17 BENIGN RECTAL, ANAL, AND PERINEAL PROBLEMS

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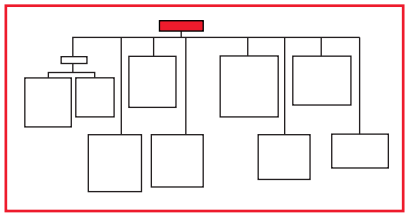
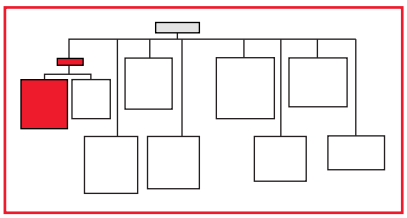


In this chapter, I briefly review the evaluation and management of common benign conditions affecting the rectum, the anus, and the perineum: hemorrhoidal disease, anal fissures, anorectal abscesses and fistulas, pilonidal disease, hidradenitis suppurativa, pruritus ani, and solitary rectal ulcer syndrome (SRUS). For proper diagnosis and treatment of these conditions, an understanding of the relevant anatomy is essential [*see Figure 1*].[1]

The dentate line divides the rectal mucosa, which is generally insensate and is lined with columnar mucosa, from the anoderm below it, which is highly sensitive (because of the somatic innervation provided by the inferior hemorrhoidal nerve) and is lined with modified squamous mucosa. The anal canal is surrounded by two muscles. The internal anal sphincter, innervated by the autonomic nervous system, maintains resting anal tone and is under involuntary control. The external sphincter, innervated by somatic nerve fibers, generates the voluntary anal squeeze and plays the key role in maintaining anal continence. The area surrounding the anorectum is divided into four spaces—perianal, ischioanal, supralevator, and intersphincteric (intermuscular)—familiarity with which is particularly important in the evaluation of perirectal abscesses and fistulas.

Hemorrhoids



Hemorrhoids are fibromuscular cushions that line the anal canal. They are classically found in three locations: right anterior, right posterior, and left lateral.[2,3] On occasion, smaller secondary cushions may be found lying between these main cushions. Contrary to popular belief, hemorrhoids are not related to the superior hemorrhoidal artery and vein, to the portal vein, or to portal hypertension.[4] In fact, hemorrhoids are part of the normal anal anatomy. Their engorgement during straining or performance of the Valsalva maneuver is a component of the normal mechanism of fecal continence: it most likely completes the occlusion of the anal canal and prevents stool loss associated with nondefecatory straining. In the medical literature, however, the term hemorrhoid is used almost exclusively to refer to pathologic hemorrhoids, and I follow this usage throughout the remainder of the chapter.

Hemorrhoids are broadly classified as either internal or external. Internal hemorrhoids are found proximal to the dentate line, whereas external hemorrhoids occur distally [*see Figure 2*]. External hemorrhoids are redundant folds of perianal skin that generally derive from previous anal swelling; they remain asymptomatic unless they are thrombosed and are treated entirely differently from internal hemorrhoids.

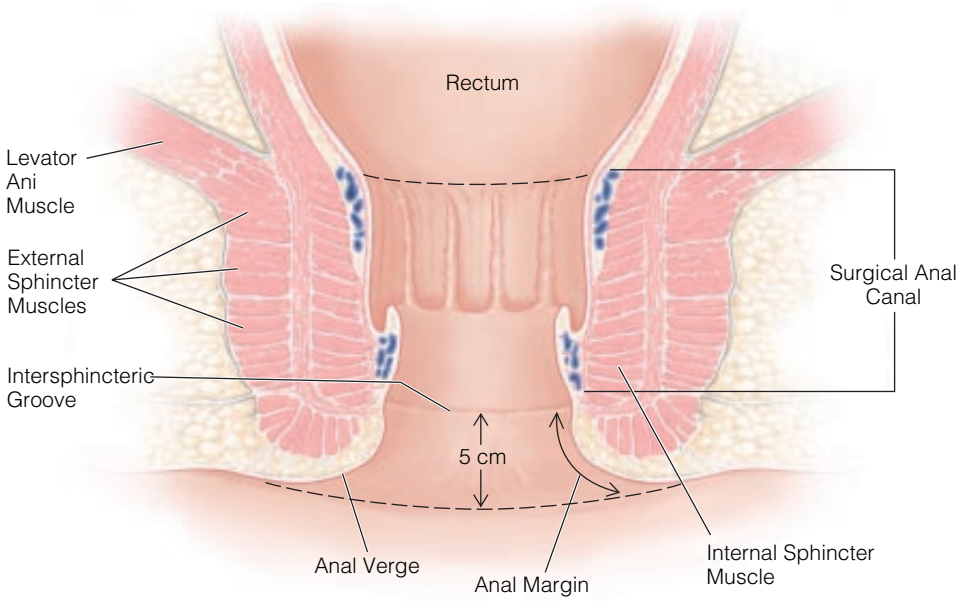
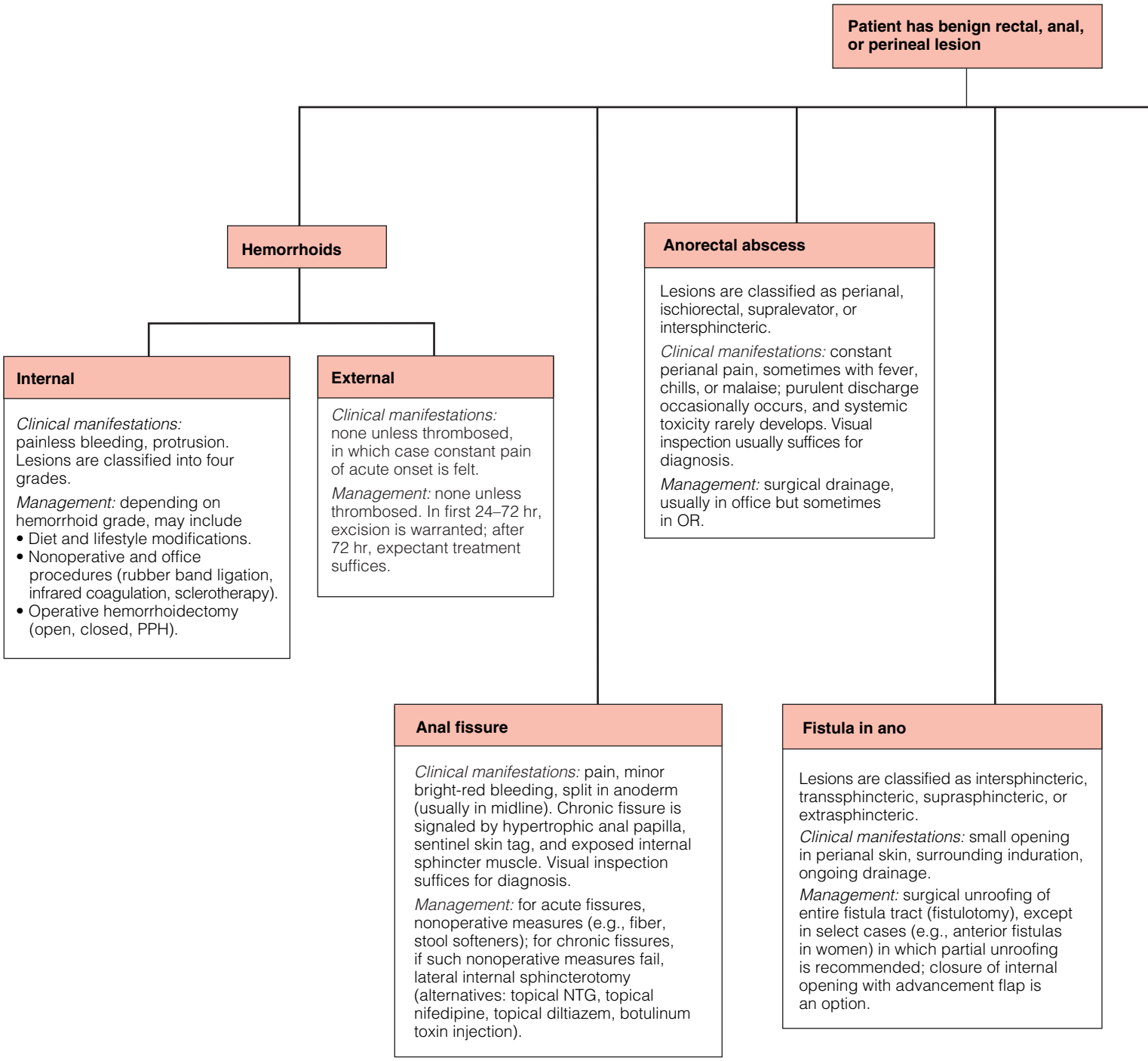
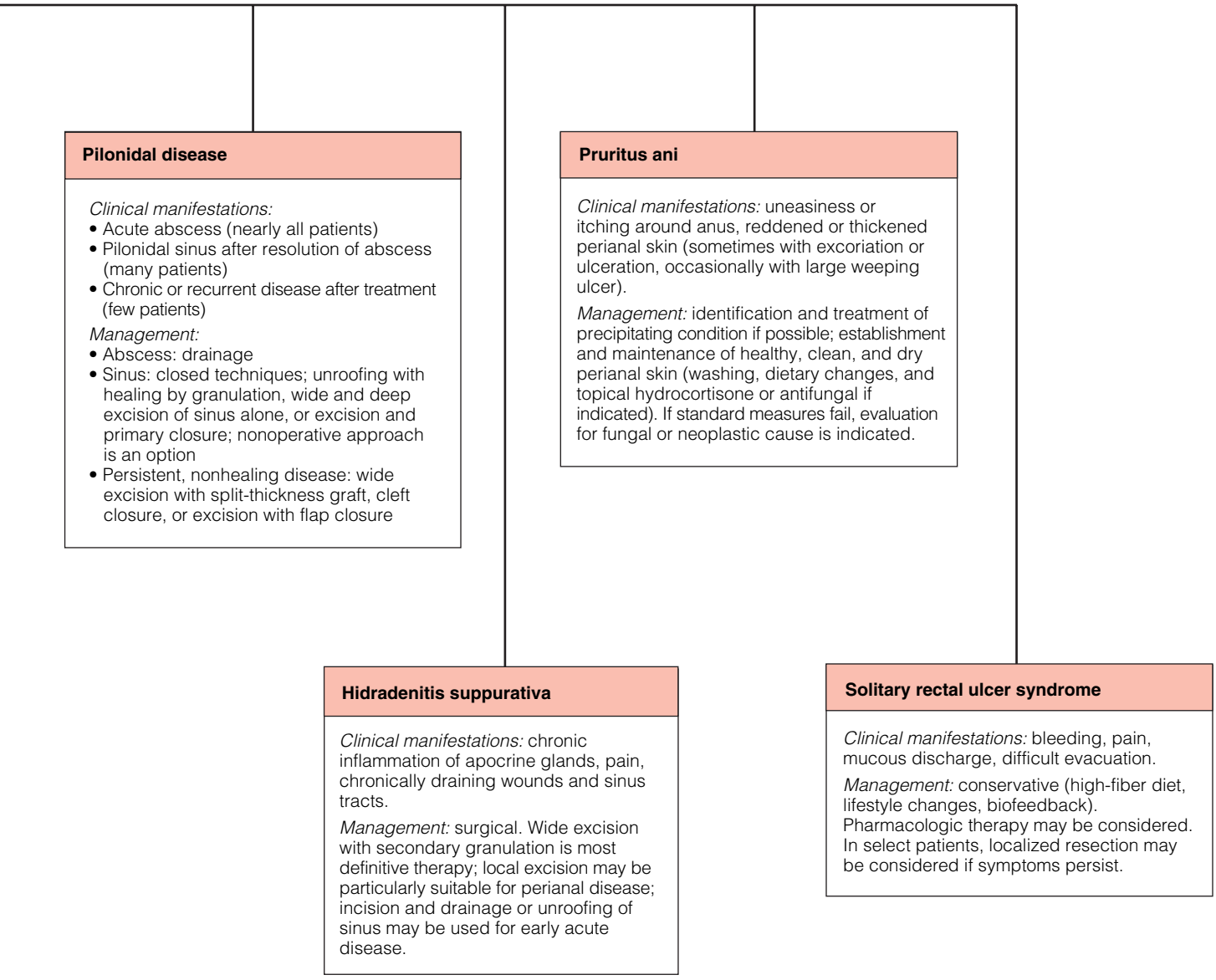


Figure 1 **Depicted is the anatomy of the anal canal.[3,5]**

Approach to Benign Rectal, Anal, and Perineal Problems





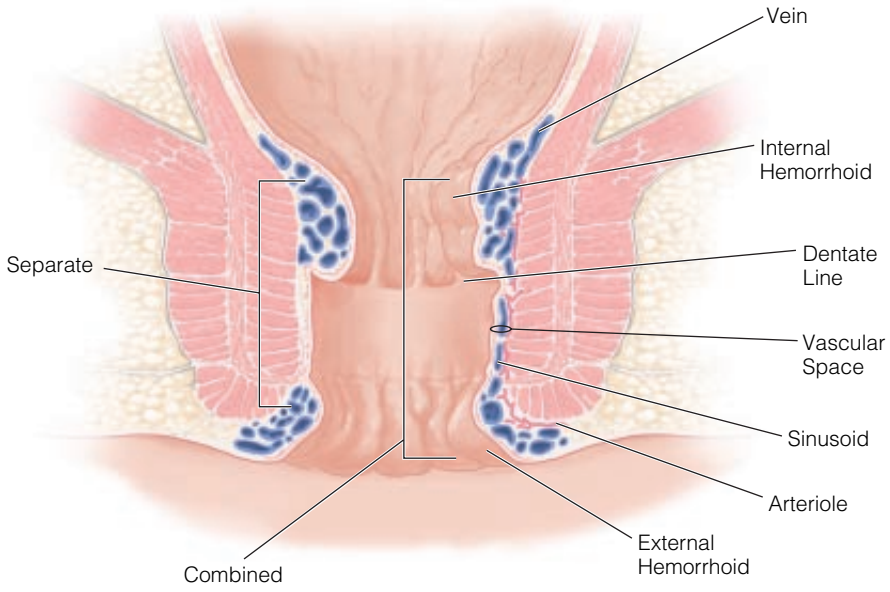


Figure 2 **External and internal hemorrhoids may occur either separately (left) or in combination (right).**

INTERNAL

Clinical Evaluation

Internal hemorrhoidal disease is manifested by two main symptoms: painless bleeding and protrusion.[4] Pain is rare because internal hemorrhoids originate above the dentate line in the insensate rectal mucosa. The most popular etiologic theory states that hemorrhoids result from chronic straining at defecation (with upright posture and heavy lifting possibly playing contributing roles as well). This straining not only causes hemorrhoidal engorgement but also generates forces that reduce the attachment between the hemorrhoids and the anal muscular wall. Continued straining causes further engorgement and bleeding, as well as hemorrhoidal prolapse. Internal hemorrhoids are classified into four grades on the basis of clinical findings and symptoms. Grade 1 represents bleeding without prolapse; grade 2, prolapse that spontaneously reduces; grade 3, prolapse necessitating manual reduction; and grade 4, irreducible prolapse.

Questioning often reveals a long history of constipation and straining at defecation. Many patients with internal hemorrhoids are extensive bathroom readers, spending many hours in the bathroom each week. Symptoms start with painless bleeding and may progress to anal protrusion. The physical examination begins with visual inspection and may reveal prolapsing hemorrhoidal tissue appearing as a rosette of three distinct pink-purple hemorrhoidal groups. Hemorrhoidal prolapse must be distinguished from true full-thickness rectal prolapse. When hemorrhoidal prolapse is not present, anoscopy typically reveals redundant anorectal mucosa just proximal to the dentate line in the classic locations.

Surgical treatment of hemorrhoids in a patient whose main disease process is Crohn disease, a pelvic floor abnormality, or fissure disease invariably yields imperfect results. It is especially important to recognize the anal pain and spasm associated with anal fissure because in patients with this problem, excision of the hemorrhoids without concomitant management of the fissure leads to increased postoperative pain and poor wound healing. Not uncommonly, patients are treated for hemorrhoids when the true primary condition is fissure disease.

Management

Treatment of symptomatic internal hemorrhoids ranges from simple reassurance to operative hemorrhoid excision, depending on hemorrhoid grade [*see Table 1*]. Therapies may be classified into three categories: (1) diet and lifestyle modifications, (2) nonoperative and office procedures, and (3) operative hemorrhoidectomies.

Diet and lifestyle modifications For all patients with grade 1 or 2 hemorrhoids and most patients with grade 3 hemorrhoids, treatment should begin with efforts to correct constipation. Recommendations should include a high-fiber diet, liberal water intake (six to eight 8 oz glasses of water daily), and fiber supplements (e.g., psyllium, methylcellulose, calcium polycarbophyl, and gum). Sitz baths are recommended for their soothing effect and their ability to relax the anal sphincter muscles. Topical creams may reduce some of the associated symptoms, though they do not affect the hemorrhoids themselves. Suppositories are not helpful, because they deliver medication to the rectum, not the anus. Patients are instructed to avoid prolonged trips to the bathroom and to read in the bathroom only when sitting atop the toilet lid.

Table 1 Treatment Alternatives for Hemorrhoids

Treatment	Internal Hemorrhoids				External Hemorrhoids
	Grade				
	1	2	3	4	
Diet modification	X				
Sclerotherapy	X	X			
Infrared coagulation	X	X	(X)		
Rubber band ligation	(X)	X	X		
Stapled hemorrhoidopexy (PPH)		X	X		
Excisional hemorrhoidectomy		(X)	X	X	X

(X)—selected patients PPH—procedure for prolapsing hemorrhoids

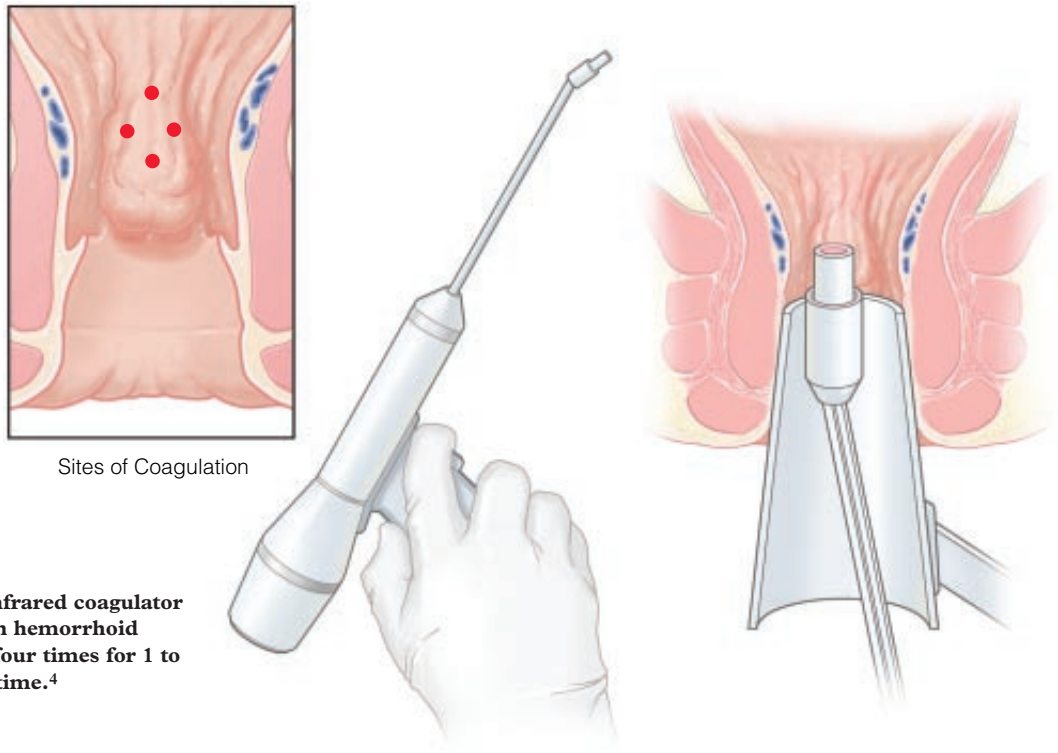


Figure 3 **The infrared coagulator is applied to each hemorrhoid bundle three or four times for 1 to 1.5 seconds at a time.[4]**

Nonoperative and office procedures If initial diet and lifestyle modifications are not effective, there are a number of nonoperative therapies that may be tried next. At present, the main recommended options are rubber band ligation, infrared photocoagulation, and sclerotherapy.

Rubber band ligation. Ligation of hemorrhoids with elastic bands is the method most commonly used in the outpatient setting [*see* 5:37 *Anal Procedures for Benign Disease*].[5] It is successful in two thirds to three quarters of all patients with grade 1 or 2 hemorrhoids.[2] Repeated banding may be necessary to resolve all symptoms. On rare occasions, patients do not respond to banding at all or cannot tolerate it. Some of these patients will respond to infrared coagulation; others may require formal hemorrhoidectomy.

Complications of rubber band ligation include bleeding, pain, thrombosis, and life-threatening perineal sepsis.[5,6] The cardinal signs of perineal sepsis are significant pain, fever, and difficult urination. Patients in whom any of these symptoms develop require urgent evaluation and treatment with broad-spectrum antibiotics, coupled with selective debridement of any necrotic anal tissue.

Infrared coagulation. The infrared coagulator generates infrared radiation, which coagulates tissue protein and evaporates water from cells.[7] The extent of the tissue destruction depends on the intensity and duration of the application. An anoscopic examination is performed, and the infrared coagulator is applied to the apex of each hemorrhoid at the top of the anal canal [*see Figure 3*]. Each hemorrhoid bundle receives three to four applications, each lasting 1 to 1.5 seconds.

The infrared coagulator was designed to decrease blood flow to the region. It is not particularly effective for treating large amounts of prolapsing tissue; it is most useful for treating grade 1 and small grade 2 hemorrhoids. Overall, it is slightly less painful than rubber banding. Infrared coagulation is especially beneficial for patients in whom rubber band ligation fails because of pain or who have symptomatic internal hemorrhoids that are too small to band.

Sclerotherapy. Sclerotherapy was once commonly employed to treat internal hemorrhoids, but with the advent of rubber band ligation, it has become less popular. The technique involves injection of a sclerosant into the anorectal submucosa to decrease vascularity and increase fibrosis. The agents used include phenol in oil, sodium morrhuate, and quinine urea.[2] Hemorrhoids are identified via anoscopy, and the sclerosant is infiltrated at the apex of the hemorrhoid (at the proximal anal rectal ring). After injection, patients occasionally experience a dull ache lasting 24 to 48 hours, but substantial bleeding and other significant complications are uncommon. Misplacement of the sclerosant, resulting in significant perianal infection and fibrosis, has been reported, albeit rarely.

Comparison of methods. A meta-analysis comparing a variety of modalities used to treat hemorrhoids found that rubber band ligation was superior to sclerotherapy for grade 1 through grade 3 hemorrhoids.[8] Patients who were treated with sclerotherapy or infrared coagulation were more likely to require further treatment than those treated with rubber band ligation. The authors concluded that hemorrhoidectomy should be reserved for patients in whom rubber band ligation or infrared coagulation fails or who have associated external hemorrhoidal disease.

Hemorrhoidectomies Various hemorrhoidectomies have been developed throughout the years. Although they differ with respect to detail, they incorporate similar basic principles—namely, reduction of blood flow to the anorectal ring, removal of redundant hemorrhoidal tissue, and fixation of redundant mucosa and anoderm.[9]

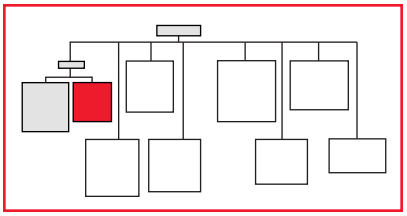
Open and closed hemorrhoidectomy. In the United Kingdom, open (Milligan and Morgan) hemorrhoidectomy, in which hemorrhoids are ligated and excised with the wound left open, is the most commonly performed operative excision procedure. In the United States, closed (Ferguson) hemorrhoidectomy is most commonly performed[10]; this procedure is described more fully elsewhere [*see* 5:37 *Anal Procedures for Benign Disease*]. With either type of hemorrhoidectomy, one, two, or three hemor-

rhoidal bundles may be excised. In performing a closed hemorrhoidectomy, it is essential to excise as little of the anoderm as possible: if large amounts are excised, closing the anal wounds or secondary healing can result in significant postoperative pain and perhaps even long-term anal stenosis.

Stapled hemorrhoidectomy. Stapled hemorrhoidectomy or anoplasty (commonly known as the procedure for prolapsing hemorrhoids [PPH]) is increasingly being performed as an alternative to standard open or closed hemorrhoidectomy with the aim of reducing the pain associated with traditional surgical techniques.[9] PPH involves transanal circular stapling of redundant anorectal mucosa with a modified circular stapling instrument (PPH-01, Ethicon Endo-Surgery Inc., Cincinnati, Ohio).[11] Because no incisions are made in the somatically innervated, highly sensitive anoderm, there should, theoretically, be significantly less postoperative pain.

Randomized, prospective trials comparing PPH with various operative hemorrhoidectomies and other therapies found it to be associated with significantly less pain then conventional treatments and to have similar complication rates.[9] PPH may, however, have a greater potential for disastrous complications (e.g., rectovaginal or rectourethral fistula from inclusion of too much tissue within the purse-string suture, perforation caused by placing the purse-string too high, and incontinence caused by placing the purse-string too low or too deep). Bleeding also remains a problem with PPH, and leaks have been reported. Finally, stapled hemorrhoidectomy has not yet been adequately compared with office procedures for grade 1 and 2 hemorrhoids and thus should not replace these techniques for treatment of minimally symptomatic hemorrhoidal disease.

EXTERNAL



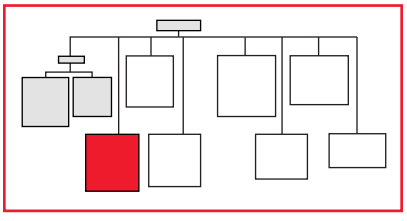
Clinical Evaluation

External hemorrhoids are asymptomatic except when secondary thrombosis occurs. Thrombosis may result from defecatory straining or extreme physical activity, or it may be a random event.[1] Patients present with constant anal pain of acute onset and often report feeling the sensation of sitting on a tender marble. Physical examination identifies the external thrombosis as a purple mass at the anal verge.

Management

Treatment depends on the patient's symptoms [*see Figure 4*].[4] In the first 24 to 72 hours after the onset of thrombosis, pain increases, and excision is warranted. After 72 hours, pain generally diminishes, and expectant treatment is all that is necessary. Patients should be advised that some drainage may occur. If operative treatment is chosen, the entire thrombosed hemorrhoid is excised with the patient under local anesthesia. Incision and drainage of the clot are avoided because they typically lead to rethrombosis and exacerbation of symptoms.

Anal Fissure



Anal fissures are tears or splits in the anoderm just distal to the dentate line.[12] They are characterized as acute or chronic. Generally, acute fissures are caused by the mechanical force generated by the passage of a large, hard bowel movement through an anal canal that is too small to accommodate it safely and easily (though they can also be caused by diarrhea). These mechanical forces usually cause a split to occur in the posterior midline: 90% of the fissures in females and 99% of those in males are located posteriorly. Either decreased local blood flow or increased mechanical stress may account for the propensity of these fissures to occur in this location.[13] Repeated injury (e.g., from hard or watery bowel movements) may result in the development of a chronic fissure.

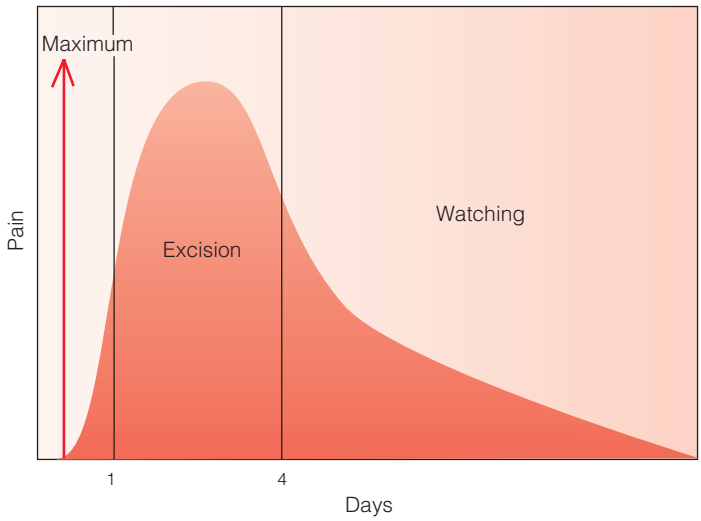


Figure 4 **Depicted is the timing of excision of thrombosed external hemorrhoids.[36]**

CLINICAL EVALUATION

Symptoms associated with anal fissures include anal pain and bright-red rectal bleeding after bowel movements. The pain is usually described as a knifelike or tearing sensation, and the associated anal sphincter spasm may persist for several hours after each bowel movement. The bleeding is usually minor and is seen mainly on the toilet paper.

Physical examination is difficult because the patient has an extremely tender anus and is fearful of further pain. Often, visual inspection with gentle eversion of the anoderm in the posterior midline is all that is required. Physical findings include a split in the anoderm approximately 1 cm long in the posterior midline just distal to the dentate line. In chronic fissures, the classic triad may be present: hypertrophy of the anal papilla, an anal fissure, and a sentinel skin tag [*see Figure 5*]. Once an anal fissure has been diagnosed, further examination is very painful, unrewarding, and unnecessary; more extensive investigations can be performed after the fissure has healed.

Multiple fissures are unusual, as are fissures that occur away from the anterior or posterior midline; either should raise suspicions that other problems may be present [*see Figure 6*].

MANAGEMENT

Acute fissures usually have been present for less than 4 to 6 weeks. As a rule, they are treated nonoperatively: fiber supplements, stool softeners, and generous intake of water, along with sitz baths and local anesthetic ointments, rapidly alleviate symptoms and usually bring about complete healing. Anal suppositories are avoided both because they are painful and because they rest in the rectum rather than the anal canal.

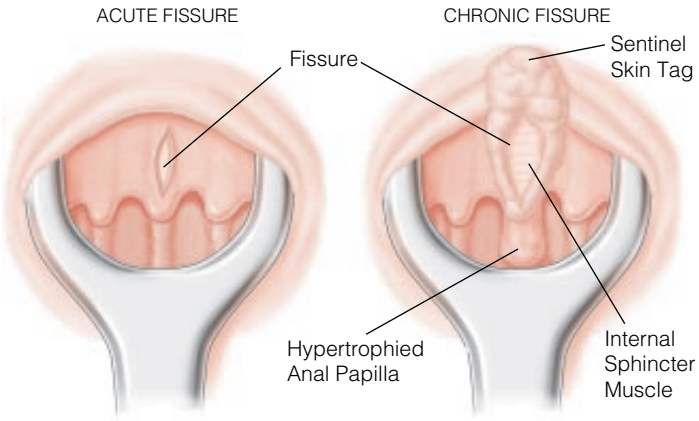


Figure 5 **Chronic anal fissures, as opposed to acute fissures, are characterized by hypertrophy of the anal papilla, a sentinel skin tag, rolled skin edges, and exposed internal anal sphincter muscle.**

Chronic fissures have been present for periods longer than 4 to 6 weeks. As noted (see above), they are typically characterized by an associated hypertrophied papilla, a skin tag, rolled skin edges, and exposed internal anal sphincter muscle at the base of the fissure. Chronic fissures respond less well to nonoperative measures than acute fissures do. The surgical procedure most frequently performed for anal fissure is lateral internal anal sphincterotomy [*see* 5:37 *Anal Procedures for Benign Disease*],[14] which results in cure in 95% to 98% of patients. Complications of this procedure include incontinence to flatus (0% to 18% of cases), soiling (0% to 7%), fecal incontinence (0% to 0.17%), and various other problems (0% to 7%).[14]

Several therapeutic alternatives for anal fissures have been proposed with the aim of avoiding the need for operation and the consequent risk of surgical complications.[15] On the basis of the theory that a fissure is actually an ischemic ulcer of the anoderm, topical nitroglycerin (NTG) ointment has been used to treat fissures, with success rates ranging from 48% to 78%. When metabolized, NTG releases nitric oxide, which is believed to be an inhibitory neurotransmitter for smooth muscle. The resulting neurogenic relaxation of the internal sphincter brings about a reduction in anal canal pressure, which diminishes pain and spasm. Typically, NTG is given in a concentration of 0.2% three to five times daily, but concentrations as high as 0.5% have been recommended. In practice, dosing is limited by NTG's side effects, which arise in as many as 88% of patients. Headaches are the predominant complaint, but dizziness, lightheadedness, and hypotension have also been reported. Caution must be exercised when NTG therapy is employed in patients receiving cardiac medications or those with sensitivities to nitrates. A meta-analysis comparing topical NTG therapy with sphincterotomy demonstrated that sphincterotomy results in better healing of chronic fissures.[16]

Another nonsurgical option is the use of nifedipine gel or ointment, which also has met with varying degrees of success. Nifedipine acts as a calcium antagonist, preventing calcium from flowing into the sarcoplasm of smooth muscle and thereby reducing local demand for oxygen and mechanical contraction of the muscle.[12] Like NTG ointment, nifedipine ointment is usually applied topically in a 0.2% concentration, but it seems to have fewer side effects than NTG does. Nifedipine should be used with caution in cardiac patients and patients who have demonstrated previous sensitivities. One multicenter study reported a 95% complete healing rate after 21 days of treatment.[12]

Topical 2% diltiazem has also been employed to treat chronic anal fissures, yielding a 67% healing rate.[12]

Botulinum toxin has been reported to facilitate healing in 78% to 90% of anal fissure patients, with an 8% recurrence rate at 6 months.[15,17] The toxin, produced by the bacterium *Clostridium botulinum,* acts by inhibiting the release of acetylcholine at the presynaptic membrane. The resulting blockage of neurotransmission diminishes or eliminates spasms and contractions of the sphincter mechanism. Typically, 2.5 to 10 units are injected at two to four sites in the internal sphincter at the level of the dentate line.[18] Pain relief is generally noted within 24 hours, though it still takes days for the fissure to heal. Postinjection incontinence is rare.[12] The major drawback is the cost of the toxin, which may be as high as $400 per 100 unit vial.

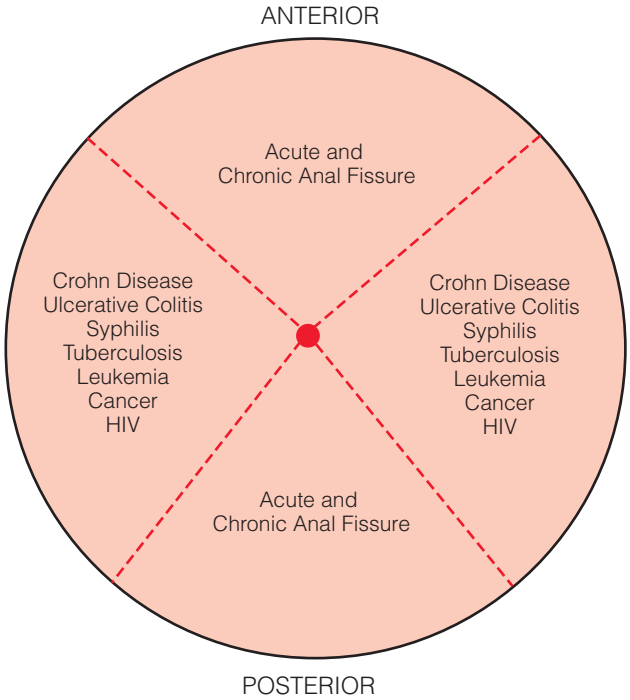
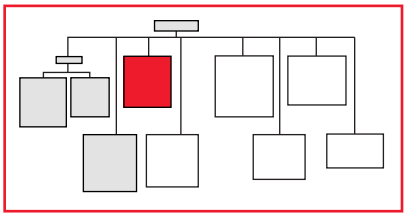


Figure 6 **Fissures occurring away from the anterior or posterior midline are likely to be associated with other conditions.[4]**

Anorectal Abscess



Anorectal abscesses, like abscesses elsewhere in the body, are the result of local, walled-off infections. Most perirectal abscesses are of cryptogenic origin—that is, they begin as infections in the anal glands that surround the anal canal and empty into the anal crypts at the dentate line.[18,19] It is thought that the ducts leading to and from these glands become obstructed by feces or traumatized tissue, and a secondary infection then develops that follows the path of least resistance, resulting in an anorectal abscess.

CLINICAL EVALUATION

Abscesses are categorized according to the space in which they occur: perianal, ischioanal, supralevator, or intersphincteric (intermuscular) [*see Figure 7*]. Perianal abscesses are the most

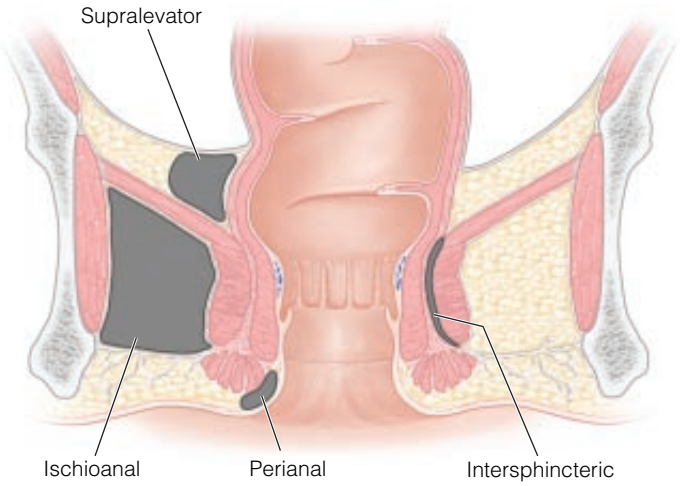


Figure 7 **Anorectal abscesses are classified according to the space in which they develop.**

common—together with ischioanal abscesses, they account for more than 90% of perianal infections. Perianal abscesses occur in the perianal space immediately adjacent to the anal verge. Ischioanal abscesses are larger and often more complex than their perianal counterparts, and they usually manifest themselves as a tender buttock mass. Supralevator abscesses occur above the levator ani muscles and are characterized by poorly localized pain; they are exceedingly rare. Intersphincteric abscesses occur in the plane between the internal and external sphincter muscles, high within the anal canal; these are also rare. The location of an abscess is important in that it dictates subsequent therapy.[20]

Regardless of location, all anorectal abscesses are associated with constant perianal pain. Accompanying symptoms may include fever, chills, and malaise. In rare cases, systemic toxicity may be evident. The history reveals rectal pain of gradual onset that progressively worsens until the time of presentation. Occasionally, spontaneous drainage decompresses the abscess, and the patient presents with a purulent discharge.

As with fissures, visual inspection of the perineum often clinches the diagnosis. A fluctuant, erythematous, tender area identifies the abscess. In the rare event of a supralevator or intersphincteric abscess, there may be no external manifestations. When this is the case, the presence of a tender mass on digital examination above the anal canal, either adjacent to the rectal ampulla (supralevator abscess) or within the anal canal (intersphincteric abscess), provides the clue to the diagnosis.

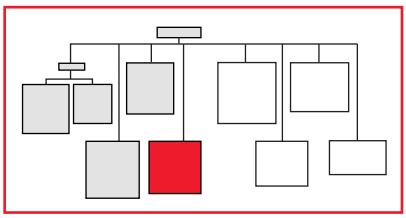
MANAGEMENT

Treatment of anorectal abscesses consists of adequate drainage performed in the office, in the emergency department, or in the operating room.[18] Most abscesses can be drained in the office, but recurrent or complex abscesses, abscesses in immunosuppressed hosts (including some diabetic patients), intersphincteric abscesses, and supralevator abscesses are more appropriately drained in the OR.

Adequate drainage is essential and may be established in several ways. One method is to place a catheter (e.g., a 10 to 16 French Pezzar catheter) through a small stab incision [*see* 5:37 *Anal Procedures for Benign Disease*].[18] This measure allows the pus to drain through the catheter as the cavity closes down. A second option involves creating a larger elliptical incision.[20] Unroofing the abscess cavity allows it to heal without any need for packing. A small incision should be avoided because it would require painful packing to keep the skin open until the abscess cavity heals.

The most difficult abscess to diagnose and manage is a deep postanal space abscess. Such abscesses are caused by fistulization from the posterior anal canal, usually in the bed of a chronic posterior fissure. The patient is highly uncomfortable and febrile, but there is no apparent sign of a problem perianally. A simple digital examination pushing posteriorly towards the coccyx and the deep postanal space will evoke severe pain, and this response should lead the examiner to suspect the diagnosis. The patient should be taken to the OR and anesthetized. On digital examination, the deep postanal space is often felt to be bulging. The diagnosis is confirmed by aspirating the space with an 18-gauge needle. Once the diagnosis is confirmed, an incision is made in the perianal skin posterior to the anal verge and deepened into the space, and the space is drained. In selected patients, a cutting seton is placed from the primary site in the posterior anal canal directly into the deep postanal space abscess. Horseshoe fistulas are dealt with as described elsewhere [*see* 5:37 *Anal Procedures for Benign Disease*].

Fistula in Ano



An anal fistula is a communication between the anal canal and the perianal skin. It usually begins in a crypt at the dentate line and follows a course either between the internal and external sphincters (the most common location), resulting in an ischioanal abscess, or above the sphincters, leading to a supralevator abscess.[18,20] After drainage of an abscess, one of three things typically occurs if a fistula is present: (1) the fistula heals spontaneously, and the patient

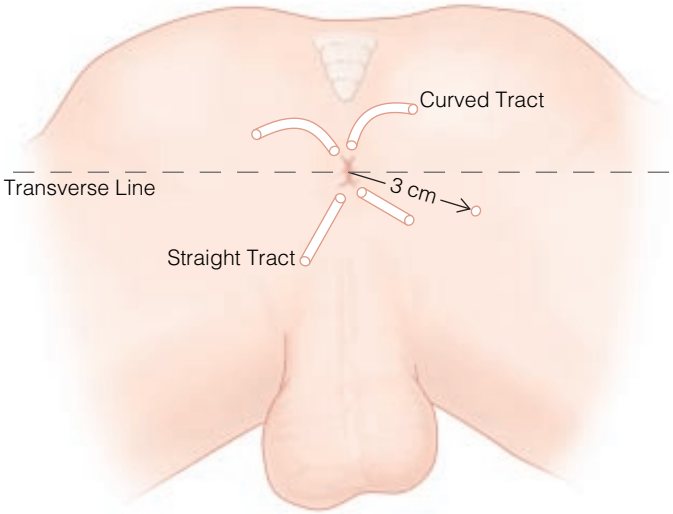


Figure 8 **The relation of the external opening of an anal fistula to the internal opening is suggested by Goodsall's rule. When the external opening is posterior to a line drawn transversely across the perineum, the fistula typically follows a curved course to an internal opening in the posterior midline. When the external opening is anterior to this line, the fistula typically follows a short, straight course to an internal opening in the nearest crypt (though this often does not hold true if the anterior exterior opening is more than 2 to 3 cm from the anus).[4]**

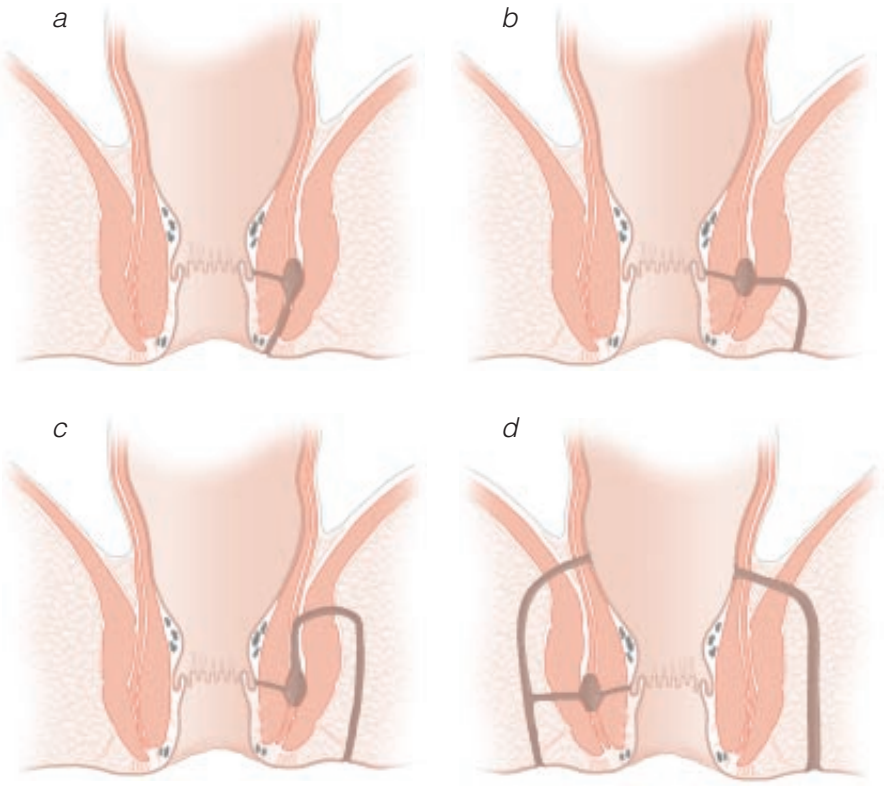


Figure 9 **Fistula in ano is classified on the basis of its relation to the anal sphincter muscles. Shown are (*a*) intersphincteric fistula, (*b*) transsphincteric fistula, (*c*) suprasphincteric fistula, and (*d*) extrasphincteric fistula.**

experiences no further symptoms; (2) the abscess heals, only to recur in the future; or (3) the abscess heals, but a chronic draining fistula remains. Only the third scenario is addressed here.

CLINICAL EVALUATION

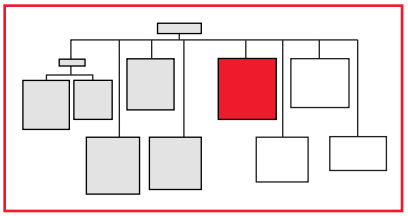
After drainage of one or more abscesses, a fistula is usually associated with chronic serosanguineous to seropurulent drainage. As long as the fistula remains open and draining, patients report little pain. If the fistula closes externally, however, an anorectal abscess may develop.

Physical examination reveals a 2 to 3 mm opening in the perianal skin, with surrounding induration. Often, a fistula tract can be palpated as a firm cord running between the external opening and the anal canal. The relationship of the external opening to the internal opening is suggested by Goodsall's rule [*see Figure 8*]. Fistulas are classified into four main categories according to their relation to the anal sphincters [*see Figure 9*]: intersphincteric, transsphincteric, suprasphincteric, and extrasphincteric.[21]

MANAGEMENT

Essentially, all chronic fistulas call for surgical treatment, which consists of unroofing the entire fistula tract (fistulotomy) and leaving the wound open to heal secondarily. Fistulas that course through significant amounts of sphincter muscle, anterior fistulas in women, and fistulas associated with inflammatory bowel disease or weakened sphincter muscles, however, cannot be opened entirely, because incontinence will result. These fistulas may be partially opened, with the anal musculature left intact and encircled with a seton [*see* 5:37 *Anal Procedures for Benign Disease*].[20] Although some surgeons sequentially tighten the seton (making it a cutting seton), I prefer not to, because of the resultant patient discomfort. Another surgical option is to close the internal fistula opening with an advancement flap.[22] Advancement flap repairs result in high success rates with minimal effects on continence.

Pilonidal Disease



The term pilonidal disease is derived from the Latin words *pilus* ("hair") and *nidus* ("nest").[23] It denotes a chronic subcutaneous infection and foreign-body reaction to hairs embedded in the skin or to abnormalities of hair follicles in the natal cleft.[23] Pilonidal disease is most commonly seen in men between the onset of puberty and 40 years of age and in obese persons.[23]

CLINICAL EVALUATION

Pilonidal disease has three common presentations. First, nearly all patients experience an episode of acute abscess formation. Second, after the abscess resolves, either spontaneously or with medical assistance, a pilonidal sinus tract develops in many cases. Third, although most of these sinus tracts resolve, chronic disease or recurrent disease after treatment develops in a small minority of cases.

Physical examination typically reveals one or more small (1 to 2 mm) dermal pits at the base of the intergluteal cleft [*see Figure 10*]. Tracking from the pits (usually proceeding in a cranial and lateral direction) appears as areas of induration. If there is an associated abscess, the diseased area may be tender and erythematous, and draining pus may be evident. The more extensive the disease, the more prominent the findings. Treatment varies according to the stage of the disease.

MANAGEMENT

Abscesses must be drained. In one study, incision and drainage in an office setting with local anesthesia led to healing in 60% of patients.[24] As many as 40% of acute pilonidal abscesses treated with incision and drainage develop into chronic sinus-

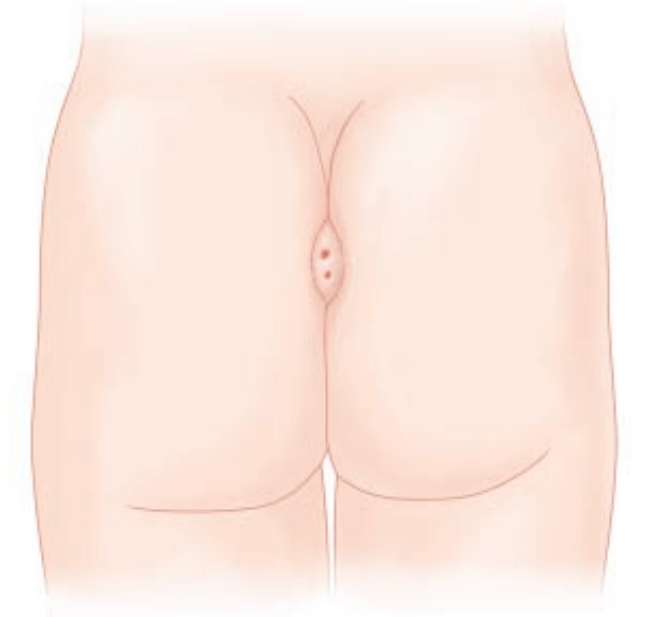


Figure 10 **Patients with pilonidal disease generally have one or two dermal pits in the intergluteal cleft, often associated with a sinus and an abscess.**

es for which additional treatment is necessary.

Several different approaches have been employed in the surgical treatment of pilonidal sinus tracts. A review of articles published over a period of 30 years on the treatment of pilonidal disease divided the procedures into the following four broad categories and reported the following findings.[25]

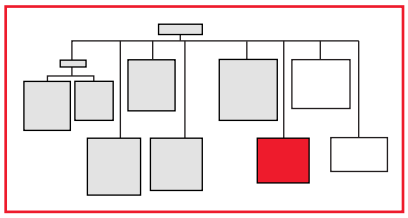
1. Closed techniques (coring out follicles and brushing the tracts). These techniques necessitated shaving of the area but could be performed on an outpatient basis. Mean healing time was about 40 days, and recurrence rates were slightly higher than those seen with other forms of treatment.
2. Laying open (unroofing) the tracts with healing by granulation. This approach resulted in average healing times of 48 days and necessitated frequent outpatient dressing changes. The incidence of recurrent sinus formation was lower than 13%.
3. Wide and deep excision of the sinus alone. This procedure resulted in an average healing time of 72 days. The recurrence rate was similar to that of simple unroofing of the sinus tract with healing by granulation.
4. Excision and primary closure. This technique resulted in wound healing within 2 weeks in successful cases (19 days overall). However, primary wound healing failed in as many as 30% of patients, and the average recurrence rate was 15%.

A nonoperative or conservative approach—involving meticulous hair control (through natal cleft shaving), improved perineal hygiene, and limited lateral incision and drainage for treatment of abscess—has been suggested as an alternative to conventional excision.[26] This approach has brought about a significant reduction in the number of excisional procedures and occupied-bed days.

Even with proper treatment of pilonidal abscesses and sinuses, a small number of patients are left with persistent, nonhealing wounds. A number of more aggressive approaches have been advocated for treatment of complex or recurrent disease, including wide excision with split-thickness skin grafting, cleft closure, and excision with flap closure.[24,27] Flap techniques, as a group, have been found to lead to primary healing within 15 days in 90% of cases.[24] These aggressive approaches nevertheless have certain disadvantages. For nearly all of them, hospitalization and general anesthesia are mandatory. In addition, as many as 50% of procedures involving the use of skin flaps for wound coverage or closure in this setting result in some loss of skin sensation or some degree of flap-tip necrosis.

My policy is to treat acute abscesses with drainage, followed by measures aimed at keeping the cleft free of hair. If a chronic sinus develops, it is managed with unroofing or, in selected patients, excision and closure. Flap procedures are reserved for cases of extensive, recurrent, and complex disease.[23]

Hidradenitis Suppurativa



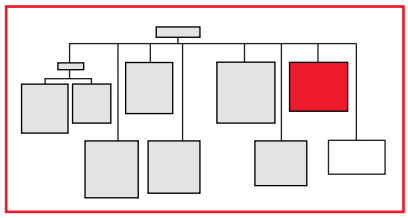
CLINICAL EVALUATION

Hidradenitis suppurativa is a chronic, recurrent inflammatory process involving the apocrine glands of the axilla, the groin, the perineum, and the perianal region.[28] The disease can result in chronically draining wounds and sinus tracts and can become quite painful and debilitating.[29] Occlusion of follicles and abnormalities of apocrine ducts are believed to be causative factors.

MANAGEMENT

Medical management may afford temporary relief of symptoms; however, most patients eventually require surgical therapy.[30] In select patients, incision and drainage or unroofing of sinuses may provide relief, but these measures should be reserved for cases of early and acute disease. Local excision provides adequate control of symptoms; however, recurrence rates higher than 50% may be anticipated. Wide excision with secondary granulation of perineal wounds constitutes the most definitive therapy and generally can be accomplished safely.[29] Perianal disease is associated with considerably lower recurrence rates than perineal disease is and thus can more often be managed with local excision alone.

Pruritus Ani



Pruritus ani is a dermatologic condition of the perianal skin characterized by uneasiness or itching in the area around the anus.[12,31] Multiple factors may predispose this area to irritation, including poor perianal hygiene (related to incontinence, diarrhea, or excessive hair), excessive moisture, irregularities of the perianal skin (from hemorrhoids, fistulas, or previous surgery), skin hypersensitivity, diet, decreased resistance to infection, and injury to the perianal skin.[31] The variety of possible causes is what often makes pruritus ani difficult to treat. Some patients improve only after the offending agents or conditions are identified and specific therapy instituted. Fortunately, many patients' symptoms can be alleviated by the application of nonspecific treatments.

CLINICAL EVALUATION

A thorough history and physical examination are necessary to suggest possible causes of pruritus. Initial inspection of the perianal skin should be conducted with gentle retraction of the buttocks under bright lighting. A characteristic finding is erythema-

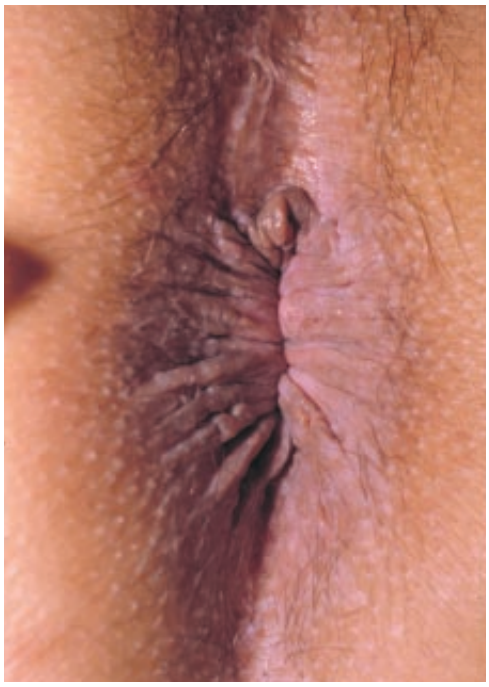


Figure 11 **A characteristic finding in idiopathic pruritus ani is erythematous or thickened perianal skin.**

tous or thickened perianal skin [*see Figure 11*]. This thickening results in a pale, whitish appearance, with accentuation of the radial anal skin creases. In addition, the skin may be excoriated or ulcerated; this process, when combined with thickening, is referred to as lichenification. Occasionally, the skin is so excoriated that a large, coalescing, weeping ulcer forms. Digital rectal examination should be performed to assess the competence of the anal sphincter both at rest and during maximal squeeze. Anoscopy and proctosigmoidoscopy may be performed after the administration of an enema.

MANAGEMENT

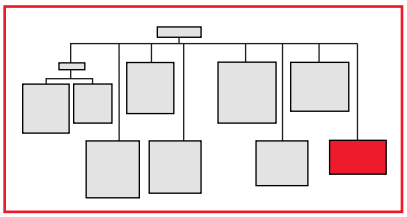
The basic principles of therapy for pruritus ani are uncomplicated. If an inciting cause can be identified, it should of course be eliminated or corrected. Frequently, the inciting cause is elusive, but even so, most patients can still be effectively managed by applying several simple measures.

Generally, patients should keep the perianal area dry and avoid further trauma to the area. The perianal area should be gently washed, never scrubbed. After showering, the area should be patted dry or dried with a hair dryer on a low heat setting. After bowel movements, the anus should be cleaned with moistened toilet paper. Excessive rubbing or wiping should be discouraged: scratching or rubbing the anal area damages the perianal skin, making it more susceptible to irritation. Patients should also be instructed to avoid irritating foods and drinks, such as tomatoes, peppers, citrus fruits and juices, coffee, colas, beer and other alcoholic beverages, milk, nuts, popcorn, and any other foodstuffs found to be associated with increased gas, indigestion, or diarrhea. After 2 weeks, food items eliminated from the diet can be reintroduced one at a time in an attempt to identify the offending agent more specifically.

A regular bowel habit should be maintained with the help of fiber supplementation or a high-fiber diet. Patients should be instructed to eschew all proprietary creams, lotions, and emollients. If prescribed and supervised by a physician, however, a hydrocortisone cream may be applied sparingly to the affected area for a period of 1 week or less to attain control of symptoms. Occasionally, when pruritus ani is refractory to treatment, a candidal yeast infection is found to be present, in which case a trial of antifungal lotion, solution, or powder is worthwhile.

The fundamental goal in the treatment of pruritus ani is to establish and maintain intact, healthy, clean, and dry perianal skin. When standard measures fail to elicit improvement, fungal and viral cultures and perhaps even biopsy may be necessary to exclude an infectious or neoplastic cause.

Solitary Rectal Ulcer Syndrome



CLINICAL EVALUATION

SRUS is a clinical condition characterized by rectal bleeding, copious mucous discharge, anorectal pain, and difficult evacuation.[32] The name of the condition notwithstanding, SRUS patients can have a single rectal ulcer, multiple ulcers, or even no ulcers at all. When present, the ulcers usually occur on the anterior rectal wall, just above the anorectal ring; less commonly, they occur from just above the dentate line to 15 cm above it. The ulcers usually appear as shallow lesions with a punched-out gray-white base that is surrounded by hyperemia.[33]

Colitis cystica profunda (CCF) is a benign condition that is related to SRUS and is characterized by mucin-filled cysts located deep to the muscularis mucosae. CCF is a pathologic diagnosis, of which the most important aspect is differentiation of CCF from colorectal adenocarcinoma.

Precisely what causes SRUS and CCF remains unclear, but both conditions are known to be associated with chronic inflammation or trauma (e.g., internal intussception or prolapse of the rectum, direct digital trauma, or the forces associated with evacuating a hard stool). Endoscopic evaluation of the distal colon and rectum reveals the lesions described. For both CCF and SRUS, the differential diagnosis includes polyps, endometriosis, inflammatory granuloma, infection, drug-induced colitis, and mucus-producing adenocarcinoma. These entities can be confirmed or excluded by means of an adequate biopsy.

MANAGEMENT

Treatment is directed at alleviating symptoms or interfering with some of the proposed etiologic mechanisms. Conservative therapy (e.g., a high-fiber diet, lifestyle changes, and biofeedback) reduces symptoms in most patients and should be tried first. Patients without rectal intussusception are offered biofeedback for retraining their bowel function. Pharmacologic therapy (e.g., anti-inflammatory enemas and suppositories) has had only limited success but nonetheless may be worth trying before the decision is made to embark on surgery.

If symptoms persist, localized resection may be considered in selected patients. Patients with prolapse are considered for perineal procedures (i.e., mucosal or perineal proctectomy) and abdominal procedures (i.e., fixation or resection and rectopexy). Patients without prolapse may be offered excision, for which the options range from a transanal excision to a major resection with coloanal pull-through. Understandably, surgeons have been hesitant to offer surgical therapy for this benign condition; the results are often unsatisfactory.[34]

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Acknowledgment

Figures 1, 2, 3, 5, 7 through 10 Tom Moore.