Eur J Med Res (2004) 9: 18-36

Review

HEMORRHOIDECTOMY: INDICATIONS AND RISKS

R. G. Holzheimer

University Halle-Wittenberg, Germany

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 primary 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 canalis 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; Shropshear 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 cavenosum 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 hemorrhoidectomy 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 preg-nancy 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 (Golighten 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 evacuation 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 determinating 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 I. 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 (Pennickx 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 interpreted 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 reinervation, 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 transphincteric plane (Johnson 1914; Morgan and Thomson 1956; Shropshear 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 supralevator 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 infralevator or ischiorectal abscess ranges from 9-38% (Hamilton 1975; Hanley et al. 1976; Parks and Stitz 1976; Aluwihare 1983; Ustynoski et al. 1990). Ischiorectal 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 treat-ment (Schouten and van Vroohoven 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 Kuijpers 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-Aquilar et al. 2000; Gustafsson and Graf 2002). Early studies have demonstrated a higher incidence of complications with ischiorectal 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 supralevator 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: transphincteric 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 supralevator or infralevator 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 ischiorectal 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 mediolegal 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-

ters were taken in patients with hemorrhoids but no case of incontinence has been reported (Teramoto et al. 1981). Khalil et al. (2000) found skeletal muscle fibers and smooth muscles fibers in histological specimens after sutured hemorrhoidectomy and stapler hemorrhoidectomy. They concluded that the histological presence of muscle fibers in excised hemorrhoidal tissue neither means sphincter injury nor incontinence (Khalil et al. 2000).

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. 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 primary 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. 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.

References

Abbasakoor F, Nelson M, Beynon J, Patel B, Carr ND. Anal endosonography in patients with anorectal symptoms after haemorrhoidectomy. Br J Surg 1998; 85:1522-4

Aguilar PS, Plasencia G, Hardy TG Jr, Hartmann RF, Stewart WR. Mucosal advancement in the treatment of anal fistula. Dis Colon Rectum 1985 ;28 :496-8

Aluwihare APR. Anterior horseshoe fistulae. Ann R Coll Surg Engl 1983;63:121-122

Andrews BT, Layer GT, Jackson BT, Nicholls RJ. Randomized trial comparing diathermy hemorrhoidectomy with the scissors dissection Milligan-Morgan operation. Dis Colon Rectum 1993;36:580-583

Arabi Y, Alexander-Williams J, Keighley MR. Anal pressures and anal fissure. Am J Surg 1977;134(5):608-10

Arbman G, Krook H, Haapaniemi S. Closed vs. open hemorrhoidectomy – is there any difference? Dis Colon Rectum 2000;43:31-34

Argov S. Ambulatory radical hemorrhoidectomy : personal experience with 1,530 Milligan-Morgan operations with follow-up of 2-15 years. Dig Surg 1999; 16:375-378

Arnold MW, Stewart WR, Aguilar PS. Rectocele repair. Four years' experience. Dis Colon Rectum 1990;33: 684-687

Asfar SK, Juma TH, Ala-Edeen T. Hemorrhoidectomy and sphincterotomy. A prospective study comparing the effectiveness of anal stretch and sphincterotomy in reducing pain after hemorrhoidectomy. Dis Colon Rectum 1988 ;31(3) :181-5

Baradnay G. Late results of hemorrhoidectomy according to Milligan and Morgan – A follow-up study of 210 patients. Am J Proctol 1974;25(5):59-62

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

Beattie GC, Wilson RG, Loudon MA. The contemporary management of haemorrhoids. Colorectal Dis 2002;4:450-454

Belliveau P, Thomson JPS, Parks AG. Fistula-in-ano. A manometric study. Dis Colon Rectum 1983;26:152-154

Bennett RC, Friedman MH, Goligher JC. Late results of haemorrhoidectomy by ligature and excision. BMJ 1963;2:216-9

Bevans DW, Westbrok KC, Thompson BW, Caldwell FT. Perirectal abscess: a potentially fatal illness. Am J Surg 1973;126:765-768

Bleday R, Pena JP, Rothenberger DA, Goldberg SM, Buls JG. Symptomatic hemorrhoids: current incidence and complications of operative therapy. Dis Colon Rectum 1992;35(5):477-81

Blessing H, Schläpfer HU, Ammann JF. Notfallmässige Hämorrhoidektomie bei akuter Hämorrhoidalthrombose. Helv chir Acta 1982;49:861-865

Brearley S, Brearley R. Perianal thrombosis. Dis Colon Rectum 1988;31:403-404

Brisinda G, Maria G. Oral nifedipine reduces resting anal pressure and heals chronic anal fissure. Br J Surg 2000;87:251

Brisinda G. How to treat haemorrhoids. BMJ 2000; 321:582-583

Brown SR, Ballan K, Ho E, Ho Fams YH, Seow-Choen F. Stapled mucosectomy for acute thrombosed circumferentially prolapsed piles: a prospective randomized comparison with conventional haemorrhoidectomy. Colorectal Dis 2001;3(3):175-8

Bruck CE, Lubowski DZ, King DW. Do patients with haemorrhoids have pelvic floor denervation? Int Colorectal Dis 1988 ;3 :210-4

Buchanan GN, Nicholls T, Solanki D, Kamm MA. Investigation of faecal incontinence. Hosp Med 2001;62(9):533-537

Buchman P, Mogg GA, Alexander-Williams J, Allan RN, Keighley MR. Relationship of proctitis and rectal capacity in Crohn's disease. Gut 1980;21:137-140

Buls JG, Goldberg SM. Modern management of hemorrhoids. Surg Clin North Am 1978;58:469-478

Burke RM, Zavela D, Kaump DH. Significance of the anal gland. Am J Surg 1951;82:659-662

Burkitt DP. Hemorrhoids, varicose veins and deep vein thrombosis: epidemiologic features and suggestive causative factors. Can J Surg 1975;18:483-8

Carapeti EA, Kamm MA, McDonald PJ, Phillips RK. Double-blind randomised controlled trial of effect of metronidazole on pain after day-case haemorrhoidectomy. Lancet 1998;351(9097):169-72

Carapeti EA, Kamm MA, Evans BK, Phillips RKS. Topical diltiazem and bethanechol decrease anal sphincter pressure without side effects. Gut 1999;45:719-722

Catto-Smith AG, Coffey CM, Nolan TM, Hutson JM. Fecal incontinence after the surgical treatment of Hirschsprung's disease. J Pediatr 1995;127:954-7

Cheetham MJ, Mortensen NJ, Nystrom PO, Kamm MA, Phillips RK. Persistent pain and faecal urgency after stapled haemorrhoidectomy. Lancet 2000; 356:730-733

Cheong DM, Vaccaro CA, Salanga VD, Wexner SD, Phillips RC, Hanson MR. Electrodiagnostic evaluation of fecal incontinence. Muscle Nerve 1995;18:612-9

Chrabot CM, Prasad ML, Abcarian H. Recurrent anorectal abscesses. Dis Colon Rectum 1983;26:105-108

Christensen A, Nilas L, Christiansen J. Treatment of transsphincteric anal fistulas by the seton technique. Dis Colon Rectum 1986;29:454-5

Chung CC, Ha JPY, Tai YP, Tsang WWC, Li MKW. Double-blind, randomized trial comparing harmonic scalpel TM hemorrhoidectomy, bipolar scissors hemorrhoidectomy, and scissors excision. Dis Colon Rectum 2002;45:789-794

Cirocco WC, Reilly JC. Challenging the predictive accuracy of Goodsall's rule for anal fistulas. Dis Colon Rectum 1992;35 :537-42

Cox SW, Senagore AJ, Luchtefeld MA, Mazier WP. Outcome after incision and drainage with fistulotomy for ischiorectal abscess. Am Surg 1997;63:686-689

Culp CE. Use of penrose drains to treat certain anal fistulas: a primary operative seton. Mayo Clin Proc 1984;59:613-7

Dayal Y, DeLellis RA. The gastrointestinal tract. In: Cotran RS, Kumar V, Robbins SL (eds.). Robbins pathologic basis of disease. WB Saunders Company Philadelphia 1989: 827-910

Dennison AR, Wherry DC, Morris DL. Hemorrhoids: Nonoperative management. Surg Clin North Am 1988;68:1401-1409

Diamant NE, Kamm MA, Wald A, Whitehead WE. AGA technical review on anorectal testing techniques. Gastroenterology 1999;116:735-60

Drossman DA, Sandler RS, Broom CM, McKee DC. Urgency and fecal soiling in people with bowel dysfunction. Dig Dis Sci 1986;31:1221-5

Duthie HL, Gairus FW. Sensory nerveendings and sensation in the anal region of man. Br J Surg 1960;47:585-595

Ebert KH, Meyer HJ. Die Klammernahtresektion bei Hämorrhoiden – eine Bestandsaufnahme nach zweijähriger Anwendung. Vergleich der Ergebnisse mit der Technik nach Milligan-Morgan. Zentralbl Chir 2002;127:9-14

Eisenhammer S. Advances of anorectal surgery with special reference to ambulatory treatment. S Afr Med J 1954;28:264-266

Eisenhammer S. The internal anal sphincter and the anorectal abscess. Surg Gynecol Obstet 1956;103:501-6

Eisenhammer S. The anorectal and anovulval fistulous abscess. Surg Gynecol Obstet 1961;113:519-20

Eisenhammer S. The anorectal fistulous abscess and fistula. Dis Colon Rectum 1966;9:91-106

El-Gendi MA, Abdel-Baky N. Anorectal pressure in patients with symptomatic hemorrhoids. Dis Colon Rectum 1986;29:388-391

Engel A, Kamm MA, Talbot IC. Progressive systemic sclerosis of the internal anal sphincter leading to passive faecal incontinence. Gut 1994;35:857-9

Engel AF, Kamm MA, Sultan AH, Bartram CL, Nicholls RJ: Anterior anal sphincter repair in patients with obstetric trauma. Br J Surg 1994;81:1231-4

Engel AF, Kamm MA, Bartram CI, Nicholls RJ. Relationship of symptoms in faecal incontinence to specific sphincter abnormalities. Int J Colorectal Dis 1995;10(3):152-5

Eu KW, Seow-Choen F, Goh HS. Comparison of emergency and elective haemorrhoidectomy. Br J Surg 1994;81(2):308-10

Fantin AC, Hetzer FH, Christ AD, Friedt M, Schwizer W. Influence of stapler haemorrhoidectomy on anorectal function and on patients's acceptance. Swiss Med Wkly 2002;132:38-42

Fazio VW. Complex anal fistulae. Gastroenterol Clin North Am 1987;16:93-114

Fazio VW. Early promise of stapling technique for haemorrhoidectomy (comment). Lancet 2000;355:768-768

Feldman M, Schiller LR. Disorders of gastrointestinal motility associated with diabetes mellitus. Ann Int Med 1983;98:378-84

Felt-Bersma RJ, Klinkenberg-Knol EC, Meuwissen SG. Investigation of anorectal function. Br J Surg 1988;75:747-56

Felt-Bersma RJF, Janssen JJWM, Klinkenberg-Knol EC, Hoitsma HFW, Meuwissen SGM. Soiling: anorectal function and results of treatment. Int J Colorectal Dis 1989 ;4 :37-40

Felt-Bersma RJ, Klinkenberg-Knol EC, Meuwissen SG. Anorectal function investigations in incontinent and continent patients: differences and discriminatory value. Dis Colon Rectum 1990;33:479-86

Felt-Bersma RJF, Cuesta MA, Koorevaar M, Strijers RLM, Meuwissen SGM, Dercksen EJ, Wesdorp RIC. Anal endosonography : relationship with anal manometry and neurophysiologic tests. Dis Colon Rectum 1992;35:944-949

Ferguson JA, Heaton JR. Closed hemorrhoidectomy. Dis Colon Rectum 1959;176:176-179

Ferguson JA, Mazier WP, Ganchrow MI, Friend WG. The closed technique of hemorrhoidectomy. Surgery 1971;70(3):480-484

Fine J, Lawes CHW. On the muscle-fibres of the anal submucosa, with special reference to the pecten band. Br J Surg 1940;27:723-727

Francombe J, Carter PS, Hershman MJ. The aetiology and epidemiology of faecal incontinence. Hosp Med 2001;62(9):529-532

Frenckner B, von Euler C. Influence of pudendal block on the function of the anal sphincters. Gut 1975;16:482-9

Ganchrow MI, Bowman HE, Clark JF. Thrombosed hemorrhoids: a clinicopathologic study. Dis Colon Rectum 1971;14(5):331-340

Ganchrow MI, Mazier WP, Friend WG, Ferguson JA. Hemorrhoidectomy revisited – a computer analysis of 2,038 cases. Dis Colon Rectum 1971;14(2):128-133

Garcia-Aguilar J, Belmonte C, Wong WD, Goldberg SM, Madoff RD. Anal fistula surgery: factors associated with recurrence and incontinence. Dis Colon Rectum 1996;39 :723-9

Garcia-Aquilar J, Belmonte C, Wong WD, Lowry AC, Madoff RD. Open vs. closed sphincterotomy for chronic anal fissure: long-term results. Dis Colon Rectum 1996 ;39 :440-3

Garcia-Aquilar J, Davey CS, Le CT, Lowry AC, Rothenberger DA. Patient satisfaction after surgical treatment for fistula-in-ano. Dis Colon Rectum 2000 ;43 :1206-1212

Garcia-Aguilar J. Invited commentary. Dis Colon Rectum 2001;44(11):1619-1621

Gass OC, Adams J. Hemorrhoids: Etiology and pathology. Am J Surg 1950;79:40-43

Gemsenjäger E. Hämorrhoidenexzision mit primärer Wundnaht. Schweiz Med Wochenschr 1989;119:259-261

Gibbons CP, Trowbridge EA, Bannister JJ, Read NW. The role of the anal cushions in maintaining continence. Lancet 1986;i:886-887

Gilliland R, Altomare DF, Moreira H Jr, Oliveira L, Gilliland JE, Wexner SD. Pudendal neuropathy is predictive of failure following anterior overlapping sphincteroplasty. Dis Colon Rectum 1998;41:1516-22

Glickman S, Kamm MA. Bowel dysfunction in spinal cord injury patients. Lancet 1996;347:1651-3

Goldberg SM. Symposium: fistula-in-ano. Discussion. Dis Colon Rectum 1976;19:520-528

Goligher JC, Graham NG, Clark CG, De Dombal FT, Giles G. The value of stretching the anal sphincters in the relief of post-haemorrhoidectomy pain. Br J Surg 1969;56:859-863

Golighten J, Graham NG, Cleark CG, De Dohmal IT, Giles G. The value of stretching the anal sphincter in the relief of post-haemorrhoidectomy pain. Br J Surg 1969;56:859-861

Golub RW, Wise WE Jr, Kerner BA, Khanduja KS, Aguilar PS. Endorectal mucosal advancement flap: the preferred method for complex crypoglandular fistula-in-ano. J Gastrointest Surg 1997;1:487-91

Grace RH, Creed A. Prolapsing thrombosed hemorrhoids: outcome of conservative management. Br Med J 1975;III:354

Graf W, Pahlman L, Ejerblad S. Functional results after seton treatment of high transsphincteric anal fistulas. Eur J Surg 1995;161:289-291

Grosz CR. A surgical treatment of thrombosed external hemorrhoids. Dis Colon Rectum 1990;33:249-250

Gustafsson UM, Graf W. Excision of anal fistula with closure of the internal opening. Functional and manometric results. Dis Colon Rectum 2002;45:1672-1678

Haas PA, Fox TA. The importance of the perianal connective tissue in the surgical anatomy and function of the anus. Dis Colon Rectum 1977;20(4):303-13

Haas PA, Fox TA. Age-related changes and scar formations of perianal connective tissue. Dis Colon Rectum 1980;23(3):160-9

Haas PA, Haas GP, Schmaltz S, Fox TA. The prevalence of haemorrhoids. Dis Colon Rectum 1983;26:435-439

Haas PA, Fox TA, Haas GP. The pathogenesis of hemorrhoids. Dis Colon Rectum 1984 ;27 :442-450

Hallan RI, Marzouk DEMM, Waldron JD, et al. Comparison of digital and manometric assessment of anal sphincter function. Br J Surg 1989;76:973-5

Hamalainen KP, Sainio AP. Cutting seton for anal fistulas: high risk of minor control defects. Dis Colon Rectum 1997;40:1443-7

Hamilton CH. Anorectal problems : the deep postanal space – surgical significance in horseshoe fistula and abscess. Dis Colon Rectum 1975;18:642-645

Hancock BD, Smith K. The internal sphincter and Lord's procedure for haemorrhoids. Br J Surg 1975;62:833-836

Hancock BD. Measurement of anal pressure and motility. Gut 1976;17(8):645-651

Hancock BD. Internal sphincter and the nature of haemorrhoids. Gut 1977;18:651-656

Hancock BD. Lord's procedure for haemorrhoids: a prospective anal pressure study. Br J Surg 1981;68:729-730

Hanley PH, Ray JE, Pennington EE, Grablowsky OM. Fistula-in-ano: a ten year study of horseshoe-abscess fistula-in-ano. Dis Colon Rectum 1976;19:507-515

Hanley PH. Anorectal abscess fistula. Surg Clin North Am 1978;58:487-503

Hanley PH. Rubber band seton in the management of abscess-anal fistula. Ann Surg 1978;187:435-7

Hanley PH. Anorectal supralevator abscess – fistula-in-ano. Surg Gynecol Obstet 1979;148:899-904

Hansen HH. Die Bedeutung des M. canalis ani für die Kontinenz und anorectale Erkrankungen. Langenbecks Arch Chir 1976;341:23-37

Hansen HH. Neue Aspekte zur Pathogenese und Therapie des Hämorrhoidalleidens. Dtsch Med Wochenschr 1977;102:1244-1248

Hansen JB, Jorgensen SJ. Radical emergency operation for prolapsed and strangulated haemorrhoids. Acta Chir Scand 1975;141:810-812

Harari D, Sarkarati M, Gurwitz JH, McGlinchey-Berroth G, Minaker KL. Constipation-related symptoms and bowel program concerning individuals with spinal cord injury. Spinal Cord 1997;35:394-401

Hassink EA, Rieu PN, Severijnen RS, Brugman-Boezeman AT, Festen C. Adults born with high anorectal atreasia – how do they manage? Dis Colon Rectum 1996 ;39 :695-9

Hayssen TK, Luctefeld MA, Senagore AJ. Limited hemorrhoidectomy: results and long term follow-up. Dis Colon Rectum 1999;42:909-914

Henrich M. Clinical topography of the proctodeum. Acta ant 1980;106:161-170

Hiltunen KM, Matikainen M. Anal manometric findings in symptomatic hemorrhoids. Dis Colon Rectum 1985;28:807-809

Hinds JP, Eidelman BH, Wald A. Prevalence of bowel dysfunction in multiple sclerosis. A population survey. Gastroenterology 1990;98:1538-42

Ho YH, Seow-Choen F, Goh HS. Haemorrhoidectomy and disordered rectal and anal physiology in patients with prolapsed haemorrhoids. Br J Surg 1995;82(5): 596-8

Ho YH, Goh HS. Unilateral anal electrosensation. Modified technique to improve quantification of anal sensory loss. Dis Colon Rectum 1995;38(3):239-244

Ho YH, Tan M. Ambulatory anorectal manometric findings in patients before and after haemorrhoidectomy. Int J Colorectal Dis 1997;12:296-7

Ho YH, Tan M, Chui CH, Leong A, Eu KW, Seow-Cheong F. Randomized controlled trial of primary fistulotomy with drainage alone for perianal abscesses. Dis Colon Rectum 1997;40 :1435-1438

Ho YH, Seow-Choen F, Tan M, Leong AF. Randomized controlled trial of open and closed haemorrhoidectomy. Br J Surg 1997;84:1729-1730

Ho YH, Ang M, Nyam D, Tan M, Seow-Choen R. Transanal approach to rectocele repair may compromise anal sphincter pressures. Dis Colon Rectum 1998;41:354-358

Ho YH, Cheong WK, Tsang C, Ho J, Eu KW, Tang CL, Seow-Choen F. Stapled hemorrhoidectomy – cost and effectiveness. Randomized, controlled trial including incontinence scoring, anorectal manometry, and endoanal ultrasound assessments at up to three months. Dis Colon Rectum 2000 ;43(12) :1666-75

Ho YH, Tsang C, Tang CL, Nyam D, Eu KW, Seow-Choen F. Anal sphincter injuries from stapling instruments introduced transanally : randomized, controlled study with endoanal ultrasound and anorectal manometry. Dis Colon Rectum 2000;43:169-173

Ho YH, Seow-Choen F, Tsang C, Eu KW. Randomized trial assessing anal sphincter injuries after stapled haemorrhoidectomy. Br J Surg 2001;88(11):1449-55

Holzheimer RG. Surgical treatment of haemorrhoids. In: Holzheimer RG, Mannick JA (eds): Surgical treatment – evidence based and problem-oriented. Zuckschwerdt Publishers Munich 2001:257-265

Howard PM, Pingree JH. Immediate radical surgery for hemorrhoidal disease with acute extensive thrombosis. Am J Surg 1968;116:777-778

Hulme-Moir M, Bartolo DC. Hemorrhoids. Gastroenterol Clin North Am 2001 ;30(1):183-197

Isler JT. Hemorrhoidectomy. Part A: Open surgical hemorrhoidectomy. In: Bailey HR, Snyder MJ (eds.). Ambulatory anorectal surgery. Springer Publishing Company Heidelberg 1999: 81-88

Jacobs PP, Scheuer M, Kipers JH, Vingerhoets MH. Obstetric fecal incontinence: role of pelvic floor denervation and results of delayed sphincter repair. Dis Colon Rectum 1990;33:494-7

Johanson JF, Sonnenberg A. The prevalence of hemorrhoids and chronic constipation. An epidemiologic study. Gastroenterology 1990;98(2):380-386

Johanson JF, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. Am J Gastroenterol 1996;91:33-6

Johnson FP. The development of the rectum in the human embryo. Am J Anat 1914;16:1-57

Jongen J, Bach S, Stubinger SH, Bach JU. Excision of thrombosed external hemorrhoid under local anesthesia: a retrospective evaluation of 340 patients. Dis Colon Rectum 2003 ;46(9) :1226-31

Kamm MA, Lennard-Jones JE. Rectal mucosal electrosensory testing – evidence for a rectal sensory neuropathy in idiopathic constipation. Dis Colon Rectum 1990;33(5):419-423

Kamm MA. Obstetric damage and fecal incontinence. Lancet 1994;344:730

Keck JO, Schoetz DJ, Roberts PL, Murray JJ, Coller JA, Veidenheimer MC. Rectal mucosectomy in the treatment of giant rectal villous tumors. Dis Colon Rectum 1995;38:233-238

Kennedy HL, Zegarra JP. Fistulotomy without external sphincter division for high anal fistulae. Br J Surg 1990;77:898-901

Khalil KH, O'Bichere A, Sellu D. Randomized clinical trial of sutured versus stapled closed haemorrhoidectomy. Br J Srg 2000;87:1352-1355

Khubchandani IT, Trimpi HD, Sheets JA. Closed hemorrhoidectomy with local anesthesia. Surg Gynecol Obstet 1972;135:955-957

Khubchandani IT, Reed JF. Sequelae of internal sphincterotomy for chronic fissure-in-ano. Br J Surg 1989; 76:431-4

Kiff ES, Swash M. Slowed conduction in the pudendal nerve in idiopathic (neurogenic) fecal incontinence. Br J Surg 1984;71:614-6

Kodner IJ. Editorial comment. Dis Colon Rectum 1990 ;33(6) :485-486

Kodner IJ, Mazor A, Shemesh EI, Fry RD, Fleshman JW, Birnbaum EH. Endorectal advancement flap repair of rectovaginal and other complicated anorectal fistulas. Surgery 1993;114:682-90

Kohlstadt CM, Weber J, Prohm P. Stapler hemorrhoidectomy. A new alternative to conventional methods. Zentralbl Chir 1999;124:238-243

Konsten J, Baeten CG. Hemorrhoidectomy vs Lord's method: 17 year follow-up of a prospective, randomized trial. Dis Colon Rectum 2000;43(4) :503-6

Kouba R. Die Hämorrhoidektomie. Gegenüberstellung der Operationsmethoden von Milligan-Morgan und Parks. Chirurg 1980;51:784-788

Kratzer GL. The anal ducts and their clinical significance. Am J Surg 1950;79:32-39

Krogh K, Nielsen J, Djurhuus JC, Mosdal C, Sabroe S, Laurberg S. Colorectal function in patients with spinal cord lesions. Dis Colon Rectum 1997 ;40 :1233-9

Kuster GG. Relationship of anal glands to lymphatics. Dis Colon Rectum 1965;8:329-333

Kuypers HC. Use of seton in the treatment of extrasphincteric anal fistula. Dis Colon Rectum 1984; 27:109-10

Lane RHS. Measurement of anal pressure in patients with haemorrhoids. Schweiz Rundsch Med Prax 1982;71:112-115

Laurberg S, Swash M, Henry MM. Delayed external sphincter repair for obstetric tear. Br J Surg 1988;75:786-8

Laurence AE, Murray AJ. Histopathology of prolapsed and thrombosed hemorrhoids. Dis Colon Rectum 1962 ;5 :56-61

Lawson JON. Pelvic anatomy II. Anal canal and associated sphincters. Ann R Coll Surg Engl 1974;54:288-300

Lestar B, Penninckx F, Kerremans R. The composition of anal basal pressure. An in vivo and in vitro study in man. Int J Colorectal Dis 1989;4 :118-122

Lestar B, Penninckx F, Rigauts H, Kerremans R. The internal anal sphincter can not close the anal canal completely. Int J Colorecal Dis 1992;7:159-161

Lin JK. Anal manometric studies in hemorrhoids and anal fissures. Dis Colon Rectum 1989 ;32 :839-842

Lockhart-Mummery HE. Anorectal problems: treatment of abscesses. Dis Colon Rectum 1975;18:650-651

Loder PB, Kamm MA, Nicholls RJ. Haemorrhoids: Pathology, pathophysiology and aetiology. Br J Surg 1994;81:946-954

Loening-Baucke V, Anuras S. Effects of age and sex on anorectal manometry. Am J Gastroenterol 1985;80: 50-3

Longo A. Treatment of hemorrhoids disease by reduction of mucosa and hemorrhoidal prolapse with a circular suturing device: a new procedure. 6th World Congress of endoscopic surgery. Rome.Manduzzi 1998:777-784

Lord PH. A day-case procedure for the cure of third-degree haemorrhoids. Br J Surg 1969;56(10):747-749

Lund JN, Scholefield JH. Aetiology and treatment of anal fissure. Br J Surg 1996;83:1335-44

Lunniss PJ, Phillips RKS. Anatomy and function of the anal longitudinal muscle. Br J Surg 1992;79:882-884

Lunniss PJ, Kamm MA, Phillips RK. Factors affecting continence after surgery for anal fistula. Br J Surg 1994;81:1382-5

MacIntyre IMC, Balfour TW. Results of the Lord non-operative treatment for haemorrhoids. Lancet 1972;1(7760):1094-1095

MacRae HM, McLeod RS. Comparison of hemorrhoidal treatment modalities: a meta-analysis. Dis Colon Rectum 1995;38:687-694

Malone PS, Wheeler RA, Williams JE. Continence in patients with spina bifida: long term results. Arch Dis Child 1994;70:107-10

Malouf AJ, Halligan S, Williams AB, Bartram CI, Dhillon S, Kamm MA. Prospective assessment of interobserver agreement for endoanal MRI in fecal incontinence. Abdom Imaging 2001;26(1):76-78

Mann CV, Clifton MA. Re-outing of the track for the treatment of high anal and anorectal fistulae. Br J Surg 1985;72:134-7

Mathai V, Ong BC, Ho YH. Randomized controlled trial of lateral sphincterotomy with haemorrhoidectomy. Br J Surg 1996;83:380-382

Matos D, Lunniss PJ, Phillips RK. Total sphincter conservation in high fistula-in-ano: results of a new approach. Br J Surg 1993;80:802-4

Mavrantonis C, Wexner SD. A clinical approach to fecal incontinence. J Clin Gastroenterol 1998;27(2):108-121

Mazier WP. Emergency hemorrhoidectomy – a worthwhile procedure. Dis Colon Rectum 1973;16(3):200-205

McHugh SM, Diamond NE. Effect of age, gender and parity on anal canal pressures. Dig Dis Sci 1987;37:726-736

Mehigan BJ, Monson JRT, Hartley JE. Stapling procedure for haemorrhoids versus Milligan-Morgan haemorrhoidectomy: randomised controlled trial. Lancet 2000;355:782-785

Melange M, Colin JF, Van Wymersch T, Vanheuverzwyn R. Anal fissure: correlation between symptoms and manometry before and after surgery. Int J Colorectal Dis 1992;7(2) :108-111

Menter R, Weitzenkamp D, Cooper D, Bingley J, Charlifue S, Whiteneck G. Bowel management outcomes in individuals with long-term sinal cord injuries. Spinal Cord 1997;35:608-12

Miller R, Bartolo DC, Cervero F, Mortensen NJ. Differences in anal sensation in continent and incontinent patients with perineal descent. Int J Colorectal Dis 1989;4(1):45-49

Milligan ETC, Morgan CN. Surgical anatomy of the anal canal with special reference to anorectal fistula. Lancet 1934;ii:1213-1217

Milligan ET, Morgan CN, Jones LE, Officer R. Surgical anatomy of the anal canal and the operative treatment of haemorrhoids. Lancet 1937;2:1119-1124

Milsom JW, Mazier WP. Classification and management of post-surgical anal stenosis. Surg Gynecol Obstet 1986;163:1-5

Molloy RG, Kingsmore D. Life threatening pelvic sepsis after stapled haemorrhoidectomy. Lancet 2000;355:810

Moore-Gillon V. Constipation: what does the patient mean? J R Soc Med 1984;77:108-10

Morgan CN. Surgical anatomy of the anal canal and rectum. Postgrad Med J 1936;12:287-300

Morgan CN, Thompson HR. Surgical anatomy of the anal canal with special reference to the surgical importance of the internal sphincter and conjoint longitudinal muscle. Ann R Coll Surg Engl 1956;19:88-114

Mortensen PE, Olsen J, Pedersen IK, Christiansen J. A randomized study on hemorrhoidectomy combined with anal dilatation. Dis Colon Rectum 1987;30:755-757

Mosley JG, Galland RB, Saunders JH, Spencer J. Haemorrhoids – objecitve measurement of proctoscopic appearances. Postgrad Med J 1980;56:30-33

Mulder W, de Jong E, Wauters I, Kinders M, Heij HA, Vos A. Posterior sagittal anorectoplasty: functional results of primary and secondary operations in comparison to the pull-through method in anorectal malformations. Eur J Paediatr Surg 1995 ;5 :170-3

Neill ME, Parks AG, Swash M. Physiological studies of the anal sphincter musculature in faecal incontinence and rectal prolaps. Br J Surg 1981;68:531-6

Nelson R, Norton N, Cautley E, Furner S. Community based prevalence of anal incontinence. JAMA 1995;274:559-61

Nieves PM, Perez J, Suarez JA. Hemorrhoidectomy – How I do it: experience with the St. Mark's Hospital technique for emergency hemorrhoidectomy. Dis Colon Rectum 1977;20:197-201

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

Oh C. Management of high recurrent anal fistula. Surgery 1983;93:330-2

Oh C. Acute thrombosed external hemorrhoids. Mt Sinai J Med 1989;56(1):30-32

Okamato GA, Lamers JV, Shurtleff DB. Skin breakdown in patients with myelomeningocele. Arch Phys Med Rehab 1983;64:20-23

Ozuner G, Hull TL, Cartmill J, Fazio VW. Long-term analysis of the use of transanal rectal advancement flaps for complicated anorectal/vaginal fistulas. Dis Colon Rectum 1996;39:10-4

Parkash S, Lakshmiratan V, Gajendran V. Fistula-in-ano: treatment by fistulectomy, primary closure and reconstruction. Aust N Z J Surg 1985;55:23-7

Parks AG. The surgical treatment of haemorrhoids. Br J Surg 1956;XLIII (January):337-351

Parks AG. Etiology and surgical treatment of fistula-in-ano. Dis Colon Rectum 1963;6:17-22

Parks AG. Haemorrhoidectomy.Surg Clin North Am 1965;45(5):1305-1315

Parks AG, Fishlock DJ, Cameron JD, May H. Catecholamine release in the lower gastrointestinal tract. Gut 1966;7:104

Parks AG, Stitz RW. Symposium: fistula-in-ano. The treatment of high fistula-in-ano. Dis Colon Rectum 1976 ;19 :487-99

Parks AG, Gordon PH, Hardcastle JD. A classification of fistula in ano. Br J Surg 1976;63:1-12

Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. Gut 1977;18:656-665

Pearl RK, Andrews JR, Orsay CP, Weisman RI, Prasad ML, Nelson RL, Cintron JR, Abcarian H. Role of the seton in the management of anorectal fistulas. Dis Colon Rectum 1993;36:573-9

Penninckx F, Lestar B, Kerremans R. The internal anal sphincter: Mechanisms of control and its role in maintaining anal continence. Baill Clin Gastroenterol 1992;6(1):193-214

Pescatori M, Marin G, Anastasio G, Rinallo L. Anal manometry improves the outcome of surgery for fistula-in-ano. Dis Colon Rectum 1989;32:588-592

Pinho M, Yoshioka K, Ortiz J, Oya M, Keighley MRB. The effect of age on pelvic floor dynamics. Int J Colo-rectal Dis 1990;5:207-8

Poen AC, Felt-Bersma RFJ, Strijers RLM, Dekker GA, Cuesta MA. Third obstetrical perineal tear : long term clinical and functional results after primary repair. Br J Surg 1998;85:1433-8

Prasad ML, Read DR, Acarian H. Supralevator abscesses: diagnosis and treatment. Dis Colon Rectum 1981;24 :456-461

Ramanujam PS, Prasad ML, Abcarian H. The role of seton in fistulotomy of the anus. Surg Gynecol Obstet 1983;157:419-22

Ramanujam PS, Prasad ML, Abcarian H, Tan AB. Perianal abscesses and fistulas: a study of 1023 patients. Dis Colon Rectum 1984;27:593-597

Read DR, Abcarian H. A prospective survey of 474 patients with anorectal abscess. Dis Colon Rectum 1979;22:566-568

Read NW, Harford WV, Schmulen AC, et al. A clinical study of patients with faecal incontinence and diarrhea. Gastroenterology 1979;76:747-56

Read MG, Read NW, Haynes WG, Donnelly TC, Johnson AG. A prospective study of the effect of haemorrhoidectomy on sphincter function and faecal continence. Br J Surg 1982;69:396-398
Read MG, Read NW. The role of anal sensation in preventing incontinence. Gut 1982;23:345-347
Read NW, Bartolo DC, Read MG. Differences in anal function in patients with incontinence to solids and in patients with incontinence to liquids. Br J Surg 1984;71:39-42
Read MG, Read NW, Haynes WG, Donnelly TG, Johnson AG. A prospective study of the effect of haemorrhoidectomy on sphincter function and faecal incontinence. Br J Surg 1992;69:396-8
Reznick RK, Bailey HR. Closure of the internal opening for treatment of complex fistula-in-ano. Dis Colon Rectum 1988;31:116-8
Roe A, Bartolo D, Vellacott K, Locke-Edmunds J, Mortensen NJ. Submucosal versus ligation excision haemorrhoidectomy: a comparison of anal sensation, anal sphincter manometry and post-operative pain function. Br J Surg 1987;74:948-995
Rowsell M, Bello M, Hemingway DM. Circumferential mucosectomy (stapled hemorrhoidectomy) versus conventional hemorrhoidectomy: randomised controlled trial. Lancet 2000;355:779-781
Rudd WWH. Hemorrhoidectomy in the office: method and precautions. Dis Colon Rectum 1970;13(6):438-440
Sainio P. Fistula-in-ano in a defined population. Incidence and epidemiological aspects. Ann Chir Gynaecol 1984;73:219-224
Sainio P, Husa A. Fistula in ano. Clinical features and long term results in surgery in 199 adults. Acta Chir Scand 1985 ;151 :169-76
Sakulsky SB, Blumenthal JA, Lynch RH. Treatment of thrombosed hemorrhoids by excision. Am J Surg 1970;120:537-538
Saleeby RG, Rosen L, Stasik JJ, Riether RD, Sheets J, Khubchandani IT. Hemorrhoidectomy during pregnancy: risk or relief? Dis Colon Rectum 1991;34:260-261
Sangwan YP, Solla JA. Internal anal sphincter. Advances and insights. Dis Colon Rectum 1998;41 :1297-1311
Sayfan J. Complications of Milligan-Morgan hemorrhoidectomy. Dig Surg 2001;18:131-133
Schouten WR, van Vroonhoven TJ. Treatment of anorectal abscess with or without primary fistulectomy: results of a prospective randomized trial. Dis Colon Rectum 1991;34:60-3
Scoma JA, Salvati EP, Rubin RJ. Incidence of fistulas subsequent to anal abscesses. Dis Colon Rectum 1974;17:357-359
Seow-Choen F, Phillips RKS. Insights gained from the management of problematical anal fistulae at St. Mark's hospital 1984-88 Br J Surg 1991;78:539-541
Seow-Choen F, Nicholls RJ. Anal fistula. Br J Surg 1992;79:197-205
Seow-Choen F, Ho YH, Ang HG, Goh HS. Prospective, randomized trial comparing pain and clinical function after conventional scissors excision/ligation vs. diathermy excision without ligatuin for symptomatic prolapsed hemorrhoids. Dis Colon Rectum 1992;35:1165-1169
Seow-Choen F, Low HC. Prospective randomized study of radical versus four piles haemorrhoidectomy for symptomatic large circumferential prolapsed piles. Br J Surg 1995;82(2):188-9
Shafik A. The pathogenesis of haemorrhoids and their treatment by anorectal bandotomy. J Clin Gastroenterol 1984;6:129-137
Shalaby R, Desoky A. Randomized clinical trial of staples versus Milligan-Morgan haemorrhoidectomy. Br J Surg 2001;88:1049-1053
Shemesh EI, Kodner IJ, Fry RD, Neufeld DM. Endorectal sliding flap repair of complicated anterior anoperineal fistulas. Dis Colon Rectum 1988;31:22-4
Shoulder PJ, Grimley MR, Alexander Williams J. Fistula in ano is usually simple to manage surgically. Int J Colorectal Dis 1986 ;1 :113-5
Shropshear G. Surgical anatomic aspects of the anorectal sphincter mechanism and its clinical significance. J Int Coll Surg 1960;33:267-287
Smith M. Early operation for acute haemorrhoids. Br J Surg 1967;54:141-144
Snooks SJ, Swash M, Setchell M, Henry MM. Injury to innervation of pelvic floor sphincter musculature. Lancet 1984;ii:546-550
Snooks SJ, Henry MM, Swash M. Faecal incontinence due to external anal sphincter division in childbirth is associated with damage to the innervation of the pelvic floor: a double pathology. Br J Obstet Gynecol 1985;92:824-8
Snooks SJ, Swash M, Henry MM, Setchell M. Risk factors in childbirth causing damage to the pelvic floor innervation: a precursor of stress incontinence. Int J Colorectal Dis 1986 ;1 :20-24
Snooks SJ, Swash M, Mathens SE, Henry MM. Effects of vaginal delivery on the pelvic floor: a 5 year follow up. Br J Surg 1990;77:1358-60
Stelzner F. Die anorectalen Fisteln. Springer-Verlag Berlin Heidelberg 1959
Stelzner F, Staubesand J, Machleidt H. Das corpus cavernosum recti – die Grundlage der inneren Hämorrhoiden. Langenbecks Arch klein Chir 1962;299:302-312
Stelzner F, Fleischhauer F, Holstein AF. Die Bedeutung des Sphincter internus für die Analkontinenz. Langenbecks Arch klein Chir 1966;314:132-136
Stelzner F. Die anorectale Inkontinenz – Ursache und Behandlung. Chirurg 1991;62:17-24
Stelzner F. Die Hämorrhoidektomie – eine einfache Operation ? Inkontinenz, Stenose, Fistel, Infektion und Todesfälle. Chirurg 1992;63:316-326
Stelzner F. Anatomisch bedingte diagnostische und operationstechnische Probleme und Komplikationen in der Chirurgie am Anorektum. Zentralbl Chir 1992;117:111-114
Stern W. Haemorrhoids. Med J Aust 1964;September: 428-429
Stern W. Thrombosed haemorrhoids: immediate surgical treatment. Med J Aust 1964;October 17:635-636
Stieve H. Über die Bedeutung der venösen Wundernetze für den Verschluß einzelner Öffnungen des menschlichen Körpers. Dtsch Med Wochenschr 1928;54:87-90,130-133
Stieve H. Über den Verschluß des menschlichen Afters. Z Mikrosk Anat Forsch 1930;21:642-653
Sultan AH, Kamm MA, Hudson CN, Bartram CI. Anal sphincter disruption during vaginal delivery. N Engl J Med 1993;329:1905-11
Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labour: prospective study before and after childbirth. Br J Obstet Gynaecol 1994;101:22-8
Sultan AH, Johanson RB, Carter JE. Occult anal sphincter trauma following randomized forceps and vacuum delivery. Int J Gynaecol Obstet 1998;61:113-19
Sun WM, Read NW, Shorthouse AJ. Hypertensive anal cushions as a cause of high anal canal pressures in patients with haemorrhoids. Br J Surg 1990;77:458-62
Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. N Engl J Med 1993;329:1905-11

Swash M, Snooks SJ, Henry MM. A unifying concept of pelvic floor disorders and incontinence. J R Soc Med 1985;78:906-911

Tajana A. Hemorrhoidectomy according to Milligan-Morgan: ligature and excision technique. Int Surg 1989;74:158-161

Talley NJ, O'Keefe EA, Zinsmeister AR, Melton LJ III. Prevalence of gastrointestinal symptoms in the elederly: a population-based study. Gastroenterology 1992;102(3):895-901

Teramoto T, Parks AG, Swash M. Hypertrophy of the external anal sphincter in haemorrhoids: a histometric study. Gut 1981;22(1):45-8

Ternent CA, Shashidharan M, Blatchford GJ, Christensen MA, Thorson AG, Sentovich SM. Transanal ultrasound and anorectal physiology findings affecting continence after sphincteroplasty. Dis Colon Rectum 1997;40:462-7

Terranova O, Battocchio F, Martella B, Celi D. Anal fistulas with recess above the anal levators. Int Surg 1989;74:267-269

Thomson H. The real nature of perianal haematoma. Lancet 1982;28:467-468

Thomson JP, Ross AH. Can the external anal sphincter be preserved in the treatment of trans-sphincteric fistula-in-ano? Int J Colorectal Dis 1989 ;4 :247-50

Thomson WHF. The nature of haemorrhoids. Br J Surg 1975;62:542-552

Thomson WHF. The anatomy and nature of piles. In: Kaufman HD (ed): The haemorrhoids syndrome. Turnbridge Wells, Kent, England, Abacus Press, 1981:15-33

Thomson WH. Haemorrhoids. In: Morris PJ and Malt RA (eds.). Oxford Textbook of Surgery. Oxford Medical Publications. Oxford University Press 1994: 1125-1136

Tinckler LF, Baratham G. Immediate haemorrhoidectomy for prolapsed piles. Lancet 1964; November 28:1145-1146

Turell R. Hemorrhoidectomy, with special reference to open versus closed technics. Surg Clin North Am 1952;32:677-686

Ustynoski K, Rosen L, Stasik J, Riether R, Sheets J, Khubchandani IT. Horseshoe abscess fistula: seton treatment. Dis Colon Rectum 1990;33 :602-5

Van Tets WF, Kuijpers HC. Continence disorders after anal fistulotomy. Dis Colon Rectum 1994;37 :1194-7

Vaizey C, Bartram CI, Kamm MA. Primary internal anal sphincter degeneration. A previously unrecognised cause of passive faecal incontinence. Lancet 1997;349:612-5

Vaizey CJ, Kamm MA, Nicholls RJ. Recent advantages in the surgical treatment of faecal incontinence. Br J Surg 1998;85:596-603

Van Tets WF, Kuijpers HC. Continence disorders after anal fistulotomy. Dis Colon Rectum 1994;37:1194-1197

Varma JS, Smith AN, Busutil A. Crrelations of clinical and manometric abnormalities of rectal function following chronic radiation injury. Br J Surg 1985;72:875-878

Varma JS, Smith AN, Busutil A. Function of the anal sphincters after chronic radiation injury. Gut 1986;27:528-533

Vasilevsky CA, Gordon PH. The incidenceof recurrent abscesses of fistula-in-ano following anorectal suppuration. Dis Colon Rectum 1984;27:126-130

Vernava AM III, Longo WE, Daniel GL. Pudendal neuropathy and the importance of EMG evaluation of fecal incontinence. Dis Colon Rectum 1993;36:23-7

Watts JM, Bennett RC, Duthie HL, Goligher JC. Healing and pain after haemorrhoidectomy. Br J Surg 1964 ;51 :808-817

Wedell J, Meier zu Eissen P, Banzhaf G, Kleine L. Sliding flap advancement for the treatment of high level fistulae. Br J Surg 1987;74:390-1

Wexner SD, Marchetti F, Salanga VD, Corredor C, Jagelman DG. Neurophysiologic assessment of the anal sphincters. Dis Colon Rectum 1991;34:606-12

Wexner SD, Marchetti F, Jagelman DG. The role of sphincteroplasty for fecal incontinence re-evaluated: a prospective physiologic and functional review. Dis Colon Rectum 1991;34:22-80

Whitehead WE, Wald A, Diamant NE, Enck P, Pemberton JH, Rao SS. Functional disorders of the anus and rectum. Gut 1999;45(Suppl II):II55-9

Whitehead WE, Wald A, Norton NJ. Treatment options for fecal incontinence. Dis Colon Rectum 2001 ;44 :131-144

Williams JG, MacLeod CA, Rothenberger DA, Goldberg SM. Seton treatment of high anal fistulae. Br J Surg 1991;78:1159-61

Wolf JS, Munoz JJ, Rosin JD. Srvey of hemorrhoidectomy practices: open versus closed techniques. Dis Colon Rectum 1979;22(8):536-538

Wunderlich M, Swash M. The overlapping innervation of the two sides of the external anal sphincter by the pudendal nerves. J Neurol Sci 1983;59:97-109

Yang YK, Wexner SD, Nogueras JJ, Jagelman DG. The role of anal ultrasound in the assessment of benign anorectal disease. Coloproctology 1993;5:260-4

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

Zuber TJ. Hemorrhoidectomy for thrombosed external hemorrhoids. Am Fam Phys 2002;65(8):1629-1632

Received: December 8, 2003 / Accepted: January 9, 2004

Address for correspondence:
René Gordon Holzheimer MD PhD
Praxisklinik Sauerlach (Center for Daycase Surgery)
Tegernseer Landstr. 8
D-82054 Sauerlach
Tel. +49-8104-887822
Fax +49-8104-887824
E-mail info@praxisklinik-sauerlach.de