

Hemorrhoids: From basic pathophysiology to clinical management

Varut Lohsiriwat

Varut Lohsiriwat, Division of Colon and Rectal Surgery, Department of Surgery, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand

Author contributions: Lohsiriwat V was the sole contributor to literature review, acquisition, analysis of data and manuscript preparation.

Supported by Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Correspondence to: Varut Lohsiriwat, MD, PhD, Division of Colon and Rectal Surgery, Department of Surgery, Faculty of Medicine Siriraj Hospital, Mahidol University, Prannok Road, Bangkok 10700, Thailand. bolloon@hotmail.com

Telephone: +66-0-24198077 Fax: +66-0-24115009

Received: September 12, 2011 Revised: January 10, 2012

Accepted: February 8, 2012

Published online: May 7, 2012

Abstract

This review discusses the pathophysiology, epidemiology, risk factors, classification, clinical evaluation, and current non-operative and operative treatment of hemorrhoids. Hemorrhoids are defined as the symptomatic enlargement and distal displacement of the normal anal cushions. The most common symptom of hemorrhoids is rectal bleeding associated with bowel movement. The abnormal dilatation and distortion of the vascular channel, together with destructive changes in the supporting connective tissue within the anal cushion, is a paramount finding of hemorrhoids. It appears that the dysregulation of the vascular tone and vascular hyperplasia might play an important role in hemorrhoidal development, and could be a potential target for medical treatment. In most instances, hemorrhoids are treated conservatively, using many methods such as lifestyle modification, fiber supplement, suppository-delivered anti-inflammatory drugs, and administration of venotonic drugs. Non-operative approaches include sclerotherapy and, preferably, rubber band ligation. An operation is indicated when non-operative approaches have failed or complications have occurred.

Several surgical approaches for treating hemorrhoids have been introduced including hemorrhoidectomy and stapled hemorrhoidopexy, but postoperative pain is invariable. Some of the surgical treatments potentially cause appreciable morbidity such as anal stricture and incontinence. The applications and outcomes of each treatment are thoroughly discussed.

© 2012 Baishideng. All rights reserved.

Key words: Hemorrhoids; Pathophysiology; Treatment; Management; Outcome

Peer reviewer: Rasmus Goll, MD, Department of Gastroenterology, University Hospital of North Norway, 9038 Tromsø, Norway

Lohsiriwat V. Hemorrhoids: From basic pathophysiology to clinical management. *World J Gastroenterol* 2012; 18(17): 2009-2017 Available from: URL: <http://www.wjgnet.com/1007-9327/full/v18/i17/2009.htm> DOI: <http://dx.doi.org/10.3748/wjg.v18.i17.2009>

INTRODUCTION

Hemorrhoids are a very common anorectal condition defined as the symptomatic enlargement and distal displacement of the normal anal cushions. They affect millions of people around the world, and represent a major medical and socioeconomic problem. Multiple factors have been claimed to be the etiologies of hemorrhoidal development, including constipation and prolonged straining. The abnormal dilatation and distortion of the vascular channel, together with destructive changes in the supporting connective tissue within the anal cushion, is a paramount finding of hemorrhoidal disease^[1]. An inflammatory reaction^[2] and vascular hyperplasia^[3,4] may be evident in hemorrhoids. This article firstly reviewed the pathophysiology and other clinical backgrounds of hemorrhoidal disease, followed by the current approaches to

non-operative and operative management.

PATHOPHYSIOLOGY OF HEMORRHOIDAL DISEASE

The exact pathophysiology of hemorrhoidal development is poorly understood. For years the theory of varicose veins, which postulated that hemorrhoids were caused by varicose veins in the anal canal, had been popular but now it is obsolete because hemorrhoids and anorectal varices are proven to be distinct entities. In fact, patients with portal hypertension and varices do not have an increased incidence of hemorrhoids^[5].

Today, the theory of sliding anal canal lining is widely accepted^[6]. This proposes that hemorrhoids develop when the supporting tissues of the anal cushions disintegrate or deteriorate. Hemorrhoids are therefore the pathological term to describe the abnormal downward displacement of the anal cushions causing venous dilatation. There are typically three major anal cushions, located in the right anterior, right posterior and left lateral aspect of the anal canal, and various numbers of minor cushions lying between them^[7] (Figure 1). The anal cushions of patients with hemorrhoids show significant pathological changes. These changes include abnormal venous dilatation, vascular thrombosis, degenerative process in the collagen fibers and fibroelastic tissues, distortion and rupture of the anal subepithelial muscle (Figure 2). In addition to the above findings, a severe inflammatory reaction involving the vascular wall and surrounding connective tissue has been demonstrated in hemorrhoidal specimens, with associated mucosal ulceration, ischemia and thrombosis^[2].

Several enzymes or mediators involving the degradation of supporting tissues in the anal cushions have been studied. Among these, matrix metalloproteinase (MMP), a zinc-dependent proteinase, is one of the most potent enzymes, being capable of degrading extracellular proteins such as elastin, fibronectin, and collagen. MMP-9 was found to be over-expressed in hemorrhoids, in association with the breakdown of elastic fibers^[8]. Activation of MMP-2 and MMP-9 by thrombin, plasmin or other proteinases resulted in the disruption of the capillary bed and promotion of angioproliferative activity of transforming growth factor β (TGF- β)^[9].

Recently, increased microvascular density was found in hemorrhoidal tissue, suggesting that neovascularization might be another important phenomenon of hemorrhoidal disease. In 2004, Chung *et al*^[4] reported that endoglin (CD105), which is one of the binding sites of TGF- β and is a proliferative marker for neovascularization, was expressed in more than half of hemorrhoidal tissue specimens compared to none taken from the normal anorectal mucosa. This marker was prominently found in venules larger than 100 μm . Moreover, these workers found that microvascular density increased in hemorrhoidal tissue especially when thrombosis and stromal vascular endothelial growth factors (VEGF)

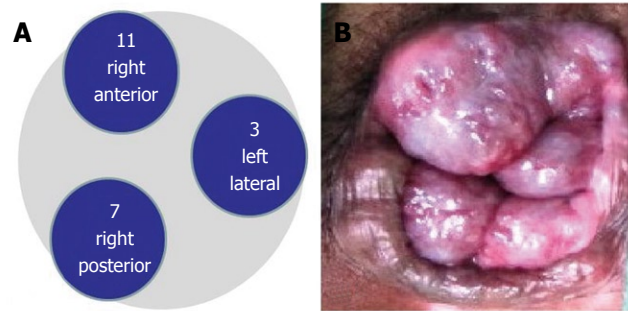


Figure 1 Diagram of common sites of major anal and internal hemorrhoids. A: Diagram of common sites of major anal cushions; B: Common sites of internal hemorrhoids.

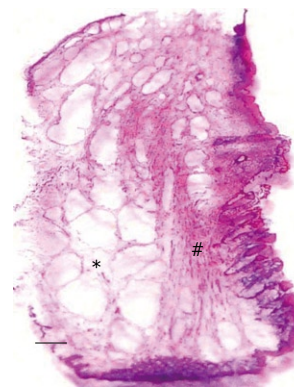


Figure 2 Pathological changes in hemorrhoids. *: Marked dilatation of hemorrhoidal venous plexus; #: Fragmented anal subepithelial muscle (the Treitz's muscle or mucosal suspensory ligament) (Scale bar = 1 mm).

were present. Han *et al*^[8] also demonstrated that there was a higher expression of angiogenesis-related protein such as VEGF in hemorrhoids.

Regarding the study of morphology and hemodynamics of the anal cushions and hemorrhoids, Aigner *et al*^[3,10] found that the terminal branches of the superior rectal artery supplying the anal cushion in patients with hemorrhoids had a significantly larger diameter, greater blood flow, higher peak velocity and acceleration velocity, compared to those of healthy volunteers. Moreover, an increase in arterial caliber and flow was well correlated with the grades of hemorrhoids. These abnormal findings still remained after surgical removal of the hemorrhoids, confirming the association between hypervascularization and the development of hemorrhoids.

Using an immunohistochemical approach, Aigner *et al*^[3] also identified a sphincter-like structure, formed by a thickened tunica media containing 5-15 layers of smooth muscle cells, between the vascular plexus within the subepithelial space of the anal transitional zone in normal anorectal specimens. Unlike the normal specimens, hemorrhoids contained remarkably dilated, thin-walled vessels within the submucosal arteriovenous plexus, with absent or nearly-flat sphincter-like constriction on the vessels. These investigators concluded that a smooth muscle sphincter in the arteriovenous plexus helps in reducing the arterial inflow, thus facilitating an effective venous drainage. Aigner *et al*^[3] then proposed that, if this mechanism is impaired, hyperperfusion of the arteriovenous plexus will lead to the formation of hemorrhoids.

Based on the histological findings of abnormal

venous dilatation and distortion in hemorrhoids, dysregulation of the vascular tone might play a role in hemorrhoidal development. Basically, vascular smooth muscle is regulated by the autonomic nervous system, hormones, cytokines and overlying endothelium. Imbalance between endothelium-derived relaxing factors (such as nitric oxide, prostacyclin, and endothelium-derived hyperpolarizing factor) and endothelium-derived vasoconstricting factors (such as reactive oxygen radicals and endothelin) causes several vascular disorders^[11]. In hemorrhoids, nitric oxide synthase, an enzyme which synthesizes nitric oxide from L-arginine, was reported to increase significantly^[8].

Several physiological changes in the anal canal of patients with hemorrhoids have been observed. Sun *et al*^[12] revealed that resting anal pressure in patients with non-prolapsing or prolapsing hemorrhoids was much higher than in normal subjects, whereas there was no significant change in the internal sphincter thickness. Ho *et al*^[13] performed anorectal physiological studies in 24 patients with prolapsed hemorrhoids and compared with results in 13 sex- and age-matched normal subjects. Before operation, those with hemorrhoids had significantly higher resting anal pressures, lower rectal compliance, and more perineal descent. The abnormalities found reverted to the normal range within 3 mo after hemorrhoidectomy, suggesting that these physiological changes are more likely to be an effect, rather than the cause, of hemorrhoidal disease.

EPIDEMIOLOGY AND RISK FACTORS OF HEMORRHOIDS

Although hemorrhoids are recognized as a very common cause of rectal bleeding and anal discomfort, the true epidemiology of this disease is unknown because patients have a tendency to use self-medication rather than to seek proper medical attention. An epidemiologic study by Johanson *et al*^[14] in 1990 showed that 10 million people in the United States complained of hemorrhoids, corresponding to a prevalence rate of 4.4%. In both sexes, peak prevalence occurred between age 45-65 years and the development of hemorrhoids before the age of 20 years was unusual. Whites and higher socioeconomic status individuals were affected more frequently than blacks and those of lower socioeconomic status. However, this association may reflect differences in health-seeking behavior rather than true prevalence. In the United Kingdom, hemorrhoids were reported to affect 13%-36% of the general population^[1,15]. However, this estimation may be higher than actual prevalence because the community-based studies mainly relied on self-reporting and patients may attribute any anorectal symptoms to hemorrhoids.

Constipation and prolonged straining are widely believed to cause hemorrhoids because hard stool and increased intraabdominal pressure could cause obstruction of venous return, resulting in engorgement of the hemorrhoidal plexus^[1]. Defecation of hard fecal material

increases shearing force on the anal cushions. However, recent evidence questions the importance of constipation in the development of this common disorder^[14,16,17]. Many investigators have failed to demonstrate any significant association between hemorrhoids and constipation, whereas some reports suggested that diarrhea is a risk factor for the development of hemorrhoids^[16]. Increase in straining for defecation may precipitate the development of symptoms such as bleeding and prolapse in patients with a history of hemorrhoidal disease. Pregnancy can predispose to congestion of the anal cushion and symptomatic hemorrhoids, which will resolve spontaneously soon after birth. Many dietary factors including low fiber diet, spicy foods and alcohol intake have been implicated, but reported data are inconsistent^[1].

CLASSIFICATION AND GRADING OF HEMORRHOIDS

A hemorrhoid classification system is useful not only to help in choosing between treatments, but also to allow the comparison of therapeutic outcomes among them. Hemorrhoids are generally classified on the basis of their location and degree of prolapse. Internal hemorrhoids originate from the inferior hemorrhoidal venous plexus above the dentate line and are covered by mucosa, while external hemorrhoids are dilated venules of this plexus located below the dentate line and are covered with squamous epithelium. Mixed (interno-external) hemorrhoids arise both above and below the dentate line. For practical purposes, internal hemorrhoids are further graded based on their appearance and degree of prolapse, known as Goligher's classification: (1) First-degree hemorrhoids (grade I): The anal cushions bleed but do not prolapse; (2) Second-degree hemorrhoids (grade II): The anal cushions prolapse through the anus on straining but reduce spontaneously; (3) Third-degree hemorrhoids (grade III): The anal cushions prolapse through the anus on straining or exertion and require manual replacement into the anal canal; and (4) Fourth-degree hemorrhoids (grade IV): The prolapse stays out at all times and is irreducible. Acutely thrombosed, incarcerated internal hemorrhoids and incarcerated, thrombosed hemorrhoids involving circumferential rectal mucosal prolapse are also fourth-degree hemorrhoids^[18].

Some authors proposed classifications based on anatomical findings of hemorrhoidal position, described as primary (at the typical three sites of the anal cushions), secondary (between the anal cushions), or circumferential, and based on symptoms described as prolapsing and non-prolapsing^[19]. However, these classifications are in less widespread use.

CLINICAL EVALUATION OF HEMORRHOIDS

The most common manifestation of hemorrhoids is painless rectal bleeding associated with bowel move-

Table 1 Current management of internal hemorrhoids by grade

Treatments	Grade I	Grade II	Grade III	Grade IV	Acute thrombosis or strangulation
Dietary and lifestyle modification	×	×	×	×	×
Medical treatment	×	×	×		
Non-operative treatment					
Sclerotherapy	×	×			
Infrared coagulation	×	×			
Radiofrequency ablation	×	×			
Rubber band ligation	×	×	×		
Operative treatment					
Plication		×	×		
DGHAL		×	×		
Hemorrhoidectomy		×	×	×	×
Stapled hemorrhoidopexy			×	×	

DGHAL: Doppler-guided hemorrhoidal artery ligation; ×: Applicable.

ment, described by patients as blood drips into toilet bowl. The blood is typically bright red as hemorrhoidal tissue has direct arteriovenous communication^[3]. Positive fecal occult blood or anemia should not be attributed to hemorrhoids until the colon is adequately evaluated especially when the bleeding is atypical for hemorrhoids, when no source of bleeding is evident on anorectal examination, or when the patient has significant risk factors for colorectal neoplasia^[18].

Prolapsing hemorrhoids may cause perineal irritation or anal itching due to mucous secretion or fecal soiling. A feeling of incomplete evacuation or rectal fullness is also reported in patients with large hemorrhoids. Pain is not usually caused by the hemorrhoids themselves unless thrombosis has occurred, particularly in an external hemorrhoid or if a fourth-degree internal hemorrhoid becomes strangulated. Anal fissure and perianal abscess are more common causes of anal pain in hemorrhoidal patients.

The definite diagnosis of hemorrhoidal disease is based on a precise patient history and careful clinical examination. Assessment should include a digital examination and anoscopy in the left lateral position. The perianal area should be inspected for anal skin tags, external hemorrhoid, perianal dermatitis from anal discharge or fecal soiling, fistula-in-ano and anal fissure. Some physicians prefer patients sitting and straining in the squatting position to watch for the prolapse. Although internal hemorrhoids cannot be palpated, digital examination will detect abnormal anorectal mass, anal stenosis and scar, evaluate anal sphincter tone, and determine the status of prostatic hypertrophy which may be the reason for straining as this aggravates descent of the anal cushions during micturition. Hemorrhoidal size, location, severity of inflammation and bleeding should be noted during anoscopy. Intrarectal retroflexion of the colonoscope or transparent anoscope with flexible endoscope also allow excellent visualization of the anal canal and hemorrhoid, and permit recording pictures^[20].

MANAGEMENT OF HEMORRHOIDAL DISEASE

Therapeutic treatment of hemorrhoids ranges from die-

tary and lifestyle modification to radical surgery, depending on degree and severity of symptoms^[21,22]. The current management of internal hemorrhoids is illustrated in Table 1. In addition, selected meta-analyses showing various treatment options of hemorrhoidal disease are shown in Table 2^[23-32].

Dietary and lifestyle modification

Since shearing action of passing hard stool on the anal mucosa may cause damage to the anal cushions and lead to symptomatic hemorrhoids, increasing intake of fiber or providing added bulk in the diet might help eliminate straining during defecation. In clinical studies of hemorrhoids, fiber supplement reduced the risk of persisting symptoms and bleeding by approximately 50%, but did not improve the symptoms of prolapse, pain, and itching^[26]. Fiber supplement is therefore regarded as an effective treatment in non-prolapsing hemorrhoids; however, it could take up to 6 wk for a significant improvement to be manifest^[33]. As fiber supplements are safe and cheap, they remain an integral part of both initial treatment and of a regimen following other therapeutic modalities of hemorrhoids.

Lifestyle modification should also be advised to any patients with any degree of hemorrhoids as a part of treatment and as a preventive measure. These changes include increasing the intake of dietary fiber and oral fluids, reducing consumption of fat, having regular exercise, improving anal hygiene, abstaining from both straining and reading on the toilet, and avoiding medication that causes constipation or diarrhea.

Medical treatment

Oral flavonoids: These venotonic agents were first described in the treatment of chronic venous insufficiency and edema. They appeared to be capable of increasing vascular tone, reducing venous capacity, decreasing capillary permeability^[34], and facilitating lymphatic drainage^[35] as well as having anti-inflammatory effects^[36]. Although their precise mechanism of action remains unclear, they are used as an oral medication for hemorrhoidal treatment, particularly in Europe and Asia. Micronized purified flavonoid fraction (MPFF), consisting of 90% dios-

Table 2 Selected meta-analyses showing various treatment options for hemorrhoidal disease (in order of publication year)

Authors	Characteristics of comparative studies	Number of trials (total cases)	Results
Johanson <i>et al</i> ^[23]	IC, IS and RBL	5 (863)	RBL had greater long-term efficacy, but led to a higher incidence of post-treatment pain. IC was associated with both fewer and less severe complications
MacRae <i>et al</i> ^[24]	IC, IS, RBL, manual anal dilation and hemorrhoidectomy	18 (1952) ¹	Hemorrhoidectomy was more effective than manual anal dilation and RBL, but more pain and complications. RBL had greater efficacy than IS for treating grade I - III hemorrhoids, with no difference in the complication rate. Patients treated with IC or IS were more likely to require further therapy
Shanmugam <i>et al</i> ^[25]	RBL vs hemorrhoidectomy	3 (202)	Hemorrhoidectomy was superior to RBL for the long-term treatment of grade III, not grade II, hemorrhoids. Although hemorrhoidectomy had more pain, higher complications and more time off work, patient satisfaction and acceptance of the two treatment modalities seems to be similar
Alonso-Coello <i>et al</i> ^[26]	Fiber vs no therapy	7 (378)	Fiber reduced the risk of bleeding and persisting by 50% and 47%, respectively, but it had no significant effect on pain and prolapse
Alonso-Coello <i>et al</i> ^[27]	Oral flavonoids vs placebo or no therapy	14 (1514)	Flavonoids reduced the risk of bleeding, pain, persisting symptoms and recurrence by 67%, 65%, 58% and 47%, respectively
Ho <i>et al</i> ^[28]	Closed vs open hemorrhoidectomy	6 (686)	Closed hemorrhoidectomy had faster wound healing but longer operating time. There was no difference in treatment efficacy, pain, complication and hospital stay between the two operations
Nienhuijs <i>et al</i> ^[29]	Conventional vs ligasure hemorrhoidectomy	12 (1142)	Ligasure hemorrhoidectomy resulted in significantly shorter operative time, less early postoperative pain, earlier recovery, without any difference in recurrent bleeding or incontinence
Burch <i>et al</i> ^[30]	Hemorrhoidectomy vs SH	27 (2279)	SH had less postoperative pain, shorter operative time, shorter hospital stay, and shorter convalescence, but a higher rate of prolapse and reintervention for prolapse
Giordano <i>et al</i> ^[31]	Hemorrhoidectomy vs SH (minimum follow-up of 1 yr)	15 (1201)	SH had a significantly higher incidence of recurrences and additional operations
Gan <i>et al</i> ^[32]	Various TCMH vs another TCMH or Western medicines	9 (1822)	TCMHs significantly improved overall symptoms and bleeding as well as decreased the inflammation of perianal mucosa

¹With available detailed data on the patients enrolled. IC: Infrared coagulation; IS: Injection sclerotherapy; RBL: Rubber band ligation; SH: Stapled hemorrhoidopexy; TCMH: Traditional Chinese medicinal herbs.

min and 10% hesperidin, is the most common flavonoid used in clinical treatment^[27]. The micronization of the drug to particles of less than 2 μm not only improved its solubility and absorption, but also shortened the onset of action. A recent meta-analysis of flavonoids for hemorrhoidal treatment, including 14 randomized trials and 1514 patients, suggested that flavonoids decreased risk of bleeding by 67%, persistent pain by 65% and itching by 35%, and also reduced the recurrence rate by 47%^[27]. Some investigators reported that MPFF can reduce rectal discomfort, pain and secondary hemorrhage following hemorrhoidectomy^[37].

Oral calcium dobesilate: This is another venotonic drug commonly used in diabetic retinopathy and chronic venous insufficiency as well as in the treatment of acute symptoms of hemorrhoids^[38]. It was demonstrated that calcium dobesilate decreased capillary permeability, inhibited platelet aggregation and improved blood viscosity; thus resulting in reduction of tissue edema^[39]. A clinical trial of hemorrhoid treatment showed that calcium dobesilate, in conjunction with fiber supplement, provided an effective symptomatic relief from acute bleeding, and it was associated with a significant improvement in the inflammation of hemorrhoids^[40].

Topical treatment: The primary objective of most topical treatment aims to control the symptoms rather than

to cure the disease. Thus, other therapeutic treatments could be subsequently required. A number of topical preparations are available including creams and suppositories, and most of them can be bought without a prescription. Strong evidence supporting the true efficacy of these drugs is lacking. These topical medications can contain various ingredients such as local anesthesia, corticosteroids, antibiotics and anti-inflammatory drugs^[41].

Topical treatment may be effective in selected groups of hemorrhoidal patients. For instance, Tjandra *et al*^[42] showed a good result with topical glyceryl trinitrate 0.2% ointment for relieving hemorrhoidal symptoms in patients with low-grade hemorrhoids and high resting anal canal pressures. However, 43% of the patients experienced headache during the treatment. Perrotti *et al*^[43] reported the good efficacy of local application of nifedipine ointment in treatment of acute thrombosed external hemorrhoids. It is worth noting that the effect of topical application of nitrite and calcium channel blocker on the symptomatic relief of hemorrhoids may be a consequence of their relaxation effect on the internal anal sphincter, rather than on the hemorrhoid tissue *per se* where one might anticipate a predominantly vasodilator effect.

Apart from topical medication influencing tone of the internal anal sphincter, some topical treatment targets vasoconstriction of the vascular channels within hemorrhoids such as Preparation-H[®] (Pfizer, United States), which contains 0.25% phenylephrine, petrola-

tum, light mineral oil, and shark liver oil. Phenylephrine is a vasoconstrictor having preferential vasopressor effect on the arterial site of circulation, whereas the other ingredients are considered protectants. Preparation-H is available in many forms, including ointment, cream, gel, suppositories, and medicated and portable wipes^[44]. It provides temporary relief of acute symptoms of hemorrhoids, such as bleeding and pain on defecation.

Non-operative treatment

Sclerotherapy: This is currently recommended as a treatment option for first- and second-degree hemorrhoids. The rationale of injecting chemical agents is to create a fixation of mucosa to the underlying muscle by fibrosis. The solutions used are 5% phenol in oil, vegetable oil, quinine, and urea hydrochloride or hypertonic salt solution^[22]. It is important that the injection be made into submucosa at the base of the hemorrhoidal tissue and not into the hemorrhoids themselves; otherwise, it can cause immediate transient precordial and upper abdominal pain^[45]. Misplacement of the injection may also result in mucosal ulceration or necrosis, and rare septic complications such as prostatic abscess and retroperitoneal sepsis^[46]. Antibiotic prophylaxis is indicated for patients with predisposing valvular heart disease or immunodeficiency because of the possibility of bacteremia after sclerotherapy^[47].

Rubber band ligation: Rubber band ligation (RBL) is a simple, quick, and effective means of treating first- and second-degree hemorrhoids and selected patients with third-degree hemorrhoids. Ligation of the hemorrhoidal tissue with a rubber band causes ischemic necrosis and scarring, leading to fixation of the connective tissue to the rectal wall. Placement of rubber band too close to the dentate line may cause severe pain due to the presence of somatic nerve afferents and requires immediate removal. RBL is safely performed in one or more than one place in a single session^[48] with one of several commercially available instruments, including hemorrhoid ligator rectoscope^[49] and endoscopic ligator^[50] which use suction to draw the redundant tissue in to the applicator to make the procedure a one-person effort.

The most common complication of RBL is pain or rectal discomfort, which is usually relieved by warm sitz baths, mild analgesics and avoidance of hard stool by taking mild laxatives or bulk-forming agents. Other complications include minor bleeding from mucosal ulceration, urinary retention, thrombosed external hemorrhoids, and extremely rarely, pelvic sepsis. The patients should stop taking anticoagulants for one week before and two weeks after RBL.

Infrared coagulation: The infrared coagulator produces infrared radiation which coagulates tissue and evaporizes water in the cell, causing shrinkage of the hemorrhoid mass. A probe is applied to the base of the hemorrhoid through the anoscope and the recommended contact time is between 1.0-1.5 s, depending on the intensity

and wavelength of the coagulator^[51]. The necrotic tissue is seen as a white spot after the procedure and eventually heals with fibrosis. Compared with sclerotherapy, infrared coagulation (IRC) is less technique-dependent and avoids the potential complications of misplaced sclerosing injection^[22]. Although IRC is a safe and rapid procedure, it may not be suitable for large, prolapsing hemorrhoids.

Radiofrequency ablation: Radiofrequency ablation (RFA) is a relatively new modality of hemorrhoidal treatment. A ball electrode connected to a radiofrequency generator is placed on the hemorrhoidal tissue and causes the contacting tissue to be coagulated and evaporized^[52]. By this method, vascular components of hemorrhoids are reduced and hemorrhoidal mass will be fixed to the underlying tissue by subsequent fibrosis. RFA can be performed on an outpatient basis and *via* an anoscope similar to sclerotherapy. Its complications include acute urinary retention, wound infection, and perianal thrombosis. Although RFA is a virtually painless procedure, it is associated with a higher rate of recurrent bleeding and prolapse^[53].

Cryotherapy: Cryotherapy ablates the hemorrhoidal tissue with a freezing cryoprobe. It has been claimed to cause less pain because sensory nerve endings are destroyed at very low temperature. However, several clinical trials revealed that it was associated with prolonged pain, foul-smelling discharge and a high rate of persistent hemorrhoidal mass^[54]. It is therefore rarely used.

There are two meta-analyses comparing outcomes among the three common non-operative treatments of hemorrhoids (sclerotherapy, RBL and IRC)^[23,24]. These two studies demonstrated that RBL resulted in the fewest recurrent symptoms of hemorrhoids and the lowest rate of retreatment, but that it led to a significantly higher incidence of pain following the procedure. Hence, RBL could be recommended as the initial non-operative modality for treatment of grade I - III hemorrhoids. In a British survey of almost 900 general and colorectal surgeons^[55], RBL was the most common procedure performed, following by sclerotherapy and hemorrhoidectomy.

Operative treatment

An operation is indicated when non-operative approaches have failed or complications have occurred. Different philosophies regarding the pathogenesis of hemorrhoidal disease creates different surgical approaches (Table 3).

Hemorrhoidectomy: Excisional hemorrhoidectomy is the most effective treatment for hemorrhoids with the lowest rate of recurrence compared to other modalities^[24]. It can be performed using scissors, diathermy^[56,57], or vascular-sealing device such as Ligasure (Covidien, United States)^[29,58] and Harmonic scalpel (Ethicon Endosurgery, United States)^[59,60]. Excisional hemorrhoidectomy can be performed safely under perianal anesthetic infiltration as an ambulatory surgery^[61,62]. Indications for hemorrhoidectomy include failure of non-operative management,

Table 3 Summary of different philosophies regarding the pathogenesis of hemorrhoids and related surgical approaches

Theory	Short description	Surgical approach
Sliding anal cushions	Hemorrhoids develop when the supporting tissues of the anal cushions disintegrate or deteriorate	Hemorrhoidectomy, plication
Rectal redundancy	Hemorrhoidal prolapse is associated with an internal rectal prolapse	Stapled hemorrhoidopexy
Vascular abnormality	Hyperperfusion of arteriovenous plexus within anal cushion results in the formation of hemorrhoids	Doppler-guided hemorrhoidal artery ligation

acute complicated hemorrhoids such as strangulation or thrombosis, patient preference, and concomitant anorectal conditions such as anal fissure or fistula-in-ano which require surgery^[18]. In clinical practice, the third-degree or fourth-degree internal hemorrhoids are the main indication for hemorrhoidectomy.

A major drawback of hemorrhoidectomy is postoperative pain^[62]. There has been evidence that Ligasure hemorrhoidectomy results in less postoperative pain, shorter hospitalization, faster wound healing and convalescence compared to scissors or diathermy hemorrhoidectomy^[63-65]. Other postoperative complications include acute urinary retention (2%-36%), postoperative bleeding (0.03%-6%), bacteremia and septic complications (0.5%-5.5%), wound breakdown, unhealed wound, loss of anal sensation, mucosa prolapse, anal stricture (0%-6%), and even fecal incontinence (2%-12%)^[66-69]. Recent evidence has suggested that hemorrhoidal specimens can be exempt from pathological examination if no malignancy is suspected^[70].

Plication: Plication is capable of restoring anal cushions to their normal position without excision. This procedure involves oversewing of hemorrhoidal mass and tying a knot at the uppermost vascular pedicle. However, there are still a number of potential complications following this procedure such as bleeding and pelvic pain^[21].

Doppler-guided hemorrhoidal artery ligation: A new technique based on doppler-guided ligation of the terminal branches of the superior hemorrhoidal artery was introduced in 1995 as an alternative to hemorrhoidectomy^[71]. Doppler-guided hemorrhoidal artery ligation (DGHAL) has become increasingly popular in Europe. The rationale of this treatment was later supported by the findings from vascular studies^[3,10], which demonstrated that patients with hemorrhoids had increased caliber and arterial blood flow of the terminal branch of the superior rectal arteries. Therefore, ligating the arterial supply to hemorrhoidal tissue by suture ligation may improve hemorrhoidal symptoms. DGHAL is most effective for second- or third-degree hemorrhoids. Notably, DGHAL may not improve prolapsing symptoms in advanced hemorrhoids. Short-term outcomes and 1-year recurrence rates of DGHAL did not differ from those of conventional hemorrhoidectomy^[72]. Given the fact that there is the possibility of revascularization and recurrence of symptomatic hemorrhoids, further studies on the long-term outcomes of DGHAL are still required^[73].

Stapled hemorrhoidopexy: Stapled hemorrhoidopexy (SH) has been introduced since 1998^[74]. A circular stapling device is used to excise a ring of redundant rectal mucosa proximal to hemorrhoids and resuspend the hemorrhoids back within the anal canal. Apart from lifting the prolapsing hemorrhoids, blood supply to hemorrhoidal tissue is also interrupted. A recent meta-analysis comparing surgical outcomes between SH and hemorrhoidectomy, which included 27 randomized, controlled trials with 2279 procedures, showed that SH was associated with less pain, earlier return of bowel function, shorter hospital stay, earlier return to normal activities, and better wound healing, as well as higher degree of patient satisfaction^[30]. However, in the longer term, SH was associated with a higher rate of prolapse^[30,31,75]. Considering the recurrence rate, cost of stapling device and potential serious complications including rectovaginal fistula^[76] and rectal stricture^[77,78], SH is generally reserved for patients with circumferential prolapsing hemorrhoids and having ≥ 3 lesions of advanced internal hemorrhoids.

These two recent surgical options, DGHAL and SH, aim to correct the pathophysiology of hemorrhoids by reducing blood flow to the anal canal (dearterialization) and eliminating anorectal mucosal prolapse (reposition), respectively. A recent retrospective study of 18-mo outcomes of DGHAL ($n = 51$) and SH ($n = 63$) for grade III hemorrhoids revealed that both procedures were safe and effective. DGHAL had less pain, shorter hospital stay, and faster functional recovery; however, it was associated with higher recurrence rate and lower patient satisfaction rating^[79]. Lately, a smaller prospective trial comparing DGHAL to SH for grade II-III hemorrhoids showed similar short-term and long-term outcomes of the two procedures^[80]. Nevertheless, patients undergoing DGHAL returned to work quicker, and had fewer complication rates than those receiving SH.

CONCLUSION

Therapeutic treatment of hemorrhoids ranges from dietary and lifestyle modification to radical surgery, depending on degree and severity of symptoms. Although surgery is an effective treatment of hemorrhoids, it is reserved for advanced disease and it can be associated with appreciable complications. Meanwhile, non-operative treatments are not fully effective, in particular those of topical or pharmacological approach. Hence, improvements in our understanding of the pathophysiology of hemorrhoids are needed to prompt the development of novel and innova-

tive methods for the treatment of hemorrhoids.

REFERENCES

- Loder PB**, Kamm MA, Nicholls RJ, Phillips RK. Haemorrhoids: pathology, pathophysiology and aetiology. *Br J Surg* 1994; **81**: 946-954
- Morgado PJ**, Suárez JA, Gómez LG, Morgado PJ. Histoclinical basis for a new classification of hemorrhoidal disease. *Dis Colon Rectum* 1988; **31**: 474-480
- Aigner F**, Gruber H, Conrad F, Eder J, Wedel T, Zelger B, Engelhardt V, Lametschwandner A, Wienert V, Böhler U, Margreiter R, Fritsch H. Revised morphology and hemodynamics of the anorectal vascular plexus: impact on the course of hemorrhoidal disease. *Int J Colorectal Dis* 2009; **24**: 105-113
- Chung YC**, Hou YC, Pan AC. Endoglin (CD105) expression in the development of haemorrhoids. *Eur J Clin Invest* 2004; **34**: 107-112
- Goenka MK**, Kochhar R, Nagi B, Mehta SK. Rectosigmoid varices and other mucosal changes in patients with portal hypertension. *Am J Gastroenterol* 1991; **86**: 1185-1189
- Thomson WH**. The nature of haemorrhoids. *Br J Surg* 1975; **62**: 542-552
- Thomson WH**. The nature and cause of haemorrhoids. *Proc R Soc Med* 1975; **68**: 574-575
- Han W**, Wang ZJ, Zhao B, Yang XQ, Wang D, Wang JP, Tang XY, Zhao F, Hung YT. [Pathologic change of elastic fibers with difference of microvessel density and expression of angiogenesis-related proteins in internal hemorrhoid tissues]. *Zhonghua Weichang Waike Zazhi* 2005; **8**: 56-59
- Yoon SO**, Park SJ, Yun CH, Chung AS. Roles of matrix metalloproteinases in tumor metastasis and angiogenesis. *J Biochem Mol Biol* 2003; **36**: 128-137
- Aigner F**, Bodner G, Gruber H, Conrad F, Fritsch H, Margreiter R, Bonatti H. The vascular nature of hemorrhoids. *J Gastrointest Surg* 2006; **10**: 1044-1050
- Stankevicius E**, Kevelaitis E, Vainorius E, Simonsen U. [Role of nitric oxide and other endothelium-derived factors]. *Medicina* (Kaunas) 2003; **39**: 333-341
- Sun WM**, Peck RJ, Shorthouse AJ, Read NW. Haemorrhoids are associated not with hypertrophy of the internal anal sphincter, but with hypertension of the anal cushions. *Br J Surg* 1992; **79**: 592-594
- Ho YH**, Seow-Choen F, Goh HS. Haemorrhoidectomy and disordered rectal and anal physiology in patients with prolapsed haemorrhoids. *Br J Surg* 1995; **82**: 596-598
- Johanson JF**, Sonnenberg A. The prevalence of hemorrhoids and chronic constipation. An epidemiologic study. *Gastroenterology* 1990; **98**: 380-386
- Gazet JC**, Redding W, Rickett JW. The prevalence of haemorrhoids. A preliminary survey. *Proc R Soc Med* 1970; **63** Suppl: 78-80
- Johanson JF**, Sonnenberg A. Constipation is not a risk factor for hemorrhoids: a case-control study of potential etiological agents. *Am J Gastroenterol* 1994; **89**: 1981-1986
- Pigot F**, Siproudhis L, Allaert FA. Risk factors associated with hemorrhoidal symptoms in specialized consultation. *Gastroenterol Clin Biol* 2005; **29**: 1270-1274
- American Gastroenterological Association medical position statement: Diagnosis and treatment of hemorrhoids. *Gastroenterology* 2004; **126**: 1461-1462
- Lunniss PJ**, Mann CV. Classification of internal haemorrhoids: a discussion paper. *Colorectal Dis* 2004; **6**: 226-232
- Harish K**, Harikumar R, Sunilkumar K, Thomas V. Videoaoscopy: useful technique in the evaluation of hemorrhoids. *J Gastroenterol Hepatol* 2008; **23**: e312-e317
- Acheson AG**, Scholefield JH. Management of haemorrhoids. *BMJ* 2008; **336**: 380-383
- Kaidar-Person O**, Person B, Wexner SD. Hemorrhoidal disease: A comprehensive review. *J Am Coll Surg* 2007; **204**: 102-117
- Johanson JF**, Rimm A. Optimal nonsurgical treatment of hemorrhoids: a comparative analysis of infrared coagulation, rubber band ligation, and injection sclerotherapy. *Am J Gastroenterol* 1992; **87**: 1600-1606
- MacRae HM**, McLeod RS. Comparison of hemorrhoidal treatment modalities. A meta-analysis. *Dis Colon Rectum* 1995; **38**: 687-694
- Shanmugam V**, Thaha MA, Rabindranath KS, Campbell KL, Steele RJ, Loudon MA. Rubber band ligation versus excisional haemorrhoidectomy for haemorrhoids. *Cochrane Database Syst Rev* 2005: CD005034
- Alonso-Coello P**, Mills E, Heels-Ansdell D, López-Yarto M, Zhou Q, Johanson JF, Guyatt G. Fiber for the treatment of hemorrhoids complications: a systematic review and meta-analysis. *Am J Gastroenterol* 2006; **101**: 181-188
- Alonso-Coello P**, Zhou Q, Martinez-Zapata MJ, Mills E, Heels-Ansdell D, Johanson JF, Guyatt G. Meta-analysis of flavonoids for the treatment of haemorrhoids. *Br J Surg* 2006; **93**: 909-920
- Ho YH**, Buettner PG. Open compared with closed haemorrhoidectomy: meta-analysis of randomized controlled trials. *Tech Coloproctol* 2007; **11**: 135-143
- Nienhuijs S**, de Hingh I. Conventional versus LigaSure hemorrhoidectomy for patients with symptomatic Hemorrhoids. *Cochrane Database Syst Rev* 2009: CD006761
- Burch J**, Epstein D, Sari AB, Weatherly H, Jayne D, Fox D, Woolacott N. Stapled haemorrhoidopexy for the treatment of hemorrhoids: a systematic review. *Colorectal Dis* 2009; **11**: 233-243; discussion 243
- Giordano P**, Gravante G, Sorge R, Ovens L, Nastro P. Long-term outcomes of stapled hemorrhoidopexy vs conventional hemorrhoidectomy: a meta-analysis of randomized controlled trials. *Arch Surg* 2009; **144**: 266-272
- Gan T**, Liu YD, Wang Y, Yang J. Traditional Chinese Medicine herbs for stopping bleeding from haemorrhoids. *Cochrane Database Syst Rev* 2010: CD006791
- Moesgaard F**, Nielsen ML, Hansen JB, Knudsen JT. High-fiber diet reduces bleeding and pain in patients with hemorrhoids: a double-blind trial of Vi-Siblin. *Dis Colon Rectum* 1982; **25**: 454-456
- Labrid C**. Pharmacologic properties of Daflon 500 mg. *Angiology* 1994; **45**: 524-530
- Labrid C**. A lymphatic function of Daflon 500 mg. *Int Angiol* 1995; **14**: 36-38
- Struckmann JR**, Nicolaidis AN. Flavonoids. A review of the pharmacology and therapeutic efficacy of Daflon 500 mg in patients with chronic venous insufficiency and related disorders. *Angiology* 1994; **45**: 419-428
- La Torre F**, Nicolai AP. Clinical use of micronized purified flavonoid fraction for treatment of symptoms after hemorrhoidectomy: results of a randomized, controlled, clinical trial. *Dis Colon Rectum* 2004; **47**: 704-710
- Misra MC**. Drug treatment of haemorrhoids. *Drugs* 2005; **65**: 1481-1491
- Tejerina T**, Ruiz E. Calcium dobesilate: pharmacology and future approaches. *Gen Pharmacol* 1998; **31**: 357-360
- Menteş BB**, Görgül A, Tatlicioğlu E, Ayoğlu F, Unal S. Efficacy of calcium dobesilate in treating acute attacks of hemorrhoidal disease. *Dis Colon Rectum* 2001; **44**: 1489-1495
- Johanson JF**. Nonsurgical treatment of hemorrhoids. *J Gastrointest Surg* 2002; **6**: 290-294
- Tjandra JJ**, Tan JJ, Lim JF, Murray-Green C, Kennedy ML, Lubowski DZ. Rectogesic (glyceryl trinitrate 0.2%) ointment relieves symptoms of hemorrhoids associated with high resting anal canal pressures. *Colorectal Dis* 2007; **9**: 457-463
- Perrotti P**, Antropoli C, Molino D, De Stefano G, Antropoli M. Conservative treatment of acute thrombosed external

- hemorrhoids with topical nifedipine. *Dis Colon Rectum* 2001; **44**: 405-409
- 44 **Sneider EB**, Maykel JA. Diagnosis and management of symptomatic hemorrhoids. *Surg Clin North Am* 2010; **90**: 17-32, Table of Contents
- 45 **Mann CV**, Motson R, Clifton M. The immediate response to injection therapy for first-degree haemorrhoids. *J R Soc Med* 1988; **81**: 146-148
- 46 **Guy RJ**, Seow-Choen F. Septic complications after treatment of haemorrhoids. *Br J Surg* 2003; **90**: 147-156
- 47 **Adami B**, Eckardt VF, Suermann RB, Karbach U, Ewe K. Bacteremia after proctoscopy and hemorrhoidal injection sclerotherapy. *Dis Colon Rectum* 1981; **24**: 373-374
- 48 **Chaleoykitti B**. Comparative study between multiple and single rubber band ligation in one session for bleeding internal, hemorrhoids: a prospective study. *J Med Assoc Thai* 2002; **85**: 345-350
- 49 **Budding J**. Solo operated haemorrhoid ligator rectoscope. A report on 200 consecutive bandings. *Int J Colorectal Dis* 1997; **12**: 42-44
- 50 **Jutabha R**, Jensen DM, Chavalitdhamrong D. Randomized prospective study of endoscopic rubber band ligation compared with bipolar coagulation for chronically bleeding internal hemorrhoids. *Am J Gastroenterol* 2009; **104**: 2057-2064
- 51 **Ricci MP**, Matos D, Saad SS. Rubber band ligation and infrared photocoagulation for the outpatient treatment of hemorrhoidal disease. *Acta Cir Bras* 2008; **23**: 102-106
- 52 **Gupta PJ**. Radiofrequency ablation and plication: a non-resectional therapy for advanced hemorrhoids. *J Surg Res* 2005; **126**: 66-72
- 53 **Gupta PJ**. Radiofrequency coagulation versus rubber band ligation in early hemorrhoids: pain versus gain. *Medicina (Kaunas)* 2004; **40**: 232-237
- 54 **Smith LE**, Goodreau JJ, Fouty WJ. Operative hemorrhoidectomy versus cryodestruction. *Dis Colon Rectum* 1979; **22**: 10-16
- 55 **Beattie GC**, Wilson RG, Loudon MA. The contemporary management of haemorrhoids. *Colorectal Dis* 2002; **4**: 450-454
- 56 **Ibrahim S**, Tsang C, Lee YL, Eu KW, Seow-Choen F. Prospective, randomized trial comparing pain and complications between diathermy and scissors for closed hemorrhoidectomy. *Dis Colon Rectum* 1998; **41**: 1418-1420
- 57 **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 ligation for symptomatic prolapsed hemorrhoids. *Dis Colon Rectum* 1992; **35**: 1165-1169
- 58 **Chen JS**, You JF. Current status of surgical treatment for hemorrhoids--systematic review and meta-analysis. *Chang Gung Med J* 2010; **33**: 488-500
- 59 **Haveran LA**, Sturrock PR, Sun MY, McDade J, Singla S, Paterson CA, Counihan TC. Simple harmonic scalpel hemorrhoidectomy utilizing local anesthesia combined with intravenous sedation: a safe and rapid alternative to conventional hemorrhoidectomy. *Int J Colorectal Dis* 2007; **22**: 801-806
- 60 **Kwok SY**, Chung CC, Tsui KK, Li MK. A double-blind, randomized trial comparing Ligasure and Harmonic Scalpel hemorrhoidectomy. *Dis Colon Rectum* 2005; **48**: 344-348
- 61 **Lohsiriwat V**, Lohsiriwat D. Ambulatory anorectal surgery under perianal anesthetics infiltration: analysis of 222 cases. *J Med Assoc Thai* 2007; **90**: 278-281
- 62 **Lohsiriwat D**, Lohsiriwat V. Outpatient hemorrhoidectomy under perianal anesthetics infiltration. *J Med Assoc Thai* 2005; **88**: 1821-1824
- 63 **Milito G**, Cadeddu F, Muzi MG, Nigro C, Farinon AM. Haemorrhoidectomy with Ligasure vs conventional excisional techniques: meta-analysis of randomized controlled trials. *Colorectal Dis* 2010; **12**: 85-93
- 64 **Tan EK**, Cornish J, Darzi AW, Papagrigroriadis S, Tekkis PP. Meta-analysis of short-term outcomes of randomized controlled trials of LigaSure vs conventional hemorrhoidectomy. *Arch Surg* 2007; **142**: 1209-1218; discussion 1218
- 65 **Mastakov MY**, Buettner PG, Ho YH. Updated meta-analysis of randomized controlled trials comparing conventional excisional haemorrhoidectomy with LigaSure for haemorrhoids. *Tech Coloproctol* 2008; **12**: 229-239
- 66 **Sayfan J**. Complications of Milligan-Morgan hemorrhoidectomy. *Dig Surg* 2001; **18**: 131-133
- 67 **Cintron JR**, Abcarian H. Benign Anorectal: Hemorrhoids. In: Wolff BG, Flashman JW, Beck DE, Pemberton JH, Wexner SD, editors. *The ASCRS Textbook of Colon and Rectal Surgery*. New York: Springer, 2007: 156-177
- 68 **Sielezneff I**, Salle E, Lécuyer J, Brunet C, Sarles JC, Sastre B. [Early postoperative morbidity after hemorrhoidectomy using the Milligan-Morgan technic. A retrospective studies of 1,134 cases]. *J Chir (Paris)* 1997; **134**: 243-247
- 69 **Pattana-arun J**, Wesarachawit W, Tantiphlachiva K, Atithansakul P, Sahakitrungruang C, Rojanasakul A. A comparison of early postoperative results between urgent closed hemorrhoidectomy for prolapsed thrombosed hemorrhoids and elective closed hemorrhoidectomy. *J Med Assoc Thai* 2009; **92**: 1610-1615
- 70 **Lohsiriwat V**, Vongjirad A, Lohsiriwat D. Value of routine histopathologic examination of three common surgical specimens: appendix, gallbladder, and hemorrhoid. *World J Surg* 2009; **33**: 2189-2193
- 71 **Morinaga K**, Hasuda K, Ikeda T. A novel therapy for internal hemorrhoids: ligation of the hemorrhoidal artery with a newly devised instrument (Moricorn) in conjunction with a Doppler flowmeter. *Am J Gastroenterol* 1995; **90**: 610-613
- 72 **Bursics A**, Morvay K, Kupcsulik P, Flautner L. Comparison of early and 1-year follow-up results of conventional hemorrhoidectomy and hemorrhoid artery ligation: a randomized study. *Int J Colorectal Dis* 2004; **19**: 176-180
- 73 **Faucheron JL**, Gangner Y. Doppler-guided hemorrhoidal artery ligation for the treatment of symptomatic hemorrhoids: early and three-year follow-up results in 100 consecutive patients. *Dis Colon Rectum* 2008; **51**: 945-949
- 74 **Longo A**. Treatment of hemorrhoids disease by reduction of mucosa and haemorrhoidal prolapse with a circular suturing device: A new procedure. *Proceedings of the 6th World Congress of Endoscopic Surgery*; 1998 June 3-6; Rome, Italy
- 75 **Shao WJ**, Li GC, Zhang ZH, Yang BL, Sun GD, Chen YQ. Systematic review and meta-analysis of randomized controlled trials comparing stapled haemorrhoidopexy with conventional haemorrhoidectomy. *Br J Surg* 2008; **95**: 147-160
- 76 **Angelone G**, Giardiello C, Protta C. Stapled hemorrhoidopexy. Complications and 2-year follow-up. *Chir Ital* 2006; **58**: 753-760
- 77 **Dowden JE**, Stanley JD, Moore RA. Obstructed defecation after stapled hemorrhoidopexy: a report of four cases. *Am Surg* 2010; **76**: 622-625
- 78 **Ravo B**, Amato A, Bianco V, Boccasanta P, Bottini C, Carriero A, Milito G, Dodi G, Mascagni D, Orsini S, Pietroletti R, Ripetti V, Tagariello GB. Complications after stapled hemorrhoidectomy: can they be prevented? *Tech Coloproctol* 2002; **6**: 83-88
- 79 **Avital S**, Itah R, Skornick Y, Greenberg R. Outcome of stapled hemorrhoidopexy versus doppler-guided hemorrhoidal artery ligation for grade III hemorrhoids. *Tech Coloproctol* 2011; **15**: 267-271
- 80 **Giordano P**, Nastro P, Davies A, Gravante G. Prospective evaluation of stapled haemorrhoidopexy versus transanal haemorrhoidal dearterialisation for stage II and III haemorrhoids: three-year outcomes. *Tech Coloproctol* 2011; **15**: 67-73