer a platform for transanal excision of early rectal cancers with acceptable results. Heintz et al. did not find any significant difference in 5 year survival rate between TEM and radical surgical therapy in patients with low risk T1 carcinoma [22]. A recent meta-analysis of local treatment for T1N0M0 rectal cancer showed that TEM subgroup did not have significantly lower overall survival than radical resection, whereas TAE was associated with significantly lower 5-year overall survival. Additionally, TEM was associated with lower post-operative complications and need for permanent stoma as compared to radical resection [23]. Transanal minimally invasive surgery (TAMIS) was introduced in 2009 and has emerged as a viable alternative to both TEM and TAE [24]. At present there are no studies comparing TAMIS versus standard transanal excision, TEM or radical anterior resection. Systematic review of TAMIS procedures ($n = 390$ reported a positive margin rate of 4.36% and tumor fragmentation rate of 4.1% [25]. Trials comparing TAMIS versus standard resection are awaited.

Urinary Retention

Urinary retention is one of the most common complication after anorectal surgery. The incidence varies between 1 and 50% [26–28]. The exact etiology of urinary retention following anorectal procedures is not completely understood. Dysfunction of detrusor muscle or the trigone in response to pain or distention of the anal canal or perineum has been postulated as a cause of urinary retention [29]. Zaheer et al. reported that urinary retention developed in 16% of patients following surgery for benign anorectal conditions [27]. The incidence varied from 34% following hemorrhoidectomy to 4% after lateral internal sphincterotomy and 2% after fistulotomy [29]. The reported rate of urinary retention following TEM, TAMIS, and TATME varies from 5 to 10% [30–32], 0 to 6% [11, 33–35] and 2 to 4%, respectively [46, 47].

Pelvic Sepsis

Pelvic sepsis following anorectal surgery is fortunately rare, with majority of cases being reported following treatment for hemorrhoids. There are a few case reports of pelvic sepsis following injection sclerotherapy [36–38]. Severe sepsis is more common after rubber band ligation as compared to injection sclerotherapy. A systematic review of life threatening sepsis following treatment for hemorrhoids reported 38 patients with severe sepsis. 17 patients had undergone rubber band ligation of which 6 patients died, 3 patients had undergone excisional hemorrhoidectomy of which 1 patient died and finally, 7 patients with stapled hemorrhoidopexy of which 1 patient died [39]. Kam et al. reported their experience with 7302 stapled hemorrhoidectomy operations in a single center in 2010. They reported 4 cases of perianal sepsis with no deaths [40]. Butterworth et al. treated 118 patients over a period of 4 years with stapled hemorrhoidopexy and reported 1 patient with severe sepsis who eventually died [41]. There are no reliable reports of pelvic sepsis following various methods of TAE.

Anal Stricture/Stenosis

Anal strictures and stenosis are most commonly seen after hemorrhoidectomy, with an incidence ranging from 1.5 to 3.8% [42]. Multiple systematic review of trials comparing conventional hemorrhoidectomy to stapled hemorrhoidectomy have not shown any statistical difference in the incidence of early and late anal stricture between the two methods [43–45]. None of the larger series of TAE reported anorectal stenosis/stricture. This could be related to the excision and closure of defects in the rectum rather than the anal canal.

Urethral Injury

Although uncommon urethral injury has been reported following TATME. Rouanet et al. [48] reported 2 urethral injuries in 25 procedures, whereas Burke et al. [47] had one urethral injury in 50 patients. In the largest series of TATME, there were no reported urethral injuries [46].

Miscellaneous Complications

Scrotal/subcutaneous emphysema, exacerbation of COPD, intraperitoneal entry and loss of pneumorectum are procedure specific complication of TAMIS. [11].

Strategies for Prevention of Complications in Transanal Surgery

Bleeding

- Keeping in the correct planes of dissection will prevent majority of the bleeding
- Submucosal injection of 1:100,000 epinephrine
- Liberal and early use of stay sutures for traction and retraction
- Use of an energy device like Harmonic Ace + Shears® (Ethicon) or LigaSure™ (Covidien-Medtronic)
- Early control of bleeding to prevent vessel retraction and decreased visualization with hematoma in a limited field of view.

Fragmentation of Lesion

- The margins of the lesion (1–2 cm) should be marked with electrocautery prior to start of dissection
- The lesion should not be grasped directly, but instead the submucosa or the muscular wall should be grasped gently for retraction
- Liberal and early use of stay sutures for traction and retraction.

Urinary Retention

- Appropriate pain control
- Minimize perioperative fluids.

Pelvic Sepsis

- Use of prophylactic IV antibiotics
- Clean and empty rectum
- Meticulous closure of full thickness defects.

Anal Stricture/Stenosis

- Avoid damage to internal sphincter muscle fibers
- Avoid circumferential resection of anoderm.

References

1. Parks AG. A technique for excising extensive villous papillomatous change in the lower rectum. Proc R Soc Med. 1968;61(95):441–2.
2. Nivatvongs S, Balcos EG, Schottler JL, Goldberg SM. Surgical management of large villous tumors of the rectum. Dis Col & Rectum. 1973;16(6):508–14.
3. Thomson JP. Treatment of sessile villous and tubulovillous adenomas of the rectum: experience of St. Mark's Hospital. Dis Colon Rectum. 1977;20:467–72.
4. Sakamoto GD, MacKeigan JM, Senagore AJ. Transanal excision of large, rectal villous adenomas. Dis Colon Rectum. 1991;31(10):880–5.
5. Winde G, Nottberg H, Keller R, et al. Surgical cure for rectal carcinomas (T1). Transanal endoscopic microsurgery vs. anterior resection. Dis Colon Rectum. 1996;39(9):969–79.
6. Moore JS, Cataldo PA, Osler T, Hyman NH. Transanal endoscopic microsurgery is more effective than traditional transanal excision for resection of rectal masses. Dis Colon Rectum. 2008;51:1026–30.
7. Langer C, Liersch T, Suss M, et al. Surgical cure for early rectal carcinoma and large adenoma: transanal endoscopic microsurgery (using ultrasound or electrosurgery) compared to conventional local and radical resection. Int J Colorectal Dis. 2003;18:222–9.
8. Said S, Stippel D. Transanal endoscopic microsurgery in large, sessile adenomas of the rectum. A 10-year experience. Surg Endosc. 1995;9:1106–12.
9. Guerrieri M, Baldarelli M, Mornio M, et al. Transanal endoscopic microsurgery in rectal adenomas: Experience of six Italian centres. Digestive and Liver disease. 2006;38:202–7.
10. Atallah S, Albert M, Larach S. Transanal minimally invasive surgery: a giant leap forward. Surg Endosc. 2010;24:2200–5.
11. Albert MR, Atallah SB, Debeche-Adams T, et al. Transanal minimally invasive surgery (TAMIS) for local excision of benign neoplasms and early stage rectal cancer: Efficacy and outcomes in first 50 patients. Dis Colon Rectum. 2013;56:301–7.
12. Lev-Chelouche D, Margel D, Goldman G, Rabau MJ. Transanal endoscopic microsurgery: experience with 75 rectal neoplasms. Dis Colon Rectum. 2000;43:662–7.
13. Neary P, Makin GB, White TJ, et al. Transanal endoscopic microsurgery: a viable operative alternative in selected patients with rectal lesions. Ann Surg Oncol. 2003;10:1106–11.
14. Muldoon JP. Treatment of benign tumors of the rectum. Clin Gastroenterol. 1975;4:563–70.
15. Christoforidis D, ChoHM DixonMR, et al. Transanal endoscopic microsurgery versus conventional transanal excision for patients with early rectal cancer. Ann Surg. 2009;249 (5):776–82.

16. You YN, Baxter NN, Stewart A, Nelson H. Is the increasing rate of local excision for stage I rectal cancer in the United States justified? A nationwide cohort study from the National Cancer Database. Ann Surg. 2007;245:726–33.
17. Garcia-Aguilar J, Mellgren A, Sirivongs P, et al. Local excision of rectal cancer without adjuvant therapy: a word of caution. Ann Surg. 2000;231:345–51. M.
18. Madbouly KM, Remzi FH, Erkek BA, et al. Recurrence after transanal excision of T1 rectal cancer: Should we be concerned? Dis Colon Rectum. 2005;48:711–21.
19. Mellgren A, Sirivongs P, Rothenberger DA, et al. Is local excision adequate therapy for early rectal cancer? Dis Colon Rectum. 2000;43:1064–74.
20. Chorost MI, Petrelli NJ, McKenna M, et al. Local excision of rectal carcinoma. Am Surg. 2001;67:774–9.
21. Clancy C, Burke JP, Albert MR, O'Connell PR, Winter DC. Transanal endoscopic microsurgery versus standard transanal excision for the removal of rectal neoplasms: A systematic review and meta-analysis. Dis Colon Rectum. 2015;58:254–61.
22. Heintz A, Morschel M, Junginger T. Comparison of results after transanal endoscopic microsurgery and radical resection for T1 carcinoma of the rectum. Surg Endosc. 1998;12:1145–8.
23. Kidane B, Chadi SA, Knaters S, et al. Local resection compared with radical resection in the treatment of T1N0M0 rectal adenocarcinoma: a systematic review and meta-analysis. Dis Colon Rectum. 2000;43:1064–71.
24. Atallah S, Albert M, Larach S. Transanal minimally invasive surgery: a giant leap forward. Surg Endosc. 2010;24:2200–5.
25. Martin-Perez B, Andrade-Ribeiro GD, Hunter L, Atallah S. A systematic review of transanal minimally invasive surgery (TAMIS) from 2010 to 2013. Tech Coloproctol. 2014;18:775–88.
26. Petros JG. BradleyTM. Factors influencing postoperative urinary retention in patients undergoing surgery for benign anorectal disease. Am J Surg. 1990;159(4):374–6.
27. Zaheer S, Reilly WT, Pemberton JH, Ilstrup D. Urinary retention after operations for benign anorectal diseases. Dis Colon Rectum. 1998;41(6):696–704.
28. Toyonaga T, Matsushima M, Sogawa N, et al. Postoperative urinary retention after surgery for benigh anorectal disease: potential risk factors and stratergy for prevention. Int J Colorectal Dis. 2006;21(7):676–82.
29. Chik B, Law WL, Choi HK. Urinary retention after haemorrhoidectomy: Impact of stapled haemorrhoidectomy. Asian J Surg. 2006;29(4):233–7.
30. Tsai BM, Finne CO, Nordenstam JF, et al. Transanal endoscopic microsurgery resection of rectal tumors: outcomes and recommendations. Dis Colon Rectum. 2010;53(1):16–23.
31. Bignell MB, Ramwell A, Evans JR, Dastur N, Simson JN. Complications of transanal endoscopic microsurgery (TEM): a prospective audit. Colorectal Dis. 2010;12:e99–103.
32. Kumar AS, Coralic J, Kelleher DC, et al. Complications of transanal endoscopic microsurgery are rare and minor: a single institution's analysis and comparison to existing data. Disc Colon rectum. 2013;56(3):295–300.
33. Hahnloser D, Cantero R, Salgado G, et al. Transanal minimal invasive surgery for rectal lesions: should the defect be closed? Colorectal Dis. 17:397–402.
34. Haugvik SP, Groven S, Bondi J, et al. A critical appraisal of transanal minimally invasive surgery (TAMIS) in the treatment of rectal adenoma: a 4 year experience with 51 cases. Scand J Gastroenterol. 2016;51(7):855–9.
35. Sumrien H, Dadnam C, Hewitt J, McCarthy K. Feasibility of transanal minimally invasive surgery (TAMIS) for rectal tumors and its impact on quality of life- The Bristol Series. Anticancer Res. 2016;36(4):2005–9.
36. Ribbans WJ, Radcliffe AG. Retroperitoneal abscess following sclerotherapy for hemorrhoids. Dic Colon Rectum. 1985;28:188–9.
37. Barwell J, Watkins RM, Lloyd-Davies E, Wilkins DC. Life-threatening sepsis after hemorrhoid injection sclerotherapy: report of a case. Dis Colon Rectum. 1999;42:421–3.

38. Kaman L, Aggarwal S, Kumar R, et al. Necrotizing fasciitis after injection sclerotherapy for hemorrhoids: report of a case. Disc Colon Rectum. 1999;42:419–20.
39. McCloud JM, Jameson JS, Scott AND. Life threatening sepsis following treatment for hemorrhoids: a systematic review. Colorectal Dis. 2006;8(9):748–55.
40. Kam MH, NG KH, Lim JF, et al. Results of 7302 stapled hemorrhoidectomy operations in a single center: a seven year review and follow up questionnaire survey. ANZ J Surg. 81:253–6.
41. Butterworth JW, Peravali R, Anwar R, et al. A four-year retrospective study and review of selection criteria and post-operative complications of stapled hemorrhoidopexy. Tech Coloproctol. 2012;16(5):369–72.
42. Eu KW, Teoh TA, Seow-Choen F, Goh HS. Anal stricture following hemorrhoidectomy: Early diagnosis and treatment. Aust N Z J Surg. 1995;65(2):101–3.
43. Tjandra JJ, Chan MK. Systematic review on the procedure for prolapse and hemorrhoids (stapled hemorrhoidopexy). Dis Colon Rectum. 2007;50(6):878–92.
44. Shao WJ, Li GC, Zhang ZH, et al. Systematic review and meta-analysis of randomized controlled trials comparing stapled hemorrhoidopexy with conventional hemorrhoidectomy. Br J Surg. 2008;95(2):147–60.
45. Sutherland LM, Burchard AK, Matsuda K, et al. A systematic review of stapled hemorrhoidectomy. Arch Surg. 2002;137(12):1395–406.
46. Lacy AM, Tasende MM, Delgado S, et al. Transanal total mesorectal excision for rectal cancer: Outcomes after 140 patients. J Am Coll Surg. 2015;221(2):415–23.
47. Burke JP, Martin-Perez B, Khan A, et al. Transanal total mesorectal excision for rectal cancer: early outcomes in 50 consecutive patients. Colorectal Dis. 2016;18(6):570–7.
48. Rouanet P, Mourregot A, Azar CC, et al. Transanal endoscopic proctectomy: An innovative procedure for difficult resection of rectal tumors in men with narrow pelvis. Dis Colon Rectum. 2013;56:408–15.

Anal Stenosis

13

Jennifer Blumetti

Introduction

Anal stenosis is defined as an abnormal narrowing of the anal canal with loss of the anoderm, secondary to scarring and fibrosis [1, 2].The most common causes by far are from surgical removal or destruction of the anoderm, with anorectal surgery, and more specifically hemorrhoidectomy, being the leading causes of anal stenosis in adults [3–9]. Incidence of anal stenosis after hemorrhoidectomy can be as high as 5% [10, 11], with approximately 90% of all anal stenosis caused by hemorrhoidectomy [12, 13]. Coloanal and ileoanal pull-through procedures can also result in anal stenosis, with up to 16% of ileoanal pouches developing postoperative stenosis [14, 15]. In children, anal stenosis is most commonly the result of pull-through procedures [16, 17]. Causes of anal stenosis are listed in Table 13.1.

Anal stenosis has been classified by severity and location, and treatment can be tailored by this classification (Tables 13.2 and 13.3). Anal stenosis is typically diagnosed based on symptoms, with difficulty in evacuation and narrow stool most common. Table 13.4 lists common symptoms of anal stenosis. Examination typically reveals narrowing or the inability to pass a finger without discomfort. The constellation of difficulty with evacuation and inability to pass an examining finger are diagnostic [1, 18]. Exam under anesthesia may be necessary to delineate the extent of the disease if unable to examine in the office setting.

J. Blumetti (✉)
Colon and Rectal Surgery Residency Program, Stroger Hospital of Cook County, 1900 W. Polk St, Room 406, Chicago, IL 60612, USA
e-mail: jblumetti5@gmail.com

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_13

Table 13.1 Causes of anal stenosis

Anorectal surgery Hemorrhoidectomy/Whitehead amputative hemorrhoidectomy Excision of low lying tumors Extensive debridement/fulguration of condyloma Wide excision of Paget's disease or Bowen's disease
Anastomotic stricture from coloanal or ileoanal anastomosis Pull-through procedures in children with Hirschsprung's disease/imperforate anus
Trauma
Inflammatory Bowel disease
Radiation
Infections Sexually transmitted disease Tuberculosis
Chronic laxative abuse
Neoplasia
Congenital abnormalities

Table 13.2 Classification of anal stenosis

Classification by severity	Classification by location	Classification by extent
Mild: Exam can be completed with finger or medium Hill Ferguson retractor	**Low:** At least 0.5 cm distal to dentate line	**Localized:** one level or quadrant of the anal canal
Moderate: Dilation need to examine with finger or medium Hill Ferguson retractor	**Mid:** 0.5 cm distal to 0.5 cm proximal to dentate line	**Diffuse:** more than one level or quadrant
Severe: Unable to examine with little finger or small Hill Ferguson unless forcefully dilated	**High:** At least 0.5 cm proximal to dentate line	**Circumferential:** entire circumference

Table 13.3 Treatment options for anal stenosis

	Low stenosis	Mid stenosis	High stenosis
Mild/Moderate stenosis	Dilation Y-V anoplasty	Dilation Stricturotomy/stricturoplasty[a] Mucosal advancement flap U-Flap House Flap Diamond Flap	Endoscopic Dilation Transanal stapled reanastomosis[b] Mucosal Advancement flap U-Flap House Flap
Severe stenosis	U-flap House flap Diamond flap	U-Flap House Flap Diamond Flap	S-Plasty U-Flap House Flap

[a]For short strictures and high-risk patients
[b]For stricture less than 1 cm from colo/ileoanal anastomosis and after stapled hemorrhoidopexy

Table 13.4 Symptoms of anal stenosis

Constipation
Decrease in stool caliber
Difficulty initiating evacuation
Incomplete evacuation
Tenesmus
Diarrhea
Bleeding
Seepage and wetness (if associated with ectropion)

Treatment

Treatment of anal stenosis will vary depending on the location, severity, and cause of the stenosis (Tables 13.2 and 13.3). Patients with stenosis from infectious causes or inflammatory bowel disease should undergo appropriate medical treatment for the underlying condition.

Non-operative Treatment

For patients with mild/moderate low stenosis, nonoperative treatment should be instituted, with stool softeners/bulking agents and dilation. Dilation is appropriate for stenoses from coloanal or ileoanal pull-through procedures, from crohn's disease and radiation [19]. In children, dilation is routinely performed after pull-through procedures for Hirschsprung's disease and anorectal malformations in order to prevent the development of anastomotic stenosis [20, 21].

For strictures from coloanal or ileoanal anastomoses, dilation may be successful, and should be initiated within the first several weeks after surgery, and digital dilation by the examiner may be all that is required [22].

In adults, there are few published standardized methods for dilation as there are in children [3, 20]. Several authors advocate performing the first dilation in the operating room using Hegar dilators followed by daily dilation at home [3, 19]. Success will therefore require a compliant and motivated patient. For those patients with mild stenosis from Crohn's disease, about half will respond to dilation [19]. Shorter strictures will respond better to dilation than longer strictures [14].

For anastomotic strictures or those from stapled hemorrhoidopexy procedures that are located slightly higher, endoscopic balloon dilation can also be performed. Dilation for stricture is relatively safe, however, complications such as perforation can occur [23, 24]. Pain from repeated dilation may lead to decrease in success of treatment, especially in children [21]. Sphincter damage leading to fecal incontinence is also a concern with repeated dilations [1, 24].

Operative Treatment

Operative treatment is indicated for patients with moderate to severe stenosis, with stenosis associated with ectropion, and for those with mild stenosis who fail non-operative treatment.

A variety of operative procedures has been described for the treatment of anal stenosis. These should be tailored to the individual patient and the surgeon's familiarity with the procedures. Preoperative workup prior to surgical repair is typically minimal as many patients will not tolerate an exam in the clinic. Adjuncts such as endoanal ultrasound or manometry, although helpful in determining the status of the sphincters, will not be tolerated by most patients. Examination under anesthesia in the operating room is the most important for preoperative planning [3, 19].

Flaps

There are a several flaps that have been described in the treatment of anal stenosis (Table 13.3) which are described below. Flaps can be sliding (mucosal advancement, V-Y), island (Diamond, U, House), or rotational (S-plasty).

Mucosal Advancement flaps are best for mid- or high stenosis [19]. The procedure is performed laterally, and can be performed bilaterally if necessary. A radial incision is made through the scar and extending to the anal verge. The scar is excised, sphincterotomy performed, and a mucosal flap raised for 2–5 cm in length. The flap is then sutured to the intersphincteric groove, with a resultant small external wound [1]. Advantages of the mucosal advancement flap are minimal morbidity [25], small perianal wounds, and the ability to perform bilateral flaps if needed. Disadvantages include mucosal ectropion if the suture line is too distal and a higher rate of restenosis in treating distal severe disease [25].

Y-V anoplasty is another sliding flap which involves the use of a Y-shaped incision which is then sutured as a V [26]. See Fig. 13.1. The base of the Y incision (medial most aspect) should be shorter than the top of the Y (lateral aspect) to ensure that the flap has enough mobility to cover the entire defect. Care must be taken to raise a full thickness flap, as the blood supply is maintained from the most lateral aspect of the flap. Ischemia of the flap can occur if there is tension or if the flap is not the full thickness, with resultant dehiscence or restenosis [10, 27]. Benefits of this flap are its ease of performance, and no open wounds.

Island flaps are fully mobilized from the surrounding skin, which can allow further mobilization into the anal canal, making them useful in the treatment of higher stenoses. The blood supply to these flaps is through the subcutaneous tissue and allows for full mobilization and a tension-free anastomosis [28–30]. The diamond flap as described by Caplin and Kodner [4] begins with release of the scar via a lateral incision, and internal sphincterotomy can be performed if needed. This

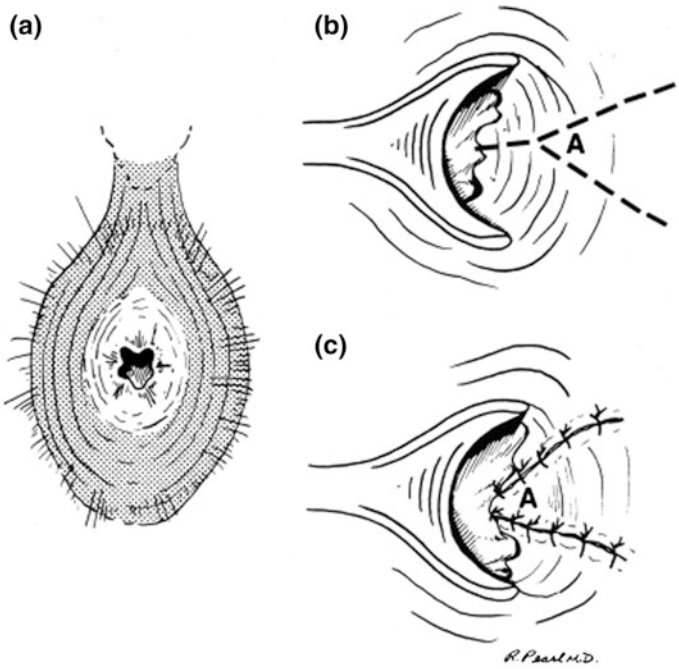


Fig. 13.1 Y-V Anoplasty—(from Fig. 41.1, Blumetti and Abcarian, Anal canal resurfacing in Anal stenosis, Chap. 41, pp 437–445, Zbar AP, Madoff RD, Wexner SD, eds. Reconstructive Surgery of the Rectum, Anus and Perineum Springer London 2013. **a** Anal Canal. **b** Line of Incision for Y-V anoplasty. The distance between the arms of the Y should be equal or greater to the length of the Y to allow a tension free closure. **c** Completed Y-V anoplasty

results in a diamond-shaped defect (Fig. 13.2). The flap is then drawn laterally to the incision, with the half of the flap closest to the anus being the size of the previously made incision. The full thickness flap is then created, with care taken to avoid undermining the flap, which can result in ischemia. The flap is then sutured into place and all the defects closed. This flap can also be performed bilaterally if necessary, and can be performed after failed Y-V.

The U-flap was initially described for the treatment of anal stenosis with associated mucosal ectropion [18]. The scar overlying the sphincters is excised, and a U-shaped incision is made in the perianal skin. The full thickness flap is then mobilized into the anal canal and it is sutured into place (Fig. 13.3). The donor site is left open. The benefits of this flap are that it is easy to perform, it can be adapted to any severity of stenosis up to 50% of the circumference, and can be performed bilaterally. The disadvantage is that there will be longer healing times due to the open donor site.

House flaps are a combination of a rectangle flap and the Y-V flap [29]. The flap is created by first incising from the dentate line to the distal end of the stenosis. The length of the "walls" of the house flap will be equal to the length of this initial

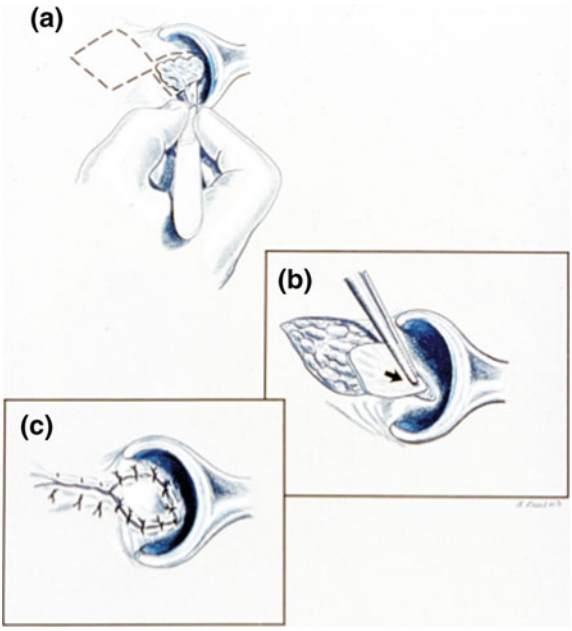


Fig. 13.2 Diamond Flap Anoplasty (from Fig. 41.2, Blumetti and Abcarian, Anal canal resurfacing in Anal stenosis, Chap. 41, pp 437–445, Zbar AP, Madoff RD, Wexner SD, eds. Reconstructive Surgery of the Rectum, Anus and Perineum Springer London 2013). **a** Line of incision for Diamond Flap Anoplasty. **b** The flap is brought into the wound. **c** Appearance after closure

incision (Fig. 13.4). The walls of the flap should be parallel and lateral to the initial incision, and the base of the house is the width of the mucosal defect, but should not be more than 25% of the circumference. The roof of the house is approximately the length of the walls. The flap is then mobilized, and the defects are all closed. The benefits of the house flap are that it is a well vascularized and broad-based flap, and it is relatively easy to perform. The house flap has been studied in a randomized trial comparing surgical techniques, and was noted to have higher clinical improvement in symptoms than either Y-V or rhomboid flaps [28]. A disadvantage is that longer operating time is needed for this flap. Also, since the flap is limited to 25% of the circumference (50% if performed bilaterally), it is less useful for more severe disease involving the majority of the anal canal.

The S-plasty rotational flap was initially described for the treatment of stenosis and ectropion associated with the Whitehead hemorrhoidectomy [6, 7]. It involves circumferential excision of the scar. The flap is then created in an S shape centered on the excision (Fig. 13.5b). The base of the flap, which corresponds to the lateral width of the incision, should be longer than the height of the flap, measured at the mid-portion of the incision (Fig. 13.5b). The full thickness flaps are mobilized and the superior flap (A) is rotated and sutured inferiorly, while the inferior flap (B) is

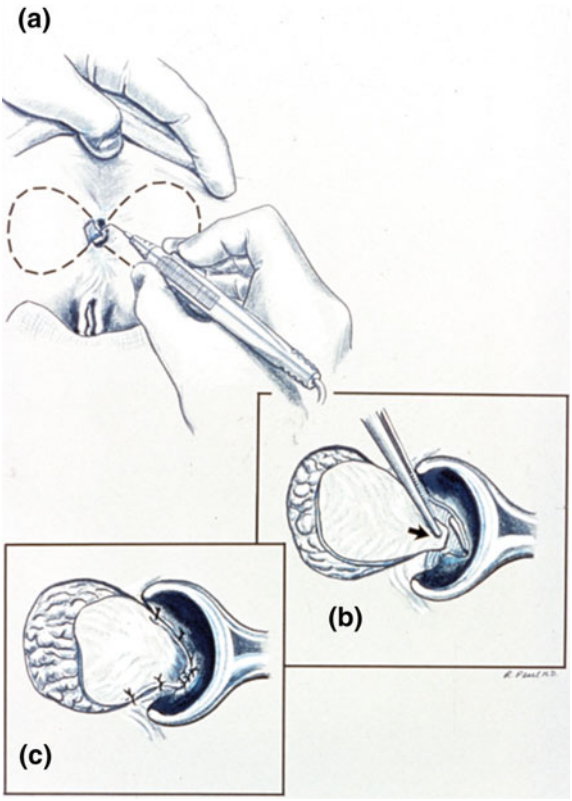


Fig. 13.3 U-Flap anoplasty (from Fig. 41.3, Blumetti and Abcarian, Anal canal resurfacing in Anal stenosis, Chap. 41, pp. 437–445, Zbar AP, Madoff RD, Wexner SD, eds. Reconstructive Surgery of the Rectum, Anus and Perineum Springer London 2013). **a** Outline of incision for bilateral U-flap anoplasty. **b** The fully mobilized flap is brought into the wound. **c** The flap sutured in place. The lateral donor site is left open to heal by secondary intention

sutured superiorly. The wounds are then closed completely (Fig. 13.5d). This flap is designed to cover large defects from circumferential stenosis. It is the most complex of the described flaps. As a rotational flap, it derives its blood supply from the tethered base of the flap, which puts it at risk for tension, ischemia, or dehiscence [30, 31]. This technique is typically utilized after other procedures have failed.

Other Techniques

Patients with mild/moderate short strictures, such as those resulting from a stapled hemorrhoidopexy, stapled transanal rectal resection (STARR), coloanal or ileoanal anastomoses, or those which are high surgical risk, may be candidates for stricturotomy with or without stricturoplasty [2, 32]. The technique involves incision of

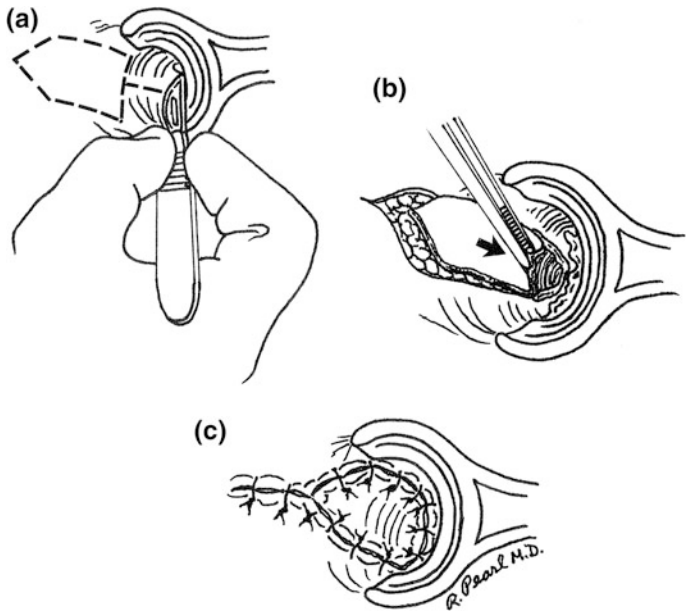


Fig. 13.4 House Flap Anoplasty (from Fig. 41.4, Blumetti and Abcarian, Anal canal resurfacing in Anal stenosis, Chap. 41, pp. 437–445, Zbar AP, Madoff RD, Wexner SD, eds. Reconstructive Surgery of the Rectum, Anus and Perineum Springer London 2013). **a** Incision for house flap. **b** Mobilized flap moved into anal canal. **c** Flap after closure

the stricture longitudinally in 3–4 quadrants, without incising muscle. The incisions can be left open to heal, or can be closed transversely as a Heineke Mikulicz type stricturoplasty. The types of strictures that can be successfully treated with this technique appear to involve mucosa, rather than anoderm [2]. Recurrence is common, and dilation can be utilized as an adjunct. If stricturotomy/stricturoplasty fails, then a flap procedure can be performed.

A relatively newer technique for the treatment of anastomotic strictures has been described, which utilizes transanal reanastomosis with a circular stapler. The stapler allows for complete excision of the stenosed segment, without the morbidity of repeat pelvic surgery [33]. The technique involves dilation of the stricture to allow passage of the anvil cephalad to the stricture, which is then coupled to the stapler and fired. Passage of the anvil can also be via a proximal stoma if the stricture is very severe or if the entire lumen is obliterated [16]. This technique is limited to short strictures less than one centimeter in length [16].

For those with anastomotic strictures from colo or ileoanal pouches, pouch advancement with reanastomosis is also an option if other procedures fail.

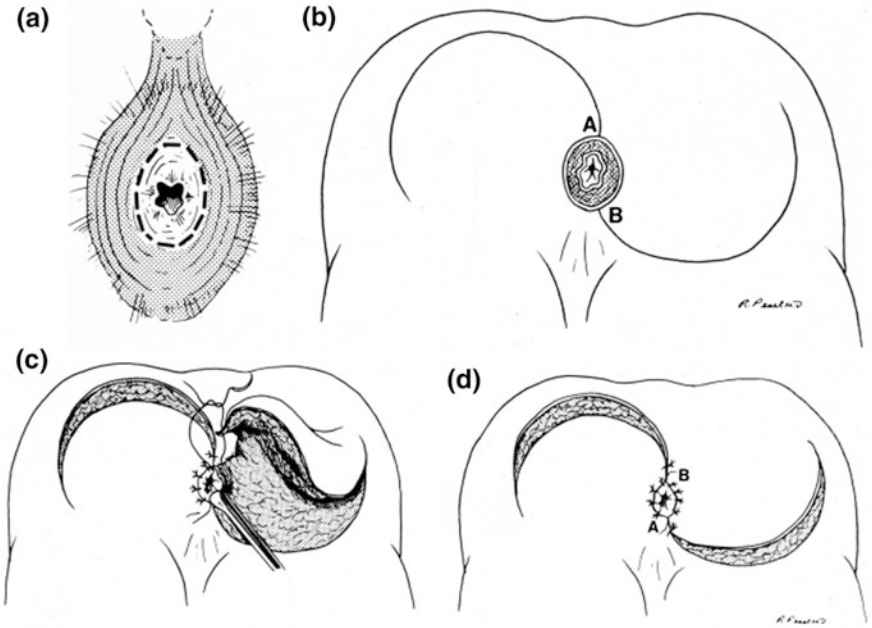


Fig. 13.5 S-Plasty (from Fig. 41.5, Blumetti and Abcarian, Anal canal resurfacing in Anal stenosis, Chap. 41, pp. 437–445, Zbar AP, Madoff RD, Wexner SD, eds. Reconstructive Surgery of the Rectum, Anus and Perineum Springer London 2013). **a** Line of excision of stenosis and ectropion. **b** Line of incisions for S-Plasty. The distance from A to the left lateral edge is the base of the superior flap. Note that this distance is longer than the height of the flap from superior to inferior. **c** Mobilization of the inferior flap is demonstrated. The superior flap has already been completed **d** Final appearance after completion. Note that the tip of the superior flap (A) has been rotated and sutured to the inferior aspect of the wound, and the tip of the inferior flap (B) now lies at the superior aspect. The donor sites are *left open*, but may also be closed primarily

Special Consideration: Stenosis in Children

Congenital abnormalities resulting in anal stenosis are extremely rare, and in children, anal stenosis is most commonly the result of pull-through/coloanal procedures performed for imperforate anus or Hirshsprung's disease [16, 17]. For these patients, prophylactic dilation is performed to avoid the formation of anastomotic strictures [21, 30, 34]. Levitt and Pena have suggested a standardized approach to dilation in these children [20]. Dilations are started 2 weeks postoperatively, starting with a dilator that fits snugly into the anal canal. Dilation is performed by parents twice daily, with increase in size of the dilator weekly until the desired size is reached. If the patient has a colostomy, it is then closed, and postoperatively the frequency of dilation is lessened in a stepwise fashion over the next several months. This dilation can extend up to 7 months [20]. The rate of stenosis with prophylactic dilation varies, but can range from less than 5–16% [35, 36]. In older infants and

children, dilation can be psychologically troubling for both the child and the parents, with increased risk of psychosocial problems in the child, which can be severe [30, 35]. Novel adjuncts to dilation include the use of topical mitomycin C, which is thought to decrease fibroblast proliferation and decrease scarring. This has been shown to be successful in one small study [21].

If a child develops a stenosis, the primary treatment is dilation prior to surgical intervention [16]. Once dilation fails, surgical intervention is indicated. Any of the procedures described above may be adapted for children. For those rare children with congenital anal stenosis, surgery is the first line therapy with Y-V flap as the procedure of choice [30]. For short ring-like strictures, stricturoplasty is in the manner of Heineke-Mikulicz [36]. Diamond flap anoplasty has been performed in children, and has been advocated as a second line procedure once others have failed [30]. The authors also note that postoperative dilations were easier after this procedure, with two children not requiring any dilation [30]. Transanal reanastomosis using a circular stapler has also been described in children and is performed in a similar fashion as adults, as described above [16]. Redo pull through can also be performed for longer strictures [36].

Choice of Procedure for Treatment (Table)

There is no one procedure of choice for anal stenosis, and treatment will depend on the location and severity as described in Tables 13.2 and 13.3. Any of the treatments described above may be utilized. Mild to moderate stenosis may be treated with non-operative measures, with or without dilation. If these conservative measures fail, then flap procedures are typically utilized. The choice of operative procedure should be tailored to the patient's disease, and the comfort level of the surgeon in performing the individual procedures.

Conclusion

Anal stenosis is a complex disease process that typically occurs after anorectal surgery in adults, and after coloanal pull-through procedures in children. Various surgical techniques have been described to treat anal stenosis, but no one procedure is ideal for every patient; the surgeon should therefore be familiar with the surgical options so that treatment may be tailored to the individual patient.

References

1. Khubchandani IT. Mucosal advancement anoplasty. Dis Colon Rectum. 1985;28:194–6.
2. Shawki S, Costedio M. Anal fissure and stenosis. Gastroenterol Clin N Am. 2013;42:729–58.

3. Liberman H, Thorson AG. Anal Stenosis. Am J Surgery. 2000;179:325–9.
4. Caplin DA, Kodner IJ. Repair of anal stricture and mucosal ectropion by simple flap procedures. Dis Colon Rectum. 1986;29:92–4.
5. Rosen L. V-Y Advancement for anal ectropion. Dis Colon Rectum. 1986;29:596–8.
6. Ferguson JA. Repair of "Whitehead deformity" of the anus. Surg, Gynec, Obst. 1959;108:115.
7. Hudson AT. S-Plasty repair of Whitehead Deformity of the Anus.
8. Faulconer HT, Ferguson JA. Anal S-Plasty for "Whitehead Deformity". Dis Colon Rectum. 1973;16:388–91.
9. Oh C, Zinberg J. Anoplasty for anal stricture. Dis Colon Rectum. 1982;25:809–10.
10. Maria G, Brisinda G, Civello IM. Anoplasty for the treatment of anal stenosis. Am J Surg. 1998;175:158–60.
11. Bonello JC. Who's Afraid of the Dentate line? The whitehead hemorrhoidectomy. Am J Surg. 1988;156:182–6.
12. Brisinda G, Vanella S, Cadeddu F, et al. Surgical treatment of anal stenosis. World J Gasroenterol. 2009;15:1921–8.
13. Milsom JW, Mazier WP. Classification and management of postsurgical anal stenosis. Surg, Gynec Obst. 1986;163:60–4.
14. Sagar PM, Pemberton JH. Intraoperative, postoperative and reoperative problems with ileoanal pouches. Br J Surg. 2012;99:454–68.
15. Kumar A, Daga R, Vijayaragavan P, et al. Anterior resection for rectal carcinoma-Risk factors for anastomotic leaks and strictures. World J Gastroenterol. 2011;17(11):1475–9.
16. Couto RA, Zequeira JJ, Vicente HL. Recalcitrant coloanal strictures in children managed by transanal circular stapled anastomosis. J Pediatric Surg. 2014;49:1686–8.
17. Shimada S, Matsuda M, Uno K, et al. A new device for the treatment of coloproctostomic stricture after double stapling anastomosis. Ann Surg. 1996;224:603–8.
18. Pearl RK, Hooks VH, Abcarian H, Orsay CP, Nelson RL. Island flap anoplasty for the treatment of anal stricture and mucosal ectropion. Dis Col Rectum. 1990;33:581–3.
19. Katdare MV, Ricciardi R. Anal stenosis. Surg Clin N Am. 2010;90:137–45.
20. Levitt MA, Pena A. Chapter 20: Operative management of anomalies in males. In: Holschneider AM, Hutson JM, editors. Anorectal Malformations in children. Berlin, Heidelberg, New York: Springer; 2006. p. 295–302.
21. Mueller CM, Beaunoyer M, St-Vil D. Topical Mitomycin-C for the treatment of anal stricture. J Pediatr Surg. 2010;45:241–4.
22. Dietz DW. Ch 10: Postoperative complications, In: Beck DE, Roberts PL, Saclarides TJ, et al, eds. The ASCRS textbook of Colon and Rectal Surgery, 2nd edition, Springer New York, 2011, pp 157–173.
23. Kanellos I, Blouhos K, Demetriades H, et al. Pneumomediastinum after dilation of anal stricture following stapled hemorrhoidopexy. Tech Coloproctol. 2004;8:185–7.
24. MacDonald A, Smith A, McNeill AD, et al. Manual dilation of the anus. Br J Surg. 1992;79:1381–2.
25. Rakhmanine M, Rosen L, Khubchandani I, Stasik J, Riether RD. Lateral mucosal advancement anoplasty for anal stricture. Br J Surg. 2002;89:1423–4.
26. Gingold BS, Arvanitis M. Y-V anoplasty for treatment of anal stricture. Surg Gynec Obst. 1986;162:241–2.
27. Angelchik PD, Harms BA, Starling JR. Repair of anal stricture and mucosal ectropion with Y-V or pedicle flap anoplasty. Am J Surg. 1993;166:55–9.
28. Farid M, Youssef M, El Nakeeb A, Fikry A, El Awady S, Morshed M. Comparative study of the house advancement flap, rhomboid flap and Y-V anoplasty in treatment of anal stenosis: a prospective randomized study. Dis Colon Rectum. 2010;53:790–7.
29. Christensen MA, Pitsch RM, Cali RL, Blatchford GJ, Thorson AG. "House" Advancement pedicle flap for anal stenosis. Dis Colon Rectum. 1992;35:201–3.

30. Anderson KD, Newman KD, Bond SJ, Sherman NJ. Diamond flap anoplasty in infants and children with an intractable anal stricture. J Pediatr Surg. 1994;29:1253–7.
31. Gonzalez AR, De Oliveira O, Verzaro R, Nogueras J, Wexner SD. Anoplasty for stenosis and other anorectal defects. Am Surg. 1995;61:526–9.
32. Guerra F, Crocetti D, Giuliani G, et al. Surgery for anorectal strictures following stapled procedure (letter). Colorectal Dis. 2015;17:265–70.
33. Rees JRE, Carney L, Gill TS, Dixon AR. Management of recurrent anastomotic stricture and iatrogenic stenosis by circular stapler. Dis Colon Rectum. 2004;47:944–7.
34. Brisighelli G, Morandi A, Di Cesare A, Leva E. The practice of anal dilations following anorectal reconstruction in patients with anorectal malformation: An international survey. Eur J Pediatr Surg. 2016; Jan 14 (e pub ahead of print).
35. Jenetzky E, Reckin S, Schmiedeke E, et al. Practice of anal dilatation after surgical correction in anorectal malformations. Pediatr Surg Int. 2012;28(11):1095–9.
36. Levitt MA, Pena A. Chapter 24: Complications after the treatment of anorectal malformations and redo operations. In: Holschneider AM, Hutson JM, editors. Anorectal Malformations in children. Berlin, Heidelberg, New York: Springer; 2006. p. 319–28.

Retrorectal Cyst

14

Kristen Donohue and Nell Maloney Patel

Introduction

The retrorectal, or presacral space is a potential space that may contain a diverse group of rare tumors. The incidence is cited to be from 0.0025 to 0.015% [1] or 1 in 40,000 admissions to major referral centers [2]. The most common of these tumors are benign developmental cystic lesions which account for 55–81% [2–4]. This varied group of tumors often produces little to no symptoms until they are locally advanced, and potentially impinging upon or invading adjacent structures. Due to their indolent course, they often are misdiagnosed and mistreated, leading to complications before a proper diagnosis has been made. This chapter examines the anatomy of the retrorectal space, differential diagnosis, and embryology of this diverse group of tumors, diagnosis, surgical treatment, and complications of treating such a rare and varied entity.

Anatomy

The retrorectal space is bound by the posterior wall of the rectum anteriorly and the presacral fascia posteriorly. The inferior border is the levator ani, while superiorly it extends to the peritoneal reflection (Fig. 14.1). The lateral borders are the ureters and the iliac vessels (Fig. 14.2). During early embryological development this

K. Donohue · N. Maloney Patel (✉)
Department of Surgery, Rutgers: Robert Wood Johnson Medical School, New Brunswick, NJ, USA
e-mail: Malonene@rwjms.rutgers.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_14

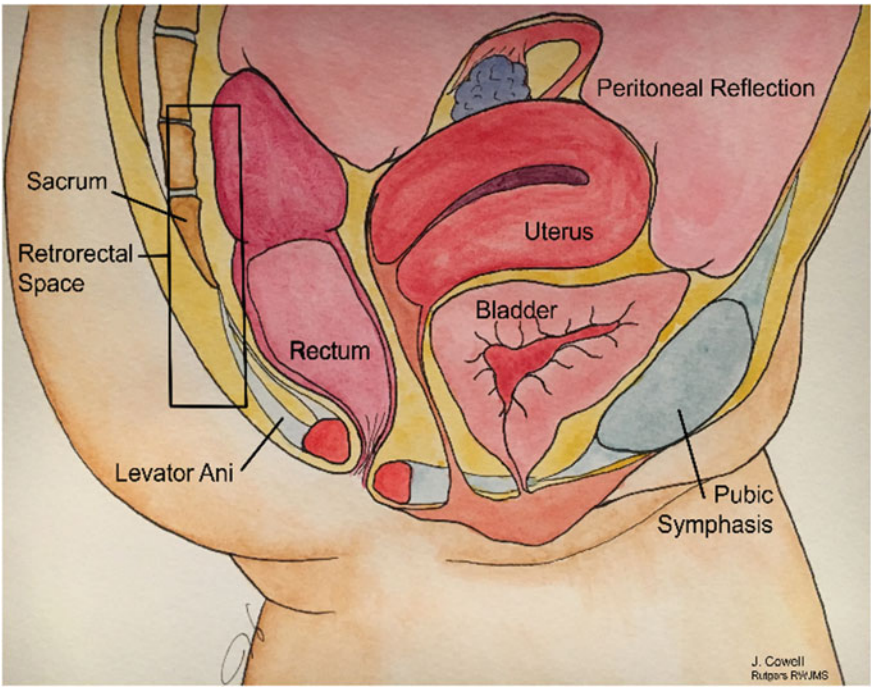


Fig. 14.1 Borders of the retrorectal space

space contains all three germinal layers, allowing for the development of various tumor types. Numerous important vascular and neurological structures are found within this space, and thus make operating in this area potentially wrought with complications. These include the hypogastric nerves, sacral nerve roots, middle sacral artery, superior rectal artery, middle rectal arteries, and lymphatics [5]. The majority of the blood supply to benign retrorectal tumors is from these nearby vessels, such as the middle sacral artery and internal iliac arteries, with smaller neighboring vessels contributing as well [5].

Differential Diagnosis and Classification

Lesions found in the retrorectal space can be broadly categorized as congenital or acquired, and benign or malignant [6]. Previously, classification systems have used these categories to give four distinct groups with similar clinical presentation, diagnosis, treatment, and prognosis, despite the variety of histologic subtypes still within each category [6]. Other classifications previously divided tumors into congenital, neurogenic, osseous, and miscellaneous [7]. Table 14.1 demonstrates

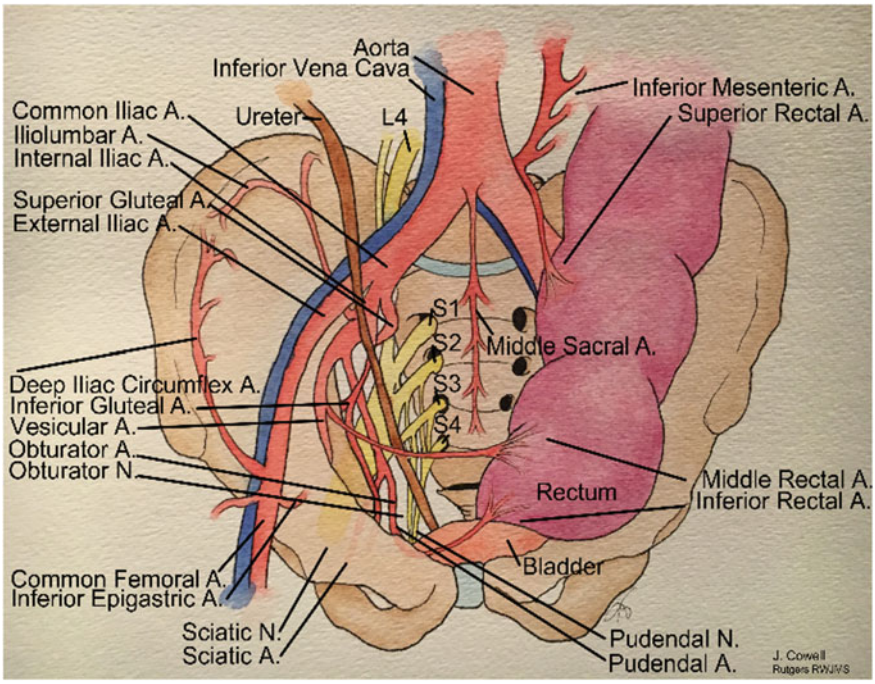


Fig. 14.2 Anatomy of the pelvis with surrounding structures, mesentery removed

the breadth of tumors that can be found in the presacral region, categorized by a combination of the above two classification systems.

Developmental Cysts

The majority of retrorectal or presacral tumors is congenital cystic lesions, also referred to as developmental cysts, and originate from developmental abnormalities such as the persistence of embryologic remnants, embryologic sequestration, or failure of midline fusion [9]. These include epidermoid cysts, dermoid cysts, enterogenous cysts, tailgut cysts, and teratomas and can make up 55–81% of retrorectal tumors [2].

Epidermoid and dermoid cysts are both noted to form due to failure of fusion or defect of the ectoderm, with the difference being skin appendages within epidermoid cysts, while dermoids contain only squamous cells [5, 10]. They are both generally benign, well-circumscribed lesions that can communicate with the skin and may be associated with a post anal dimple. Abel reported up to 1/3 of all cysts will become infected [11]. This complicates diagnosis, and they may be confused for perirectal abscesses and patients have been found to undergo multiple prior procedures before the correct diagnosis is made [10, 12].

Table 14.1 Classification of retrorectal tumors

	Benign	Malignant
Congenital	Epidermoid cyst	Chordoma
	Dermoid cyst	Teratocarcinoma
	Anterior sacral meningocele	
	Enterogenous cysts	
	Tailgut cysts (cystic hamartomas)	
	Teratomas	
Neurogenic	Ganglioneuroma	Ependymoma
	Neurofibroma	Neuroblastoma
	Neurolemmoma	Neurofibrosarcoma
	Schwannoma	Ganglioneuroblastoma
Osseous	Bone cysts	Osteogenic Sarcoma
	Chondroma	Ewing's Sarcoma
	Osteochondroma	Chondromyxosarcoma
	Osteoma	Myeloma
	Giant cell tumor	Chondrosarcoma
Miscellaneous (Includes Inflammatory)	Foreign body granuloma	Desmoid tumor (locally aggressive)
	Abscess/fistula	Lymphoma
	Angiomyxoma	Angiosarcoma
	Leiomyoma	Soft tissue sarcoma
	Hemangioma	Carcinoid
		Metastatic disease

Source Modified from Uhlig and Johnson [7], Lev-Chelouche et al. [6], and Szmulowicz and Hull [8]

Enterogenous cysts are endodermal in origin. They often present as multilobular cysts with a variety of epithelial contents. While predominantly benign, malignant degeneration has been reported within rectal duplications [13]. Tailgut cysts, or cystic hamartomas, are also multicystic and contain a range of epithelial contents.

Teratomas are derived from totipotential cells and include all three germ cell layers. They can be categorized as immature, mature, or malignant (teratocarcinoma) depending on the degree of differentiation of their contents. Teratomas are more common in the pediatric population with adult incidence ranging from 1 in 30,000–43,000 live births [14–16]. Malignant degeneration can occur in up to 50% of cases [17].

Malignant Tumors

Malignant retrorectal tumors have been reported to account for 8.7–50% of cases [7, 18, 19]. Some series demonstrate a male predilection for malignant tumors with

up to 86% being found in men [20], while other series report no difference in malignancy by sex [7, 12]. Cystic masses are less frequently found to be malignant (10%), while solid tumors are malignant in up to 60% of cases [12]. Chordomas are the most commonly reports malignant retrorectal tumors, and develop from a persistent mesodermic notochord.

Other Entities

Anterior sacral meningoceles can also be found in this presacral space. These congenital cystic lesions are made up of herniated dural and arachnoid membranes of the spinal cord through a defect in the sacrum. These may be associated with other congenital anomalies. Care must be taken not to enter these tumors and contaminate the cerebral spinal fluid for risk of meningitis given their direct connection to the subdural space.

Neurogenic and osseous tumors are also located in the retrorectal space along with a variety of miscellaneous entities including inflammatory conditions and metastatic disease from other sites (Table 14.1). Osseous tumors can often be differentiated on imaging due to their association with sacral destruction.

Diagnosis and Preoperative Management

Presacral tumors often present very indolently with nonspecific symptoms that make them difficult to diagnose. Many are found incidentally during pelvic or gynecologic examinations. Benign lesions have been reported to be more common in females, though women of childbearing age often undergo pelvic and rectal exams more often than their male counterparts, therefore this selection bias may partly explain this difference. Malignant lesions tend to have an equal distribution among the sexes [7, 12].

The most common symptom reported by patients is pain, which is vague, difficult to localize, and longstanding. Pain has been reported to be associated more frequently with malignant tumors than benign (88% vs. 39%) [2]. Other symptoms reported in the literature include perineal discharge, constipation, incontinence of urine or stool, sexual dysfunction, limb weakness, perineal pressure, or rectal bleeding [21–23]. These vague symptoms and presentations can often lead to misdiagnoses including perianal abscess, fistula in ano posttraumatic pain, pilonidal cyst, presacral abscess, postpartum pain, and psychogenic pain [12]. Average symptom duration is reported at up to 4.9 years in patients with misdiagnoses, and patients undergo an average of 4.7 invasive procedures in order to diagnose or treat their disease [12].

A thorough history and physical exam with careful attention to the sacral nerves, presence of a post anal dimple, or fullness in the precoccygeal area should be performed. Digital rectal exam should also be performed and these lesions are

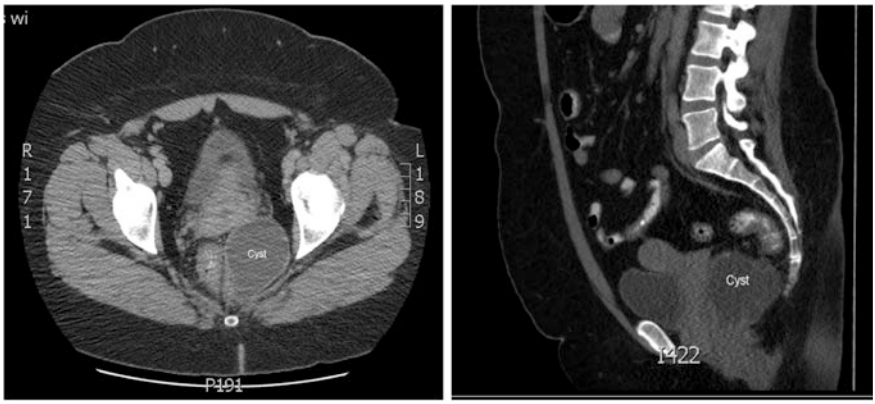


Fig. 14.3 CT imaging of cystic hamartoma. Axil view, sagittal view

palpable on DRE in anywhere from 35 to 97% of patients [2, 20]. Sigmoidoscopy can also be performed to determine invasion of the rectal wall. A high index of suspicion is necessary in patients with multiple prior incision and drainage procedures and recurrent infections or any of the above physical exam findings.

Imaging has improved diagnosis and management of this wide array of disorders in recent years. CT scan and MRI are most often used, and both are reported to have 100% sensitivity for diagnosing the presence of a retrorectal mass [20] (Fig. 14.3). Imaging determines whether a lesion is cystic or solid, the relationship to surrounding structures, and the boundaries of the tumor. While imaging modalities can be helpful for planning operative approach, they often are not sufficient for diagnosis. Correct diagnosis is made by CT alone in only 15% of one reported cohort, and 67% via MRI [24] (Fig. 14.4). Of these patients, 49% could not be given a diagnosis based on imaging alone, and 35% of those cases had malignancy on final

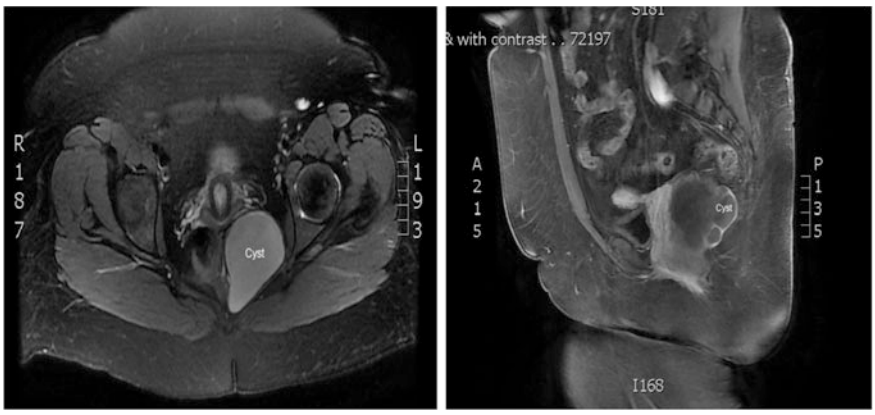


Fig. 14.4 MRI of retrorectal tailgut cyst; T2 weighted axial view, T2 fat sat sagittal view

pathology [24]. Transrectal ultrasound has also been used in combination with rigid proctoscopy with a reported sensitivity of 100% visualization of retrorectal tumors for this combined modality [12, 20].

Preoperative Biopsy

The role of preoperative biopsy of retrorectal tumors has been controversial in the literature. Older literature reports significant risks including seeding the biopsy tract causing increased recurrence rates, increased risks of infection, and risk of injury to surrounding structures including but not limited to the anococcygeal ligament, presacral nerves and sympathetic ganglion, bowel, bladder, vasculature, uterus, and adnexae [25]. With the increased use of high resolution CT guided biopsy these risks have become minimized. Studies have shown minimal complications. One such study reported 76 biopsies performed on 73 patients with only two complications (both hematomas with no clinical sequelae) [24]. These biopsies were 96% sensitive, 100% specific, and had 91% final pathology correlation, overturning diagnoses in 29% of their patients who had been given definitive diagnoses based on imaging alone [24]. Additionally, in 39 biopsies of malignant tumors in another series, there were no complications [23].

Moreover, the importance of obtaining a tissue diagnosis prior to surgical excision lies in the potential to change treatment, as some solid tumors may be better managed with preoperative radiation, chemotherapy or both. Shrinking a large pelvic tumor can alleviate potential operative complications by decreasing the surgical burden. Messick et al. biopsied 32% of their patients preoperatively, with no reported complications, and five patients had a management change as a result of the biopsy [4]. They also reported no change in local recurrence rates (21% benign, 41% malignant in their series) when compared with prior case series, despite the increased use of preoperative biopsy. Other authors advocate that biopsy should be reserved for tumors that appear unresectable to aid in planning for adjuvant therapy [20].

There is little to no role of biopsy in a purely cystic lesion, as many of these are benign, and even with malignant transformation, the treatment approach does not change. There is, however, an increased risk of infection with transrectal and transvaginal biopsy, and thus these routes should also be avoided [10]. Finally, if biopsied, it should be done so the tract can be excised with the tumor if it is malignant; therefore, biopsy approach should be incorporated into the operative plan [10].

Surgical Management

All retrorectal tumors should be surgically excised, with the exception of presumed malignancies such as Ewing's sarcomas, desmoid tumors, osteogenic sarcomas, or neurofibrosarcomas, which benefit from preoperative radiation or chemotherapy

[26]. All other tumors should be removed because even with presumed benign lesions, malignant transformation can occur, despite imaging and even biopsy [2]. Additionally, about 1/3 of cystic lesions will become infected over time, thus making eventual operative resection more difficult and fraught with complication as tissue planes become inflamed [11], and once infected up to 30% recurrence rate is reported in such cases [2]. Moreover, anterior meingoceles are at risk for life threatening meningitis if infected. Additionally all woman of childbearing age should have retrorectal masses excised due to their potential to obstruct the vaginal canal and cause dystocia during childbirth [7, 27, 28].

Surgical Approach

Preoperative planning taking into consideration the extent of the tumor, involvement of adjacent structures, and the relationship to the sacrum is important in deciding how to proceed. Most authors also advocate preoperative bowel preparation to reduce infectious complications in the event that the rectum is entered inadvertently. The operative approach varies depending on the relationship of the tumor to the sacrum.

Low-lying tumors, such as those in which the superior most aspect can be palpated on digital rectal exam, should be approached by a posterior, or perineal approach [19] (Fig. 14.5). This approach is also useful for infected cysts, though some advocate draining infected cysts via CT guidance prior to operative resection, and proceeding with definitive resection once the acute inflammation has resolved [29]. A paracoccygeal incision is made, and the coccyx may be removed. Of note, vascular control is difficult via this approach, and if the tumor extends superiorly and proximal control cannot be obtained, it may be necessary to convert to laparotomy to control bleeding.

If the tumor extends above S-3, a combined abdominosacral approach is advocated. This method primarily allows for control of the iliac vessels, and anterior mobilization of the ureters and rectum from the tumor, while also allowing the tumor to be dissected from the sacrum and the tenuous blood supply posteriorly.

If the entirety of the tumor lies at or above S-4 [12] the abdominal approach is preferred, though some authors report S-3 to be their cutoff [9]. More recently, there have been case reports describing laparoscopic approach to these tumors, citing the excellent visualization of the deep pelvic anatomy [30].

Finally, a transanal approach is rarely used to resect these lesions. There are reported excisions of tailgut cysts or cystic hamartomas in this fashion with no complications or recurrence [31–33]. One group used this approach for 11 patients, with only one developing a complication of a presacral abscess [3]. Though other authors prefer that this method only be used for lesions that had previously been drained through the rectum, as well as small epidermoid and dermoid cysts, noting poor visualization as a barrier to complete resection [5].

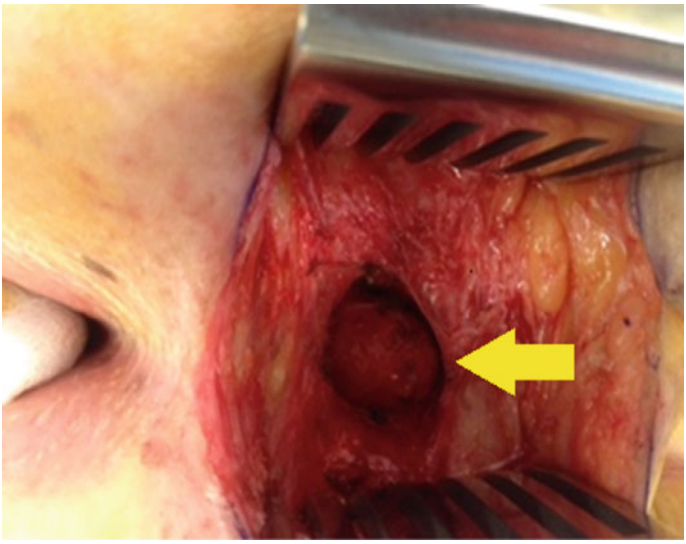


Fig. 14.5 Visualization of tailgut cyst intraoperatively via posterior "Kratske" approach, *arrow* identifies cyst

Complications

Preoperative Complications

As demonstrated thus far, this heterogeneous group of tumors faces many complications even prior to diagnosis. Preoperatively patients face the risk of delayed and missed diagnosis, infection, recurrent procedures, and malignant degeneration of previously benign lesions. Biopsy continues to be debated, with the risks of bleeding, injury to surrounding structures, and possible spread of malignant or infected tissues.

Intraoperative and Postoperative Complications

Complication rates for the operative intervention vary in the reported literature. The most devastating intraoperative and postoperative complications include bleeding, infection, rectal perforation, and incomplete excision/recurrence. Other frequently reported complications include bowel and bladder dysfunction (incontinence, retention), sexual dysfunction, ureteral injury, pelvic floor dysfunction, neurologic symptoms, and low incidence of various other common surgical complications, such as DVT, pneumonia, and bowel obstruction (Table 14.2). Table 14.2 shows

Table 14.2 Complications during and after surgery for various retrorectal tumors

	Cases	Mortality	Morbidity	Bleed	Infection	Rectal injury	Bowel or bladder	Sexual	Neurologic	Other
Localio et al. [22]	20	1 (5%) (sepsis)	12 (60%)	4 (20%) 4 (>1500 mL)	1 (5%) urosepsis wound, death	0	6 (30%) 5 UR 1 UI	0	2 (10%) 2 LE weakness	0
Cody et al. [23]	27	1 (3.7%) (bleed MI)	8 (30%)	4 (15%) 1 MI/death 1 re-op 2 hematoma	0	0	3 (11 N %) 3 UR	0	1 (3.7%) 1 foot drop	1 (3.7%) 1 SBO
Jao et al. [2]	102	0	48 (47%)	4 (4%)	15 (15%) 11 wound 3 abscess 1 fistula	0	22 (22%) 15 UR 7 FI	0	7 (7%) 7 dysthesia	0
Pidala et al. [3]	14	0	2 (14%)	0	1 (7%) 1 abscess	1 (7%)	0	0	0	0
Lev-Chelouche et al. [6]	42	0	28 (67%)	11 (26%) 10 transfusion 1 hematoma	7 (17%) 5 wound 2 abscess	3 (7%)	2 (5%) 2 UR	0	1 (2%) 1 LE weakness	4 (10%) 1 UTI 1 PNA 1 SBO 1 DVT
Glasgow et al. [20]	34	0	7 (21%)	2 (6%) 2 reoperation	1 (3%) 1 wound	0	0	2 (6%) 2 impotency	0	2 (6%) 1 MI 1 UI
Buchs et al. [34]	16	0	1 (6%)	0	0	0	0	0	0	1 (6%) 1 PE

(continued)

Table 14.2 (continued)

	Cases	Mortality	Morbidity	Bleed	Infection	Rectal injury	Bowel or bladder	Sexual	Neurologic	Other
Canelles et al. [35]	20	0	5 (25%)	0	2 (10%) 1 wound 1 mesh	0	0	0	1 (5%) 1 LE pain	2 (10%) 2 Seroma
Woodfield et al. [9]	27	0	9 (33%)	5 (19%) 5 transfusion	3 (11%) 3 wound	0	0	0	0	1 (4%) 1 PTX
Mathis et al. [36]	31	0	8 (26%)[a]	NR	2 (6%) 2 wound	NR	NR	1 (3%)	NR	2 (6%) 2 PF
Lin et al. [37]	62	0	9 (14.5%)	3 (5%) 3 (>800 mL) (transfusion)	3 (5%) 3 presacral	3 (5%)	0	0	0	0
Messick et al. [4]	45	0	10 (22%)	5 (11%)	3 (7%) 1 wound 2 pelvic abscess	0	0	0	0	2 (4%) 1 CSF 1 C Diff
Patsouras et al. [38]	17	0	6 (35%)	0	3 (18%) 3 wound	3 (18%)	0	0	0	0
Total	457	2 (0.4%)	153 (33%)	50 (10.9%)	48 (10.5%)	10 (2.2%)	33 (7.2%)	3 (0.7%)	12 (2.6%)	15 (3.3%)

Adapted from Szmulowicz and Hull [8] with additions and edits

UR Urinary Retention, *UI* Urinary Incontinence, *LE* Lower Extremity, *MI* Myocardial Infarction, *SBO* Small Bowel Obstruction, *IR* Incomplete Resection, *UTI* Urinary Tract Infection, *PNA* Pneumonia, *DVT* Deep Vein Thrombosis, *UI* Ureteral Injury, *PE* Pulmonary Embolus, *PTX* Pneumothorax, *NR* Not Reported, *PF* Pelvic Floor Dysfunction, *CSF* Cerebral Spinal Fluid leak, *C Diff* Clostridium Difficile

[a]8 reported in paper, but only 5 described in detail

reported complication data and interpreted blood loss from many of the larger case series discussing retrorectal tumor excisions.

These studies represent a broad range of time (1964–2013), diagnoses, and operative approaches. Some included only benign cystic lesions, while others included all retrorectal tumors in their reviews. Overall a total of 457 cases were included in the above analysis. The reported morbidity from these larger selected case series in the literature range from 6 to 67%, with an average of 33% across all studies. The mortality associated with resection of retrorectal tumors is minimal (0.4%), and this decreased to 0 after 1981. Complications were more common among malignant than benign tumors, likely due to greater extent of disease, without any differences among operative approach [18]. Moreover, recurrent tumors are often found to be more difficult to approach surgically due to altered anatomical planes from fibrosis after prior interventions [39].

Bleeding

The most significant morbidity tends to arise from bleeding, and this was reported in 10.9% of cases in the above literature, with a range from 0 to 26%. Many authors discuss the potential for significant blood loss during these procedures.

Risk factors for increased bleeding include previous radiation, vascular tumors, sacrectomy [26], and adherence of the tumor to the presacral plexus [14]. Preoperative angioembolization for particularly vascular tumors has been described to aid in reduction of intraoperative blood loss, but is not routinely done [40]. Many authors advocate access through the abdomen for control of the vasculature to decrease operative blood loss; however, some report less blood loss and morbidity from a posterior approach [18]. This is likely due to the fact that posteriorly approached tumors are often smaller and benign compared with those approached anteriorly.

Certain situations may require a more aggressive approach to prevent bleeding complications. One group described three patients with chordomas approached posteriorly with an average estimated blood loss (EBL) of 5L. Their next five patients underwent abdominosacral approach with control of the iliac vessels and EBL decreased to 400–1500 in 4/5 of these patients [22]. Many authors also advocate ligating of the internal iliac vessels on the side of the tumor, as well as the middle sacral artery and vein if necessary to control blood loss [23, 41]. Sharp as opposed to blunt dissection has also been described to minimize operative blood loss [37].

Rectal Injury/Perforation

Rectal injury or perforation is a much rarer complication, and was reported in only 2.2% of the 457 cases in Table 14.2. Additionally, many tumors may be adherent or previously draining through the rectum, making avoiding entering the rectum

impossible. It is for this reason that most authors advocate a bowel preparation for all patients undergoing surgery for retrorectal tumors [2, 12, 34].

Preoperative imaging with endorectal ultrasound (ERUS) as well as proctoscopy has been recommended in order to better delineate the tumors relationship with the rectal wall [12, 42], and MRI allows visualization of tissue planes between the tumor and surrounding structures [14]. Using the posterior approach can be difficult to differentiate the levator ani from the rectum. For small cystic lesions approached this way, a doubly gloved finger can be inserted into the rectum to deliver the tumor up away from the rectum, and facilitate exposure [39, 43]. Infiltration of dilute epinephrine solution in the plane between the cyst and rectum may facilitate dissection.

If a rectal injury does occur, it may be primarily repaired or that area may be excised and an anastomosis made. The decision of whether to place a protective loop ostomy is based largely on patient factors including overall nutrition and health [3, 37]. Therefore, all patients should be counseled on the potential for a colostomy with planned resection of presacral tumors.

Infection

In the above review of the literature, 10.5% of patients had local infectious complications. These ranged from wound infections, abscesses, fistulas, wound breakdown, and dehiscence. This is largely in part due to a high infectious rate of the tumors themselves with up to 1/3 of congenital cystic lesions being previously infected. Authors have advocated draining these infected lesions prior to definitive operative intervention [11]. Additionally, while biopsy of these lesions at all remains controversial, it is prudent to avoid transrectal or transvaginal biopsy for risk of seeding the previously sterile collection [10]. Finally, avoidance of rectal injury may prevent wound and pelvic infections.

Incomplete Resection/Recurrence

Recurrence is common both for benign and malignant retrorectal tumors, however prognosis is improved with complete surgical excision [26, 44]. In Table 14.3, the recurrence rates for benign and malignant tumors across case series are reviewed. In these studies benign tumor recurrence occurred in 5–15%, where malignant tumors recurred at much higher rates, anywhere from 6 to 100%. Overall recurrence was 23.8%. Chordomas, the most common malignant retrorectal tumor, have a reported recurrence rate of 40–100% [6, 8]. One author reported that 100% of partially resected malignant tumors recurred, compared with only 1 benign mass that had positive margins [6].

Prior literature cited coccygectomy to be protective against recurrence, specifically against chordoma; however, later reviews have shown no difference in recurrence when coccygectomy was performed [2, 22]. Authors advocate

Table 14.3 Compilation of recurrence rates of retrorectal tumors

	Benign	Malignant	Total
Localio et al. [22]	0/9 (0%)	5/11 (45%)	5 (25%)
Cody et al. [23]			13 (48%)
Jao et al. [2]	10/66 (15%)	18/21 malignant (86%) 20/30 chordomas (67%)	48 (41%)
Pidala et al. [3]	1 (7%) Incomplete resection		1 (7%)
Lev-Chelouche et al. [6]	0/18 (0%)	12/24 (50%)	12 (29%)
Glasgow et al. [20]	0/26 (0%)	8/8 (100%)	8 (24%)
Buchs et al. [34]	[a]	1 (6%) Incomplete Resection	1 (6%)
Canelles et al. [35]	1/15 (6.7%)	2/5 (40%) Incomplete Resection	4 (20%)
Woodfield et al. [9]	1/20 (5%)	2/7 (28.5%)	3 (11%)
Mathis et al. [36]	NR	NR	1 (3%)
Lin et al. [37]	4/48 (8.3%)	3/14 (21%)	7 (11%)
Messick et al. [4]	4/36 (11%)	5/9 (56%)	9 (20%)
Patsouras et al. [38]	1/17 (5.8%)	1 (6%)	1 (6%)
Total			109 (23.8%)

[a]Pathology of this case not reported

performing coccygectomy only when the tumor is involving the coccyx, or when it is needed to facilitate exposure. Complete resection is fundamental for malignant disease, as most of these tumors are poorly responsive to adjuvant therapy. There are reports of adjuvant treatment with radiotherapy for incompletely resected malignant tumors, but there is no strong data for survival benefit [10].

Re-excision is often done for recurrent disease that is amenable to surgery, but these operations are much more difficult as planes have been destroyed [39] (Fig. 14.6).

Bowel/Bladder/Sexual/Neurologic

The surgical excision of any retrorectal tumor may also be complicated by neurologic sequelae. The incidence of bowel of bladder dysfunction is around 7.2%, with the most common complication being urinary retention. However, the reporting of this complication has decreased in recent years (Table 14.2). It is common to disrupt the abdominal and pelvic nerve plexuses with posterior rectal resection [37]. The reported urinary disturbance rate in the literature is 7–70%, while sexual dysfunction is 25–100% [37, 45]. In the 457 cases in Table 14.2, sexual dysfunction was cited only in three patients, or 0.7%.

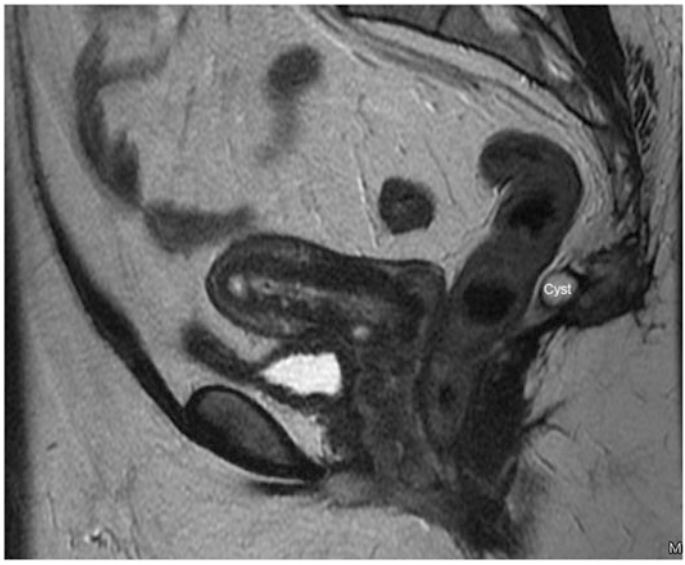


Fig. 14.6 MRI of recurrent retrorectal mass. Sagittal view of T2 weighted image, cyst appears hyperintense

An important landmark for neurological sequelae is the S2/S3 vertebral levels. In patients with mid S2 vertebral involvement, increased urinary symptoms were identified, requiring self-catheterization or indwelling foley catheters [22]. Fecal incontinence is also possible, though much less likely. An intersphincteric excision for distal, benign tumors may be used in order to avoid injury to the anal sphincter and preserve fecal continence [34]. This approach also prevents urinary retention by avoiding the sacral nerves [34, 46]. In Table 14.2, 2.6% of patients reported some disturbance in lower extremity neurologic function. These included weakness, foot drop, or dysthesias (Table 14.3). In order to prevent neurologic sequelae, it is prudent when approaching the level of S2/S3 to expose both sciatic nerve trunks prior to dissection of the sacrum [9, 23]. This ensures the ability to protect one or both sides, and minimize injury.

Conclusions

Retrorectal masses remain a rare and diverse group of tumors. Proper diagnosis is important for correct and prompt treatment, and avoiding early complications such as infection, unnecessary biopsies, and seeding of potentially malignant tumors. Avoiding preoperative biopsy of cystic lesions, especially transvaginal or transrectal biopsies will help avoid infectious complications. Choosing the best operative approach, based on the tumor location and characteristics, will enable the

easiest access to the tumor, blood supply, and exposure of surrounding structures. Operative complications are rare, and mortality is low. These complications include bleeding, infection, rectal injury, recurrence, and neurologic sequelae. It is imperative to be aware of surrounding structures such as nerves, blood supply, the rectum, ureters, and sacrum in order to avoid injury. Multidisciplinary teams may be necessary to address these masses safely. An unfortunately common complication of these tumors is recurrence, and complete microscopic R-0 excision when possible can help minimize this, as few effective adjuvant therapies are available.

References

1. Bohm B, Milsom JW, Fazio VW, Lavery IC, Church JM, Oakley JR. Our approach to the management of congenital presacral tumors in adults. Int J Colorectal Dis. 1993;8:134–8.
2. Jao S-W, Beart RW, Spencer RJ, Reiman HM, Ilstrup DM. Retrorectal tumors: mayo clinic experience, 1960-1979. Dis Colon Rectum. 1985;28:644–52.
3. Pidala MJ, Eisenstat TE, Rubin RJ, Salvati EP. Presacral cysts: transrectal excision in select patients. Am Surg. 1999;65:112–5.
4. Messick CA, Londono JM, Hull T. Presacral tumors: how do they compare in pediatric and adult patients? Pol Przegl Chir. 2013;85:253–61.
5. Hannon J, Subramony C, Scott-Connor CEH. Benign retrorectal tumors in adults: the choice of operative approach. Am Surg. 1994;60:267–72.
6. Lev-Chelouche D, Gutman M, Goldman G, Even-Sapir E, Meller I, Issakov J, et al. Presacral tumors: a practical classification and treatment of a unique and heterogeneous group of diseases. Surgery. 2003;133(5):473–8.
7. Uhlig BE, Johnson RL. Presacral tumors and cysts in adults. Dis Colon Rectum. 1975;18:581–96.
8. Szmulowicz UM, Hull TL. Retrorectal Tumors. In: Zbar PA, Madoff DR, Wexner S, editors. Reconstructive surgery of the rectum, anus and perineum. London: Springer; 2013. pp. 517–49.
9. Woodfield JC, Chalmers AG, Phillips N, Sagar PM. Algorithms for the surgical management of retrorectal tumors. Br J Surg. 2007;95:214–21.
10. Dozois EJ, Jacofsky DJ, Dozois RR. Presacral Tumors. In: Wolff BG, Fleshman JW, Beck DE, Pemberton JH, Wexner SD, Church JM, et al., editors. The ASCRS textbook of colon and rectal surgery. New York, NY: Springer; 2007. pp. 501–14.
11. Abel ME, Nelson R, Prasad L, Pearl RK, Orsay CP, Abcarian H. Parasacrococcygeal approach for the resection of retrorectal developmental cysts. Dis Colon Rectum. 1985;28:855–8.
12. Singer MA, Cintron JR, Martz JE, Schoetz DJ, Abcarian H. Retrorectal cyst: a rare tumor frequently misdiagnosed. J Am Coll Surg. 2003;196(6):880–6.
13. Springall RG, Griffiths JD. Malignant change in rectal duplication. J R Soc Med. 1990;83:185–7.
14. Ghosh J, Eglinton T, Frizelle FA, Watson AJM. Presacral tumors in adults. Surgeon. 2007;5:31–8.
15. Bull Y Jr, Yeh KA, McDonnell D, Caudell P, Davis J. Mature presacral teratoma in an adult male: a case report. Am Surg. 1999;65(6):586–91.
16. Ng EW, Porcu P, Loehrer PJ Sr. Sacrococcygeal teratoma in adults: case repots and a review of the literature. Cancer. 1999;86(7):1198–202.
17. Waldhausen JA, Kolman JW, Vellios F, Battersby JS. Sacrococcygeal teratoma. Surgery. 1963;54:933–49.

18. Spencer J, Jackman RJ. Surgical management of precoccygeal cysts. Surg Gynecol Obstet. 1962;115:449–52.
19. Guillem P, Ernst O, Herjean M, Triboulet JP. Tumeurs retrorectales: interet de la voie abdominale isolee. Ann Chir. 2001;126:138–42.
20. Glasgow SC, Birnbaum EH, Lowney JK, Fleshman JW, Kodner IJ, Mutch DG, et al. Retrorectal tumors: a diagnostic and therapeutic challenge. Dis Colon Rectum. 2005;48 (8):1581–7.
21. Simpson PJ, Wise KB, Merchea A, Cheville JC, Moir C, Larson DW, et al. Surgical outcomes in adults with benign and malignant sacrococcygeal teratoma: a single-institution experience of 26 cases. Dis Colon Rectum. 2014;57(7):851–7.
22. Localio SA, Eng K, Ranson JHC. Abdominosacral approach for retrorectal tumors. Ann Surg. 1980;191:555–9.
23. Cody HS, Marcove RC, Quan SH. Malignant retrorectal tumors: 28 years' experience at Memorial Sloan-Kettering Cancer Center. Dis Colon Rectum. 1981;24:501–6.
24. Merchea A, Larson DW, Hubner M, Wenger DE, Rose PS, Dozois EJ. The value of preoperative biopsy in the management of solid presacral tumors. Dis Colon Rectum. 2013;56 (6):756–60.
25. Gupta S, Nguyen HL, Frank A. Morello J, Ahrar K, Wallace MJ, Madoff DC, et al. Various approaches for CT-guided percutaneous biopsy of deep pelvic lesions: anatomic and technical considerations. RadioGraphics. 2004;24(1):175-89.
26. Church JM, Raudkivi PJ, Hill GL. The surgical anatomy of the rectum—a review with particular relevance to the hazards of rectal mobilization. Int J Colorectal Dis. 1987;2:158–66.
27. Hobson KG, Ghaemmaghami V, Roe JP, Goodnight JE, Khatri VP. Tumors of the retrorectal space. Dis Colon Rectum. 2005;48(10):1964–74.
28. Sobrado CW, Mester M, Simonsen OS, Justo CR, deAbreu JN, Habr-Gama A. Retrorectal tumors complicating pregnancy: report of two cases. Dis Colon Rectum. 1996;39:1176–9.
29. Ludwig KA, Kalady MF. Trans-sacral approaches for presacral cyst/rectal tumor. Operative Tech Gen Surg. 2005;7(3):126–36.
30. Gunkova P, Martinek L, Dostalik J, Gunka I, Vavra P, Mazur M. Laparoscopic approach to retrorectal cyst. World J Gastroenterol. 2008;14(42):6581–3.
31. Jones H, Cunningham C. Extending the indications: transanal endoscopic surgery for fistula, stricture, and rare tumors. Semin Colon Rectal Surg. 2015;26(1):45–8.
32. Zoller S, Joos A, Dinter D, Back W, Horisberger K, Post S, et al. Retrorectal tumors: excision by transanal endoscopic microsurgery. Revista espanola de enfermedades digestivas: organo oficial de la Sociedad Espanola de Patologia Digestiva. 2007;99(9):547–50.
33. Gutiérrez PC, Taghavi MK, Sosa DR, Salas AP, Ovejero VJ, Ruiz JL, Cabezas JMG, Setién AI. New surgical approach of retrorectal cystic hamartoma using transanal minimally invasive surgery (TAMIS). J Coloproc. 2014;34(4):260–4.
34. Buchs N, Taylor S, Roche B. The posterior approach for low retrorectal tumors in adults. Int J Colorectal Dis. 2007;22(4):381–5.
35. Canelles E, Roig JV, Cantos M, Armengol JG, Barreiro E, Villalba FL. Presacral tumors: analysis of 20 surgically treated patients. Cirugía Española (English Edition). 2009;85 (6):371–7.
36. Mathis KL, Dozois EJ, Grewal MS, Metzger P, Larson DW, Devine RM. Malignant risk and surgical outcomes of presacral tailgut cysts. Br J Surg. 2010;97(4):575–9.
37. Lin C, Jin K, Lan H, Teng L, Lin J, Chen W. Surgical management of retrorectal tumors: a retrospective study of a 9-year experience in a single institution. Onco Targets Ther. 2011;4:203–8.
38. Patsouras D, Pawa N, Osmani H, Phillips RK. Management of tailgut cysts in a tertiary referral centre: a 10-year experience. Colorectal Dis. 2015;17(8):724–9.
39. Sagar AJ, Tan WS, Codd R, Fong SS, Sagar PM. Surgical strategies in the management of recurrent retrorectal tumours. Tech Coloproctol. 2014;18(11):1023–7.

40. Dozois EJ, Malireddy KK, Bower TC, Stanson AW, Sim FH. Management of a retrorectal lipomatous hemangiopericytoma by preoperative vascular embolization and a multidisciplinary surgical team: report of a Case. Dis Colon Rectum. 2009;52(5):1017–20.
41. Pack GT, Miller TR. A plea for the synchronous combined abdominoperineal surgical approach for certain pelvic tumors. Surgery. 1965;57:613–4.
42. Wolpert A, Beer-Gabel M, Lifschitz O, Zbar PA. The management of presacral masses in the adult. Tech Coloproctol. 2014;6(1):43–9.
43. Miles RM, Stewart GS Jr. Sacrococcygeal teratomas in adult. Ann Surg. 1974;179(5):676–83.
44. Wang JY, Hsu CH, Changchien CR, Chen JS, Hsu KC, You YT, et al. Presacral tumor: a review of forty-five cases. Am Surg. 1995;61:310–5.
45. Mancini R, Cosimelli M, Filippini A, Tedesco M, Pugliese P, Marcellini M, Pietrangeli A, Lepiane P, Mascagni D, Cavaliere R, Di Matteo G. Nerve-sparing surgery in rectal cancer: feasibility and functional results. J Exp Clin Cancer Res. 2000;19(1):35–40.
46. Pescatori M, Brusciano L, Binda GA, Serventi A. A novel approach for perirectal tumours: the perianal intersphincteric excision. Int J Colorectal Dis. 2005;20:72–5.

York Mason Procedure

15

Ariane M. Abcarian and Herand Abcarian

The transsphincteric approach to the rectum was originally proposed by Aubrey York Mason for the surgical management of postoperative rectourethral fistulas (RUF) in 1969 [1]. Until that time most RUFs were approached through an anterior pubic splitting incision which was extremely complicated and morbid. He subsequently utilized this approach to gain access to and locally excise certain low rectal cancers [2]. Similar operations had been advocated by Arthur Dean Bavan in 1917 for small low rectal cancers "without any radial involvement [3]." As quoted by Corman "Bavan did not repair the sphincter", simply stating "I did not hope to attain anything like complete continence" and he did not comment about the risk of development of a fistula [4].

In 1970, Mason described the transsphincteric procedure as special access to the rectum and since then this operation has become popular and named the "York Mason procedure [5]." The surgical technique is described and illustrated in detail by Prasad and Abcarian in 1983 [6]. In brief, the patient receives full bowel preparation and preoperative antibiotic prophylaxis. The procedure can be performed under spinal or general anesthesia. A Foley catheter is inserted (mandatory for cases of RUF) and the patient is placed in prone jackknife position (Fig. 15.1). and the buttocks are taped apart (Fig. 15.2).

A 10 cm incision is made starting at the posterior anal verge and carried to the right or left of the coccyx (paracoccygeal) (Fig. 15.3).

A.M. Abcarian
Division of Colon and Rectal Surgery, John H. Stroger Hospital of Cook County, 1900 W. Polk Street, Chicago, ILL 60612, USA

H. Abcarian (✉)
Division of Colon and Rectal Surgery, University of Illinois at Chicago, 840 S. Wood Street, MC 958, Chicago, IL 60612, USA
e-mail: Abcarian@uic.edu

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_15

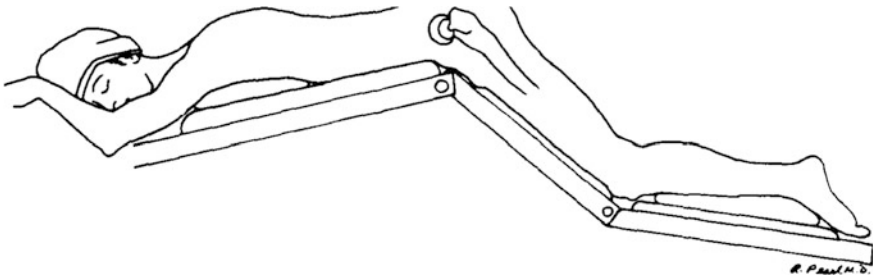


Fig. 15.1 Position of patient for repair of rectoprostatic urethral fistula

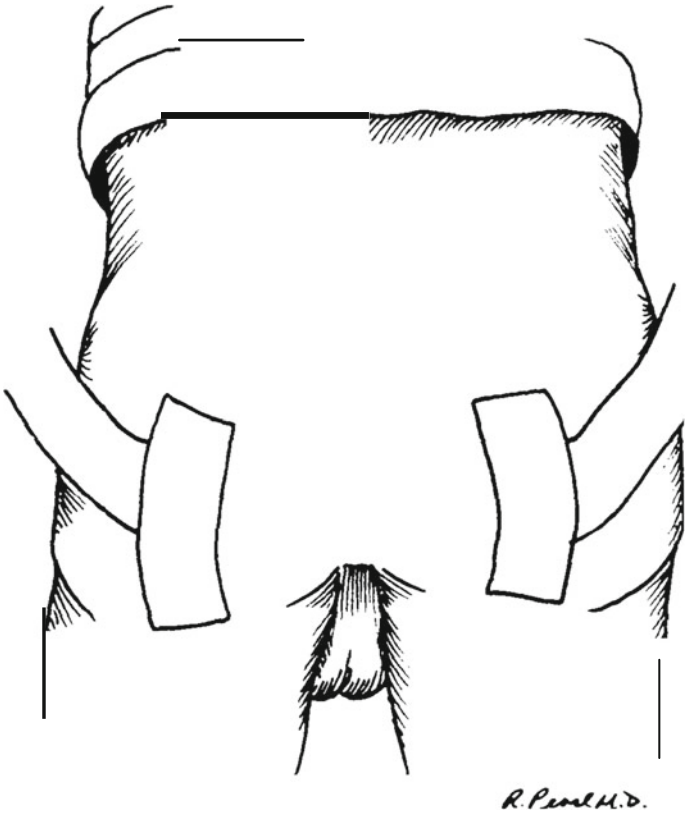


Fig. 15.2 Incision

The incision is deepened in subcutaneous fat to reach the lower border of the gluteus maximus. The fascia and lower fibers (3–4 cm) of this muscle is divided to achieve exposure to the retrorectal space. The external sphincters, levator ani, puberictalis muscle, and internal sphincter are sharply divided and marked by different colored paired sutures to facilitate identification of each muscle during

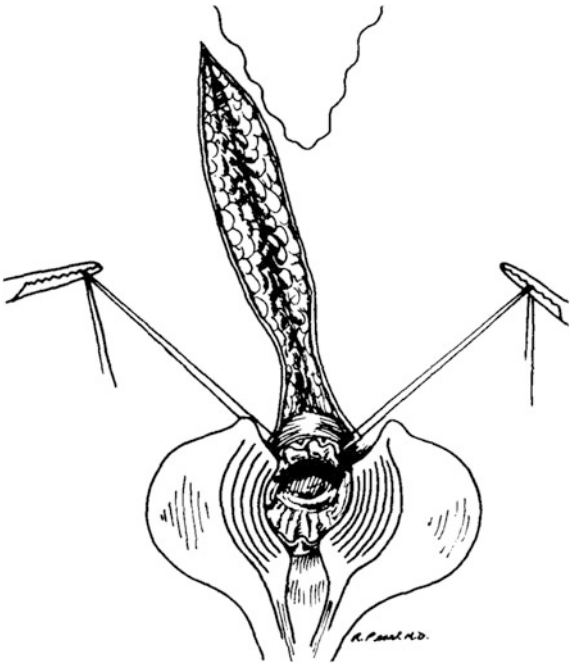


Fig. 15.3 Skin incision is made. Mucocutaneous junction is marked with sutures. Internal sphincter is exposed

closure. The posterior rectal wall is then incised longitudinally to open the rectum "like a book" and gain access to low and mid rectum (Fig. 15.4).

To repair the RUF, the fistula is cored out, the urethra repaired with 3/0 monofilament absorbable sutures over a silastic Foley catheter used as a stent. The rectal wall is mobilized 2–3 cm and repaired in vest over pants fashion using absorbable sutures (Figs. 15.5 and 15.6).

The rectal wall is closed and the individual layers of sphincter mechanism are identified using the colored paired sutures and approximated with absorbable sutures (Fig. 15.7).

The wound is irrigated and a suction drain is placed deep or superficial to the gluteus, and its fascia reapproximated. The subcutaneous tissue is irrigated and the skin closed with interrupted sutures after careful approximation of the anoderm and the anal verge [6, 7]. If the York Mason procedure is done to remove a large rectal villous adenoma, submucosal infiltration of dilute (1:200,000) epinephrine solution will elevate the lesion, assist in dissection and decrease bleeding.

The York Mason procedure was expanded in later years to include repair of suprasphincteric or extrasphincteric fistulas and approach to retrorectal (presacral) cyst. In case of an extrasphincteric or high suprasphincteric fistulas, after opening the rectum posteriorally the primary opening of the fistula is cored out the rectal wall closed with vest over pants technique and the external fistula tract is drained

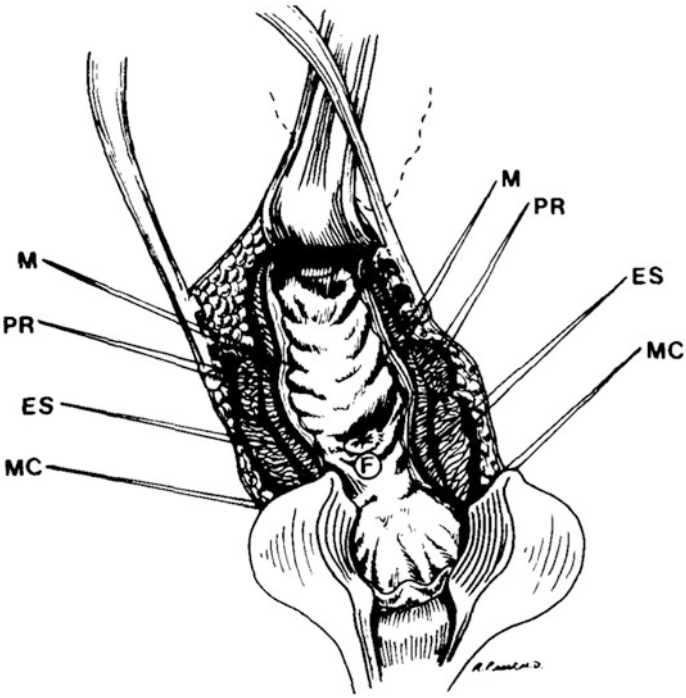


Fig. 15.4 Sphincter mechanism and posterior rectal wall divided exposing the fistula (*F*). Each sphincter muscle is tagged with color-coded sutures. (*M*) Mucosa. (*PR*) puborectalis, (*ES*) external sphincter, (*MC*) mucocutaneous junction

with a #12 or #14 mushroom or Mallicot catheter and the incision is closed. A modification of the York Mason procedure, without division of the sphincter mechanism has been used to gain access to retrorectal space for removal of presacral developmental cysts. In such cases, after division of fibers of gluteus maximus, the sphincter mechanism is retraced caudad and the presacral cyst is visualized. Infiltration of dilute epinephrine solution between the cyst and the rectal wall facilitated dissection and prevents injury to the posterior rectal wall. After placement of suction drain within the dead space, the wound is closed per perineum [8, 9].

Results of the York Mason procedure are difficult to assess due to the paucity of reports of large series in the literature. Mason reported recurrence rate of 13% in his original series of rectal cancers treated with this procedure [2]. Allogower and associates reported 36 patients treated for rectal cancer through sphincter splitting transsphincteric approach [10]. There were no operative deaths and nine recurrences (25%). He recommended frozen section examination of margins and depth of invasion in "superficial" cancers. Allogower and colleagues reported a larger series of parasacral approach to the rectum. These included 116 patients with various indications, with nearly 50% done for malignancies [11]. There is little information

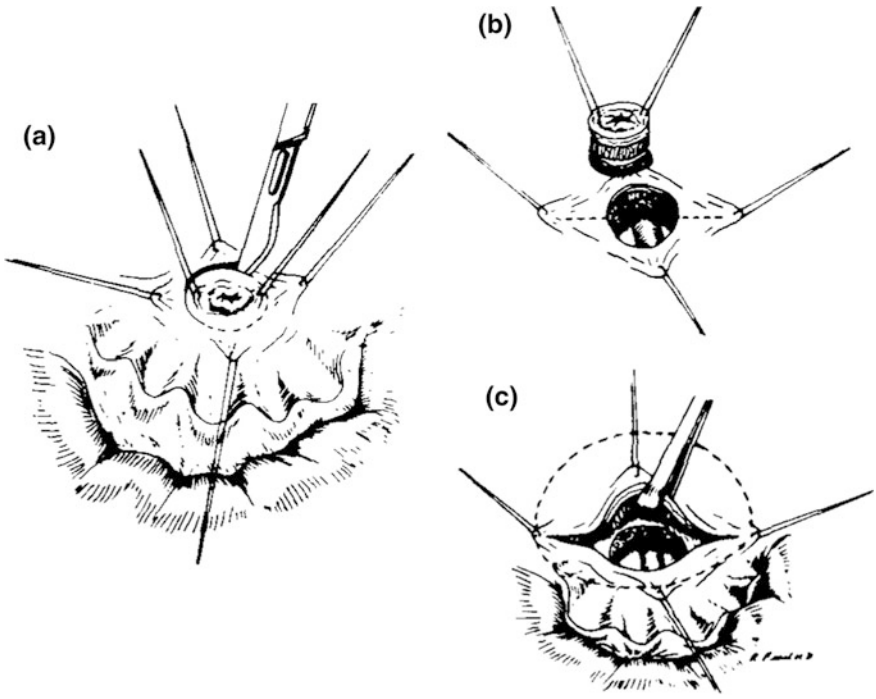


Fig. 15.5 Incision around fistula (**a**). Excised fistulous tract exposing catheter in prostatic urethra (**b**). Undermining of rectal wall. *Dotted line* represents the extent of rectal wall mobilization (**c**)

available on the outcomes of the patients. The same authors subsequently published on the anatomy of the pelvic floor for translevator-transsphincter operations [12].

Huber reported on 106 cases of sphincter splitting parasacral approach performed between 1974 and 1985. The procedure was done in deep lithotomy position and "very good results" were obtained when the technique was applied for benign rectal tumors (villous adenomas), fistulas and traumatic lesions [13]. Radical resection of the bowel wall could be accomplished and the prolapsed rectosigmoid could be resected and the lax pelvic floor tightened through this approach. He concluded that "transsphincteric" approach is a highly desirable technique in the treatment of high fistulas and traumatic lesions. "Severe complications are rare among accurate preliminaries and surgical skills [13]."

Arnaud and colleagues reported on 35 patients (20 ♂, 15 ♀) who had posterior transsphincter approach for villous adenoma, rectal prolapse, rectal stricture, or high fistula [14]. No complications were seen in 20 patients, but delayed fistula occurred in seven patients, four of whom healed spontaneously, and three needed colostomy and surgical repair. Pathology of villous tumors showed invasive malignant changes in three patients requiring proctectomy and end-to-end coloanal anastomosis. Two patients had mild incontinence and were treated with biofeedback. Two

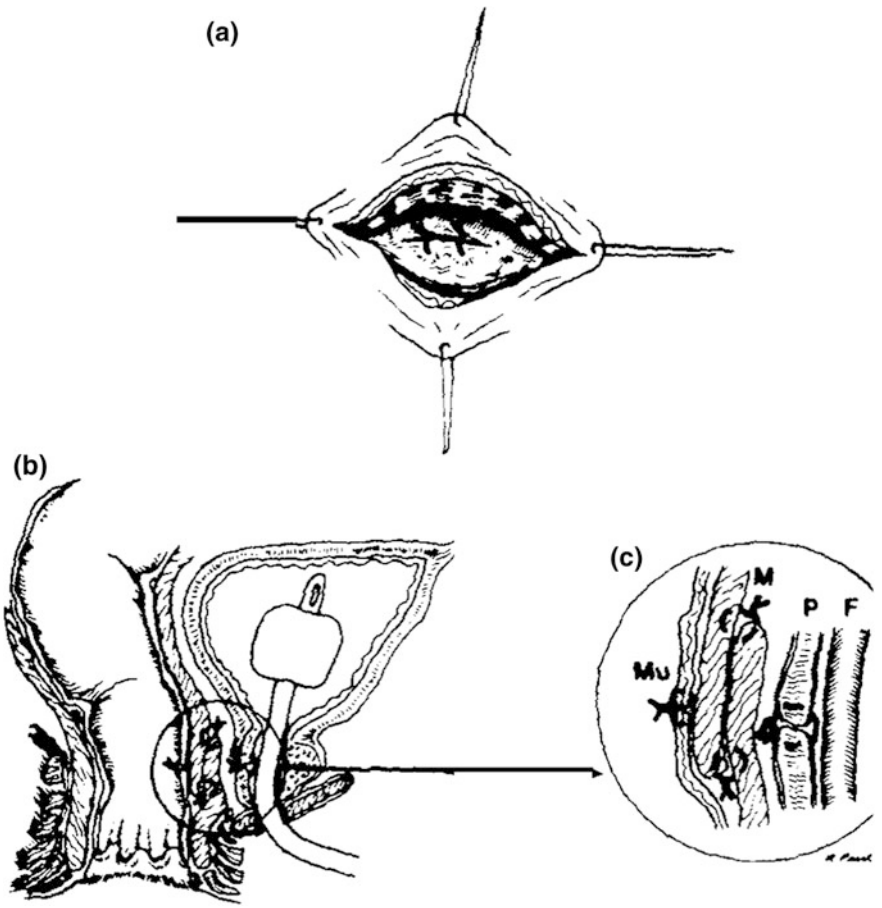


Fig. 15.6 *Closw·e* of prostatic urethra (**a**). Sagittal section showing sumre line after repair of fistula (**b**). Magnified view of suture lines. (*F*) Foley catheter, (*P*) prostatic urethra, (*M*) full-thickness rectal wall flaps sutured "vest over pants" technique. Note that the suture lines do not overlie each other (**c**)

patients developed sacrococcygeal hernia and delayed recto-perineal pain was reported in another two patients [14].

Recently, Qui and colleagues reported their experience of 102 patients with mid to low lying rectal neoplasms treated between 1990 and 2006, (40 ♂, 62 ♀ average age 55.5) [15]. Surgical indications were: rectal villous adenoma 36, early rectal cancer 43, advanced rectal cancer 10, and submucosal rectal wall neoplasm 13. Operating time was 75 min, blood loss average 60 ml, and hospital stay was 8 days. All 102 rectal neoplasms were resected completely with partial proctectomy in 96 and segmental proctectomy in 6 all with clear resected margins. Three patients (2.5%) had postoperative infection and 4 (3.9%) had fecal fistula. The authors used

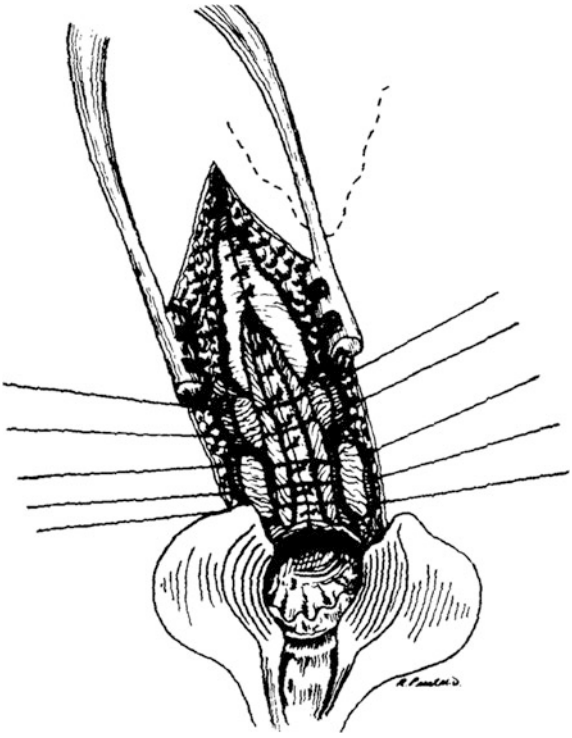


Fig. 15.7 Suture of rectal wall completed. Sphincter muscle being approximated

Williams incontinence score and reported 33 patients (32.4%) with postoperative incontinence to flatus (26) and liquid stool (7) within 1 week. Three months postoperatively 94 patients (92.2%) achieved grade 1 continence and only eight had occasional episodes of flatus incontinence (grade 2). There were no operative deaths and no incidence of rectal stricture. Three patients (2.9%) developed local recurrence during median follow-up of 76.8 months [15].

Poirier and Abcarian collected their experience of 28 patients (18 ♂; 10 ♀) operated in a 14 year period. Indications in decreasing frequency were rectal villous adenoma 36%, retrorectal cyst 29%, rectourethral fistula 21%, suprasphincteric fistula 7% and low coloanal/ileoanal anastomotic leak 7%. All patients underwent the standard York Mason procedure with the exception of those with retrorectal (presacral) cyst who had modified, (sphincter sparing) York Mason procedure. Twenty-seven of 28 patients completed the follow-up. Twenty-three patients had successful outcomes, four failed and one was lost to follow-up. A total of ten complication occurred in six patients. They conclude that morbidity occurred in 21% of the patients and success in 85% of completely followed patients. There were no deaths in this series [15].

Complications and Management

Wound Infections

Despite all precautions (oral antibiotics, laxative bowel preparation, perioperative antibiotics, and intraoperative rectal irrigation with dilute Betadine solution), would infections can never be totally eliminated. John Alexander Williams of Birmingham, UK is credited with the famous quote "the only way to sterilize the bowel is to remove it and boil it for 1 hour." Despite this, the wound infection quoted in various series is Smell 3/102 (3.5%) [15] and 4/28 (14%) [16]. Interestingly two of the four patients who developed wound infection in the latter series had excision of presacral cyst without opening the rectum (sphincter sparing modified York Mason procedure) [16].

This complication is usually treated with opening the wound and packing with wet to dry dressings. A short course of intravenous antibiotics may be indicated in febrile patients who have leukocytosis and most patients can be discharged to home care with oral antibiotics for 10–14 days.

Fecal Fistula

Usually follow an apparent wound infection and it is caused by disruption of the rectal wall closure in the classical York Mason procedure. In the sphincter sparing operation it is related to the injury and repair of the rectal wall during excision of infected presacral cysts especially after missed diagnosis and open or CT guided drainage. This is the dreaded complication which has discouraged many surgeons from attempting this approach. However, the incidence of this complication is quite low, 3.9% in 102 patients reported by Qui [15] and 7 patients (20%) reported by Around and colleges Four of whom healed spontaneously and the other three needed colostomy and surgical repairs [14]. In the report by Poirier, of the four patients with wound infections, two healed without any consequences, one needed two debridement in the operating room and healed. Only one patient (1/27) developed a rectocutaneous fistula together with sphincter defect. This patient had temporary fecal diversion, followed by overlapping repairs of the sphincter and closure of the rectal wall defect. Six weeks later when complete healing was documented, the colostomy was closed. The patient remained well and fully continent in later follow-up.

Bleeding

This operation can be bloody due to sharp division of various muscle layers needed to gain access to the rectum. Liberal use of 1:200,000 epinephrine solution aids in obtaining dry field and reduce bleeding. To prevent postoperative bleeding, the

wound must be thoroughly irrigated and hemostasis secured before proceeding to the next level of closure. A soft suction catheter may be used either deep to or superficial to the gluteus especially in cases of presacral cyst excision where a dead space is inevitably left behind. Most minor bleeding will stop within 2–3 days of continuous suction but if the patient passes clots from the rectum, return to the operating room, evacuation of the clots and inspection of the rectal wall closure for bleeding is mandatory.

Fecal Incontinence

Even though fecal incontinence remains a major concern due to the nature of this procedure, its actual incidence is quite small. A transient weakness in the sphincter in the immediate postoperative period is to be expected and most will resolve spontaneously (92.2%) in 3 months, [15] and 2/35 patients with post York Mason fecal incontinence in the Arnaund series were treated with biofeedback [14]. If major incontinence (to solid stool) persists, the patient should have an endoanal ultrasound to document the potential sphincter defect and be taken back to the operating room for overlapping sphincteroplasty. In the Poirier series one patient need overlapping sphincter repair with simultaneous rectal wall closure and remained continent afterwards [16].

Recurrences

The various indicators for York Mason procedure each require careful scrutiny of the pathologic specimen and close follow-up. Preoperative staging using ERUS and MRI are invaluable.

Recurrence following excision of rectal neoplasm In cases of rectal villous adenoma, pathologic report must exclude malignancy. If a T1 malignancy is identified, the patient must be offered radiation therapy, as was successfully used with excellent long-term outcome [16]. Adequacy of margins during surgery can be ascertained using frozen section examination of the specimen [10, 11]. None of the Poirier's patients had any evidence of recurrence in up to 74 months followup [16] In the Qui series 3 patients (2.9%) developed recurrence during the median follow-up of 76.8 months [15].

Recurrence following excision of retrorectal (presacral) cyst Complete excision of the lesion is imperative because of the potential for malignancy. Every effort should be made to excise these lesions in total and obtain pathologic confirmation. In such cases there should be no recurrence [16].

Recurrence after repair of rectourethral fistula is clearly dependent on the etiology of the fistula. In cases when the RUF results from prostatectomy, whether open or minimally invasive route, the closure is usually highly successful. On the other hand, if the tissues surrounding the fistula are significantly radiated, the closure is more apt to breakdown [16–18]. If the patient receives radiation therapy

but the RUF is caused by subsequent prostatectomy, the outcome is better than those in which the fistula occurs after external beam radiation or brachytherapy [19].

Recurrence after repair of high suprasphincteric/extrasphincteric is uncommon due to virgin territory for surgery in patients who have had multiple prior unsuccessful operations. The important point is to maintain the external drainage via mushroom or Mallicot catheter for up to 2 weeks allowing the rectal wall repair to heal before removing the catheter.

Recurrence after coloanal or ileoanal anastomotic repair can be minimized with attention to meticulous technical detail. Other techniques such as pouch advancement advocated by surgeons in Cleveland Clinic can be utilized for such recurrences or as an alternative to the York Mason approach.

Sacoccygeal hernia has been reported by Arnaud and colleagues and is anatomically related to disruption of the pelvic floor (levator ani) repair. This complication has not been reported in any other series but if it were to occur, documentation of the defect with pelvic CT or MRI is important. The defect can then be repaired using biologic or synthetic mesh.

Personal View Due to the complexity of the operation, there is no extensive body of literature reporting large patient series. Also in recent years, TEM, TAMIS, and other platforms have offered alternative interventions to York Mason procedure. However, the procedure is still ideal for rectourethral fistulas, extrasphincteric fistulas, and low anastomotic leaks. Also the sphincter sparing (modified York Mason) procedure offers excellent exposure and is ideal for excision of retrorectal (presacral) developmental cyst. The York Mason procedure is an excellent addendum to the armamentarium of a colorectal surgeon. It is associated with reasonably low complication, no significant long-term morbidity and no mortality. With appropriate of patient selection the success rate can be very high (85%).

References

1. Kilpatrick FR, Mason AY. Post-operative recto-prostatic fistula. Br J Urol. 1969;41(4):649–54.
2. Mason AY. The place for local resection in the treatment of rectal carcinoma. Proc R Soc Med. 1970;63(12):1259–62.
3. Bevan AD. Carcinoma of the rectum: treatment by local excision. Surg Clin North Am. 1917;1:1223–39.
4. Corman ML. Carcinoma of the rectum. In: Colon and Rectal Surgery. 6th ed. p. 963.
5. Mason AY. Surgical access to the rectum: a transsphincter exposure. Proc R Soc Med. 1970;63:91–4.
6. Prasad ML, Nelson R, Hambrick E, Abcarian H. York Mason procedure for repair of postoperative rectoprostatic urethral fistula. Dis Colon Rectum. 1983;26:716–20.
7. Wood TW, Middleton RG. Singe-stage transrectal transsphicteric (modified York-Mason) repair of rectourinary fistulas. Urol. 1990;35(1):27–30.
8. Abel ME, Nelson RL, Prasad ML, Abcarian H, et al. Parasacrococcygeal approach for the resection of retrorectal developmental cysts. Dis Colon Rectum. 1985;28:855–8.

9. Singer MA, Cintron JR, Schoetz DJ, Abcarian H, et al. Retrorectal cyst: rare tumor frequently misdiagnosed. J Am Coll Surg. 2003;196:880–6.
10. Allgöwer M. Sphincter-splitting approach to the rectum. Am J Surg. 1983;145(1):5–7.
11. Allgöwer M, Dürig M, van Hoschstetter A, Huber A. The parasacral sphincter-splitting approach to the rectum. World J Surg. 1982;6(5):539–48.
12. Huber A, von Hochstetter A, Allgöwer M. Anatomy of the pelvic floor for translevatoric-transsphincteric operations. Am Surg. 1987;53(5):247–53.
13. Huber A. Transsphincteric approach to the rectum. Ann Chir Gynaecol. 1986;75(2):106–10.
14. Arnaud A, Fretes IR, Joly A, Sarles JC. Posterior approach to the rectum for treatment of benign lesions. Int J Colorectal Dis. 1991;6(2):100–2.
15. Qui HZ, Guo-Le L, Xiao Y, Wu B. The use of posterior transsphincteric approach in surgery of the rectum: A Chinese 16 year experience. World J Surg. 2008;32(8):1976–82.
16. Poirier M, Abcarian H. Transsphincteric (York Mason) parasacrococcygeal approach: outcomes of an old procedure with expanded indications. Submitted.
17. Fengler SA, Abcarian H. The York Mason approach to repair of iatrogenic rectourinary fistulae. Amer J Surg. 1998;173:213–7.
18. Munoz M, Nelson H, Harrington J, et al. Management of acquired rectourinary fistulas: outcome according to cause. Dis Colon Rectum. 1998;41:1230–8.
19. Dal Moro F, Mancini M, Pinto F, et al. Successful repair of iatrogenic rectourinary fistulas using the posterior sagittal transrectal approach (York-Mason): 15 year experience. World J Surg 1006;30(1):107–113.

Pull-Through Procedures

16

Kristin Vercillo and Jennifer Blumetti

Introduction

Operations that include complete or distal proctectomy with restoration of gastrointestinal continuity at or below the level of the anorectal junction are referred to as pull-through procedures. These resections may include mucosectomy or intersphincteric resection of the proximal anal canal. Depending on the length of the patient's anal canal and the most distal extent of the resection, these anastomoses typically occur less than 5 cm from the anal verge.

Indications and types of pull-through procedures are shown in Table 16.1. The operations can be performed through a transabdominal or transanal approach, depending on the disease process, resection, and intended reconstruction. In adults, the most common pull-through procedures are coloanal and ileal pouch-anal anastomoses (IPAA), typically done for rectal cancer and inflammatory bowel disease, respectively. Perineal proctectomy is performed less commonly. In infants and young children, the transanal endorectal pull-through, introduced in the late 1990s, has become standard for the management of Hirschsprung's disease [1–3]. This one-stage procedure involving resection of the aganglionic segment and pull-through of the normal ganglionic colon has also been shown to be safe and effective in adolescents and adults diagnosed with Hirschsprung's disease as well [4]. Although the traditional transabdominal endorectal pull-through procedures, such as Swenson, Duhamel, and Soave, for Hirschsprung's disease are less

K. Vercillo
Colon & Rectal Surgery Resident, Stroger Hospital of Cook County, Chicago, USA

J. Blumetti (✉)
Colon & Rectal Surgery Residency Program, Stroger Hospital of Cook County, 1900 W. Polk St., Room 406, Chicago, IL 606012, USA
e-mail: jblumetti5@gmail.com

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_16

Table 16.1 Pull-through procedures and their indications

Procedure	Indications
Coloanal anastomosis Straight Colonic J pouch	Rectal cancer Large rectal polyp Rectovaginal fistula Rectourethral fistula Radiation proctitis Hirschsprung's disease (adult) Slow transit constipation with megarectum
Ileal pouch-anal anastomosis	Ulcerative colitis Familial adenomatous polyposis Crohn's disease (select cases) Hereditary colon cancer syndrome Congenital defects of colonic motility
Perineal proctectomy	Rectal prolapse
Transanal endorectal pull-through	Hirschsprung's disease (children and adult)
Transabdominal endorectal pull-through Swenson Duhamel Soave	Hirschsprung's disease (children) Severe/high imperforate anus (children)

common today, they remain the procedure of choice for high imperforate anus in children, and many adults will present to a colorectal surgeon with complications from these operations later on [5–8].

Standard techniques to fashion the pull-through include both hand-sewn and stapled anastomoses. Historically, a hand-sewn anastomosis was the standard practice. Hand-sewn anastomoses can be interrupted or running, single- or double-layered, and with a variety of absorbable and nonabsorbable sutures. Intersphincteric resection or mucosectomy require a hand-sewn technique [9, 10]. Procedures such as the perineal proctectomy for rectal prolapse and the transanal endorectal pull-through for Hirschsprung's disease are also typically hand-sewn, although stapled techniques have been described [11]. A recent Cochrane review [12], analyzed 1233 patients who underwent colorectal resections with colorectal or coloanal anastomoses and found no differences in all clinically relevant parameters, including anastomotic leak, both clinically and radiographically, between hand-sewn and stapled techniques.

The integrity of any anastomosis results from a complex interaction between the surgeon, the patient, and the disease process. Ultimately, any one factor or a combination of several may lead to a significant anastomotic complication after a pull-through procedure (Table 16.2).

Complications following pull-through procedures may be acute (bleeding, anastomotic disruption) or more insidious (chronic anastomotic sinus, stricture, prolapse, incontinence, emptying issues). To manage acute and chronic complications appropriately, the surgeon must take into account the clinical acuity and severity of the complication. A current awareness of nonoperative, as well as

Table 16.2 Factors influencing anastomotic complications following a pull-through procedure, (Adapted from [108])

Surgeon factors	
Intestinal blood supply Tension on the anastomosis Perioperative factors Hypoxia Resuscitation Hypothermia	Intraoperative factors Blood loss Blood transfusion Duration of surgery Choice of minimally invasive approach Manipulation of tissue
Patient factors	
Age Smoking Alcohol and illicit drug use Body mass index Visceral obesity Anesthesia severity assessment	Anesthesia severity assessment (ASA) Nutritional status Prior abdominal surgery Medications Antiplatelet therapy Systemic anticoagulation
Disease factors	
Inflammatory bowel disease Metastatic cancer Medications Steroids Immunomodulators and biologics	Radiation therapy Emergency surgery Extraperitoneal anastomosis

operative techniques, and their suitable indications to treat these complications is crucial to minimize risk to the patient and the integrity of the original anastomosis, while maintaining the best chance for gastrointestinal continuity.

Bleeding

Most bleeding after a gastrointestinal anastomosis is relatively minor and self-limited, and does not require intervention. Rarely, clinically significant hemorrhage from an anastomosis can occur, ranging from 0.3 to 3.5% [13–18]. Transfusion requirements are typically less than 5% [14]. In a study of 1389 stapled colorectal anastomoses, severe bleeding necessitating intervention occurred in only seven patients (0.5%) [13]. Six (85.7%) of these seven patients' bleeding resolved with nonoperative measures, including endoscopy. No patient developed an anastomotic leak. In another series, transfusion alone with observation was successful in 6 of 17 bleeding patients (43%) [14]. Diagnostic and therapeutic modalities for the bleeding patient may include observation, endoscopy, and transanal or abdominal reoperative surgery.

Gentle endoscopic evaluation can be attempted in the stable patient with anastomotic site bleeding. A simple endoscopic washout of the anastomotic site may be sufficient to stop the bleeding. Martinez-Serrano and colleagues [13] achieved

success in 5 of 6 patients (85.7%) with proctoscopy and washout with 2000–5000 ml of saline. All six patients presented with significant bleeding from the colorectal anastomosis within the first postoperative day. Another valid option is irrigation of the anastomosis with an enema of 1:200,000 epinephrine solution. This method controlled bleeding in 80% of cases of J pouch bleeding following ileal pouch-anal anastomosis in a series of over 1000 patients [15]. This method is preferred when there is generalized oozing from the anastomosis rather than a distinct bleeding point. Endoscopic submucosal injection of 10 ml of diluted epinephrine (1:200,000) in saline at a discrete bleeding site along the anastomosis can also be performed with good results [19].

The use of endoscopic hemoclips has been well described for both upper gastrointestinal pathology as well as colon diverticular bleeding [20, 21]. Endoscopic clipping can be an alternative treatment modality for anastomotic hemorrhage, although it has been described only in small case series [17, 18, 22]. One case report describes the successful use of an over-the-scope clip for severe bleeding from a gastroenteral anastomosis [22]. Over-the-scope clips have also been successfully utilized for anastomotic dehiscence in low colorectal anastomoses [23, 24]. These clips should be able to be applied in the case of bleeding from an anastomosis as well.

Endoscopic electrocoagulation using hot biopsy forceps has been utilized in the treatment of anastomotic bleeding, although care must be taken in the early postoperative period [18]. Cirocco and Golub [14] successfully applied endoscopic electrocoagulation in six patients with unremitting bleeding from a colorectal anastomosis. However, one patient did develop an anastomotic fistula following this technique. Lou et al. [17] reported the endoscopic management of anastomotic bleeding in six patients following low anterior resection for rectal cancer, four of which were successfully treated with electrocoagulation alone.

Most patients with anastomotic bleeding can be managed successfully with nonoperative therapies. Lian and colleagues [16] reported a 96% success rate in the setting of bleeding ileal pouch-anal anastomoses using cauterization, clips, or epinephrine injection. If nonoperative measures fail, then surgical intervention will be necessary. Transanal oversewing of the anastomosis is the ideal option for surgical control of bleeding in the setting of pull-through procedures. If hemostasis and a secure anastomosis cannot be maintained, then anastomotic revision with resection and re-stapling is an option [18]. In the setting of significant intra-abdominal bleeding, transabdominal exploration and hemostasis with resection of the anastomosis may be necessary. The surgeon should always consider the possibility that postoperative bleeding may be secondary to a disrupted suture or staple line of the coloanal or ileoanal anastomosis. If this separation is caught early before pelvic sepsis has supervened, it may be controlled with transanal placement of sutures to repair the defect [25].

Anastomotic Disruption

Anastomotic leak remains a major complication of intestinal surgery that increases postoperative morbidity, mortality, and resource utilization [26, 27]. Overall incidence varies widely in the literature, occurring in 3–23% of patients, with low colorectal and coloanal anastomoses posing the highest risk [28–30].

The presentation and severity of anastomotic leak following a pull-through procedure is diverse. Some patients present with hemodynamic instability and peritonitis, while others have a more insidious course. Management is guided by the patient's clinical picture and type of leak, with the goal being preservation of the anastomosis, if possible, and restoration of gastrointestinal continuity with good functional outcomes.

Operative Interventions

Hartmann's procedure is no longer considered the treatment of choice for anastomotic leak after a pull-through procedure, with the focus now on preservation of the anastomosis [31–33]. Although a Hartmann's procedure may still be required in the unstable patient with profound sepsis or ischemia [34], the likelihood that the patient will undergo subsequent reversal of the colostomy is less than 50% [35–37].

Many contemporary surgeons now advocate the use of a "divert and drain" approach for those patients requiring reoperation for a leaking extraperitoneal anastomosis [27, 33, 38–40]. This strategy involves proximal fecal diversion with loop ileostomy, if not already present, and pelvic drain placement without manipulation of the anastomosis. Healing rates with this technique have ranged from 54 to 100% [31, 41]. Further repair of the anastomosis is not typically required. This treatment modality results in a much higher likelihood of stoma reversal than resection [42]. Diversion and external drainage can be supplemented as needed with additional nonoperative interventions, which are described below.

Although a simple transanal suture repair of the anastomotic defect may seem appealing, this method is not well supported in the literature. The opposition to this technique is based on possible exacerbation of the problem by creating further ischemia of the disrupted segment [43]. However, single case reports have been described with either the standard transanal technique or transanal endoscopic microsurgery [44, 45].

With the increasing incidence of laparoscopic colorectal operations today, a laparoscopic approach to reoperation may be performed. 16 of 18 patients who required reoperation for anastomotic leak were managed laparoscopically with ileostomy and operative drainage in one study [32]. Eighty percent of those patients were able to undergo subsequent stoma reversal. Should reoperation be necessary for an anastomotic leak, the procedure must minimize manipulation of the anastomosis, which will limit morbidity and increase the chance of successful restoration of gastrointestinal continuity.

Nonoperative Interventions

Nonoperative interventions can be employed in the vast majority of patients with proximal fecal diversion, and in select patients without proximal diversion [31, 38, 39]. In the setting of a contained pelvic leak, treatment options include transanal or percutaneous drainage of the pelvic fluid collection along with antibiotics, and/or newer endoscopic therapies.

Transanal drainage through the anastomosis is a well-described technique in the management of low colorectal, coloanal, or ileoanal anastomotic leaks. A Foley catheter may be placed into the leaking anastomosis, secured, and subsequently irrigated every 6 h [46]. Over the next 1–2 weeks, the cavity ideally decreases in size and the catheter is removed. Sirois-Giguere et al. [47] reported their experience with 37 symptomatic anastomotic leaks following low anterior resection for rectal cancer. The majority of patients (58%) with diverting stomas were managed with transanal drainage alone, compared with 9% without a diverting stoma. In those patients, Malecot catheters or closed suction drains were placed across the anastomotic defect. No patients who underwent transanal drainage required a transabdominal intervention, although 50% required an additional local intervention. Of the treatment modalities applied, transanal drainage was associated with the highest rate of stoma closure (93%) [47].

With advances in interventional radiology, computer tomography guided percutaneous drainage is now a common approach to manage contained pelvic leaks [47, 48]. A transgluteal or transabdominal drain can be placed, depending on the location of the fluid collection. Judicious management of drainage catheters may improve clinical outcomes. Ideally, the catheter should be flushed several times a day to maintain patency. When comparing transanal and percutaneous drainage, one study found no difference in success rates between the two techniques in patients with ileoanal anastomoses [49]. However, in contrast to internal transanal drainage, external percutaneous drainage carries the risk of developing an enterocutaneous fistula, although this occurs rarely [50].

Endoscopic therapies allow for minimally invasive management of anastomotic defects, and may be used independently or in conjunction with the above drainage procedures. The application of endoclips may close a leaking anastomosis. Over-the-scope clips are preferred to standard clips, as standard clips have a low closure force and are limited in size [51]. Over-the-scope clips employ newer technology using a nitinol clip loaded at the tip of the endoscope (OTSC, Ovesco, endoscopy, Tubingen, Germany) [52]. The bowel wall is anchored with the device and then suctioned as the clip is released. These clips are larger with increased compression, allowing for more complete closure in the setting of an inflamed, fibrotic anastomosis.

In a series of 188 patients with gastrointestinal defects, of which 50 involved the colon and rectum, clinical success with OTSC placement was 92.7%. Twelve of 15

lower gastrointestinal tract leaks healed using OTSC [24]. A smaller series of 14 patients with colorectal anastomotic leaks showed healing in 86% after OTSC. Only two patients had a diverting stoma at the time of clip placement [23]. The OTSC system should be used in anastomotic defects less than 1.5 cm in size and the absence of a pelvic collection [23]. Percutaneous drains can be used as an adjunct to clip application in the setting of a pelvic abscess, and a diverting stoma is not required for successful treatment [52].

Endoscopic stenting across the anastomosis has also been used to treat colorectal anastomotic leaks. Covered metal, plastic, and biodegradable stents have all been used with 80–100% clinical success [30, 53–55]. They can be left in place for up to 50–60 days, and are removed once the anastomosis heals [30, 54]. However, this technique is not typically useful following pull-through operations, as the distal end of the stent must be 5 cm or more from the anal verge [54].

The latest endoscopic technique to manage a colorectal or coloanal anastomotic leak is a corollary to the application of negative pressure wound vacuum devices for subcutaneous wound closure. The endosponge is a small vacuum device placed endoscopically into a defect or cavity. Weidenhagen et al. [56] pioneered this method, which utilizes an open pored polyurethane sponge (B Braun Medical BV, Melsungen, Germany), with an attached evacuation tube that is connected to a vacuum drainage system. The sponge is placed via an introducer sleeve that is fitted over an endoscope and placed through the anastomotic defect into the pelvic cavity. The sponge is exchanged every 48–72 h, downsizing the sponge as the cavity size decreases [56, 57]. The initial series consisted of 29 patients who underwent endosponge therapy over a median of 34 days. The endosponge was discontinued when the cavity was less than 1 cm in size. Overall, 28 patients (96.6%) healed the anastomosis [56].

As transanal and percutaneous drainage may need to be coupled with an endoscopic technique, combinations of different endoscopic therapies may lead to successful healing of the anastomotic leak [55, 57]. If one endoscopic modality fails, additional treatment with another technique is an option. Chopra proposed an algorithm for endoscopic closure of anastomotic defects [53]. For those patients with a defect greater than 2 cm, diverting ileostomy with endosponge therapy is preferred. Treatment of choice for defects less than 2 cm in the mid-rectum is endoscopic stenting with or without percutaneous drainage of the collection. Fibrin sealant is preferred for small defects less than 3 ml without abscess. For those with an abscess only, percutaneous drainage is preferred. Using this algorithm, 77% of patients had restoration of bowel continuity compared to 57% of surgically managed patients (Hartmann's procedure or diverting ileostomy alone) [53].

Proponents of early intervention and closure of the leaking anastomosis, such as those described above, believe that the function of the neorectum will be improved by earlier healing and less fibrosis. This approach prevents a persistent anastomotic sinus, and also leads to increased stoma closure rates [2, 4, 5].

Chronic, Non-healing Cavity

Despite control of leak-associated sepsis with transanal or percutaneous drainage of the fluid collection, there are still some patients whose anastomoses will not heal or will develop a chronic sinus. These chronic tracts and/or cavities have been shown to occur in up to 36% of anastomotic leaks [58]. Broder and colleagues [59] recommend a contrast study prior to removal of a drain to evaluate for persistent leak. Some patients, up to 8%, are asymptomatic and the sinus is found on contrast enema prior to diverting ileostomy closure [31, 39]. For those patients with a diverting stoma in place, a "watch and wait" approach can be used to manage these sinuses. Some of these chronic sinuses will heal with time. However, the sequelae of scarring and fibrosis may lead to impaired functional outcomes [60], resulting in permanent stoma for many patients [58]. Up to 63% of patients with chronic anastomotic sinuses will require multiple interventions [48, 58].

If the "watch and wait" approach is not successful, additional techniques may be attempted to salvage the anastomosis. A transanal advancement flap may be used to close the sinus. The technique of endorectal flap advancement is well described in the treatment of ileoanal anastomotic sinuses [61, 62]. In a small series of patients with persistent leaks after surgery for rectal cancer, four patients underwent delayed repair using an advancement flap [45]. Three endorectal flaps and one dermal flap were utilized after the sinus opening was excised. 50% had successful local treatment and underwent subsequent ileostomy reversal.

Marsupialization of the anastomotic sinus can be effective in the setting of a large residual cavity. A common lumen is created using an endoscopic stapler, electrocautery, or laparoscopic electrocautery scissors to incorporate the sinus into the bowel itself [63, 64]. This procedure results in epithelialization of the cavity, and the diverting stoma can then be reversed [63]. This technique has been utilized successfully in coloanal and ileal pouch anastomoses. Fibrin glue injection can also be effective in the treatment of chronic presacral sinuses, although only effective for diminutive, narrow tracts [65].

Reconstruction

If the above methods fail to resolve the leak despite diversion, or if an operative excision of the anastomosis was already urgently necessary, then a new reconstruction is the final treatment option to restore gastrointestinal continuity. Patients should be counseled extensively on the risks of reoperation including the possibility of permanent stoma. Most patients with coloanal anastomoses have already undergone extensive splenic flexure mobilization to allow the proximal colon to reach the pelvic floor without tension during their initial operations. After excision of the leaking anastomosis, the remaining proximal bowel is unlikely to reach to the pelvic floor without tension. Therefore, those with a failed coloanal anastomosis

who require excision may face a completion colectomy with an ileoanal anastomosis.

Alternatively, a salvage technique for the colorectal or coloanal anastomosis is the Deloyers procedure. The proximal colon is completely mobilized and rotated, while preserving the ileocolic junction and the ileocolic artery. An anastomosis is then created between the right or proximal transverse colon and the rectum or anus (Fig. 16.1). In one series from 1998 to 2011, Manceau et al. [66] performed this technique on 48 patients, 11 of which had previous failed colorectal or coloanal anastomoses. Results were excellent. No patients developed anastomotic leakage, and more than 80% of patients had good functional results with fewer than four bowel movements per day. As confirmed by others [67–69], the Deloyers procedure represents a safe and valid alternative to total colectomy with ileorectal anastomosis.

In a subsequent series of 50 patients who underwent redo surgery specifically after failed colorectal or coloanal anastomoses, all patients were able to have a successful reanastomosis. The authors note that this may require full mobilization of the remaining colon, with ligation of the middle colic vessels and a right colon to

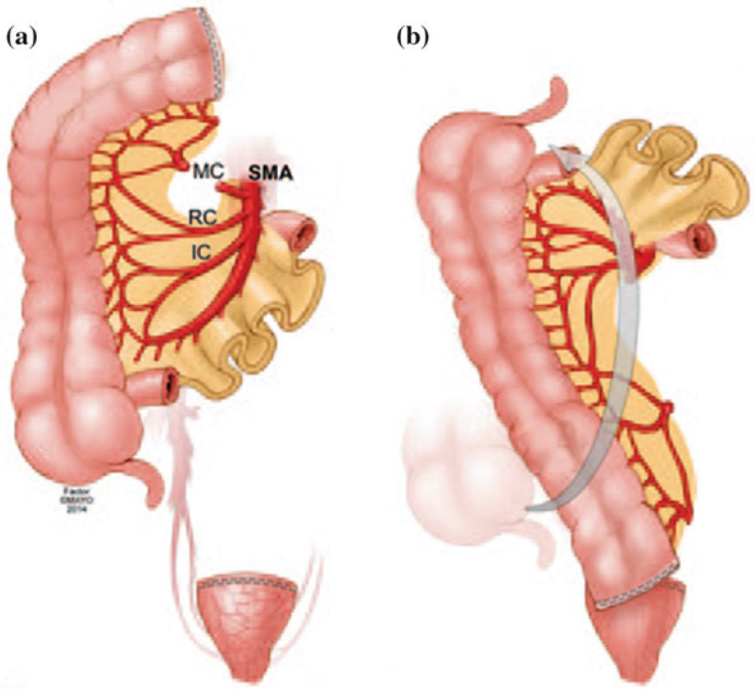


Fig. 16.1 The Deloyers Procedure (Adapted from [109]). **a** The proximal colon is available for a colorectal or coloanal anastomosis, but without mobilization will not reach the pelvis. **b** After mobilization of the *right colon* and preservation of the ileocolic pedicle, the colon is rotated 180° to place the cecum in the *right upper* quadrant and the proximal transverse colon into the pelvis for a tension-free anastomosis

rectal or anal anastomosis (Deloyers procedure) in order to create a tension-free anastomosis [70].

In summary, an early, expeditious diagnosis and treatment of anastomotic disruption may allow for local, less invasive methods to adequately treat the leak, ultimately preventing long-term anastomotic failure. Resection and reanastomosis should be considered the treatment of last resort for a persistent extraperitoneal anastomotic leak or chronic sinus.

Anastomotic Stricture

Anastomotic strictures usually occur in the setting of pelvic sepsis, but may also develop from anastomotic tension, ischemia, or Crohn's disease. Symptoms range from mild difficulty evacuating to near complete obstruction. A recent large study of 2361 consecutive colorectal anastomoses over 17 years revealed a 3.2% incidence of symptomatic stricture. Ileal pouch-anal strictures, on the other hand, are more common (10–40%) [15, 71–75]. Surgical technique may lead to differences in the type and length of the stricture. Stapled IPAA are typically associated with shorter, web-like strictures, whereas mucosectomy with hand-sewn anastomoses produce longer, more fibrotic strictures [74]. Successful transanal drainage to treat a colorectal anastomotic leak is associated with future stricture in up to 33% [47].

For those patients who have proximal fecal diversion, evaluation of the anastomosis prior to ileostomy reversal with a contrast enema study is crucial to assess for subclinical persistent leak or stricture. Endoscopy should be considered as well; it is not only diagnostic, but also potentially therapeutic.

In the setting of ileal pouch-anal reconstruction for presumed ulcerative colitis, an anastomotic stricture should raise suspicion for rectal cuff inflammation secondary to colitis or undiagnosed Crohn's disease, particularly in the setting of adjacent pouchitis. If Crohn's disease is diagnosed, strictures may respond to medical therapy with immunomodulators or biologics. Additionally, patients with a history of ulcerative colitis suffering from "cuffitis" as a component of their stricture will often respond to steroid or mesalamine enemas [76].

In general, most strictures respond well to nonoperative therapy. For the low distal anastomoses that accompany a pull-through procedure, the ease of access to the stricture site makes digital exam and the use of Hegar dilators relatively simple and often successful, either at home or under general anesthesia. Were and colleagues [77] reported 21 of 256 (8.2%) consecutive patients who underwent low anterior resection and developed an anastomotic stricture. Stricture symptoms presented after a mean of 7.7 months. This group utilized endoscopic Savary dilators, with bougies of increasing diameter (10–19 mm), over a series of sessions. Of 15 patients available for follow-up, ten achieved normal defecation with complete resolution of symptoms. Five patients had only partial improvement in symptoms, with three requiring reintervention. No complications occurred. A normal defecation pattern was never regained if more than three dilations were necessary [77].

Dilation can also be performed with the aid of endoscopic pneumatic balloons with high success rates (80–97%) [78–81]. Arauko and Costa [78] used pneumatic balloon dilation in 24 symptomatic patients with benign colorectal anastomotic stricture using a through-the-scope balloon technique. Dilation was successful in 22 (91.7%) patients, with a mean number of 2.3 treatment sessions. No complications occurred. A larger study over 17 years revealed a 97.4% success rate with endoscopic balloon dilation in 76 patients with a symptomatic colorectal anastomotic stenosis [81].

Successful pneumatic balloon dilation of ileal pouch inlet and outlet strictures has been reported as well [79, 80]. A large series of 150 patients with IPAA and stricture were followed after endoscopic balloon dilation [79]. A total of 646 strictures were identified and endoscopically dilated over 406 pouchoscopies from 2002 to 2010. Technical success of dilation was achieved in over 87%, with 80% having symptom improvement over a mean of 9.6 years. Major complications were low, with two perforations (0.46%) and four bleeds requiring transfusion (0.98%). Overall, balloon dilation was demonstrated to be reasonably safe in this patient population, although pouches with multiple strictures or acute angulations were technically more challenging [79].

Similar to the management of anastomotic leak, endoscopic stents may be used in the treatment of strictures, but have limited utility as pull-through anastomoses are too distal to allow placement of a stent. Other reported options that have proved successful include the combination of electroincision (radial incisions of the scar) with pneumatic balloon dilation [82], and dilation with concomitant corticosteroid injection [83].

If nonoperative treatments fail, or if the stricture is severe, surgical approaches such as mucosal or dermal advancement flaps should be considered if technically feasible. Ileal mucosal advancement flaps have been advocated for short pouch strictures that appear as a fibrous ring [84]. Further details on specific procedures or indications for surgery for anastomotic stricture can be found in the chapter on treatment of Anal Stenosis.

Prolapse

Patients may develop recurrent rectal prolapse following perineal proctectomy, or de novo prolapse as a complication of a coloanal or ileoanal anastomosis. Prolapse of the ileoanal pouch, either mucosal or full thickness, is uncommon. Joyce et al. [85] reported an incidence of 0.3% in 3176 patients who underwent ileal pouch surgery at a large tertiary referral center. Full-thickness prolapse was more common (63.6%) than mucosal prolapse (36.4%). In contrast to primary rectal prolapse, there was no female predominance [85]. Most pouch prolapses occur within two years of the original procedure [86].

Patients with pouch prolapse present with a sense of obstructed defecation, seepage, pain, and external prolapse of tissue [25]. If prolapse is suspected, asking the patient to sit on the toilet and strain may assist with diagnosis. The first line of

treatment for minor mucosal prolapse is stool bulking agents and biofeedback therapy to avoid excessive straining. If this fails, then the surgeon should attempt a local perineal procedure analogous to the Delorme's procedure, in the form of pouch advancement with excision of the redundant mucosal tissue.

Full-thickness pouch prolapse requires definitive surgery and is associated with a risk of pouch loss [85]. Surgical options include transanal repair, abdominal pouchopexy, and transabdominal revision. The abdominal pouchopexy can be supplanted with a biologic mesh sutured posterior to the pouch with subsequent fixation of the mesh to the sacrum [85]. Patients who fail definitive pouch prolapse surgery may elect to undergo ileostomy placement with or without pouch excision. Similar to an ileoanal anastomosis, a coloanal anastomosis may develop a symptomatic prolapse and the above algorithm should be employed for this situation as well.

Recurrent rectal prolapse following perineal proctectomy ranges from 0 to 10% with a follow-up of 6 months to 5 years; series with longer follow-up reveal higher recurrence rates, ranging from 16 to 18% [87–95]. The median time to recurrence ranges from 14 to 24 months [96, 97]. In this setting, repeat perineal proctectomy can be safely performed. In one series of 10 patients, procedures performed for recurrent rectal prolapse after perineal proctectomy included five repeat perineal proctectomies and levatoroplasty, three transabdominal sacral rectopexies, one anterior resection with rectopexy, and one anal encirclement. Average follow-up was 50 months, with no full-thickness recurrences during this period [97].

When selecting an operation for recurrent rectal prolapse, the surgeon must consider the initial operation. Unless the previous anastomosis is resected during the second operation, resectional procedures should be avoided. Abdominal rectopexy with sigmoid colectomy after perineal proctectomy can result in ischemia of the retained rectal segment, leading to mucosal slough, stricture, necrosis, and anastomotic dehiscence. Given that recurrence after perineal proctectomy will invariably prolapse the previous anastomotic line, no ischemic segment will be present if a repeat perineal proctectomy is performed.

In the setting of recurrent full-thickness rectal prolapse following a pull-through perineal proctectomy, redo perineal proctectomy or abdominal rectopexy without resection should be considered for complete full-thickness prolapse. In the setting of a recurrence with mild mucosal/submucosal prolapse, a Delorme's procedure should suffice.

Long-Term Results for Continence and Emptying in Children

Following pull-through procedures for Hirschsprung's disease, a large percentage of patients suffer from impaired continence or constipation. The key factors during the initial procedure to best prevent incontinence are preservation of the dentate line and not overstretching the sphincters. In the absence of the rectal reservoir inherent to a pull-through, high amplitude contractions propagate down to the coloanal

anastomosis, which may overcome insufficient anal sphincter pressure and lead to soiling. Inadvertent surgical removal of the transitional epithelium, which is responsible for anal sensation and anal sampling reflexes, often leads to fecal incontinence due to abnormal anal sensation. This complication highlights the importance of performing the coloanal anastomosis above the dentate line so that the transitional epithelium is not damaged. As the Soave procedure involves a submucosal dissection near the dentate line, these patients may suffer from fecal incontinence more often than other procedures for Hirschsprung's disease [98].

When fecal incontinence has been independently studied, the incidence exceeds 50% during childhood following these pull-through procedures [5–8, 99–101]. However, a recent study from Romero et al. [102] evaluating incontinence in children after the one-stage transanal pull-through quotes a much lower incidence of 5.2% with incontinence at 5 years following surgery. Although most patients suffer from soiling rather than frank incontinence, even mild fecal soiling can lead to troubling social problems [36].

Constipation with difficulty in emptying may also occur following a pull-through for Hirschsprung's disease. During childhood, the incidence of constipation in recent studies at 5-year follow-up is 21.2–30.2% [99, 102]. Long-term follow-up from the newer one-stage transanal pull-through demonstrates no statistically significant difference between the transanal and abdominal procedures with respect to long-term incontinence or constipation in children [99, 103].

Constipation is more common in patients in whom the aganglionic segment was resected but a dilated portion of colon was used to create the anastomosis [104]. Most cases of constipation can be avoided by resecting not only the ganglionic segment but also the dilated portion of colon. Another anatomic factor is a large, dilated, aganglionic Duhamel pouch, which can compress the ganglionic pull-through and cause obstructive symptoms [105]. Anastomotic stricture may be the sole cause.

In contrast to Hirschsprung's disease, those patients operated on for high imperforate anus do not typically suffer from constipation following a pull-through procedure, but do exhibit a high incidence of incontinence and soiling. Hassink et al. [106] reported long-term follow-up on a series of 58 patients at a median age of 26 years who underwent surgical repair of high imperforate anus as an infant. 7 patients (12.1%) had a permanent ileostomy or colostomy due to severe incontinence. Of the other 51 patients, 78.4% suffered from soiling; only 11 patients (21.6%) exhibited no soiling. Although soiling interferes with quality of life, 35 patients (69%) reported only occasional soiling. The use of the full-thickness terminal rectal wall to perform the pull-through procedure has the best fecal continence results in those with high imperforate anus [107].

To evaluate these complications in patients, anal manometry should be performed, which commonly reveals high or low resting pressure reflecting internal sphincter dysfunction. Prior to corrective surgery, manometry on a Hirschsprung's patient reveals an absent anorectal inhibitory reflex (RAIR), in which the internal sphincter does not relax with rectal distension. An absent RAIR is also a common finding on manometry after the pull-through procedure, which reiterates the

persistent sphincter dysfunction postoperatively. Endoanal ultrasound can also be performed to assess the nature of the sphincter muscles, including any defects. For those with constipation, the surgeon must distinguish between an anatomic (stricture, obstructing cuff, kink/twist of pull-through) or functional (aganglionic pull-through, transition zone pull-through) problem.

For the patient with incontinence, treatment depends on whether or not the sphincter muscles are intact. If the sphincters are intact, then a constipating diet and/or loperamide should be considered for those with hypermotility, while laxatives can be used with hypomotility. If the sphincters are not intact, then enemas can be used, and potentially supplanted with a constipating diet and loperamide for components of hypermotility. Severe fecal incontinence impairing quality of life may result in a permanent ostomy.

In a child with a primary symptom of constipation, laxatives or enemas can be attempted, which are primarily used in functional constipation without an anatomic mechanical obstruction. In the setting of an anatomic problem or severe functional constipation, reoperation with corrective surgery is an option. The most common indications for reoperations for Hirschsprung's disease are stricture (38.7%), megarectal pouch post-Duhamel (29.3%), and aganglionosis (20%) [104]. Distal anastomotic strictures may be managed with mucosal or dermal advancement flaps, as for any pull-through anastomosis complicated by stricture. Resection of a large Duhamel pouch, long stricture, or aganglionic segment with reanastomosis may be required. Symptoms may also be controlled with a diverting ostomy prior to definitive corrective surgery.

Conclusion

Pull-through procedures share the common goal of restoring gastrointestinal continuity following a complete or distal proctectomy down to or below the level of the anorectal junction (pelvic floor). Although these operations may result in a myriad of complications, there are several nonoperative and operative management strategies to not only treat the acute complication but also achieve an acceptable functional result without a permanent stoma.

References

1. Somme S, Langer JC. Primary vs staged pull-through for the treatment of Hirschsprung disease. Semin Pediatr Surg. 2004;13:249–55.
2. Elhalaby EA, Hashish A, Elbarbary MM, et al. Transanal one-stage endorectal pull-through for Hirschsprung's disease: a multicenter study. J Pediatr Surg. 2004;39:345–51.
3. Zhang SC, Bai YZ, Wang W, Wang WL. Clinical outcome in children after transanal 1-stage endorectal pull-through operation for Hirschsprung disease. J Pediatr Surg. 2005;40:1307–11.
4. Ammar SA, Ibrahim IA. One-stage transanal endorectal pull-through for treatment of Hirschsprung's disease in adolescents and adults. J Gastrointest Surg. 2011;15:2246–50.

5. Reding R, de Ville de Goyet J, Gosseye S. Hirschsprung's disease: a 20-year experience. J Pediatr Surg. 1997;32:1221–5.
6. Bai Y, Chen H, Hao J, et al. Long-term outcome and quality of life after the Swenson procedure for Hirschsprung's disease. J Pediatr Surg. 2002;37:639–42.
7. Baillie CT, Kenny SE, Rintala RJ, et al. Long-term outcome and colonic motility after Duhamel procedure for Hirschsprung's disease. J Pediatr Surg. 1999;34:325–9.
8. Yanchar NL, Soucy P. Long-term outcome after Hirschsprung's disease: patient's perspectives. J Pediatr Surg. 1999;34:1152–60.
9. Nivatvongs S. Ulcerative colitis. Principles and practice of surgery for the colon, rectum, and anus, 3rd ed. New York: Informa Health Care, 2007.
10. Wexner SD, Stollman N. Ulcerative colitis. Disease of the Colon. Boca Raton: CRC Press; 2007.
11. Hedlund H. Colorectal resection and anal anastomosis with an intraluminal stapler in Hirschsprung's disease. Pediatr Surg Int. 1997;12:142–4.
12. Neutzling CB, Lucosta SA, Proenca IM, et al. Stapled versus hand-sewn methods for colorectal anastomosis surgery. Cochrane Database Syst Rev. 2012;2:CD003144.
13. Martinez-Serrano MA, Pares D, Pera M, et al. Management of lower gastrointestinal bleeding after colorectal resection and stapled anastomosis. Tech Coloproctol. 2009;13:49–53.
14. Cirocca WC, Golub RW. Endoscopic treatment of postoperative hemorrhage from a stapled colorectal anastomosis. Am Surg. 1995;61:460–3.
15. Fazio VW, Ziv Y, Church JM, et al. Ileal-pouch anal anastomoses complications and function in 1005 patients. Ann Surg. 1995;222:120–7.
16. Lian L, Serclova Z, Fazio VW, et al. Clinical features and management of postoperative pouch bleeding after ileal pouch-anal anastomosis (IPAA). J Gastrointest Surg. 2008;12:1991–4.
17. Lou Z, Zhang W, Yu E, et al. Colonoscopy is the first choice for early postoperative rectal anastomotic bleeding. World J Surg Oncol. 2014;12:376.
18. Malik AH, East JE, Buchanan GN, Kennedy RH. Endoscopic haemostasis of staple-line hemorrhage following colorectal resection. Colorectal Dis. 2008;10:616–8.
19. Perez RO, Sousa A Jr, Bresciani C, et al. Endoscopic management of postoperative stapled colorectal anastomosis hemorrhage. Tech Coloproctol. 2007;11:64–6.
20. Baron TH, Norton ID, Herman L. Endoscopic hemoclip placement for post-sphincterotomy bleeding. Gastrointest Endosc. 2000;52:662.
21. Prakash C, Chokshi H, Walden DT, et al. Endoscopic hemostasis in acute diverticular bleeding. Endoscopy. 1999;31:460–3.
22. Tontini GE, Naegel A, Albrecht H, et al. Successful over-the-scope clip (OTSC) treatment for severe bleeding due to anastomotic dehiscence. Endoscopy 2013;45 Suppl 2 UCTN: E343–4.
23. Arezzo A, Verra M, Reddavid R, et al. Efficacy of over-the-scope clip (OTSC) for treatment of colorectal postsurgical leaks and fistulas. Surg Endosc. 2012;26:3330–3.
24. Haito-Chavez Y, Law JK, Kratt T, et al. International multicenter experience with an over-the-scope clipping device for endoscopic management of GI defects (with video). Gastrointest Endosc. 2014;80:610–22.
25. Sagar PM, Pemberton JH. Intraoperative, postoperative, and reoperative problems with ileoanal pouches. Br J Surg. 2012;99:454–68.
26. Hammon J, Lim S, Wan Y, et al. The burden of gastrointestinal anastomotic leak: an evaluation of clinical and economic outcomes. J Gastrointest Surg. 2014;18:1176–85.
27. Midura EF, Hanseman D, Davis BR, et al. Risk factors and consequences of anastomotic leak after colectomy: a national analysis. Dis Colon Rectum. 2015;58(3):333–8.
28. Slieker JC, Daams F, Mulder IM, et al. Systematic review of the technique of colorectal anastomosis. JAMA Surg. 2013;148(2):190–201.

29. Rahbari NN, Weitz J, Hohenberger W, et al. Definition and grading of anastomotic leakage following anterior resection of the rectum: a proposal by the International Study Group of Rectal Cancer. Surgery. 2010;147:339–51.
30. Cooper CJ, Morales A, Othman MO. Outcomes of the use of fully covered esophageal self-expandable stent in the management of colorectal anastomotic strictures and leaks. Diagn Ther Endosc. 2014;2014:187541.
31. Blumetti J, Chaudhry V, Cintron JR, et al. Management of anastomotic leak: lessons learned from a large colon and rectal surgery training program. World J Surg. 2014;38:985–91.
32. Joh YG, Kim SH, Hahn KY, et al. Anastomotic leakage after laparoscopic proctectomy can be managed by a minimally invasive approach. Dis Colon Rectum. 2009;52:91–6.
33. Hedrick RL, Sawyer RG, Foley EF, et al. Anastomotic leak and the loop ileostomy: friend or foe? Dis Colon Rectum. 2006;49:1167–76.
34. Phitayakorn R, Delaney CP, Reylonds HL, et al. Standardized algorithms for management of anastomotic leaks and related abdominal and pelvic abscesses after colorectal surgery. World J Surg. 2008;32:1147–56.
35. Lindgren R, Hallbook O, Rutegard J, et al. What is the risk of permanent stoma after low anterior resection of the rectum for cancer? A six-year follow up study of a multicenter trial. Dis Colon Rectum. 2011;54:41–7.
36. Khan AA, Wheeler JM, Cunningham C, et al. The management and outcomes of anastomotic leaks in colorectal surgery. Colorectal Dis. 2008;10:587–92.
37. Mala T, Nesbakken A. Morbidity related to the use of a protective stoma in anterior resection for rectal cancer. Colorectal Dis. 2008;10:785–8.
38. Thornton M, Joshi H, Vimalachandran C, et al. Management and outcome of colorectal anastomotic leaks. Int J Colorectal Dis. 2011;26:313–20.
39. Lim M, Akhtar S, Sasapu K, et al. Clinical and subclinical leaks after low colorectal anastomosis: a clinical and radiologic study. Dis Colon Rectum. 2006;49:1611–9.
40. Ikeda T, Kumashiro R, Oki E, et al. Evaluation of techniques to prevent colorectal anastomotic leakage. J Surg Res. 2015;194:450–7.
41. Parc Y, Frileux P, Schmitt G, et al. Management of postoperative peritonitis after anterior resection: experience from a referral intensive care unit. Dis Colon Rectum. 2000;43:579–87.
42. Krarup PM, Jorgensen LN, Harling H. Management of anastomotic leakage in a nationwide cohort of colonic cancer patients. J Am Coll Surg. 2014;218:940–9.
43. Smallwood N, Mutch MG, Fleshman JW. The failed anastomosis. Complexities in colorectal surgery: decision-making and management. New York: Springer, 2014.
44. Beunis A, Pauli S, van Cleemput M. Anastomotic leakage of a colorectal anastomosis treated by transanal endoscopic microsurgery. Acta Chir Belg. 2008;108:474–6.
45. Blumetti J, Chaudhry V, Prasad L, Abcarian H. Delayed transanal repair of persistent coloanal anastomotic leak in diverted patients after resection for rectal cancer. Colorectal Dis. 2012;14:1238–41.
46. Thorson AG, Thompson JS. Transrectal drainage of anastomotic leaks following low colonic anastomosis. Dis Colon Rectum. 1984;27:492–4.
47. Sirois-Giguère E, Boulanger-Gobeil C, Bouchard A, et al. Transanal drainage to treat anastomotic leaks after low anterior resection for rectal cancer: a valuable option. Dis Colon Rectum. 2013;56:586–92.
48. Vermeer TA, Orsini RG, Daams F, et al. Anastomotic leakage and presacral abscess formation after locally advanced rectal cancer surgery: incidence, risk factors, and treatment. Eur J Surg Oncol. 2014;40:1502–9.
49. Kirat HT, Remzi FH, Shen B, Kiran RP. Pelvic abscess associated with anastomotic leak in patients with ileal pouch-anal anastomosis (IPAA): transanastomotic or CT-guided drainage? Int J Colorectal Dis. 2011;26:1469–74.
50. Khurrum Baig M, Hua Zhao R, Batista O, et al. Percutaneous postoperative intra-abdominal abscess drainage after elective colorectal surgery. Tech Coloproctol. 2002;6:159–4.

51. Prasad LM, de Souza AL, Blumetti J, et al. Endoscopic-assisted closure of a chronic colocutaneous fistula. Gastrointest Endosc. 2010;72:662–4.
52. Kobayashi H, Kikuchi A, Okazaki S, et al. Over-the-scope-clipping system for anastomotic leak after colorectal surgery: report of two cases. World J Gastroenterol. 2014;20:7984–7.
53. Chopra SS, Mrak K, Hunerbein M. The effect of endoscopic treatment on healing of anastomotic leaks after anterior resection of rectal cancer. Surgery. 2009;145:182–8.
54. DiMaio CJ, Dorfman MP, Gardner GJ, et al. Covered esophageal self-expandable metal stents in the nonoperative management of postoperative colorectal anastomotic leaks. Gastrointest Endosc. 2012;76:431–5.
55. Perez Roldan F. Gonzalez Carro P, Villafanez Garcia MC, et al. Usefulness of biodegradable polydioxanone stents in the treatment of postsurgical colorectal strictures and fistulas. Endoscopy. 2012;44:297–300.
56. Weidenhagen R, Gruetzner KU, Wiecken T, et al. Endoscopic-vacuum-assisted closure of anastomotic leakage following anterior resection of the rectum: a new method. Surg Endosc. 2008;22:1818–25.
57. Verlaan T, Bartels SA, van Berge Henegouwen, et al. Early, minimally invasive closure of anastomotic leaks: a new concept. Colorectal Dis. 2011;13 Suppl 7:18–22.
58. Van Koperen PJ, van der Zaag ES, Omloo JM, et al. The persisting presacral sinus after anastomotic leakage following anterior resection or restorative proctocolectomy. Colorectal Dis. 2011;13:26–9.
59. Broder JC, Tkacz JN, Anderson SW, et al. Ileal-pouch anal anastomosis surgery: imaging and intervention for postoperative complications. Radiographics. 2010;30:221–33.
60. Nesbakken A, Nygaard K, Lunde OC. Outcome and late functional results after anastomotic leakage following mesorectal excision for rectal cancer. Br J Surg. 2001;88:400–4.
61. Fleshman JW, McLeod RS, Cohen Z, Stern H. Improved results following use of an advancement flap technique in the treatment of ileoanal anastomotic complications. Int J Colorectal Dis. 1988;3:161–5.
62. Wexner SD, Rothenberger DA, Jensen L, et al. Ileal pouch vaginal fistulas: incidence, etiology, and management. Dis Colon Rectum. 1989;32:460–5.
63. Stewart BT, Stitz RW. Marsupialization of presacral collections with use of an endoscopic stapler. Dis Colon Rectum. 1999;42:264–5.
64. Whitlow CB, Opelka FG, Gathright JB, Beck DE. Treatment of colorectal and ileoanal anastomotic sinuses. Dis Colon Rectum. 1997;40:760–3.
65. Swain BT, Ellis CN. Fibrin glue treatment of low rectal and pouch-anal anastomotic sinuses. Dis Colon Rectum. 2004;47:253–5.
66. Manceau G, Karoui M, Breton S, et al. Right colon to rectal anastomosis (Deloyers procedure) as a salvage technique for low colorectal or coloanal anastomosis: postoperative and long-term outcomes. Dis Colon Rectum. 2012;55:363–8.
67. Deloyers L. Suspension of the right colon permits without exception preservation of the anal sphincter after extensive colectomy of the transverse and left colon (including rectum): technic-indications- immediate and late results. Lyon Chir. 1964;60:404–13.
68. Bonnard A, de Lagausie P, Leclair MD, et al. Definitive treatment of extended Hirschsprung's disease or total colonic form. Surg Endosc. 2001;15:1301–4.
69. Tang ST, Yang Y, Wang GB, et al. Laparoscopic extensive colectomy with transanal Soave pull-through for intestinal neuronal dysplasia in 17 children. World J Pediatr. 2010;6:50–4.
70. Genser L, Manceau G, Karoui M, et al. Postoperative and long-term outcomes after redo surgery for failed colorectal or coloanal anastomosis: retrospective analysis of 50 patients and review of the literature. Dis Colon Rectum. 2013;56:747–55.
71. MacLean AR, Cohen Z, MacRae HM, et al. Risk of small bowel obstruction after the ileal pouch-anal anastomosis. Ann Surg. 2002;235:20–206.
72. Hahnloser D, Pemberton JH, Wolff BG, et al. Results at up to 20 years after ileal pouch-anal anastomosis for chronic ulcerative colitis. Br J Surg. 2007;94:333–40.

73. Michaelassi F, Lee J, Rubin M, et al. Long-term functional results after ileal pouch-anal restorative proctocolectomy for ulcerative colitis: a prospective observational study. Ann Surg. 2003;238:433–41.
74. Lewis WG, Kuzu A, Sagar PM, et al. Stricture at the pouch-anal anastomosis after restorative proctocolectomy. Dis Colon Rectum. 1994;37:120–5.
75. Fleshman JW, Cohen Z, McLeod RS, et al. The ileal reservoir and ileoanal anastomosis procedure: factors affecting technical and functional outcomes. Dis Colon Rectum. 1988;31:10–6.
76. Shen B, Lashner BA, Bennett AE, et al. Treatment of rectal cuff inflammation (cuffitis) in patients with ulcerative colitis following restorative proctocolectomy and ileal pouch-anal anastomosis. Am J Gastroenterol. 2004;99:1527–31.
77. Werre A, Mulder C, van Heteren C, et al. Dilation of benign strictures following low anterior resection using Savary-Gilliard bougies. Endoscopy. 2000;32:385–8.
78. Araujo SE, Costa AF. Efficacy and safety of endoscopic balloon dilation of benign anastomotic strictures after oncologic anterior rectal resection: report on 24 cases. Surg Laparosc Endosc Percutan Tech. 2008;18:565–8.
79. Shen B, Lian L, Kiran RP, et al. Efficacy and safety of endoscopic treatment for ileal pouch strictures. Inflamm Bowel Dis. 2011;17:2527–35.
80. Shen B, Fazio VW, Remzi FH, et al. Endoscopic balloon dilation of ileal pouch strictures. Am J Gastroenterol. 2004;99:2340–7.
81. Biraima M, Adamina M, Jost R, et al. Long-term results of endoscopic balloon dilation for treatment of colorectal anastomosis stenosis. Surg Endosc 2016; Feb 19. [Epub ahead of print].
82. Truong S, Willis S, Schumpelick V. Endoscopic therapy of benign anastomotic strictures of the colorectum by electroincision and balloon dilation. Endoscopy. 1997;29:845–9.
83. Lucha PA, Fticsar JE, Francis MJ. The strictured anastomosis: successful treatment by corticosteroid injections: report of three cases and review of literature. Dis Colon Rectum. 2005;48:862–5.
84. Prudhomme M, Dozois RR, Godlewski G, et al. Anal canal strictures after ileal pouch-anal anastomosis. Dis Colon Rectum. 2003;46:20–3.
85. Joyce MR, Fazio WV, Hull TT, et al. Ileal pouch prolapse: prevalence, management, and outcomes. J Gastrointest Surg. 2010;14:993–7.
86. Ehsan M, Isler JT, Kimmins MH, Billingham RP. Prevalence and management of prolapse of the ileoanal pouch. Dis Colon Rectum. 2004;47:885–8.
87. Gopal FA, Amshel AL, Shonberg IL, Eftaiha M. Rectal procidentia in elderly and debilitated patients. Experience with the Altemeier procedure. Dis Colon Rectum. 1984;27:376–81.
88. Finlay IG, Aitchison M. Perineal excision of the rectum for prolapse in the elderly. Br J Surg. 1991; 78: 687–9.
89. Williams JG, Rothenberger DA, Madoff RD, Goldberg SM. Treatment of rectal prolapse in the elderly by perineal rectosigmoidectomy. Dis Colon Rectum. 1992;34:209–16.
90. Johansen OB, Wexner SD, Daniel N, et al. Perineal rectosigmoidectomy in the elderly. Dis Colon Rectum. 1993;36:767–72.
91. Kim D, Tsang C, Wong W, et al. Complete rectal prolapse: evolution of management and results. Dis Colon Rectum. 1999;42:460–9.
92. Azimuddin K, Khubchandani I, Rosen L, et al. Rectal prolapse: a search for the "best" operation. Am Surg. 2001;67:622–7.
93. Zbar A, Takashim S, Hasegawa T, Kitabayashi K. Perineal rectosigmoidectomy (Altemeier's procedure): a review of physiology, technique and outcome. Tech Coloproctol. 2002;6:109–16.
94. Habr-Gama A, Jacob CE, Jorge JM, et al. Rectal procidentia treatment by perineal rectosigmoidectomy combined with levator ani repair. Hepatogastroenterology. 2006;53:213–7.
95. Altomare DF, Binda G, Ganio E, et al. Long-term outcome of Altemeier's procedure for rectal prolapse. Dis Colon Rectum. 2009;52:698–703.

96. Hool GR, Hull TL, Fazio VW. Surgical treatment of recurrent complete rectal prolapse: a thirty-year experience. Dis Colon Rectum. 1997;40:270–2.
97. Fengler SA, Pearl RK, Prasad ML, et al. Management of recurrent rectal prolapse. Dis Colon Rectum. 1997;40:832–4.
98. Levitt M, Martin C, Olesevich M, et al. Hirschsprung's disease and fecal incontinence: diagnostic and management strategies. J Pediatr Surg. 2009;44:271–277.
99. Stensrud KJ, Emblem R, Bjornland K. Functional outcome after operation for Hirschsprung disease: transanal versus transabdominal approach. J Pediatr Surg. 2010;45:1640–4.
100. Heij HA, de Vries X, Bremer I, et al. Long-term anorectal function after Duhamel operation for Hirschsprung's disease. J Pediatr Surg. 1995;30:430–2.
101. Shankar KR, Losty PD, Lamont GL, et al. Transanal endorectal coloanal surgery for Hirschsprung's disease: experience in two centers. J Pediatr Surg. 2000;35:1209–13.
102. Romero P, Kroiss M, Chmelnik M, et al. Outcome of transanal endorectal vs. transabdominal pull-through in patients with Hirschsprung's disease. Langenbecks Arch Surg. 2011;396:1027–33.
103. Kim AC, Langer JC, Pastor AC, et al. Endorectal pull-through for Hirschsprung's disease-a multicenter, long-term comparison of results: transanal vs. transabdominal approach. J Pediatr Surg. 2010;45:1213–20.
104. Levitt MA, Dickie B, Pena A. Evaluation and treatment of the Hirschsprung's patient who is not doing well after a pull-through procedure. Semin Pediatr Surg. 2010;19:146–53.
105. Bax KN. Duhamel lecture: the incurability of Hirschsprung's disease. Eur J Pediatr Surg. 2006;16:380–4.
106. Hassink EA, Rieu PN, Severijnen RS, et al. Are adults content or continent after repair for high anal atresia? A long-term follow-up study in patients 18 years of age or older. Ann Surg. 1993;218:196–200.
107. Templeton JM Jr, Ditesheim JA. High imperforate anus: quantitative results of long-term fecal continence. J Pediatr Surg. 1985;20:645–52.
108. Davis B, Rivadeneira DE. Complications of colorectal anastomoses: leaks, strictures, and bleeding. Surg Clin N Am. 2013;93:61–87.
109. Chu DI, Dozois EJ. Pearls for the small bowel and colon that will not reach. Gastrointestinal surgery: management of complex perioperative complications. New York: Springer, 2015.

Perineal Wound Post APR

17

Torbjörn Holm

Introduction

The first surgical operations including removal of the rectum and anus were performed for rectal cancer and the procedures were mainly extra-peritoneal through a posterior parasacral approach via the perineum. An important step in the development of the surgical treatment for rectal cancer was taken by Ernest Miles, who in 1908 published a paper entitled "*A method of performing abdominoperineal excision for carcinoma of the rectum and of the terminal portion of the pelvic colon*" in The Lancet [1].

For over a century, abdominoperineal resection (APR) has been a standard procedure in the treatment of rectal cancer and despite the development of sphincter sparing procedures for high, mid, and early low rectal cancers there still exists multiple indications for APR. These include primary and recurrent low rectal and anal cancer, Crohn's disease, radiation injury, and after anorectal trauma. The extent of removal of perianal tissues and pelvic floor structures varies considerably depending on the indication for APR and must therefore be tailored to the individual patient.

When W. Ernest Miles first described APR for cancer of the rectum in 1908, he advocated primary closure of the perineal wound and the use of two large drains in the posterior and anterior part of the sutured incision. Due to the high rate of perineal wound complications, with related morbidity and mortality, Miles later

T. Holm (✉)
Department of Molecular Medicine and Surgery, Karolinska Institutet, 171 77 Stockholm, Sweden
e-mail: torbjorn.holm@ki.se; Torbjorn.holm@karolinska.se

T. Holm
Karolinska University Hospital, 171 76 Stockholm, Sweden

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8_17

changed to packing the open wound [2]. The practice of leaving the perineal wound open with packs was followed on a regular basis for several decades and is still occasionally used today. However, due to the significant problems associated with an open perineal wound such as pain, fluid discharge, foul order, painful dressing changes, and long hospital stay this practice gradually became unpopular. Today, the primary goal after an APR is to reconstruct the pelvic floor and to close the perineal wound.

Like the extent of excision depends on the indication, the reconstruction depends on the size of the defect. Small defects after an inter-sphincteric APR may be closed primarily by suturing the levator muscles, subcutaneous fat, and skin. The recent development of extralevator abdominoperineal excision (ELAPE) has been reported to improve oncological outcomes in patients with low rectal cancer [3]. This potential benefit comes at the expense of a large cylindrical open wound extending from the pelvic floor to the perineal skin. The technical nature of ELAPE does not allow for primary closure in many cases. Special closure or reconstructive procedures are often required after more extensive excisions, resulting in large wounds, such as in ELAPE [4]. Obviously, the more extensive the wounds, the more complicated their closure and these are also more prone to infection and breakdown.

The incidence of perineal wound complications after primary closure ranges from 20–50% [5]. Successful healing after pelvic floor reconstruction depends on, but is not limited to factors such as patient characteristics, neoadjuvant therapy, size of the defect, contamination of the wound, type of reconstruction and experience of the surgical team.

Nutritional status of the patient, smoking habits, comorbidities and different types of medication may influence wound healing and must influence the treatment plan.

Neoadjuvant radiotherapy significantly increases the risk of perineal wound complications. In one series by Ballard and colleagues the overall complication rate was 41% and major wound complications inclusive of infection and delayed healing was 35%. Neoadjuvant radiation therapy had a major influence on the complication rate. The complication rate following radiation therapy was 47 versus 23% in non-radiated patients ($p = 0.005$) [5]. Thus, preoperative radiation therapy adds a significant challenge in the management of perineal defects.

The size of the defect is the main determinant in deciding on how to reconstruct the pelvic floor and to close the perineal wound but is also related to the risk of wound complications. After small excisions, including only the anus and the internal sphincter or the internal and external sphincter, the perineum can usually be reconstructed by primary closure of the levator muscles, subcutaneous fat and skin, especially if the patient has not received radio-chemotherapy (Fig. 17.1). Additional procedures including reconstruction of the pelvic floor are usually required after more extensive procedures, such as ELAPE with a more or less wide removal of the pelvic floor or ischioanal APE with less radical clearance of the pelvic floor and ischioanal fat (Fig. 17.2).

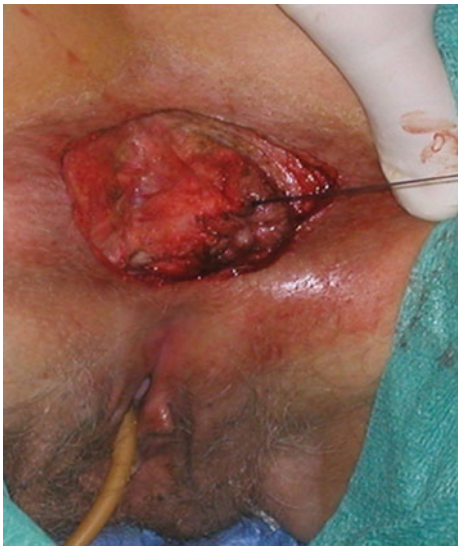


Fig. 17.1 Small perineal defect after proctectomy

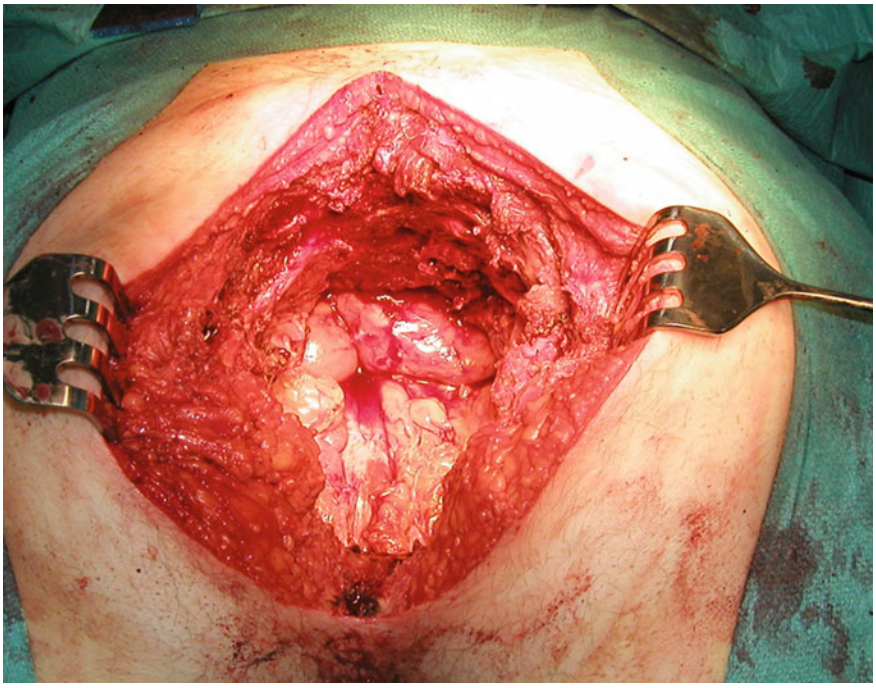


Fig. 17.2 Large perineal defect after ischioanal APR

The risk of contamination of the wound by perianal or ischioanal abscesses and/or fistulae from infections or a perforated cancer is also an important factor to consider before planning the procedure. If the abscess or fistula is caused by a perforated cancer the whole affected area must be removed en bloc with the cancer to prevent seeding of cancer cells into the wound. If the abscess or fistula is not related to cancer there is still a risk of bacterial contamination of the wound with subsequent wound healing problems.

The experience of the surgical team is clearly an important factor in the management of the defect after APR. Small defects can easily be handled by colorectal surgeons but with more advanced reconstructions, including musculocutaneous flaps, the competence of a plastic surgeon may be required.

Type of Reconstruction

A variety of surgical alternatives to primary closure have been used in order to reconstruct the pelvic floor and to reduce the wound healing problems after APR. These procedures include different rotational musculocutaneous flaps, reconstruction with biological mesh, and omental pedicle flaps (omentoplasty).

Simple Closure

As mentioned above, a simple closure of the perineal wound after an APR is associated with a high risk of major wound complications. The rate may be 40% or even higher, especially in patients who have received neoadjuvant radio- or radio-chemotherapy and where the levator muscle has been more or less entirely removed. In addition, closure of skin and fat alone provides a weak pelvic floor and the patient may develop a perineal hernia as a late complication after an APR. However, simple closure may be considered in patients where an inter-sphincteric APR has been performed and the perineal defect is small; for example in mid- and upper rectal tumors, as an alternative to Hartmann's procedure if incontinence precludes a sphincter saving procedure with a low anastomosis or in benign disease, such as Crohn's disease, where removal of the anal canal is necessary. However, in many cases a primary simple closure is insufficient and some type of flap is often used in this situation. Indications for the use of musculocutaneous flaps include coverage of large perineal defects, vaginal reconstruction, and secondary repair of non-healing wounds.

The Rectus Abdominis Musculocutaneous Flap (Figs. 17.3 and 17.4)

Shukla et al. first published on the use of the rectus abdominis muscle flap for reconstruction of perineal wounds in three patients in 1984 [6]. Tobin and coworkers later reported on its use for vaginal and pelvic floor reconstruction and since then several series in the medical literature have demonstrated good results with relatively low morbidity associated with the use of these flaps [7–10].

The rectus abdominis myocutaneous (RAM) flap may be harvested as a transverse rectus abdominis muscle flap (TRAM) or as a vertical rectus abdominis muscle flap (VRAM), depending on its variable skin paddle orientation. There are no comparative studies on the relative merits of either orientation but the VRAM flap has been used most often for reconstruction of large perineal wounds.

One of the largest series examining the VRAM flap for perineal reconstruction was published by Buchel et al. This was a retrospective review of 73 patients and reported that primary healing occurred in 85% of patients and that 95% obtained a healed perineal wound within 30 days [11]. Another study compared 19 patients with anorectal cancer treated with external beam pelvic radiation followed by APR and RAM flap reconstruction of the perineum with a control group of 59 patients

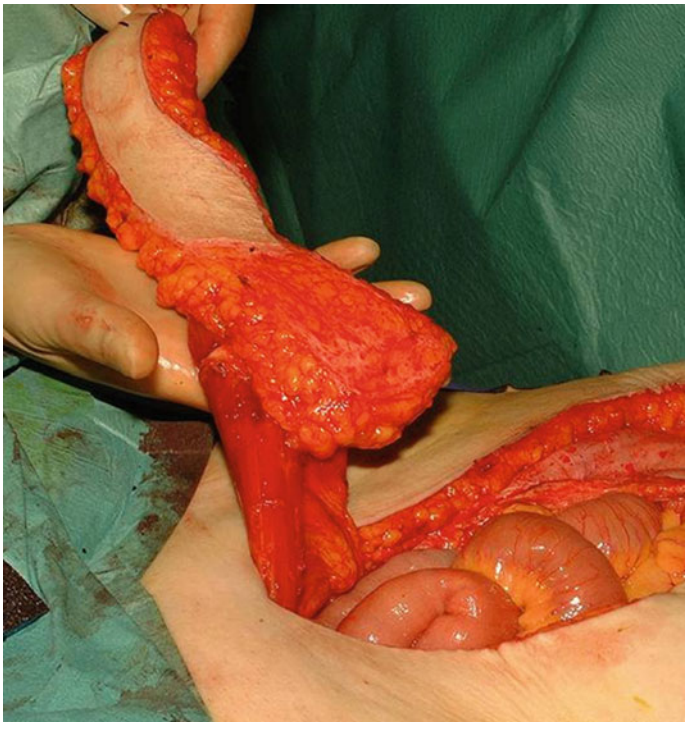


Fig. 17.3 VRAM flap

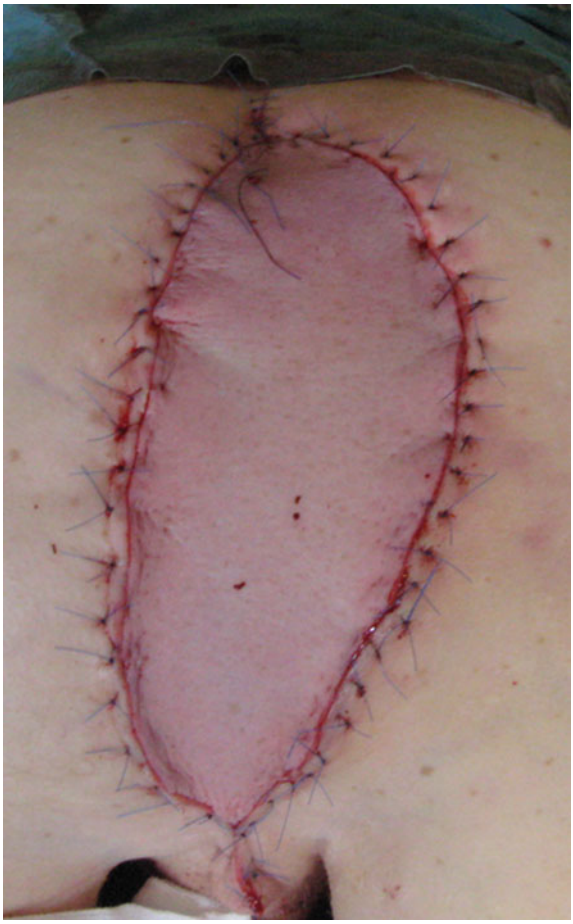


Fig. 17.4 Reconstruction of the pelvic floor and perineum with VRAM flap

treated with similar radiation doses that subsequently underwent an APR without a RAM flap during the same time period. Perineal wound complications occurred in 16% of the RAM flap patients and in 44% of the control patients, which suggests that perineal closure with a flap significantly decreases the incidence of perineal wound complications in patients undergoing external beam pelvic radiation and APR [12].

Although the RAM flap is probably the most frequently used tissue transfer to promote perineal wound healing and decrease the risk of complications, there are some concerns to be mentioned. The dissection of this flap is technically demanding and great care must be taken not to injure the inferior epigastric artery as the circulation may otherwise be compromised. The RAM flap is denervated, not contractile and thus prone to loss of volume with time. Also, donor site morbidity, such as abdominal wall weakness and an increased risk of incisional hernia, has to be considered.

The Gluteus Maximus Flap

This flap has mainly been used for pressure wound surgery but has recently been used also for reconstruction after APR for rectal cancer [13, 14]. A unilateral gluteus maximus flap is usually sufficient after ELAPE (Fig. 17.5) but with more extensive excisions, resulting in substantial loss of tissue, bilateral flaps may be necessary (Figs. 17.6 and 17.7). Most papers reporting on outcomes after gluteus maximus flap reconstruction include small numbers of patients and there is no randomized controlled comparison between the RAM and gluteus maxumus flaps. In a report by Anderin et al. 65 patients were studied after ELAPE and a one-sided musculocutaneous gluteus flap for low or locally recurrent rectal cancer. Fifty-nine had received neoadjuvant radio- or radio-chemotherapy. Twenty-seven (41.5%) patients had one or more perineal wound complications. A minor wound infection occurred in 15, while 12 had either a more severe infection with dehiscence or a pelvic abscess. The reconstruction was completely healed in 91% of the patients at 1 year [13].

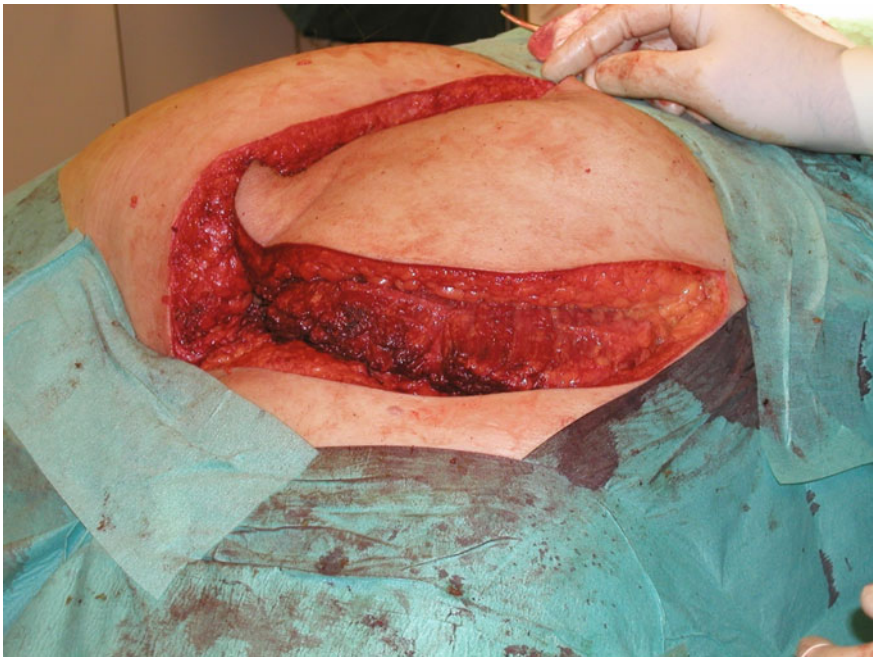


Fig. 17.5 Unilateral gluteus maximus musculocutaneous flap

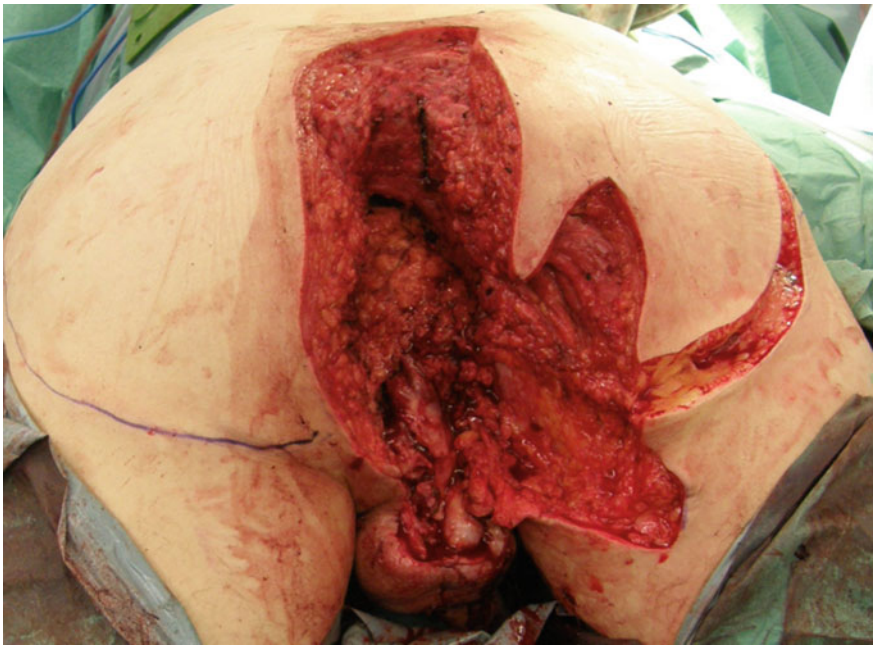


Fig. 17.6 Extensive resection of pelvic floor and perineum in a patient with perforated rectal cancer and complex ischioanal fistulae

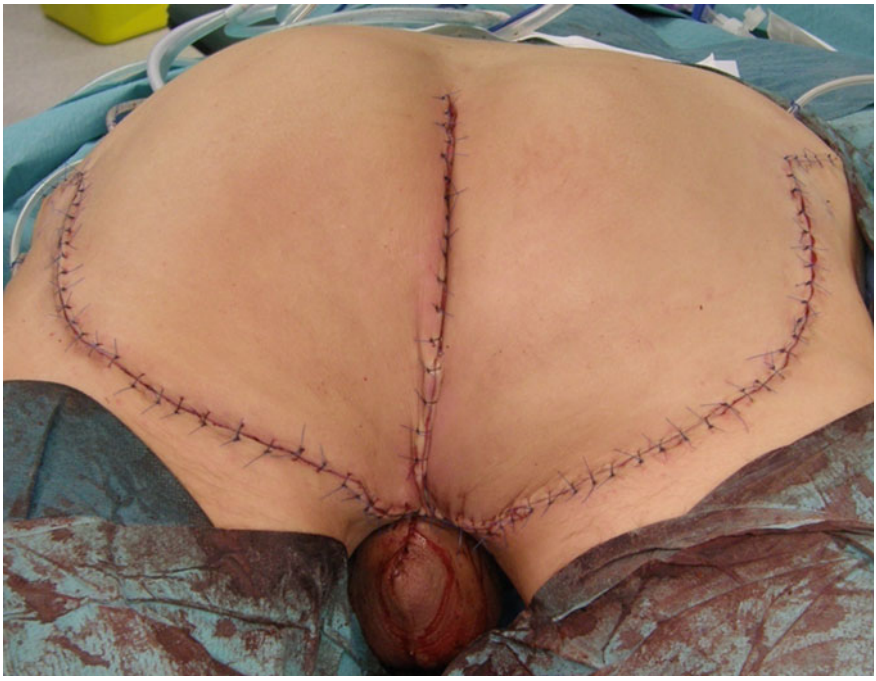


Fig. 17.7 Bilateral gluteus maximus musculocutaneous flaps

Advantages of the gluteus maximus flap include that it is well vascularized and innervated and does not shrink with time and that it does not cause donor site morbidity in the abdominal wall, which is especially attractive after minimally invasive surgery. The disadvantage of this flap is that is does not fill the pelvic cavity to the same degree as the RAM flap and that a combination of vaginal wall reconstruction makes the procedure more complicated.

The Gracilis Musculocutaneous Flap

Utilizing the gracilis myocutaneous flap to repair persistent perineal sinuses was described in 1975 by Bartholdson et al. [15]. This flap has mainly been used in patients with delayed healing or persistent sinuses after previous APR with primary closure, or as a primary reconstruction in patients with recurrent rectal cancer after radiochemotherapy. Shibata and colleagues investigated perineal wound healing in patients who all received neoadjuvant radiotherapy and subsequently had an APR for recurrent rectal cancer. Sixteen patients underwent either unilateral or bilateral gracilis muscle flap closure, while 24 patients had a primary perineal closure alone. The results dramatically favored the gracilis flap closure; only 12% of the patients closed with gracilis flaps had major complications compared to 46% of the patients who underwent primary closure. In 63% of the patients closed with gracilis flaps the perineum healed without incident, but only 33% of the patients with primary closure had an uneventful recovery [16]. The drawbacks of the gracilis flap include its relatively small muscle bulk and skin fragility but despite these limitations, its role in preventing postoperative and post-irradiation perineal complications is well established [17].

Pelvic Floor Reconstruction with Biological Mesh

The different musculocutaneous flap solutions to reconstruct the pelvic floor are valuable in order to reduce complications but many colorectal surgeons have been hesitant to use flaps routinely due to the more extensive procedure, the prolongation of operation time and often limited access to plastic surgeons. Instead of reconstructing the pelvic floor by flaps it has been suggested to apply a biological mesh in the pelvic defect. This method is quick, easy to perform, and not dependent on the availability of plastic surgeons (Fig. 17.8). In addition, it seems feasible with a reasonable complication rate. In one report the use of a biological mesh also significantly reduced the risk of perineal hernia [18]. However, the number of reports is still limited and substantial, long term results from biological mesh reconstruction of the pelvic floor are lacking.

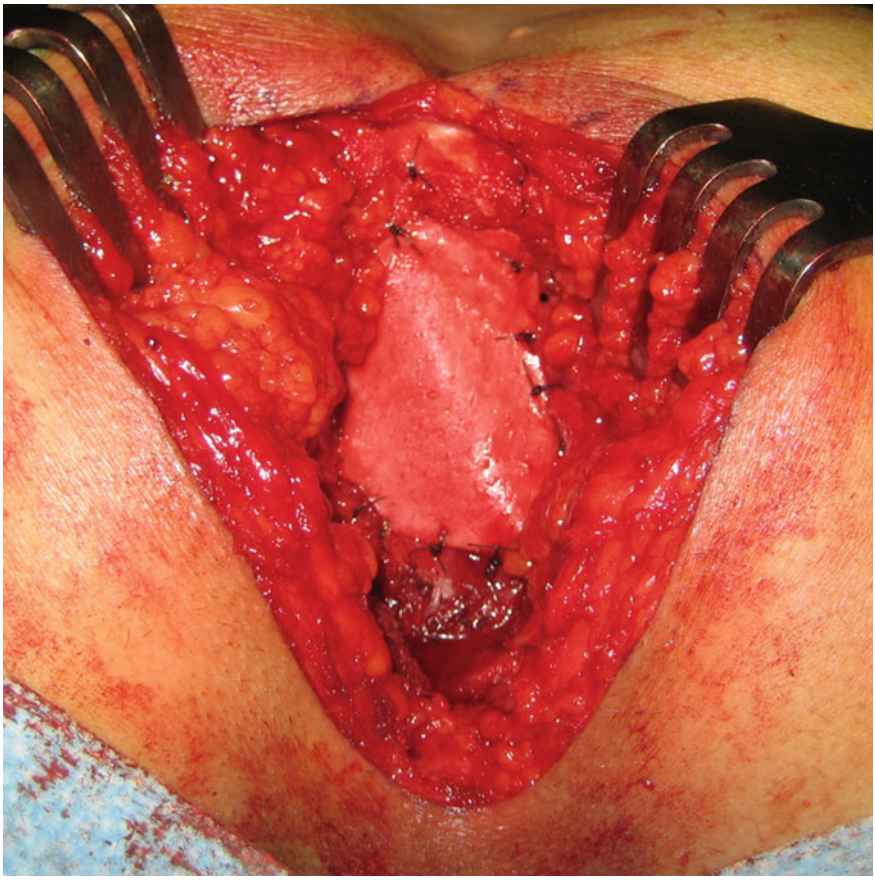


Fig. 17.8 Reconstruction of pelvic floor with biological mesh

Omentoplasty

Bowel obstruction, due to entrapment of the small bowel in the pelvic cavity, is not infrequent after an APR. An omentoplasty filling out the pelvic cavity may reduce this cause of postoperative small bowel obstruction. Therefore, and if the patient has a large omentum, it is feasible to mobilize it from the transverse colon and from the greater curvature of the stomach and to prepare an omentoplasty which can fill out the empty pelvic cavity. Mobilization of the omentum and its placement in the pelvic cavity to prevent injury to the small bowel during postoperative pelvic irradiation is well known [19, 20]. Killeen and colleagues published a systematic review on the use of omental pedicle flaps following proctectomy. They collected

data from 14 studies totaling 891 patients with a median follow-up of 13.5 months. Mean rate of primary healing with omentoplasty was 67 versus 50% with no omentoplasty. Mean time to healing in the former group was 24 versus 79 days in the latter group. The authors concluded that: "Omental mobilization and buttressing of primary perineal repair reduced perineal wound morbidity" [21].

Is There an Optimal Way to Reconstruct the Pelvic Floor and Perineum After an APR?

Butt and colleagues preformed a systematic review of ELAPE including 27 series and 963 patients. They compared the results of biomesh closures (149 patients) with musculocutaneous flap closures (201 patients) and 578 patients with primary closure. Minor and major wound complications and perineal hernias were compared. The results are shown in Table 17.1. The authors found no significant differences regarding minor or major wound complications or perineal hernias in relation to biomesh, muscle flaps, or primary closure and concluded that: "Despite several techniques currently employed for perineal construction, it remains unclear as to which is optimal" [22]. This systematic review does not include randomized controlled trials and it is highly likely that the size and nature of the defect might have affected the choice of the closure technique and resultant complication rates.

Another review compared 255 patients undergoing flap repair to 85 patients undergoing biological mesh repair and also found no significant difference in the rates of perineal wound complications or perineal hernia formation [23].

In fact, there is no standard solution for pelvic floor reconstruction after APR and as mentioned above the method used must be tailored according to the patient and the extent of excision. It is recommended to assess each patient carefully within a multidisciplinary team approach before surgery to determine the suitable type of pelvic floor reconstruction and to establish collaboration with a plastic surgeon team for reconstruction after the more wide excisions [24].

Table 17.1 Wound complications in relation to type of pelvic floor reconstruction after ELAPE

	Wound complication		
	Minor (%)	Major (%)	Perineal Hernia (%)
Biomesh	27.5	13.4	3
Muscle flap	29.4	19.4	0
Primary closure	17.1	6.4	1

Type of Complications

The goal after a more or less extensive excision of the perineum and pelvic floor after an APR is to achieve a closed perineal wound without complications. Despite improvements in surgical techniques, wound and patient care, perineal wound complications are still common and account for significant morbidity. The main complications from the perineal wound are superficial and deep infections, often resulting in wound rupture and occasionally septicemia (Figs. 17.9, 10 and 11). Delayed healing is a significant problem and the management of an open, deep perineal wound is difficult with severe morbidity for the patient and with high costs for the health care system. Features of the perineal wound predisposing to such complications have been mentioned above and in addition, the vast area of dead space in the pelvic cavity and the location of the wound in a pressure zone make it more susceptible to necrosis and subsequent infection.

There are numerous reports with different outcomes concerning wound healing after APR. A recent report from the LOREC group mirrors current practice for patients with low rectal cancer in the UK [25]. Forty-two units entered 266 patients. Of these 172 (65%) underwent extralevator APE (ELAPE) and 94 non-ELAPE. After ELAPE the perineal wound was closed primary with mesh in 55%, without mesh in 15% and with a flap in 21% of cases. After non-ELAPE 54% of wounds

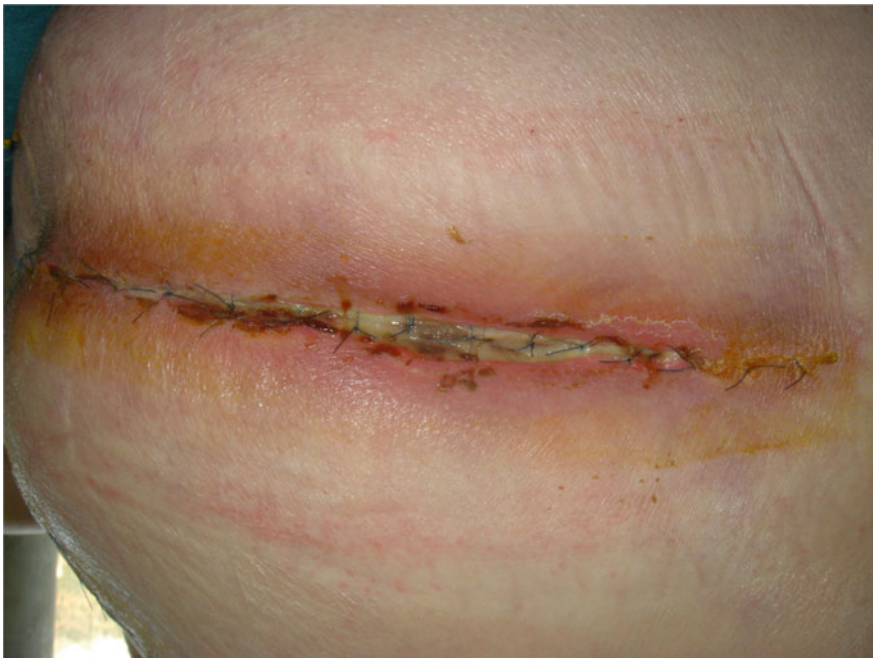


Fig. 17.9 Superficial perineal infection

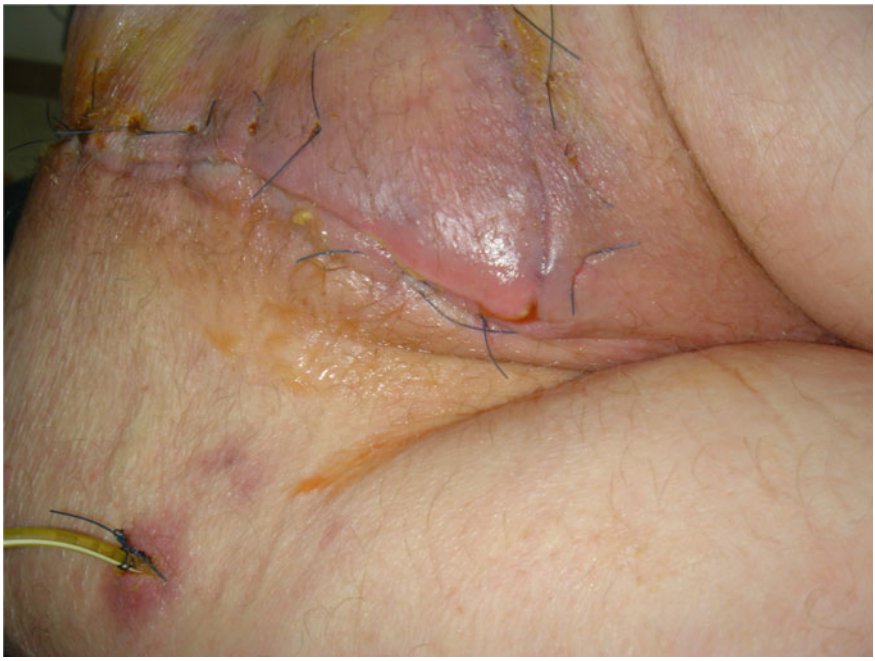


Fig. 17.10 Deep perineal infection with abscess

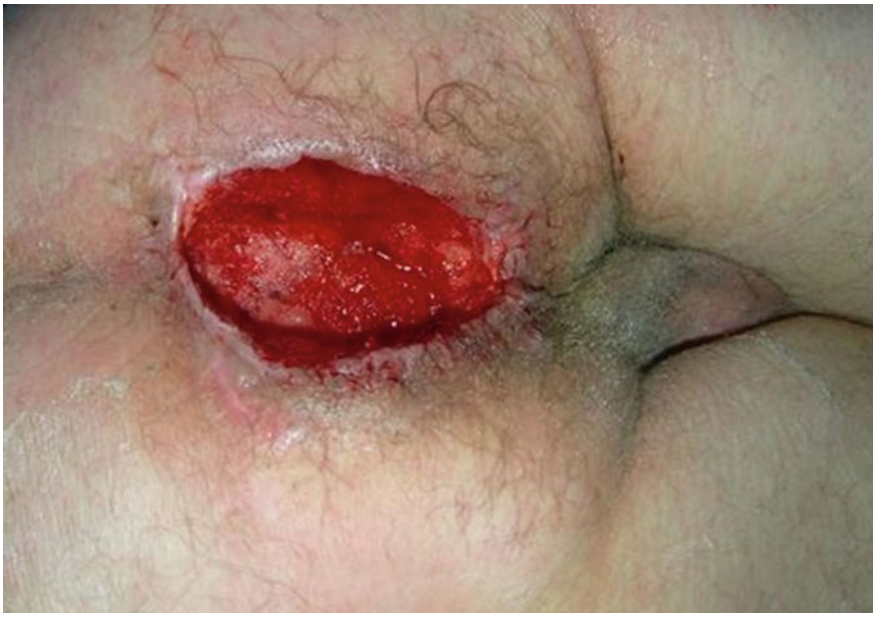


Fig. 17.11 Perineal wound rupture

were closed primarily without mesh, 29% primarily with mesh and 5% by a flap. Wound breakdown occurred in 30% after ELAPE and 31% after non-ELAPE. It was more common after neoadjuvant radiotherapy. Donor site complications occurred in 17% of flap cases. Perineal morbidity remained at 12 months in 11% of the patients.

Perineal hernia is a late complication after APR and was reported originally by Gregory and Muldoon in 1969 [26]. The extensive pelvic floor resection in ELAPE is probably more conducive to perineal herniation (Fig. 17.12). The risk may increase even further if the abdominal part of the operation is done by laparoscopy. Sayers et al. recently reported a small study including 56 patients who underwent ELAPE. Perineal hernia was the commonest complication (26%) and occurred in nine (45%) of 20 patients who had a laparoscopic ELAPE [27]. These results differ from those reported by Christensen et al. who did not see any perineal hernia in 24 patients after biological mesh repair [18].

Perineal entero-cutaneous bowel fistula is another late complication (Fig. 17.13). Fortunately, this is rare and is difficult to treat when it occurs. The fistula most often develops from the small bowel and usually in irradiated patients. It may occur as an early or late complication, sometimes several years after the APR.

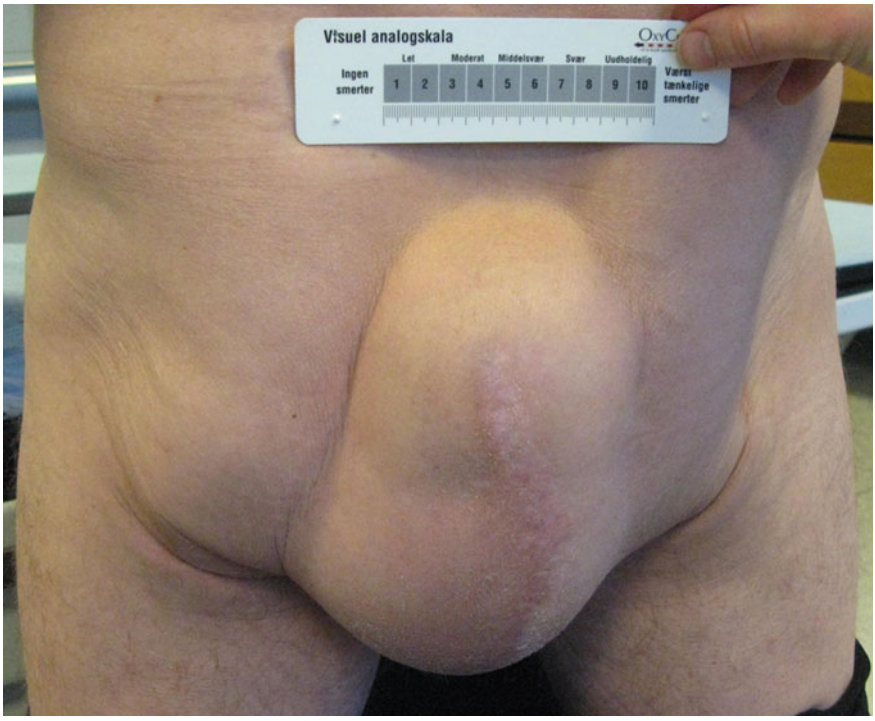


Fig. 17.12 Large perineal hernia

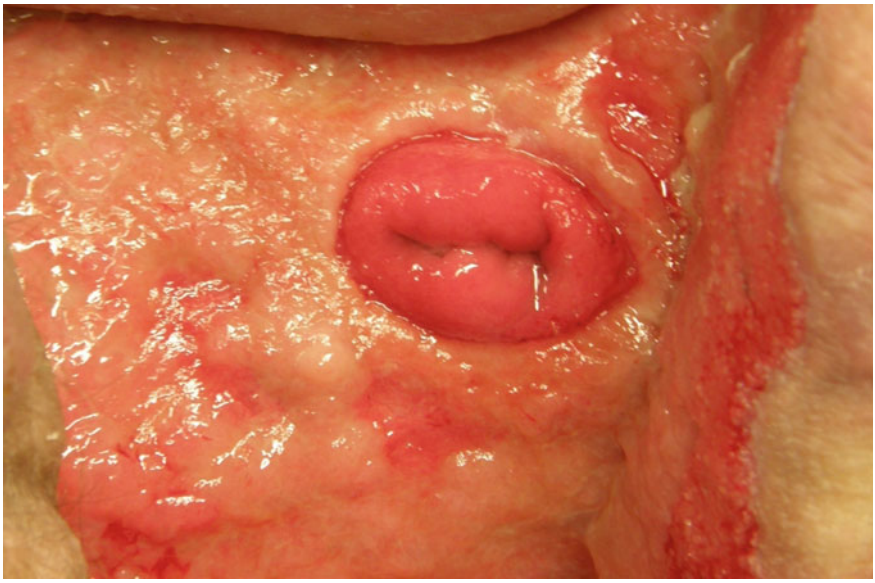


Fig. 17.13 Small bowel fistula to perineum

Management of Complications

Perineal wound complications after ELAPE differ significantly in severity and the need for treatment is completely different in different situations. Table 17.2 gives a brief summary of the different complications and their treatment.

Superficial infections (Fig. 17.9) are very common and can usually be treated with simple debridement, cleansing, and wound dressings. Antibiotic therapy is usually not indicated. The vast majority of these infections will heal within a few weeks without persistent problems.

Deeper infections may appear as a deep subcutaneous infection or a pelvic abscess with or without perineal wound breakdown (Fig. 17.14). The first priority is to control infection. This may occasionally be done by drainage of the pelvic abscess and antibiotics but usually the best treatment is to open up the wound and apply intensive cleansing and debridement of the pelvic cavity and subcutaneous

Table 17.2 Perineal wound complications and treatment

Complication	Treatment
Simple Infection	Cleansing and Dressing Changes
Severe Infection/Wound Dehiscence	Cleansing, debridement, lay open ± Vac treatment
Persistent Fistula/Sinus	Surgical treatment
Perineal Hernia	Surgical treatment

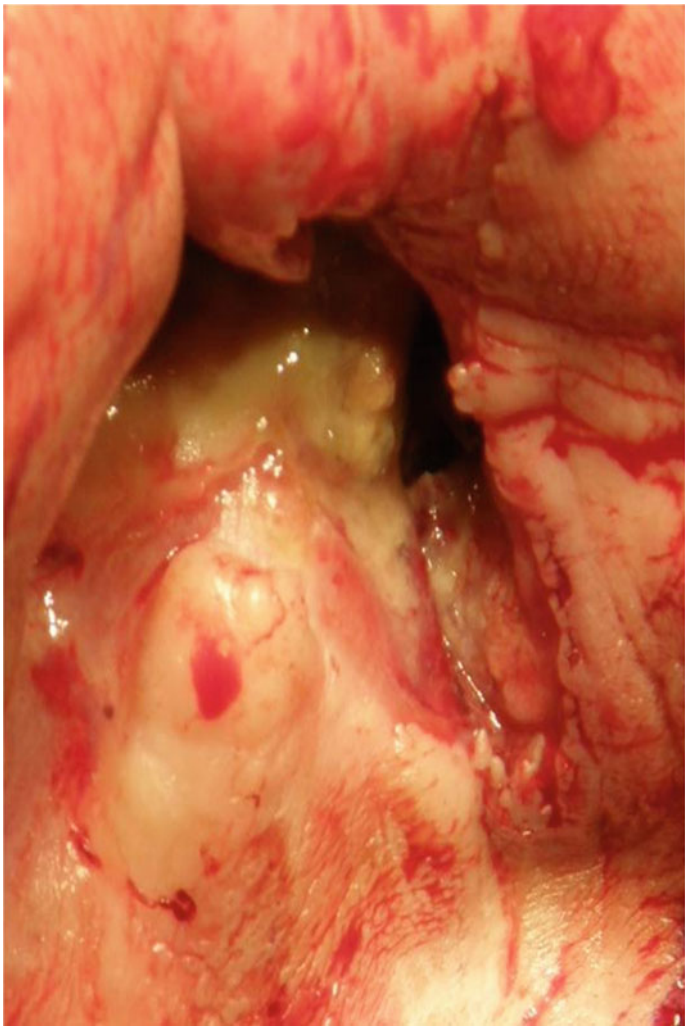


Fig. 17.14 Deep infection with necrosis and breakdown of perineal wound

tissues. Vac therapy may be applied in this situation in order to improve cleansing and induce tissue granulation (Fig. 17.15). When infection is under control, the best approach is to wait for 3–6 months for secondary healing. Early secondary closure after a deep infection and wound breakdown is generally futile and may induce a new infection. However, surgery should be considered if the wound fails to heal completely or a persistent sinus remains after six months. In this situation it is crucial to evaluate the type and extent of the wound healing problem and to plan the reconstructive procedure in detail. Timing of the operation is also very important.

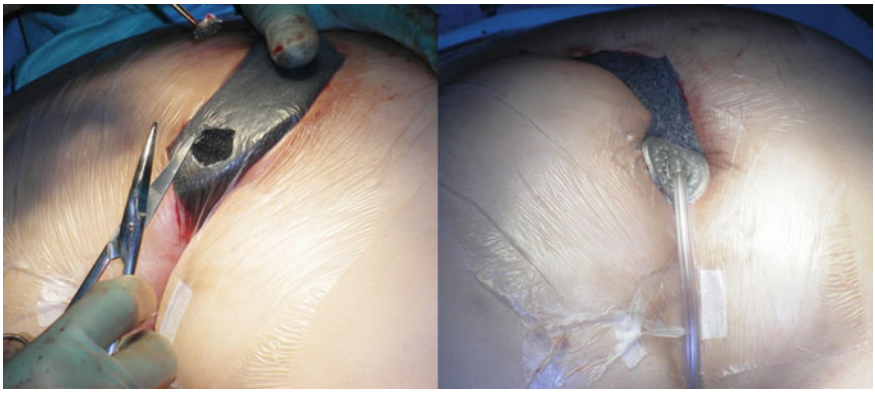


Fig. 17.15 Vac therapy of perineal wound

High resolution magnetic resonance imaging (MRI) is an important adjunct to clinical examination in assessing the extent of the complication. This is analogous to its utility in preoperative staging of rectal cancers [28]. MRI is very useful to distinguish an isolated perineal problem from a more complex problem, which may involve small bowel adhesions to the pelvic floor or an enteric fistula to the perineal wound or other organs. Such fistulae may also involve other organs, such as vagina, bladder and urethra. The extent of the surgical procedure depends on the extent of the problem and may involve only a perineal approach or a combined abdominal and perineal approach.

A perineal approach is appropriate if the wound healing problem is confined to the perineum. When the wound is clean with healthy granulating tissue one may consider revision with primary suture or reconstruction with some form of musculocutaneous flap. Gluteus flaps are practical in this situation and uni- or bilateral flaps may be used depending on the size of the unhealed defect. This type of reconstruction is usually successful and the cosmetic result acceptable (Fig. 17.16). In patients with a smaller and deeper unhealed sinus a gracilis muscle flap may be more suitable. Reconstruction with a gluteus flap is best done with the patient in the prone jack-knife position while the supine position often is better for a gracilis flap reconstruction. It is important to take great care not to injure the small bowel when a

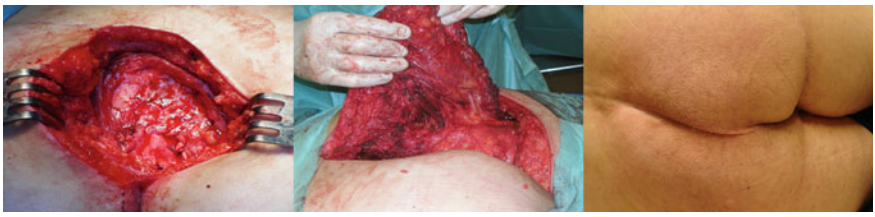


Fig. 17.16 Clean perineal wound, reconstruction with gluteus flap, and healed perineum

perineal approach only is used and the relation of the bowel to the bottom of the wound must be established by MRI before surgery. If the distance from bowel to wound is very short it may be safer to use a combined abdominal and perineal technique.

The combined abdominoperineal operation must be used in situations where an enteric fistula is present together with an unhealed perineal wound or if an entero-cutaneous fistula to the perineum develops as a late complication (Fig. 17.17). The extent of the abdominal surgery depends on the pathology and may include simple adhesiolysis, small bowel resection, resection of the vagina and all the way to pelvic exenteration in patients with complex fistulae, involving bladder or urethra. Segments of fistulating bowel must be removed and anastomoses made on healthy bowel. When the "neo-pelvic floor" is cleared from adhesions and

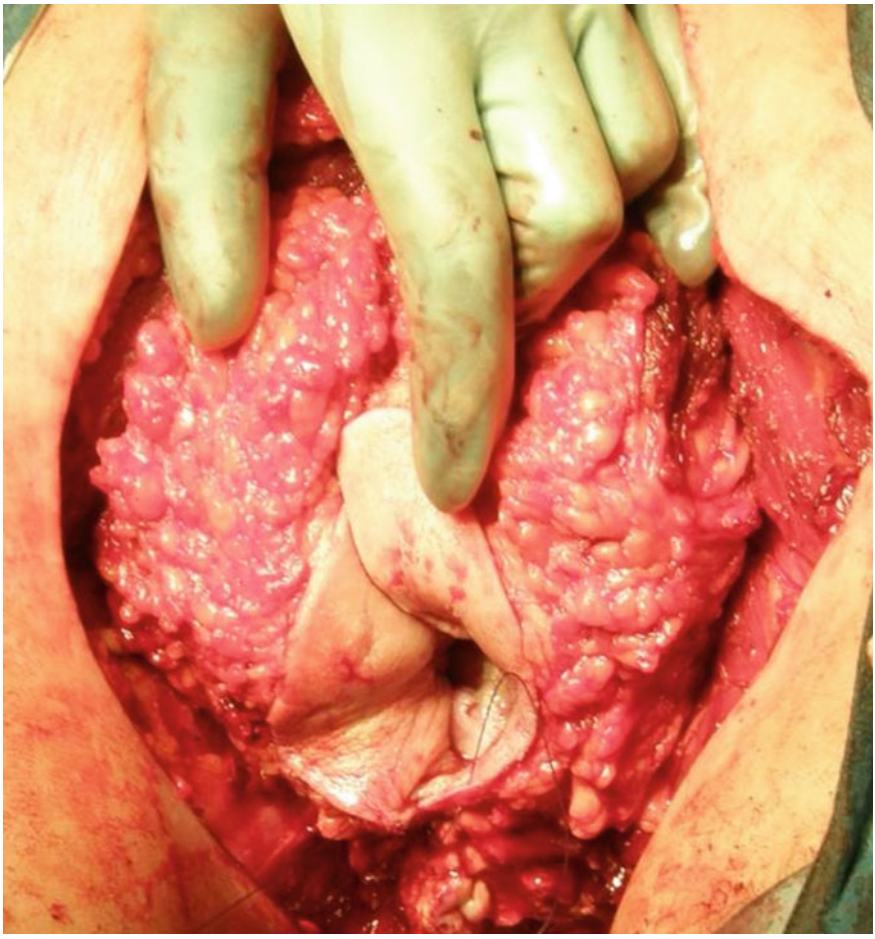


Fig. 17.17 Persistent perineal sinus with complex entero-cutaneous fistula

fistula tracts, and scar tissues excised, it is recommended to fill the pelvis with an omental flap as described above. When the abdominal surgery is completed the perineal reconstruction can be performed using a VRAM flap or uni- or bilateral gluteus flaps.

Perineal hernias may develop as an early or late complication after APR and the risk is probably related to the extent of pelvic floor removal. If the hernia is symptomatic, which is often the case, it may cause pain and severely disable everyday activities. Repair of perineal hernia remains challenging and there is no consensus in the literature on the best approach. These patients should also be examined clinically and by MRI to assess the extent of the perineal defect and the contents of the hernia and the surgical approach must be tailored to the individual patient. Our current approach is to repair small hernia without bowel involvement by a perineal approach using mesh or gluteus flap. In hernias with bowel involvement we use an abdominal approach with adhesiolysis, mesh repair of the pelvic floor and omentoplasty. For large hernias with bowel involvement a combined abdominoperineal approach is used, combining adhesiolysis, omentoplasty and gluteus muscle flap reconstruction. Most papers on perineal hernia are case reports but one pooled analysis on 40 patients was published by Mjoli et al. in 2012. They report a median time interval of 8 months between APR and surgical repair of perineal hernia. The surgical approaches were perineal in 22 patients, open abdominal in 11, open abdominoperineal in three, laparoscopic in five, and laparoscopic-perineal in two patients. The recurrence rate was 5/25 for synthetic or biological mesh, 6/12 for primary closure, and 2/6 for the remaining techniques. The authors conclude that that the recurrence rates after primary perineal hernia repair is lower with the use of a mesh or other assisted closure than with primary suture repair.

Summary

Perineal complications after APR, conventional or ELAPE, are common and vary significantly in complexity. The perineal wound is particularly prone to infectious complications and wound rupture due to its location, size, and loss of tissue which makes a tension-free repair difficult. The risk of wound complications increases substantially after neoadjuvant radio- and radio-chemotherapy and as these treatments are increasingly used in patients with low rectal cancer it can be anticipated that more patients will develop perineal wound problems after APR. Primary closure may still be an option in nonirradiated patients with small defects but as the calculated risk for complications increases this is not sufficient and the surgeon must plan for alternative procedures. The type of pelvic floor reconstruction is best individualized and the decision should be based on several factors including patient comorbidity, neoadjuvant treatment, and the extent of the pelvic floor defect. Good collaboration with a skilled plastic and reconstructive surgeon team is invaluable. Most perineal wound infections heal with proper local treatment. In patients with

wound breakdown and a persistent open wound a thorough evaluation clinically and with MRI is crucial. Treatment must be tailored to the patient and the complexity of the complication and with appropriate timing.

References

1. Miles WE. A method of performing abdomino-perineal excision for carcinoma of the rectum and of the terminal portion of the pelvic colon. The Lancet. 1908;2:1812–3.
2. Miles W. Technique of the radical operation for cancer of the rectum. Br J Surg. 1914;2 (6):292–305.
3. West NP, Anderin C, Smith KJ, Holm T, Quirke P. Multicentre experience with extralevator abdominoperineal excision for low rectal cancer. Br J Surg. 2010;97(4):588–99.
4. Shihab OC, Heald RJ, Holm T, How PD, Brown G, Quirke P, et al. A pictorial description of extralevator abdominoperineal excision for low rectal cancer. Colorectal Dis: Official J Assoc Coloproctol G B Irel. 2012;14(10):e655–60.
5. Bullard KM, Trudel JL, Baxter NN, Rothenberger DA. Primary perineal wound closure after preoperative radiotherapy and abdominoperineal resection has a high incidence of wound failure. Dis Colon Rectum. 2005;48(3):438–43.
6. Shukla HS, Hughes LE. The rectus abdominis flap for perineal wounds. Ann R Coll Surg Engl. 1984;66(5):337–9.
7. Erdmann MW, Waterhouse N. The transpelvic rectus abdominis flap: its use in the reconstruction of extensive perineal defects. Ann R Coll Surg Engl. 1995;77(3):229–32.
8. Jain AK, De Franzo AJ, Marks MW, Loggie BW, Lentz S. Reconstruction of pelvic exenterative wounds with transpelvic rectus abdominis flaps: a case series. Ann Plast Surg. 1997;38(2):115–22 (discussion 22–3).
9. McAllister E, Wells K, Chaet M, Norman J, Cruse W. Perineal reconstruction after surgical extirpation of pelvic malignancies using the transpelvic transverse rectus abdominal myocutaneous flap. Ann Surg Oncol. 1994;1(2):164–8.
10. Tobin GR, Day TG. Vaginal and pelvic reconstruction with distally based rectus abdominis myocutaneous flaps. Plast Reconstr Surg. 1988;81(1):62–73.
11. Buchel EW, Finical S, Johnson C. Pelvic reconstruction using vertical rectus abdominis musculocutaneous flaps. Ann Plast Surg. 2004;52(1):22–6.
12. Chessin DB, Hartley J, Cohen AM, Mazumdar M, Cordeiro P, Disa J, et al. Rectus flap reconstruction decreases perineal wound complications after pelvic chemoradiation and surgery: a cohort study. Ann Surg Oncol. 2005;12(2):104–10.
13. Anderin C, Martling A, Lagergren J, Ljung A, Holm T. Short-term outcome after gluteus maximus myocutaneous flap reconstruction of the pelvic floor following extra-levator abdominoperineal excision of the rectum. Colorectal Dis: Official J Assoc Coloproctol G B Irel. 2012;14(9):1060–4.
14. Holm T, Ljung A, Haggmark T, Jurell G, Lagergren J. Extended abdominoperineal resection with gluteus maximus flap reconstruction of the pelvic floor for rectal cancer. Br J Surg. 2007;94(2):232–8.
15. Bartholdson L, Hulten L. Repair of persistent perineal sinuses by means of a pedicle flap of musculus gracilis. Case report. Scand J Plast Reconstr Surg. 1975;9(1):74–6.
16. Shibata D, Hyland W, Busse P, Kim HK, Sentovich SM, Steele G Jr, et al. Immediate reconstruction of the perineal wound with gracilis muscle flaps following abdominoperineal resection and intraoperative radiation therapy for recurrent carcinoma of the rectum. Ann Surg Oncol. 1999;6(1):33–7.
17. Small T, Friedman DJ, Sultan M. Reconstructive surgery of the pelvis after surgery for rectal cancer. Semin Surg Oncol. 2000;18(3):259–64.

18. Christensen HK, Nerstrom P, Tei T, Laurberg S. Perineal repair after extralevator abdominoperineal excision for low rectal cancer. Dis Colon Rectum. 2011;54(6):711–7.
19. DeLuca FR, Ragins H. Construction of an omental envelope as a method of excluding the small intestine from the field of postoperative irradiation to the pelvis. Surg, Gynecol Obstet. 1985;160(4):365–6.
20. Granai CO, Gajewski W, Madoc-Jones H, Moukhtar M. Use of the omental J flap for better delivery of radiotherapy to the pelvis. Surg, Gynecol Obstet. 1990;171(1):71–2.
21. Killeen S, Devaney A, Mannion M, Martin ST, Winter DC. Omental pedicle flaps following proctectomy: a systematic review. Colorectal Dis: Official J Assoc Coloproctol G B Irel. 2013;15(11):e634–45.
22. Butt HZ, Salem MK, Vijaynagar B, Chaudhri S, Singh B. Perineal reconstruction after extra-levator abdominoperineal excision (eLAPE): a systematic review. Int J Colorectal Dis. 2013;28(11):1459–68.
23. Foster JD, Pathak S, Smart NJ, Branagan G, Longman RJ, Thomas MG, et al. Reconstruction of the perineum following extralevator abdominoperineal excision for carcinoma of the lower rectum: a systematic review. Colorectal Dis: Official J Assoc Coloproctol G B Irel. 2012;14 (9):1052–9.
24. Peirce C, Martin S. Management of the perineal defect after abdominoperineal excision. Clin Colon Rectal Surg. 2016;29(2):160–7.
25. Jones HJ, Moran BJ, Crane S, Hompes R, Cunningham C, group L. The LOREC APE registry—Operative technique, oncological outcome and perineal wound healing after abdominoperineal excision. Colorectal Dis: Official J Assoc Coloproctol G B Irel. 2016.
26. Gregory JS, Muldoon JP. Perineal herniation–a late complications of adominoperineal resection of the rectum: report of a case. Dis Colon Rectum. 1969;12(1):33–5.
27. Sayers AE, Patel RK, Hunter IA. Perineal hernia formation following extralevator abdominoperineal excision. Colorectal Dis: Official J Assoc Coloproctol G B Irel. 2015;17 (4):351–5.
28. Shihab OC, Heald RJ, Rullier E, Brown G, Holm T, Quirke P, et al. Defining the surgical planes on MRI improves surgery for cancer of the low rectum. Lancet Oncol. 2009;10 (12):1207–11.

Index

Note: Page numbers followed by f and t indicate figures and tables respectively

H. Abcarian et al. (eds.), *Complications of Anorectal Surgery*,
DOI 10.1007/978-3-319-48406-8