

Review

HEMORRHOIDECTOMY: INDICATIONS AND RISKS

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Abstract: Hemorrhoids are a common cause of perianal complaints and affect 1-10 million people in North-America and with similar incidence in Europe. Symptomatic hemorrhoids are associated with nutrition, inherited predisposition, retention of feces with or without chronic abuse of laxatives or diarrhea. Increased pressure and shearing force in the anal canal may lead to severe changes in topography with detachment of the hemorrhoids from the internal sphincter and fibromuscular network resulting in bleeding, itching, pain and disordered anorectal function, even incontinence. The significance of hemorrhoids for anal continence (corpus cavernosum) is recognized. In most instances, hemorrhoids are treated conservatively; the surgeon is contacted when conservative measures have failed or complications, e.g., thrombosis, have occurred. 4° prolapsed internal hemorrhoids are the main indication for hemorrhoidectomy: high (Parks) or low (Milligan-Morgan) ligation with excision, closed hemorrhoidectomy (Ferguson) or stapler hemorrhoidectomy. Thrombosed external hemorrhoids are primarily treated by incision and secondary by excision. Complications after operative treatment of external thrombosed hemorrhoids are rare. After standard hemorrhoidectomy for internal hemorrhoids approximately 10% may have a complicated follow-up (bleeding, fissure, fistula, abscess, stenosis, urinary retention, soiling, incontinence); there may be concomitant disease, e.g., perianal cryptoglandular infection, causing complex fistula/abscess, which is associated with an increased risk (30-80%) for complications, e.g., incontinence. Other treatment options, e.g., sphincterotomy, anal stretch, have been accused to cause more complications, e.g., incontinence in 30-50% of cases. However, incontinence is a complex phenomenon; it is evident that an isolated single injury is normally not a sufficient cause, e.g., injury of the internal sphincter. The majority of patients may present with prior obstetric injury, perianal infection or Crohn's disease and other comorbidity. Therefore all systemic and regional disorders, causing incontinence, should be excluded before starting manometric, neurophysiological and sonographic investigations. Variation and overlap in test results, patient-, instrument- or operator-dependent factors ask for cautious interpretation. There is vast evidence that the demonstration of muscle fibers in

hemorrhoidectomy specimens is a normal feature. In conclusion, standard hemorrhoidectomy with proper indication is a safe procedure. If complications occur, it is in the interest of the patient and surgeon to perform a thorough investigation.

Key words: hemorrhoids - hemorrhoidectomy - fecal incontinence - external thrombosed hemorrhoids - anal fistula - complication - anal manometry - endoanal sonography - internal anal sphincter - ischiorectal abscess

Hemorrhoids affect many human beings in North-America and Europe. It is estimated that one to ten million Americans suffer from hemorrhoidal complaints per year. The incidence ranges from 58 % to 86 % (Haas et al. 1983; Dennison et al. 1988; Johanson and Sonnenberg 1990; Bleday et al. 1992).

Hemorrhoids are classified into external and internal hemorrhoids. Internal hemorrhoids are further subclassified into first, second, third and fourth degree internal hemorrhoids (Thomson 1981; Hulme-Moir and Bartolo 2001). Thickened cushions of mucosa and submucosa appear to the right anterior, right posterior, and left posterior position with possible variations and secondary cushions. Hemorrhoids consist of venous plexus and arterial supply embedded in a stroma of connective tissue, smooth muscle and nerves (Thomson 1975; Mosley et al. 1980).

The epithelial lining of the anal canal has been divided into three zones: cutaneous, middle, and mucosa. The cutaneous zone consists of true skin and squamous stratified epithelium. The transition zone from cutaneous to middle zones is called mucocutaneous junction and contains the anal crypts. The mucosal zone consists of true columnar epithelium and continues upward as rectal mucosa. On palpation a depression is felt between the internal and external sphincter, which should not be confused with a dip formed by the mucocutaneous junction. According to Parks Milligan and others confused the visible depression (the line of anal crypts) with the palpable groove lower in the anal canal (Parks 1956). This intersphincteric groove may be misleading in the diagnosis of sphincter defect (Sangwang and Solla 1998).

In relation to the anal canal, there are two anatomical spaces that are of clinical importance: the submucous space, lying between the mucous membrane and the internal sphincter, including the internal hemorrhoidal plexus, covered by glandular rectal epithelium, and the perianal space which contains the external hemorrhoid plexus and the subcutaneous external sphincter (Milligan et al. 1937).

The internal sphincter forms a wall of the whole anal canal though the subcutaneous external sphincter may occupy the terminal position. Fibers of the conjoined longitudinal muscle, which acts as supportive structure for the venous plexus and the anal submucosa and mucosa penetrate the internal and external sphincter forming a fibroelastic network (Thomson 1975; Haas and Fox 1977; Mosley et al. 1980). The longitudinal muscle is attached to the anal skin and anal margin. Through the gap between the two sphincter muscles passes the most medial of the terminating strands of the longitudinal muscle, which gains firm attachment to the skin over the anal verge. The groove between the hemorrhoidal plexuses is caused by the adherence of a fibromuscular band to the mucosa, which was called mucosal suspensory ligament by Parks (1956). It consists of muscularis mucosae, muscle-fibers from the internal sphincter, fibrous tissue from the fascia surrounding the internal sphincter, gains attachment to the mucosa of the anal crypts and divides the subepithelial space into the superior compartment containing the internal hemorrhoidal plexus and the inferior compartment, marginal or perianal space which contains the external hemorrhoidal plexus (Parks 1956). Thomson (1975) noted that this smooth muscle (corrugator cutis ani, musculus submucosae ani, mucosal suspensory ligament, musculus canal-is ani) was always present in normal cadavers and hemorrhoidectomy specimens. The anatomical description of the conjoined longitudinal muscle layer and the musculus canalis ani with its relation to the sphincter muscles, rectal mucosa suggested a role in normal defecation and internal sphincter contraction (Fine and Lawes 1940; Shropshire 1960; Lawson 1974; Thomson 1975; Hansen 1976; Haas and Fox 1977; Lunniss and Phillips 1992; Loder et al. 1994). Stelzner (1992) emphasized that with regard to the anorectal continence we are focused on the sphincter muscles not recognizing the importance of the corpus cavernosum and the network of the longitudinal muscle mesh. Stieve reported that the internal sphincter might not completely close the anal canal (Stieve 1928; Stieve 1930). Stelzner has introduced the term corpus cavernosum with his description of the arteriovenous shunts in the hemorrhoids (Stelzner 1962). The importance of the cushions for the continence has been elucidated in further studies and it is now suggested that the vascular filling is contributing 15 to 20 % to the resting anal pressure (Stelzner et al. 1966; Thomson 1975; Hansen 1977; Gibbons et al. 1986; Lestar et al. 1989). This has been supported by the finding that excision of the piles in he-

morrhoidectomy may impair continence to rectal saline infusion (Read et al. 1982).

ETIOLOGY

The pathogenesis of hemorrhoids is not yet finally elucidated. It has been suggested by several authors that there is a genetic link, which would explain the association between hemorrhoids and hernia and prolapse of the genitourinary system or varicose veins (Stelzner 1962; Burkitt 1975; Loder et al. 1994). Environmental factors, e.g., low-fiber diet, constipation, repeated and prolonged straining, hard stool, chronic use of laxatives, have been identified to support the development of hemorrhoids (Burkitt 1975; Haas et al. 1984). In fact, straining associated with constipation, gynecological prolapse and prolapse of the anal mucosa with loose stools, diarrhea or ulcerative colitis can be found in the patient history (Stern 1964). Chronic abuse of laxatives has been observed in 22.2% of males and 76.4% of females prior to hemorrhoidectomy (Kouba 1980). In recent reports it has been proposed that hemorrhoids are caused by pathologic slippage of the normal lining of the anal canal together with changes in connective tissue, e.g., loss of organization, muscular hypertrophy, fragmentation of the muscle and elastin components which may be aggravated by stress during defecation (Gass and Adams 1950; Thomson 1975; Haas and Fox 1980; Haas et al. 1983; Haas et al. 1984; Loder et al. 1994). Venous stasis, ischemia, edema, clot formation may be responsible for complications of the hemorrhoidal disease, e.g., superficial ulceration, fissure formation, hemorrhagic infarction, external thrombosed hemorrhoids (Dayal and DeLellis 1989; Jongen et al. 2003).

SYMPTOMS

Patients with symptomatic hemorrhoids may have bleeding, prolapse, pain, itching, mucous discharge, leakage of anal contents, soiling, rectal dysfunction, incontinence, feeling of a lump and constipation (Stern 1964; Ganchrow et al. 1971; Thomson 1994).

PATHOPHYSIOLOGY

The anal cushions resemble erectile tissue containing large blood spaced fed by arterioles (Thomson 1975). In normal subjects they may help to preserve continence by forming an expansive seal (Gibbons et al. 1986). Forty % of patients with hemorrhoids, especially non-prolapsing hemorrhoids, complain of obstructed defecation and vigorous straining (Hancock 1977; Sun et al. 1990) leading to increased anal pressure, lower rectal compliance and more perineal descent (Arabi et al. 1977; Hiltunen and Matikainen 1985; el-Gendi and Abdel-Baky 1986; Lin 1989; Sun et al. 1990; Ho et al. 1995). It has been assumed that the abnormal high anal pressures are caused by an increased tonic activity of the internal and external

sphincter (Hancock 1977; Teramoto et al. 1981; Lane 1982), which may hinder defecation, and cause expansion of the anal cushions by impairing venous drainage (Sun et al. 1990). Teramoto demonstrated by biopsies taken from anal sphincters in patients with hemorrhoids that these were in a state of increased tonic contraction, which causes muscle hypertrophy and may contribute to increased resting pressure (Teramoto et al. 1981). More recently, the anal cushions were rediscovered as main cause for the high resting and residual pressures in the outer canal by demonstrating abnormally high vascular pressure in anal cushions themselves (Sun et al. 1990). Hancock (1976) considered ultraslow waves to represent a synchronous contraction of the whole internal sphincter. Roe et al. (1987) observed that ultraslow waves were associated with high pressures and often a pulse wave was superimposed indicating the vascularity of the hemorrhoidal tissue. Ultraslow waves may represent some form of peristaltic action in the sphincters by the presence of hemorrhoids (Roe et al. 1987). During defecation, when sphincters are relaxed to facilitate expulsion of stool, dilated anal cushions could cause anal resistance, which could only be overcome by increased abdominal pressure (straining). This pressure may create a shearing force with detrimental effect on venous drainage of the cushions, connective tissue supporting the anal lining finally leading to intermittent or permanent prolapse of hemorrhoids and influencing the continence (Hancock 1977; Hancock 1981; Shafik 1984; Gibbons et al. 1986; Sun et al. 1990). The external sphincter in patients with hemorrhoids may remain in a state of increased tonic contraction, inducing muscle hypertrophy; the role of the internal sphincter for the pressure in the anal canal when a bolus is present may be overestimated (Teramoto et al. 1981). Especially females with a history of constipation and pregnancy were recognized to be prone to develop spontaneous incontinence (Stelzner 1992). Despite controversy on the occurrence of increased resting anal pressure in patients with symptomatic hemorrhoids (Fantin et al. 2002) it is well accepted that there is distal displacement of anal cushions with loosening and fragmentation of the subepithelial connective tissue (conjoined longitudinal muscle, musculus canalis ani, fibromuscular ligament) with a significant change of the topography of the anal canal which may affect continence (Parks 1956; Stelzner 1962; Thomson 1975; Hansen 1976; Hansen 1977; Haas et al. 1984; Stelzner 1992; Loder et al. 1994).

DIAGNOSIS

For diagnosis of hemorrhoids a detailed history with local and general examination may be necessary. Inspection and proctoscopy may reveal acute thrombosed external or internal hemorrhoids; however, perianal abscess, submucous abscess, and even ischio-rectal abscess should be ruled out. Sigmoidoscopy, colonoscopy or further radiologic

examinations (barium enema, MRT) may be necessary in individual cases. (Stern 1964). The sphincter ani externus and internus can be palpated well and their functional status checked. The inferior part of the sphincter ani internus has been described by Stelzner as "hard as cartilage" (Stelzner et al. 1966). Sensitivity of the anal canal, which is also important for continence, is highest in the lower part (Stelzner 1992).

TREATMENT

Evidence-based treatment modalities include injection sclerotherapy, photocoagulation, cryotherapy, diathermy, banding, laser, open or closed hemorrhoidectomy and stapled hemorrhoidectomy (Holzheimer 2001). The indication for hemorrhoidectomy should be based on the patient's symptoms and the condition of rectal outlet (Ferguson and Heaton 1959). Associated anorectal disease may be present at least in 22 % of patients (Bleday et al. 1992). Patients with long-standing hemorrhoids may have impaired anal sphincter pressures associated with perineal descent and pudendal nerve injury (Hancock 1976; Read et al. 1982; Bruck et al. 1988; Ho et al. 1995). External sphincters may be hypertrophied probably from hyperactivity in response to an irritating anal mass as well as the constant fear of soiling from discharge associated with piles (Teramoto et al. 1981). Most patients with symptomatic hemorrhoids may be treated conservatively (45.2%) or by rubber band ligation (44.8%) (Rudd 1970; Bleday et al. 1992). The most common indication for surgery are persistent grade IV hemorrhoids after failure of conservative management with the conventional excision-ligation (Milligan-Morgan) hemorrhoidectomy being the most common technique in Britain and Ireland (Beattie et al. 2002), but the best treatment for hemorrhoids is prevention (Brisinda 2000). Patients seek surgical attention often after onset of complication or when medical therapies have failed. Surgical intervention is required when clear signs and significant discomfort are present (Tajana 1989).

HIGH AND LOW LIGATION WITH EXCISION VERSUS CLOSED TECHNIQUE

The operation should aim at the removal of the dilated veins, ligation of the hemorrhoidal arteries and fixation of the anal mucosa to the underlying muscle (internus) to prevent prolapse and to obliterate the submucous space (Parks 1956). Ligation of the hemorrhoidal artery may be done as high ligation (Parks 1956) or low ligation (Milligan et al. 1937). Closed hemorrhoidectomy is supposed to be less painful and may preserve anal sensory function better and lead to faster wound healing (Ferguson and Heaton 1959; Khubchandani et al. 1972) but it has a reputation in Europe for dehiscence and infection (Turell 1952; Watts et al. 1964). However, Ferguson insisted that complications such as abscess or para-anal cellulitis practi-

cally never occur after closed hemorrhoidectomy (Ferguson and Heaton 1959). In Europe the Milligan-Morgan procedure is preferred whereas in the United States the closed hemorrhoidectomy as described by Ferguson is more popular (Milligan et al. 1937; Ferguson and Heaton 1959; Wolf et al. 1979; Arbman et al. 2000). However, only few studies have compared closed and open techniques (Gemsenjäger 1989; Ho et al. 1997; Arbman et al. 2000); nevertheless, both operations may have an impact on the maximum resting pressure leaving the recto-anal inhibitory reflex unchanged which would infer that the hemorrhoidal tissue itself is responsible for the increased pressure (Roe et al. 1987). In most descriptions of the hemorrhoidectomy technique the dissection of the mucosal ligament of the internal sphincter opens up a plane between the superior hemorrhoidal plexus and the internal sphincter – the plane of the M. canalis ani or M. corrugator ani (Milligan et al. 1937; Parks 1956; Ferguson et al. 1971; Stelzner 1992). Stelzner has emphasized that only hemorrhoids which have been disconnected from the rectum and/or sphincter ani internus or a fibrotic/thrombotic at the anal verge fixated segment of the hemorrhoids (external hemorrhoid) may be securely resected (Stelzner 1992). Parks' observations support this notion: adherent mucosa is difficult to rise from the underlying muscle unless continued prolapse has resulted in attenuation of the mucosal ligament. In third degree piles the mucosal ligament has disappeared and the mucosa flaps have to be fixed to the muscle because otherwise the slipping down of rectal mucosa may cause discharge and irritation (Parks 1956).

SPHINCTEROTOMY AND STAPLER PROCEDURE

Some authors have recommended an additional sphincterotomy for surgical treatment of hemorrhoids, although this may be associated with a risk for incontinence (Tajana 1989; Arbman et al. 2000). Baradnay (1974) stated that the high recurrence rate after the Langenbeck (1852) operation, and the frequent incontinence, stricture and mucosal prolapse after the Whitehead (1887) operation, has been a stimulus for the surgeons to look for better procedures (Milligan et al. 1937, Parks 1956; Ferguson and Heaton 1959; Baradnay 1974), but neither closed nor open techniques are perfect. Postoperative pain, a frequent nuisance to the patient, led to the introduction of the stapler procedure as described by Longo for the treatment of third and fourth degree hemorrhoids (Longo 1998; Kohlstadt 1999; Rowsell et al. 2000; Fazio 2000).

TREATMENT OF THROMBOSED EXTERNAL HEMORRHOIDS

Thrombosed external hemorrhoid, located in the marginal or perianal space, is probably one of the most frequently diagnosed anorectal emergencies

and as a result excision is a frequently performed anorectal operation (Jongen et al. 2003). Symptomatic isolated thrombosed external hemorrhoids may ask for a much higher excision rate (84 %) than internal disease, which may be due to the increased occurrence and recurrence of external thrombosed hemorrhoids (Bleday 1992). Although acute thrombosed external hemorrhoids are usually obvious, according to Stern (1964) they should be differentiated from perianal abscess, submucous abscess or even ischiorectal abscess. The treatment strategy from that performed in internal non-thrombosed hemorrhoids differs: in acute thrombosed hemorrhoids it is not necessary to perform a standard three-position hemorrhoidectomy but rather just to remove symptomatic tissue (Hayssen et al. 1999; Hulme-Moir and Bartolo 2001).

COMPLICATIONS

"Poor results of hemorrhoidectomy have nearly always been reported by surgeons among cases referred to them in which the initial treatment was given elsewhere. Those who have attempted to follow-up their own cases have all reported satisfactory results" Parks (1965) was quite aware that it is difficult to evaluate postoperative complications due to flaws in subjective – there is a group of patients with hemorrhoidal symptoms who have either weak anal sphincters or whose muscle relax easily when they strain is leading to a descent of the perineum and a shallow anal canal – and objective factors – assessing the results of operations performed elsewhere may be difficult as one never can be sure what procedure was carried out (Parks 1965). 13-18 % of treated patients had preoperative incontinence, soiling was reported in 82-84 %, bleeding in 95-97 % and prolapse in 79-82 % of patients (Arbman et al. 2000). Even histological examination may not be reliable as the fixed tissue lost its elasticity. The interpretation of the actual topography is finally left to the attentive surgeon (Stelzner 1992). On the other hand, surgical hemorrhoidectomy is known to have an impact on continence – continence to saline infusion and anal canal pressures are reduced – which may be due to anal dilatation (Read et al. 1982; Ho and Tan 1997; Shalaby and Desoky 2001). Hemorrhoidectomy is the most definitive way of treating prolapsing piles. Postoperative pain is a major concern, and surgery itself is not without complications, including notably bleeding and anal stricture (Golighetti et al. 1969; Milsom and Mazier 1986; MacRae and McLeod 1995). Recurrence of hemorrhoids after hemorrhoidectomy has been observed in 0.5 % to 26 % of cases (Bennett et al. 1963; Tajana 1989; Konsten and Baeten 2000).

Complications occurring within the first two postoperative days include urinary retention, bleeding, soft fecal impaction, and itching. Later complications include urinary tract infection, secondary bleeding, wound infection, fissure and incontinence. Up to 50% of patients complain of

soiling in the early postoperative period (Roe et al. 1987; Isler 1999).

COMPLICATIONS AND EMERGENCY SURGERY

Emergency surgery, usually performed by junior doctors, did not result in a higher incontinence rate when compared to elective operation (4.4 % versus 5.2 %) (Eu et al. 1994).

COMPLICATIONS AND SPECIFIC SURGICAL TECHNIQUE

Some types of techniques seem to be associated with a higher complication rate. Four piles hemorrhoidectomy may have an increased risk of incontinence when compared to modified radical hemorrhoidectomy (Seow-Choen and Low 1995). There is some controversy with regard to additional sphincterotomy. When anal stretch was used in the treatment of hemorrhoids 57.3 % of patients suffered from fecal soiling for ten weeks postoperatively when compared to 6.4 % of patients with additional subcutaneous external sphincterotomy (Asfar et al. 1988; Arbman et al. 2000) whereas Goligher observed impairment of anal control for 6-12 months postoperatively (Goligher et al. 1969). It seems fair to say that in general anal dilatation and lateral sphincterotomy in combination with open hemorrhoidectomy may not offer an advantage and carries the risk of incontinence; however, the interpretation of the studies remains open as different techniques, e.g., open partial sphincterotomy, subcutaneous external sphincterotomy, partial lateral internal sphincterotomy (LIS), were used (Goligher et al. 1965; Mortensen et al. 1987; Asfar et al. 1988; Bleday et al. 1992; Mathai et al. 1996; Arbman et al. 2000), and some investigators saw no incontinence in patients treated with excision-ligation procedure, anal dilatation and open partial sphincterotomy (Arbman et al. 2000). Chung et al. (2002) found no difference in postoperative complications after harmonic scalpel, bipolar scissors hemorrhoidectomy or scissors excision (Chung et al. 2002).

GENERAL COMPLICATIONS AFTER HIGH/LOW LIGATION HEMORRHOIDECTOMY

In a survey among the members of the American Society of Colon and Rectal surgeons (ASCRS) comparing open and closed techniques incontinence has been observed after both procedures (0.18 % - 14.6 %) (Wolf et al. 1979). A high incidence of temporary soiling and leakage was produced by both procedures, with half of the patients in each group (submucosal hemorrhoidectomy; excision/ligation hemorrhoidectomy) affected (Roe et al. 1987). These figures are supported by Arbman et al. who reported that six weeks after closed and open hemorrhoidectomy 28-52 % had soiling, 9-15 % incontinence. More than one year after hemorrhoidectomy 24-30 % had soiling

and 8-15 % incontinence (Arbman et al. 2000). Urinary retention may occur in 4-12 %, early hemorrhage in 1-3 %, late hemorrhage in 1 %, fissure in 2-4 %, inflammatory complications in 3-6 % of patients treated with the excision/ligation technique. Pain may be severe in 4-8 %, mild in 55-65 %. Disturbances of continence can be present even a long time after operation and is usually due to muscular strains, diarrhea, imperfect closure of the anus or change in sphincter pressure (Baradnay 1974; Tajana 1989; Argov 1999). It became obvious that a definite assessment of the surgical results cannot be done prior to the end of a year (Kouba 1980).

COMPLICATIONS AFTER CLOSED HEMORRHOIDECTOMY

The introduction of the closed hemorrhoidectomy by Ferguson as opposed to the open technique by Milligan-Morgan did not eliminate the risk of delayed bleeding. Obviously, both the classical open and the closed hemorrhoidectomy techniques are far from optimal: both operations seem to be followed by protracted convalescence period and significant prevalence of complications although the fear of infection caused by primary closure of hemorrhoidectomy incisions is unfounded (Ganchrow et al. 1971; Buls and Goldberg 1978; Sayfan 2001).

PAIN AFTER HEMORRHOIDECTOMY

In the period immediately following operation not only retention of urine, hemorrhage, occasionally incontinence but also pain may occur (Parks 1956). Pain sometimes increases a few days after hemorrhoidectomy and should be differentiated from infection (Carapeti et al. 1998). Watts et al. (1964) have compared five different forms of hemorrhoidectomy (excision with high ligation, excision with low ligation, excision with primary suture, submucosal excision, excision with clamp and cautery). Despite the extensive intra-anal wounds, healing of the mucosal part of the anal canal proceeded rapidly. There was no difference in pain following the different types of operation, with the exception of excision with primary suture, which was more painful (Watts et al. 1964). Recognition of postoperative pain has been a powerful stimulus to surgeons in introducing various modifications of technique for this operation (Watts et al. 1964). Diathermy excision of hemorrhoids has not been shown to reduce postoperative pain compared with scissors excision (Seow-Choen et al. 1992; Andrews et al. 1993).

COMPLICATIONS AFTER STAPLER HEMORRHOIDECTOMY

Stapler hemorrhoidectomy has been compared in several studies against standard hemorrhoidectomy techniques. Data from randomized studies showed that continence score, anorectal mano-

metric and endoanal ultrasonographic findings were not different after stapler hemorrhoidectomy from those after open hemorrhoidectomy (Ho et al. 2000). Complications observed were urinary retention, hemorrhage in up to 50% of cases, anal fissure, anal stenosis and incontinence (Mehigan et al. 2000; Ho et al. 2001). The development of postoperative pain after stapler hemorrhoidectomy has been a matter of controversy recently. Cheetham et al. reported that patients had severe rectal pain and fecal urgency after stapler hemorrhoidectomy (2000), whereas Fantin et al. (2002) observed only mild rectal pain in 68% and intermittent anal bleeding in 50% of patients. Also others have reported less pain and less urinary retention after stapler operation (Longo 1998; Kohlstadt et al. 1999; Mehigan et al. 2000; Rowsell et al. 2000; Cheetham et al. 2001; Ebert and Meyer 2002; Fantin et al. 2002). Ebert and Meyer were concerned about the high rate of incontinence in their patients treated with stapler hemorrhoidectomy, although they admitted that these were subjective complaints and preoperative incontinence was not recorded (Ebert and Meyer 2002). Rarely, septic complications after stapler hemorrhoidectomy were observed (Molloy and Kingsmore 2000).

COMPLICATIONS AFTER EXCISION OF EXTERNAL HEMORRHOIDS

External hemorrhoids represent distended vascular tissue distal to the dentate line. They may result from straining with stools, childbirth, long car trips or prolonged sitting, constipation or diarrhea. 50% of patients treated for acute thrombosed external hemorrhoids had previous bleeding/prolapse of hemorrhoids and diarrhea in 54% of cases (Turell 1952; Sakulsky et al. 1970; Oh 1989). A prior history of hemorrhoids was nearly always obtained (Stern 1964). Previous anal symptoms (wet anus, itching, bleeding at defecation, prolapsing hemorrhoids) were recorded in 74-88% of patients (Nieves et al. 1977; Saleeby et al. 1991). External hemorrhoids are covered by anoderm and perianal skin richly innervated with somatic pain fibers, which explains why thrombosis may cause intensive pain (Zuber 2002). Perianal thrombosis results from thrombosis within the rich external venous plexus constituting the most distal part of the hemorrhoidal mechanism, mostly caused by stasis and local trauma (Ganchrow et al. 1971; Thomson 1982; Brearly and Brearly 1988; Oh 1989). Pain will often prevent a full assessment of other local conditions (Stern 1964). *"Traditionally, it has been taught, for safety's sake, hemorrhoidal crisis should be treated with heat or ice, bed rest, ointments, analgesics, suppositories, antibiotics, and in some countries, with prayers to St. Fiacre, patron saint of proctologists and hemorrhoid sufferers"* (Nieves et al. 1977). However, a review of the literature has failed to substantiate the widely held belief that operation at this time carries with it a risk of infection and subsequent portal pyemia (Smith 1967). Usually a simple evacua-

tion of the blood clot through a small incision is performed (Stern 1964). In up to 70% of cases, however, a secondary hemorrhoidectomy may be necessary (Grace and Creed 1975). Hemorrhoidectomy is performed through an elliptic or circumferential incision over the site of the thrombosis with removal of the entire diseased hemorrhoidal plexus. Infection after suture closure is rare secondary to the rich vascular network in the anal area (Turell 1952; Sakulsky et al. 1970; Mazier 1973; Hansen and Jorgensen 1975; Grosz 1990; Zuber 2002). Possible complications include bleeding, excessive scarring, stenosis, fissure and fistula - incontinence has not been reported as complication after hemorrhoidectomy of external thrombosed hemorrhoids (Blessing et al. 1992; Zuber 2002). Infectious complications of the excision procedure may relate to unrecognized infectious processes, such as perianal abscess (Zuber 2002; Jongen et al. 2003). Ulceration of the overlying mucosa and not the presence of thrombosis seemed to be the determining factor in degree of inflammatory change. By histological examination of a segment of the subcutaneous portion of the external sphincter it has been demonstrated that thrombosis did not increase the occurrence of postoperative infection (Laurence and Murray 1962). In conclusion, thrombosed external hemorrhoid can be safely performed as outpatient procedure with a low complication rate (Jongen et al. 2003). (Table I)

PREVALENCE OF FECAL INCONTINENCE

Fecal incontinence is said to affect at least 2 % of the adults in the United States (Nelson et al. 1995). The prevalence may be much higher in specific groups, e.g., multiple sclerosis (51 %), irritable bowel syndrome (20 %) and diabetes (20 %) (Feldman and Schiller 1983; Okamoto et al. 1983; Drossman et al. 1986; Hinds et al. 1990; Harari et al. 1997; Krogh et al. 1997; Menter et al. 1997). However, *"reluctance to disclose incontinence is recognized to be an impediment to obtain accurate estimates of the prevalence of fecal incontinence; only one-third of individuals with fecal incontinence have discussed this with a physician"* (Johanson and Lafferty 1996). The majority of patients seen in specialized units have incontinence resulting from trauma, especially obstetrical injury, from a cryptogenic abscess/fistula, or from a sphincter disruption due to Crohn's disease (Kodner 1990).

DEFINITION OF FECAL INCONTINENCE

Despite the definition of incontinence - recurrent uncontrolled passage of fecal material for at least one month - the difficulty has been recognized to articulate a definition of fecal incontinence that reliably separates health from disease (Whitehead et al. 1999; Whitehead et al. 2001). There may be different forms of incontinence which may be associated with specific pathology: passive inconti-

Table 1. Complications after surgical treatment of thrombosed hemorrhoids.

Author	Patients	Type of hemorrhoid	Operation	Fecal impaction	Fistula	Stenosis stricture	Hemorrhage	Infection	Ulcer fissure	Incontinence
Laurence and Murray 1962	12	Prolapsed	Dissection and ligation; Whitehead	Yes	No	Yes	No	No	No	No
Tinckler and Baratham 1964	39	External prolapsed	Excision	No	No	3	No	No	No	No
Smith 1967	15	Internal	St. Marks	No	No	No	No	No	No	No
Howard and Pingree 1968	25	External	Whitehead	No	No	No	No	No	No	No
Sakulsky et al. 1970	50	Mostly external	Excision	No	No	No	1	No	1	No
Ganchrow et al. 1971	A) 100 B) 30	A) 87 external B) 23 external	Ferguson	No	No	1	3	No	No	No
Hansen and Jorgensen 1975	25	Prolapsed	Milligan- Morgan	No	No	1	2	No	No	No
Nieves et al. 1977	85	Prolapsed	St. Marks	No	No	No	1	No	1	No
Saleeby et al. 1991	25	Thrombosed or gangrenous	Closed hemorrhoidectomy	No	No	No	1	No	No	No
Jongen et al. 2003	340	External	Excision	No	7 (2.1%)	No	1 (0.3%)	See fistula	No	No

nence (unwanted stool without patient awareness), urge incontinence (unwanted stool despite active attempts to inhibit defecation), post-defecation soiling (stool postvoiding with normal continence), nocturnal fecal incontinence or incontinence associated with increased bowel frequency; 80% of patients with rectal prolapse report some degree of incontinence (Talley et al. 1992; Engel et al. 1995; Buchanan et al. 2001).

PATHOGENESIS OF FECAL INCONTINENCE

"Fecal continence is maintained by the integrated action of anal sphincteric function, the puborectalis, the levator plate, and intact sensory pathways. It is dependent on rectal reservoir function, colonic motility, the volume and consistency of stool, anorectal sensation, the anorectal angle, normal resting anal tone and resistance to opening. As continence depends on several factors, some mechanisms may be able to compensate and maintain clinical continence when one element is abnormal. Therefore, objective knowledge of the continence state and of the function of all components is fundamental to an adequate estimate of the clinical situation". In view of the heterogeneous etiology of anal incontinence, a uniform defect in anorectal function should not be expected. (Felt-Bersma et al. 1990; Felt-Bersma et al. 1992; Penninckx et al. 1992; Mavrantonis and Wexner 1998; Francombe et al. 2001; Whitehead et al. 2001). True fecal incontinence must be distinguished from soiling, which may be caused by deformation of the anal canal, fecal mass in the rectum, mucosal prolapse, and from urgency - patients are unable to withhold the stool -, which may be related to impaired rectal compliance due to inflammatory disease of the rectum. Incontinence may be due to a sensory loss in the anal canal, which is characterized by the passage of feces without the patient being aware of it, as in rectal prolapse and in patients with neuropathic incontinence or diabetes (Felt-Bersma et al. 1989; Mavrantonnis and Wexner 1998). Incontinence

may be associated with perineal descent or descending perineum syndrome which occurs more often in women, not only because of obstetric injury, but also because of the higher incidence of straining during defecation, constipation, and outlet obstruction (Moore-Gillon 1984; Pinho et al. 1990). A thorough history, which includes the possibility of steatorrhea, neurologic information, obstetric and gynecological surgical history in female patients, all previous operations upon the anus, rectum, colon and operations for fistulae and hemorrhoids is mandatory (Mavrantonis and Wexner 1998). Causes of incontinence could be simple structural defects, weak sphincters, idiopathic incontinence, rectal prolapse, diabetes mellitus, progressive systemic sclerosis, multiple sclerosis, descending perineum syndrome, perianal infection (Penninckx et al. 1992; Vaizey et al. 1998). The cause of incontinence may be classified according to functional aspects, sphincter weakness, sensory loss (Whitehead et al. 2001), sphincter complex, neurological disorders, sensory, alterations of compliance, congenital, psychological, and miscellaneous disorders (Francombe et al. 2001) or trauma, congenital, myopathy, neurological, colorectal disease, and miscellaneous disorders (Buchanan et al. 2001). Whitehead et al. (2001) stated that diarrhea and constipation are the most common cause of incontinence. Several compounds may decrease the anal basal pressure, which may affect continence, e.g., diltiazem, ketanserin, encephalin, glucagon, somatostatin (Penninckx et al. 1992; Carapeti et al. 1999; Whitehead et al. 2001). (Table II)

DIAGNOSIS OF FECAL INCONTINENCE

Before specific investigations are performed, all intestinal and systemic disorders must be excluded as causative factors (Mavrantonis and Wexner 1998). Due to the multifactorial and complex etiology the investigation should include anorectal manometry, electromyography, pudendal nerve terminal

Table II. Common causes for fecal incontinence.

Fecal impaction	Pelvic floor dyssynergia, drug-side effect, idiopathic, spinal cord injury
Diarrhea	Irritable bowel syndrome, infections, metabolic diseases
Cognitive/psychological	Dementia, psychosis, willful soiling
Sphincter injury	Obstetrical trauma, motor vehicle accident, foreign body trauma, complex fistula and/or abscess, fistula surgery, hemorrhoidectomy
Pudendal nerve injury	Obstetrical trauma, diabetic neuropathy, multiple sclerosis, idiopathic, straining
CNS injury	Spina bifida, spinal cord injury, cerebrovascular accident, multiple sclerosis
Sensory loss	Diabetic neuropathy, spinal cord injury, multiple sclerosis, surgery
Congenital	Hirschsprungs disease, atresia, spina bifida
Colorectal disease	Rectal prolapse, rectocele, hemorrhoids, tumors, inflammatory bowel disease
Miscellaneous	Laxative abuse, constipation, straining, drugs decreasing anal pressure
Myopathy	Primary internal sphincter degeneration, systemic sclerosis

Modified according to Buchanan et al. 2001, Francombe et al. 2001, Whitehead et al. 2001

motor latency, cinedefecography, and anal ultrasound. While some authors reported that an experienced colorectal surgeon is capable of assessing the resting and squeeze pressures with physical examinations, others disagree. To make things even more difficult, the usefulness and practicality of anorectal neurophysiologic evaluation is a matter of controversy (Hallan et al. 1989; Wexner et al. 1991; Buchanan et al. 2001). The investigation normally starts with perianal inspection, which may reveal scarring from previous surgery, childbirth damage or episiotomy, hemorrhoids, skin tags or rectal prolapse, and digital rectal examination for assessment of anal sphincter tone, impaction, abnormalities of stool. Proctosigmoidoscopy is mandatory to view mucosal abnormality, hemorrhoids, fissure, or fistula (Hallan et al. 1989; Buchanan et al. 2001).

ANAL MANOMETRY, RECTAL CAPACITY, SALINE-INFUSION AND PNTML

Anorectal manometry is most commonly used to assess anal sphincter function. Maximum basal pressure (MBP) was thought for measurement of the internal sphincter and maximum squeeze pressure (MSP) for evaluation of the external sphincter (Felt-Bersma et al. 1990). Early study results falsely gave the impression that the maximum basal pressure is generated for 85 % by the internal sphincter and for 15 % by the external sphincter (Frenckner and von Euler 1975). This, however, is no longer accepted as more recent studies came up with different figures: the MBP is generated for 30 % by the external sphincter, for 55 % by the internal sphincter and for 15 % by expansion of the hemorrhoidal plexus (Lestar et al. 1989; Felt-Bersma et al. 1992). Furthermore, the anal manometry may be influenced by sex, age, prolapsing or non-prolapsing hemorrhoids, rectocele or rectal tumor presence and repair (Loening-Baucke and Anuras 1985; McHugh and Diamond 1987; Arnold et al. 1990; Felt-Bersma et al. 1990; Sun et al. 1990; Keck et al. 1995; Ho et al. 1998). *"There is no accepted standardized method of performing or interpreting this test, thus comparison of data between institutions has been problematic"* (Mavrantonis and Wexner 1998). This and the limited value of anal manometry in patients with soiling were repeatedly demonstrated (Felt-Bersma et al. 1989; Diamant et al. 1999). Several studies have analyzed differences between incontinent and continent patients with basic anorectal function investigations including anal manometry, rectal capacity, and the saline-infusion test with controversial results (Read et al. 1979; Read et al. 1984; Felt-Bersma et al. 1988). In a more recent study Felt-Bersma et al. found no significant difference in MBP between incontinent patients and normal controls. Differentiation between incontinent and continent patients was not possible with a single test (anal manometry, rectal capacity, saline-infusion), because there was complete overlap. *"Therefore, in the individual patient, an abnormal result in one test must be inter-*

preted with caution." (Felt-Bersma et al. 1990). The saline-infusion test has been developed for demonstration of fluid incontinence (Read et al. 1979), but Felt-Bersma et al. (1990) found no absolute discrimination between incontinent and continent patients, even when corrected for stool consistency. Rectal capacity may be influenced by concomitant diseases, e.g., proctitis, Crohn's disease (Buchman et al. 1980). Anal sensation has been recognized as important factor for the continence (Read et al. 1982; Read and Read 1982). If impaired it may indicate neurological or postsurgical nerve impairment (Miller et al. 1989; Kamm and Lennard-Jones 1990). However, both methods, anal manometry and determination of anal sensation, are largely dependent on the patient's cooperation (Read et al. 1982; Felt-Bersma et al. 1990) and on the absence of certain compounds which are known to influence anal pressure, e.g., diltiazem, etc. (Penninckx et al. 1992) or lignocaine. Lignocaine is known to double the electrosensitivity threshold (Roe et al. 1987). The rectoanal inhibitory reflex has been proposed as a measure of the internal sphincter function, but the interpretation of levels in continent and incontinent patients is difficult (Buchanan et al. 2001; Zbar et al. 2001). Pudendal nerve terminal motor latency (PNTML) is often used to provide additional information on the innervation of the external anal sphincter. While some authors report it may reveal unsuspected neuropathy in traumatic fecal incontinence or correlate with outcome from sphincteroplasty (Felt-Bersma et al. 1992; Engel et al. 1994; Ternent et al. 1997), the test has been criticized by others for its lack of sensitivity and specificity (Laurberg et al. 1988; Wexner et al. 1991; Vernava et al. 1993; Cheong et al. 1995; Ho and Goh 1995; Gilliland et al. 1998; Buchanan et al. 2001).

ENDOANAL ULTRASONOGRAPHY, MRT AND EVACUATION DEFECOGRAPHY

Anal ultrasonography may be used for mapping internal and external sphincter defects (Yang et al. 1993). Different structures of the upper part, midanal canal or lower part may be identified (Mavrantonis and Wexner 1998). It is primarily used for identifying morphologic anomalies (Whitehead et al. 2001). Interpretation of the ultrasonographic investigation may be difficult after hemorrhoidectomy: Ho et al. (2001) demonstrated new internal sphincter defects after hemorrhoidectomy, but none of the patients had symptoms of incontinence. *"It is conceded that operator dependency and possible false identification of anal sphincter injury at endoanal ultra-sonography is possible"* (Ho et al. 2001). Variability in manometric measurement (Ho et al. 2001) and the use of a 7 MHz probe instead of a 10 MHz probe (Buchanan et al. 2001) will make the job more difficult. Unfortunately magnetic resonance imaging, which is complimentary to endoanal ultrasonography, has shown lower inter-observer reproducibility for detecting sphincter defects (Buchanan et al. 2001;

Malouf et al. 2001). Evacuation proctography (defecography) is not of established value in patients with fecal incontinence (Diamant et al. 1999). In conclusion, *"although anorectal investigations are in widespread use, their clinical value has sometimes been doubted"* and results of tests should be interpreted with caution (Buchanan et al. 2001).

OBSTETRIC INJURY AND FECAL INCONTINENCE

The majority of patients referred to a colorectal clinic may have incontinence as a result of obstetric injury to the sphincter complex (uncontrolled second stage delivery, epidural anesthesia, instrumental delivery, episiotomy) or as results of prolonged labor with damage to the innervation (Nn. Levatorii, Ganglion pelvinum, Nn. Pudendales) (Snooks et al. 1985; Stelzner 1991; Sultan et al. 1993; Sultan et al. 1994; Kamm 1994; Poen et al. 1998; Sultan et al. 1998). Occult injuries to the sphincter are more common than previous thought and may present many years later (Sultan et al. 1998; Francombe et al. 2001). The occult denervation of the pudendal innervation is a gradually progressive phenomenon in that functional effects of the original injury together with other factors, e.g., abnormal straining patterns of defecation with pelvic floor descent, are leading to stretch-induced damage to the pelvic floor during defecation (Parks et al. 1977; Snooks et al. 1984; Swash et al. 1985; Snooks et al. 1985b; Snooks et al. 1986; Snooks et al. 1990). There may be extensive reinnervation, which may explain why in some patients the external sphincter seems to be intact despite obstetric trauma in the past (Wunderlich and Swash 1983). Childbirth causing damage to the pelvic floor innervation is considered to be a precursor of stress incontinence (Snooks et al. 1986). After first vaginal delivery endoanal ultrasonography has demonstrated sphincter defects in 30 % of women (Sultan et al. 1993). In a study by Jacobs et al. (1990) 19 % were incontinent without signs of obstetric injury (Jacobs et al. 1990). This has been supposed to be an early manifestation of idiopathic (neurogenic) incontinence, which has been associated with prolonged or difficult labor (Neill et al. 1981; Kiff and Swash 1984). There is evidence gained in recent studies that 25% of primiparous women and about 33% of multiparous women with a history of vaginal delivery have an anal sphincter defect. The probability that postpartum fecal incontinence is associated with an anal sphincter defect is 76.8-82.8 % (Oberwalder et al. 2003).

CONSTIPATION AND FECAL INCONTINENCE

Many incontinent patients were women with chronic constipation and a descending perineum. Incontinence is believed to be caused by chronic stretch injury of the pudendal nerve and the sacral branches causing a low anal pressure and an obtuse puborectalis angle (Neill et al. 1981; Bartolo et al. 1983; Read et al. 1984; Felt-Bersma et al.

1990). Anorectal incontinence due to sphincter denervation has been demonstrated in patients with rectal prolapse and fecal impaction (Parks et al. 1977). Stelzner (1991) has seen an association of the abuse of laxatives causing toxic damage to the innervation of the bowel and the consecutive incontinence.

NEUROLOGICAL DISEASE AND FECAL INCONTINENCE

In patients with neurological disease other factors than childbirth need to be identified, e.g., side effects of drugs or coexistent behavioral disorders (Hinds et al. 1990). Fecal incontinence may be observed in patients with multiple sclerosis (50%), spinal injury (61%), spina bifida (90%), or in patients with long-standing neuropathy secondary to diabetes mellitus (Hinds et al. 1990; Malone et al. 1994; Glickman and Kamm 1996). Isolated degeneration of the smooth muscle of the internal anal sphincter may affect both men and women often in the middle age and is a common cause of soiling (Vaizey et al. 1997). The resting anal pressure is usually low and endosonography shows a thin and fibrotic internal sphincter (Francombe et al. 2001). Degeneration and fibrosis of the internal anal sphincter and sensory receptors, leading to passive fecal incontinence may occur in patients with progressive systemic sclerosis, idiopathic intestinal pseudoobstruction and diarrhea may aggravate the clinical situation (Engel et al. 1994). Incontinence is seen in patients treated for anal atresia or Hirschsprung's disease (Catto-Smith et al. 1995; Mulder et al. 1995; Hassink et al. 1996). Sphincter function may also be impaired by radiation injury (Varma et al. 1985; Varma et al. 1986).

ANAL STRETCH, SPHINCTEROTOMY AND FECAL INCONTINENCE

Manual dilatation (Lord procedure) for treatment of hemorrhoids has not gained widespread acceptance since it relies on uncontrolled damage to the anal sphincter with high incontinence rates (20% and more) at long-term follow-up (Lord 1969; MacIntyre and Balfour 1977; Buls and Goldberg 1978; Konsten and Baeten 2000). Fecal incontinence after sphincterotomy may range from 1% to 5%, but 35% of patients may have a lack of control for flatus and 22% complain of soiling (Buls and Goldberg 1978; Khubchandani and Reed 1989; Lund and Scholefield 1996). However, others have performed sphincterotomy without observing any case of incontinence (Bleday et al. 1992). Arbman et al. (2000) used anal dilatation in the Milligan-Morgan group, but they found no patient with fecal incontinence after six weeks of follow-up.

ANAL FISTULA/ABSCESS AND FECAL INCONTINENCE

Anal abscess and fistula are parts of a spectrum of the same disease process (Eisenhammer 1954). The

incidence for men has been estimated to be 12.3 per 100.000 and for women 5.6 per 100.000 (Sainio 1984). Anorectal suppuration is usually caused by cryptoglandular infection. The anal glands, arising at the level of the crypts, penetrate through the internal sphincter muscle into the transsphincteric plane (Johnson 1914; Morgan and Thomson 1956; Shropshire 1960; Schouten and van Vroonhoven 1991). Anal fistula is caused by infected anal glands in the vast majority of cases (Eisenhammer 1956; Eisenhammer 1961; Parks 1963). Anal abscess or fistula may be idiopathic or non-specific. Specific causes of anorectal sepsis include Crohn's disease, tuberculosis, actinomycosis, various malignancies, gut duplication, foreign bodies, as well as intraabdominal or pelvic disease (Seow-Choen and Nicholls 1992). The site of the internal opening is relevant for the development of incontinence as is the type of fistula (high or complex fistula) (Milligan and Morgan 1934; Belliveau et al. 1983; Garcia-Aguilar et al. 1996). Most of the ducts have their orifices in the posterior portion of the anal canal, which may explain the predominance of posterior anal fistulae (6 O'clock in lithotomy position) (Morgan 1936; Burke et al. 1951; Kuster 1965; Cirocco and Reilly 1992). Intersphincteric, transsphincteric and extrasphincteric fistula were identified by Stelzner (1959). The most widely used system of classification is that of Parks: intersphincteric, transsphincteric, suprasphincteric and extrasphincteric fistula (Parks et al. 1976). The prevalence of supralelevator fistulous abscess varies from 2-22 % (Eisenhammer 1966; Bevans et al. 1973; Parks et al. 1976; Hanley 1978; Read and Abcarian 1979; Prasad et al. 1981; Terranova et al. 1989), the prevalence of infralelevator or ischio-rectal abscess ranges from 9-38% (Hamilton 1975; Hanley et al. 1976; Parks and Stitz 1976; Aluwihare 1983; Ustynoski et al. 1990). Ischio-rectal abscess, which is the second most common anatomic site, is prone to cause incontinence (Cox et al. 1997). The estimated amount of internal sphincter involved by the fistula has been identified as an independent factor for the development of incontinence by multivariate analysis (Garcia-Aguilar et al. 1996). Soiling (20.6%) and incontinence (21.8 %) was present already in one fifth of patients before surgical treatment (Schouten and van Vroonhoven 1991) and they may have already a poor resting tone and/or voluntary contraction before operation, which indicates an already compromised internal or external anal sphincter function (Pescatori et al. 1989). Affected patients may have quite subtle signs despite the development of massive anorectal suppuration (Seow-Choen and Nicholls 1992). Problematic fistula and/or abscesses may be missed or misdiagnosed even in specialized units. Endoanal ultrasonography was not considered to be as useful as digital examination (Seow-Choen and Phillips 1991). Management of anal fistula may be done by classical laying open (fistulotomy), by excision (fistulectomy) or by incision and drainage. Several studies have shown continence disturbance in 44 to 67 % of patients

(Parks and Stitz 1976; Hanley 1978; Oh 1983; Ramanujam et al. 1983; Culp 1984; Kuypers 1984; Aguilar et al. 1985; Mann and Clifton 1985; Parkash et al. 1985; Christensen et al. 1986; Wedell et al. 1987; Reznick and Bailey 1988; Shemesh et al. 1988; Thomson and Ross 1989; Kennedy and Zegarra 1990; Ustynoski et al. 1990; Schouten et al. 1991; Williams et al. 1991; Seow-Choen and Nicholls 1992; Kodner et al. 1993; Matos et al. 1993; Pearl et al. 1993; Lunniss et al. 1994; van Tets and Kuipers 1994; Graf et al. 1995; Garcia-Aguilar et al. 1996; Ozuner et al. 1996; Golup et al. 1997; Hamalainen and Sainio 1997; Ho et al. 1997; Garcia-Aguilar et al. 2000; Gustafsson and Graf 2002). Early studies have demonstrated a higher incidence of complications with ischio-rectal and intersphincteric abscesses (Chrabot et al. 1983; Ramanujam et al. 1984; Vasilevsky and Gordon 1984). Major incontinence will follow with drainage of transsphincteric fistula with translevator supralelevator extension (Parks et al. 1976). Women are particularly prone to sphincter damage during surgery for anal fistula (Goldberg 1976; Seow-Choen and Nicholls 1992). In a more recent study incontinence was associated with female sex, high anal fistula, type of surgery, and previous fistula surgery. "*Surgical treatment of fistula-in-ano is associated with a significant risk of recurrence and a high risk of incontinence*" (Garcia-Aguilar et al. 1996). Certain types of fistula may be associated with an even higher rate of incontinence: transsphincteric 54%, suprasphincteric 80%, extrasphincteric 83% (Garcia-Aguilar et al. 1996). In certain cases it may be difficult to find the internal openings and the production of false openings or passages may result in complex supralelevator or infralelevator abscesses (Kratzer 1950; Scoma et al. 1974; Lockart-Mummery 1975; Hanley 1978; Hanley 1979; Fazio 1987) enbloc resection of the scarred tissue may cause incontinence (Stelzner 1992). In conclusion, surgical treatment of fistula-in-ano frequently results in postoperative incontinence (Garcia-Aguilar et al. 2000). Even a simple division of an acceptable portion of the sphincter muscle in low anal fistula may cause varying degrees of impairment of anal control (Sainio and Husa 1985; Shoulder 1986). The risk for incontinence after treatment of ischio-rectal abscess is higher than after perianal abscess (Ho et al. 1997). The higher risk of incontinence after fistula surgery in female patients is probably attributable in part to partial anal disruption and/or traction injury to the pudendal nerve during vaginal delivery (Garcia-Aguilar et al. 1996).

HEMORRHOIDECTOMY AND INCONTINENCE

Hemorrhoidectomy is not generally regarded as cause of incontinence, but may be a predisposing factor when the patient has diarrhea (Read et al. 1979; Read et al. 1982). The evaluation is complicated by preexisting comorbidity. Bennett reported that 63% of patients with symptomatic hemorrhoids admitted that they have experienced perianal trouble for more than five years before opera-

tion. 86% complained of pain and discomfort prior to the hemorrhoidectomy (Bennett et al. 1963). The details of studies on the incidence of incontinence after hemorrhoidectomy are often unclear. Read has reported an incidence of less than 10 % of frank incontinence following hemorrhoidectomy (Read et al. 1982). Bennett is often quoted to report an incidence of 26% of incontinence after hemorrhoidectomy. However, he indicates that these were minor defects of anal control and "certainly do not amount to frank incontinence" (Bennett et al. 1963). Minor defects in anal control do also occur in normal people, according to Bennett: 10% experience inadvertent passage of flatus, 3% occasional unexpected leakage of feces and 2% frequent soiling. He concluded, that as none of the sphincters are divided in a standard hemorrhoidectomy – though the possibility of slight inadvertent damage to superficial layers of the internal sphincter cannot be entirely excluded – it might be assumed that the functional imperfections are related to removal of sensitive anal skin (Duthie and Gairus 1960; Bennett et al. 1963). "*Whether an insensitive anal-canal scar, weakened sphincter action, or yet unelucidated factors are responsible, it is apparent that minor imperfections of continence do sometimes occur after hemorrhoidectomy as well as after other anorectal operations*" (Bennett et al. 1963). The true incidence of symptomatic soiling after hemorrhoidectomy is unknown which may be due to no or short follow-up (Bennett et al. 1963; Zbar et al. 2001). Patients have been reported to present with symptoms of incontinence after hemorrhoidectomy 11 months (inter-quartile range 8-16 months) after hemorrhoidectomy (Zbar et al. 2001). Manometry or sphincter morphology has been studied only rarely in incontinent patients before and/or after hemorrhoidectomy and results are conflicting (Read et al. 1992; Abbasakoor et al. 1998; Zbar et al. 2001). It is well recognized that patients with long-standing hemorrhoids can have impaired anal sphincter pressures at rest before surgery (Bruck et al. 1988; Ho et al. 1995; Zbar et al. 2001) and after hemorrhoidectomy (Ho and Tan 1997). Several other factors may influence anal pressure. The function of the internal anal sphincter may be altered by endoanal retraction during hemorrhoidectomy (Zbar et al. 2001). Increased sphincter tone is maintained by the presence of hemorrhoids, possibly because of reflex tonic contraction of the internal and external anal sphincter caused by prolonged stimulation of the anal canal (Hancock and Smith 1975; Hancock 1976; Teramoto et al. 1981; Read et al. 1982). The fact that postoperative squeeze pressures were lower in patients who soiled after hemorrhoidectomy compared to those who did not suggests that conscious contraction of the external sphincter may be of importance in preventing leakage through a distorted anal canal (Read et al. 1982). Hemorrhoidectomy is known to result in the abolition of ultra-slow waves together with a reduction in basal and squeeze pressures. An association was found between soiling following surgery and abnormally

low postoperative squeeze pressure (Read et al. 1982), but manometry failed to correlate with clinical function after operation (Melange et al. 1992). It also has been stated that morphologic assessment of anal sphincters after surgery may not be helpful for assessment. "*The causes of incontinence after these types of surgery (lateral sphincterotomy, hemorrhoidectomy) are probably multifactorial and do not seem to rely entirely on the presence either of an occult preexisting sphincter injury or an advertent intraoperative sphincter injury*" (Zbar et al. 2001), although it has been conceded that individual patients' variations in the length and tone of the anal sphincter, preexisting sphincter injuries undetected by diagnostic modalities and differences in other anatomic and functional elements may contribute to anal incontinence (Garcia-Aguilar 2001). "*Continence is a more complex phenomenon than mere preexisting or inadvertent intraoperative sphincter injury. This of course has considerable medico-legal significance*" (Zbar et al. 2001).

INTERNAL SPHINCTER DEFECT AND FECAL INCONTINENCE

Internal sphincter defect does not necessarily lead to a state of incontinence. There may be transient incontinence and/or internal sphincter fragmentation found on endoanal ultrasound 6-8 weeks after operation, which may not be present at later controls (Ho et al. 2000). Internal anal sphincter defects seen on the endoanal ultrasound are not necessarily associated with clinical incontinence (Ho et al. 2000; Brown et al. 2001; Ho et al. 2001). Complete fecal incontinence does not usually occur with internal sphincterotomy (Mavrantonis and Wexner 1998); it is the interaction between the internal anal sphincter and anal cushions, which is essential for perfect anal control (Sanwang and Solla 1998). The anatomic topography may also be relevant for the interpretation of a possible sphincter injury: while internal hemorrhoids are in close contact with the internal sphincter, the external thrombosed hemorrhoid is located more distally in the anal canal and sphincter injury does occur less likely, actually there is none reported in the literature "*The pathologist can tell by looking at the lining of protruding hemorrhoids, if they are internal (mucosa lining) or external (anoderm)*" (Haas et al. 1984).

MUSCLE FIBERS IN HEMORRHOIDECTOMY SPECIMEN AND FECAL INCONTINENCE

Smooth muscle fibers are normally present in normal hemorrhoids (Haas et al. 1984). Their presence does not indicate injury to the sphincter. Hemorrhoids consist of a stroma with blood vessels, smooth muscles and supporting connective tissue; and there is the anchoring connective tissue system, which connects hemorrhoids to the internal sphincter and the conjoined longitudinal coat, all consisting of muscle fibers (Haas et al. 1984). Teramoto et al. reported that biopsies of sphinc-

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In summary, in most instances, hemorrhoids are treated conservatively; the surgeon is contacted when conservative measures have failed or complications, e.g., thrombosis, have occurred. Prolapsed internal hemorrhoids are the main indication for hemorrhoidectomy: high (Parks) or low (Milligan-Morgan) ligature with excision, closed hemorrhoidectomy (Ferguson) or stapler hemorrhoidectomy. Thrombosed external hemorrhoids are primarily treated by incision and secondary by excision. Complications after operative treatment of external thrombosed hemorrhoids are rare. After standard hemorrhoidectomy for internal hemorrhoids approximately 10% may have a complicated follow-up (bleeding, fissure, fistula, abscess, stenosis, urinary retention, soil-ing, incontinence); there may be concomitant disease, e.g., perianal cryptoglandular infection, causing complex fistula/abscess, which is associated with an increased risk (30-80%) for complications, e.g., incontinence. Other treatment options, e.g., sphincterotomy, anal stretch, have been accused to cause more complications, e.g., incontinence in 30-50% of cases. However, incontinence is a complex phenomenon; it is evident that an isolated single injury is normally not a sufficient cause, e.g., injury of the internal sphincter. The majority of patients may present with prior obstetric injury, perianal infection or Crohn's disease and other comorbidity. Therefore all systemic and regional disorders, causing incontinence, should be excluded before starting manometric, neurophysiological and sonographic investigations. Variations and overlap in test results, patient, instrument- or operator-dependent factors ask for cautious interpretation. There is vast evidence that the demonstration of muscle fibers in hemorrhoidectomy specimens is a normal feature. In conclusion, standard hemorrhoidectomy with proper indication is a safe procedure. If complications occur, it is in the interest of the patient and surgeon to perform a thorough investigation.

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