Operative Management

14

Antonio Longo, Adolfo Renzi, and Francesco Prete

Introduction

To be effective, surgical treatment must be based on detailed knowledge of the nature of the pathology that it aims to resolve. Therefore, the deeper and more accurate the knowledge of anatomy, physiology and pathogenesis, the higher the chances of being able to develop surgical techniques that are rational and focused on a specific disease.

Haemorrhoidal disease has been known for thousands of years for its high incidence among the human species and the relative ease with which it is diagnosed. Even the Bible mentions how God punished the Philistines making them the target of an epidemic of haemorrhoids, although this probably has to be attributed to an incorrect translation.

In the course of the millennia, countless theories have followed one another trying to clarify the anatomical and physiological nature of haemorrhoids and the dynamics of their causes and pathogenesis. Without indulging in details, which would go beyond the scope of this chapter, it seems appropriate to briefly report some concepts of anatomy and physiopathology to better understand the rationale of the various therapeutic options for the haemorrhoidal disease.

Today it is widely recognised that the haemorrhoidal cushions play a role in anal continence because of their ability to inflate and deflate rapidly [1]. This ability to adjust their volume is due to the anatomical nature of the cushions, with their numerous arterial and venous shunts that produce vascular lacunar spaces. The blood supply to the haemorrhoidal cushions through the superior, middle and inferior haemorrhoidal arteries, which undoubtedly exceeds the sole biological needs, has the ultimate purpose of allowing the haemorrhoids to quickly fill with blood to optimise, in synergy with the anal sphincters, the anal continence. It is thus a case of "hyper-vascularisation" that supports the functional role optimising the anal continence.

The haemorrhoidal cushions are kept in their position by connective tissue and smooth muscle fibres [2] and are covered with anal mucosa. The anal mucosa overlying the haemorrhoids, besides being arranged in longitudinal folds that provide for an adequate aperture of the anal canal during defecation, is specialised in the discrimination of rectal contents and therefore is an anatomical structure with a fundamental role regarding the anorectal reflexes and, thus, regarding anal continence. These simple considerations have led to two important reflections.

The first is that the scope of "hyper-vascularisation" is that of guaranteeing a hyperflow of blood to the haemorrhoidal vessels. This aspect, which some, as we will later see,

A. Longo, MD (✉)
European Center of Coloproctology
and Pelvic Diseases, Vienna, Austria
e-mail: alongo@neomedia.it

A. Renzi, MD, PhD
Department of Colorectal Surgery,
Villa delle Querce Hospital, Naples, Italy

F. Prete, FRCS
Department of GI Surgery,
University College London Hospitals, London, UK

R. Cohen, A. Windsor (eds.), *Anus*,
DOI 10.1007/978-1-84882-091-3_14,

have seen as one of the causing factors of the haemorrhoidal disease, is instead a physiological peculiarity that exists with the only aim to quickly increase haemorrhoidal volume to seal the anus.

The second reflection is that ablation or destruction of the haemorrhoids, regardless of the methods, ends up in weakening – in various degrees – anal continence and dilatability.

Among the many theories on the pathogenesis of the haemorrhoidal disease that have followed one another in centuries, some have obviously left such a long-lasting impression that they are still evoked nowadays, often wrongly, in therapeutic decisions. Among these theories we find the so-called theory of the varicose veins, dating from age of Galen and Hippocrates [1]. Going back to the observation of the frequent association between dilatation of the haemorrhoidal plexuses and symptoms, this theory, although identifying different causative factors for haemorrhoidal dilatation, ended up equating haemorrhoidal disease to rectal varices, like the ones following portal hypertension. Subsequently it has well been clarified that rectal varices caused by portal hypertension are a rare pathology and distinct from that of the haemorrhoids. As the dilatation of the haemorrhoidal veins is always associated to a prolapse, it is fundamental to understand the pathogenetic correlation between these two phenomena. Angiographic studies have demonstrated that haemorrhoidal prolapse causes a venous kinking between the middle and inferior haemorrhoidal veins and a stretching of the superior haemorrhoidal vessels that obviously obstacle outflow. This obstacle is worsened by sphincter hypertone. These factors can create a venous dilatation with blood stagnation, thrombosis and oedema. It is therefore evident that venous dilatation is a complication following prolapse and not a primary cause of haemorrhoidal pathology.

Although nowadays this theory is no longer supported among proctologists, the idea that the haemorrhoidal pathology is similar in some way to varicose veins continues to be widespread. This is proved by the fact that many drugs prescribed for haemorrhoidal pathology are the same used for lower limb varices. The good business for the pharmaceutical companies producing the so-called vessel protectors and vasoactive drugs and the natural inclination of patients to try whichever form of treatment just to avoid surgical treatment, which has always been considered an extremely painful experience, seem to be among the reasons for the survival of this theory.

The theory of the so-called vascular hyperplasia, particularly popular in Europe, probably has its origin in a certain histological similarity between the prolapsed haemorrhoidal cushions and angiomatous tissues. Although it has been abandoned, this theory deserves to be remembered here as many of the studies performed to prove its validity have contributed to clarify the anatomical structure and the physiological function of the haemorrhoids. In any case it has been clearly demonstrated histologically that haemorrhoidal specimens show no signs of tissue hyperplasia.

The theory nowadays largely accepted by proctologists is the one proposed by Gass and Adams in 1950 [3], *the sliding anal lining theory*. This theory assumes the prolapse of the anal lining as the pathogenetic cause of haemorrhoidal disease. It is based on the concept that "fragmentation" of the ligaments of Treitz and Parks, which support the haemorrhoidal cushions, causes prolapse of haemorrhoids and of anal mucosa. The prolapse is considered a predisposing cause of all the haemorrhoidal symptoms and complications.

It is useful making some considerations on Goligher's classification [4] that, as is known, is the most widespread tool to assess haemorrhoidal pathology. Although this classification, that divides haemorrhoidal prolapse into four grades, is accepted unanimously, some of its elements have to be viewed critically. The definition of the first degree of prolapse, for example, is not entirely clear, as it includes haemorrhoids that are "not prolapsing, but increased in volume, and projecting into the anal canal and bleeding". It is evident that this definition of first degree is bound to be considered critically for several reasons: (a) it is not very clear why you would include non-prolapsed haemorrhoids in a classification based on prolapse itself; (b) besides the fact that projection into the lumen of the anal canal is a normal anatomical condition, the concept of

"increased volume" appears unclear as no reference is made to either what a normal volume is or to a range of normality; it is well known that haemorrhoidal volume is very variable in the population at large and even in the same subject depending on physiological conditions; and (c) in order to define this first-degree prolapse, a symptom like bleeding is being used which is illogical in a classification otherwise based on the clinical behaviour of the prolapse (whether reducible spontaneously, manually or non-reducible). This incongruence in classification is probably due to the fact that the way the theory of prolapse was formulated is not able to sufficiently explain the clinical events and, in a certain way, the true essence of haemorrhoidal pathology.

Moreover, there is much confusion regarding the significance of the prolapse degree even among specialists – although Goligher cannot be blamed for this. It is not unusual to read endoscopic reports defining the grade of prolapse based on an evaluation of the volume of the inspected haemorrhoidal cushions rather than an evaluation based on history and clinical appearance: in fact a distinction between second and third degree can only be made by questioning the patient (whether the prolapse retracts spontaneously or requires manual manoeuvres). Grade IV is the only one that can be diagnosed by a doctor during proctological exam.

The three pathological theories outlined, even if conceptually different, have some elements in common. Varices, vascular hyperplasia and prolapse generated by wearing of supportive tissue are considered irreversible anatomical and histological alterations. Moreover, all the variations in pathogenesis delineated so far tend to indicate haemorrhoidal pathology as a primary disease, with cause and effect limited to the anal canal. Because of these convictions, the elimination of haemorrhoidal tissue was considered, until recent times, the only logical and effective therapeutic treatment. We will see that it cannot be that way.

For reasons of better readability of this chapter, we will progressively analyse the solutions that were proposed in the course of time for treatment of haemorrhoidal pathology, reporting techniques and results. We will first take into consideration outpatient procedures that, as we will see, are mainly focused on destroying haemorrhoids through activation of tissue necrosis, even though in different ways. At the end of this section, dearterialisation will be discussed. After that we will analyse the actual surgical techniques for removal of haemorrhoids and finally, following the presentation of the unitary theory of rectoanal prolapses, the techniques stapled haemorrhoidopexy (SH) and stapled transanal rectal resection (STARR).

Ambulatory Treatment

Sclerotherapy

This outpatient procedure consists in injecting chemical agents in the haemorrhoidal plexuses with subsequent fibrosis, scarring, shrinking and fixation of the haemorrhoids. These effects are secondary to the obliteration of haemorrhoidal vessels induced by the sclerosing agent solution.

The first attempt at obliterating haemorrhoids through the use of sclerosing injections was performed by Morgan, who in 1869 used a solution of ferrous persulphate to treat external haemorrhoids. In 1871 sclerotherapy with phenol and other chemical agents was introduced in the United States and a few years later indicated as "cure for haemorrhoids without pain or surgery" [5].

The absence or at least the scarce availability of anal specula at that time made only prolapsed haemorrhoids treatable that were attacked with a massive injection of sclerosing solution. The enthusiasm that this technique inspired can be imagined when thinking that even Mr. Andrews [1], then president of the Chicago Medical Society, considered this technique appropriate despite an investigation he had conducted himself on over 3,000 patients undergoing sclerotherapy (many of them treated by travelling charlatans and inexperienced doctors) and which revealed a high number of complications, including severe pain and even nine deaths. Andrews indicated both the use of phenol as sclerosing agent and bed rest for at least 8 h following

treatment as indispensable elements for a good outcome of the therapy.

In this same period also, Kelsey in the United States and Edwards in England recognised the therapeutic efficacy of sclerosing treatment. These authors found that the incidence of complications were lower when a less concentrated solution of phenol, between 5 and 7.5 %, was used [6, 7].

We have to give credit to Terrell for being the first to identify the most appropriate indication for sclerosing therapy in the treatment of internal haemorrhoids. In one of his publications in 1913, he reported on the brilliant results he obtained and with this contributed significantly to the spreading of this method [8].

In the following decade, Anderson published an interesting historical review, in which he reports on what has happened since then, with particular attention to the historical and not only the scientific implications [9].

Concerning sclerosing agents, it should be remembered that in the last 140 years, i.e. from the first injections performed by Morgan, various combinations have been proposed, very often based on alchemistic rather than truly scientific approaches. The 5 % phenol solution in almond or vegetable oil proposed since the beginning of the twentieth century though has asserted itself and to date is still the most used sclerosing agent in the world and in fact the only one used in the United Kingdom.

Indications

Internal non-prolapsing haemorrhoids are the ones that benefit most from sclerotherapy. Although occasionally a single, slightly protruding haemorrhoid plexus can be treated successfully, more commonly, in case of voluminous haemorrhoids accompanied by abundant submucosal tissue and requiring manual repositioning after defecation, sclerotherapy is completely ineffective. Also, such a procedure is not to be performed in cases of internal haemorrhoids associated with thrombi and anodermal ulcers. External haemorrhoids constitute an absolute contraindication, as treatment would not only be ineffective but also cause even more severe pain and sloughing.

Technique

Sclerotherapy is usually performed with the patient in jackknife or Sims position. Following insertion of an anoscope and identification of the haemorrhoidal plexuses that need treatment, the surgeon goes on to inject the sclerosing solution. Use of local antiseptic solutions or analgesics is not necessary. It is particularly useful to have a needle with an angled distal end that facilitates vision through the anoscope. In the absence of a dedicated needle, a spinal anaesthesia needle can be used efficiently too. The needle tip is inserted into the centre of the haemorrhoidal plexus, while avoiding going down too much towards the pectinate line. A single injection can be made in each plexus to be treated. The formation of a submucosal bulge indicates a correct execution of the manoeuvre. Depending on the type of solution, different quantities of solution can be used: with regard to the ones available on the market, based on sodium morrhuate, quinine, urea hydrochloride or Sotradecol, generally no more than 3 ml is used in total; in the case of solutions with 5 % phenol in oil, it is possible to use 3 ml for each haemorrhoidal plexus treated.

Results

In the last few years, only a few studies have been published, at least in English. In 1985, Khoury reported the results of a randomised study aiming at comparing efficacy and safety of single vs. multiple injections, showing that a single session of sclerotherapy (with more than one plexus treated) is as effective as a treatment consisting of multiple sessions [10].

In a study by Dencker [11], sclerotherapy resulted scarcely effective if compared to internal haemorrhoid ligation or to the excision as described by Milligan. Meagre long-term results, at 4 years, are also reported by Santos, who describes the technique as effective only in the short term [12]. Also Alexander-Williams came to the same conclusions in 1975 [13].

Unfavourable outcomes of sclerotherapy also emerge from other studies, e.g. those published by Ambrose et al. and Gartell et al. [13, 14], in which sclerotherapy is compared, respectively, to photocoagulation and to rubber band ligation.

Concerning complications, the most frequent one is sloughing which however can almost always be ascribed to an incorrect technical performance of the procedure with a too superficial or too abundant injection or to a second injection done too soon after the previous one. Other, less frequent complications, which are however typical of the method used, are necrosis and haemorrhoidal thrombosis, the treatment of which is always conservative. Cases of "chemical stenosis" of the anal canal [15] have also been described for which anoplasty may be necessary.

Development of a burning sensation in the anal canal is a late complication which, although uncommon, is seen following repeated sclerotherapies. This complication is only partially responsive to topical medications, and its symptoms can be very debilitating.

Also episodes of bacteraemia may represent a complication of sclerotherapy. In 1981 Adami et al. [16] observed this complication with a frequency of 8 %; although this almost never leads to a sepsis, prophylactic antibiotic therapy is indicated, especially in subjects at risk.

Other very rare complications, such as urological sepsis, prostate and seminal vesicles abscess, epididymitis, uro-perineal fistula, necrotising fasciitis involving the perineum and scrotum, rectal perforation and septic shock, were described by both Ribbans and Radcliffe [17] in 1985 and Guy and Seow-Cohen [18] in 2003.

Rubber Band Ligation

This is an outpatient procedure for the treatment of haemorrhoidal pathology based on the effect of fixation-necrosis determined by ligation of the haemorrhoidal plexuses with elastic rings. Although the original idea goes back to Blaisdell [19], the description of the general technical principles and the development of a dedicated instrument are the work of Barron who, in 1963, in two scientific papers published within a few weeks, reported the satisfactory results obtained in 150 patients, almost all of them treated as outpatients [20, 21].

Currently rubber band ligation is one of the most widely used outpatient procedure in the United States. In the fifth edition of his textbook, Corman [22] states that the results of this procedure are so gratifying that they have induced him to prefer this technique over surgical haemorrhoidectomy in about 80 % of his patients.

Indications

This technique is usually indicated in first- and second-degree haemorrhoids.

Technique

This technique can be performed safely with the patient in different positions, without anaesthesia and with the optional use of an enema. After introducing an anoscope, the physician proceeds by placing one or more often two elastic bands (in case one breaks) at the base of the haemorrhoidal plexus to be treated, on which he previously applied gentle traction; this causes strangulation of the blood vessels and cuts off the blood supply to the which eventually falls off after 5–7 days. The remaining small ulcer heals off if the mucosa fixed on the underlying muscular layer is left in place.

The original technique by Barron implies the use of an instrument designed by himself, consisting of a simple metallic rod with a hollow cylinder fixed at its end whose main axis runs parallel to that of the metallic rod; some elastic rings are mounted on this cylinder. It is a bimanual manoeuvre: with one hand the physician leads the distal end of a grasping forceps through the cylinder that he holds in place with his other hand placed on the rod; with the forceps he grasps the haemorrhoidal plexus and drags it through the cylinder from which the elastic bands are released by means of a sliding mechanism.

The biggest haemorrhoidal plexus is treated first, taking care that the elastic bands are positioned 1–3 cm cranially to the dentate line. Although it is appropriate to treat only one nodule per session, it is not uncommon that bands are applied on more than one plexus simultaneously.

Nowadays different instruments can be used to perform problem-free elastic ligations. Also suction devices as the McGown and Lurz-Goltner

instruments are available which use the force of an aspirator instead of a forceps to pull the haemorrhoidal tissue into the cylinder. The advantage offered by this type of instruments is that it is possible to perform the ligation procedure with one hand only, while the other hand holds the anoscope: it is therefore not necessary then having an assistant. When compared to those requiring manual traction, however, these instruments have a disadvantage in that the cylinder is generally smaller, so less haemorrhoidal tissue is involved in each ligature. Among manual traction instruments, the McGivney ligator is particularly appreciated for the possibility to adjust the position of the cylinder and handle with respect to the rod thanks to a system of joints that can be rotated 360°, thus always guaranteeing an optimal vision for the surgeon.

More recently a single-use instrument, the O'Regan System, was launched. Apart from the single-use design of the device, its main advantage is in the suction modality, which is generated with a system similar to a syringe that eliminates the need for tubes and aspirators.

For some days after the procedure patients can experience tenesmus that usually is easily controlled with warm washes and/or common analgesics. Causing the patient constipation should be avoided as it has been reported to worsen results [23].

Results

The reported success rate of this method varies depending on the length of follow-up, grade of haemorrhoidal pathology and the criteria used to define success and failure [24–27]. In the majority of cases, two-thirds to three-quarters of patients with either first- or second-degree haemorrhoids respond positively to rubber band ligation, although in a significant number of these patients repeating the procedure is necessary [28].

In a study conducted by Savioz et al. [29] on 92 patients, aiming at detecting the rate of recurrence, the author reports that 23 % of patients needed a new session of ligation at 5 years and 32 % at 10 years.

From analysis of a sample of almost 3,000 patients undergoing rubber band ligation, followed for 12 years, Bayer et al. [30] states that 79 % did not need any further therapy, 18 % underwent a new session of rubber band ligation and 2.1 % were treated with surgical haemorrhoidectomy because of persistence of symptoms.

As already noted pathological haemorrhoidal plexuses can be ligated in one or more sessions. In an interesting retrospective study, Lee et al. [25] compares the results of sessions with single band ligation with those of sessions with multiple ligations. The author identifies multiple ligation sessions as a cause of increased patient discomfort (29 % vs. 4.5 %) and a higher incidence of vagal symptoms (12.3 % vs. 0 %). This opinion is not shared by other authors who do not detect a significant increase of morbidity when sessions with multiple ligatures are performed [28, 31–35].

Possible complications of rubber band ligation of haemorrhoids are pain, thrombosis and pelvic and perineal sepsis. The most frequent complication is pain, reported in 5–60 % of patients [34–37]. Pain is generally mild to moderate and controllable with warm local washes and analgesics. Severe pain is less frequent and often caused by positioning the elastic bands too close to the dentate line; in these cases early removal of a ligation is the only solution. Thrombosis of the internal haemorrhoids or more often of the corresponding external ones (2–3 %) is far less frequent. In these cases warm washes and painkillers can facilitate spontaneous resolution, whereas surgical excision is rarely indicated [22].

Pelvic and perineal sepsis is an infrequent but very dramatic adverse event after rubber band ligation. Documented for the first time in 1980 by O'Hara [38], it has since been reported also by other authors [39–44]. It seems that young men are the "ideal" subjects for this complication, presenting with progressive increase in anorectal and then perineal pain, with worsening difficulty in micturition, scrotal swelling and fever. Aetiology remains uncertain. Treatment obviously requires admission, often intensive care, massive antibiotic therapy, surgical debridement and possibly hyperbaric oxygen therapy.

In 1993 Bat et al. [45] published a prospective study conducted on 513 patients with the aim of detecting complications from rubber band ligation. In this series 4.6 % of patients developed minor complications including pain, dislocation of the rubber band, mucosal ulceration, priapism and urinary retention. Serious complications requiring admission, such as massive haemorrhage, severe pain and perianal sepsis, occurred in 2.5 % of patients.

Cryotherapy

Cryotherapy is based on the concept that in a tissue undergoing rapid freezing, intracellular water crystallises, cell membranes are destroyed and cell necrosis sets in. Freezing also destroys nerve terminations and induces immediate anaesthesia so cellular destruction is painless.

In the early 1970s, this procedure spread fairly rapidly and, as it had happened a 100 years before with Morgan's sclerotherapy, was indicated by various authors [46–50] as "effective and painless". However, the volume increase of the surviving adjacent cells and the oedema occurring with the increasing temperature in the tissue, together with a considerable increase in cellular tension, are the cause of a profuse serous secretion and pain. Secretion is particularly abundant if treatment is performed distally to the dentate line with the aim of treating external haemorrhoids or anal tags, with some patients being obliged to change pads constantly in order to avoid surrounding skin maceration. For this reason after the first experiences with "expanded" indications, treatment with cryotherapy is reserved only to internal haemorrhoids.

Technique

The patient is placed in Sims or jackknife position. With the aid of a plastic speculum (this material does not conduct cold), the cylindrical cryoscopic probe is positioned in contact with the part to treat or the whole length of the haemorrhoidal plexus. Time of contact is about 2 min but can vary depending on the type of probe adopted. Carbon dioxide probes, the most widely used ones due to their relatively low cost, have a lower cooling power (−89 °C)) than the much more expensive ones based on liquid nitrogen (−196 °C). The advantage of the latter is exclusively in the higher speed with which they generate the freezing of a haemorrhoidal plexus, reducing procedure time. Freezing, i.e. the transformation of the haemorrhoidal plexus into an "ice ball", occurs at −22 °C and is therefore obtainable with both types of probe. Once 2–3 min has elapsed, the probe is removed, and in a few minutes the tissue defrosts returning apparently identical to its initial state. Some authors, among them Kaufman [51], suggest a second application after a defrosting period of 5–10 min.

Results

Between the late 1970s and the early 1980s, Kaufman [51], Southam [52] and Berry and D'Acosta [53] reported very convincing results in their studies, with a degree of patient satisfaction varying from 75 to 97 %.

Subsequently, both because of longer follow-up and the enrolment of patients in wider case series and in randomised studies, the results of this technique appeared gradually less brilliant: Smith et al. [54], in a randomised study of cryotherapy vs. haemorrhoidectomy, identifies the latter as a technique which offers faster healing without residual disease. Besides describing the frequent presence of bothersome signs left on skin and thrombosis in the haemorrhoidal vessels not involved in cryo-destruction, the author also reports cases of incontinence apparently due to damages to the internal sphincter because of the impossibility of limiting the depth of cryo-generated necrosis.

In addition O'Callaghan et al. [55] and Goligher [56] both refer to the procedure as time-consuming and too frequently associated with foul-smelling and profuse secretion, irritation of perianal skin and severe pain. These authors also report of how inappropriate applications can cause stenosis and/or incontinence due to sphincter involvement.

Keighley and Williams [57] are straightforward in describing cryotherapy as a painful

therapy frequently associated with profuse secretion and sick leave for at least 1 week. These authors state that only 50 % of patients are fully satisfied, and long-term results are little predictable. Generally, this procedure is no longer recommended.

Photocoagulation

Photocoagulation is a therapeutic technique based on the possibility of converting infrared light into heat, causing coagulation of tissue proteins, water evaporation, eschar formation and subsequent scarring and fixation of the mucosa above the haemorrhoids to the muscular wall. The technique involving infrared coagulation of small haemorrhagic areas was developed by Nath et al. [58] in 1977, with the aim of obtaining haemostasis without tissue adhesion typical of diathermic coagulation. Nieger [59], in 1979, was the first to adopt this technique in the elective therapy of haemorrhoidal disease.

Technique

After inserting a proctoscope, preferably oblique cut, the infrared light generated by a halogen lamp is applied on haemorrhoidal tissue through an instrument similar to a gun that focuses light rays through a quartz piece. Three to five impulses of light of the duration of 1–1.5 s are directly applied onto haemorrhoidal tissue, without the need for anaesthetic injection for applications above the dentate line. Whether he wants to treat one or more plexuses in the same session is left to the discretion of the surgeon. The burnt tissue reacts in the same way as that destroyed through freezing (cryotherapy) or strangulation (band ligation). Healing of the respective ulcer usually occurs in a month. Further treatments can be repeated after 2–3 weeks.

Results

When compared to other techniques of haemorrhoidal "destruction", infrared coagulation offers comparable efficacy but with less complications. In a recent randomised study against rubber band ligation, published in 2006 and conducted on a sample of 94 patients, Marques et al. [60] identifies a higher frequency of haemorrhage and higher posttreatment pain with rubber band ligation. Similar results had also been reported by Ambrose et al. [37] on a sample of 268 patients. Weinstein et al. [61] indicates a higher frequency of haemorrhoidal thrombosis and late haemorrhage in his comparative study of infrared photocoagulation and rubber band ligation.

Monopolar Diathermy

Electrotherapy with direct current, or monopolar diathermy, is a technique of haemorrhoidal tissue destruction obtained through the heat generated by monopolar current.

Technique

The technique involves the use of an instrument; the most widely used example of which is the Ultroid (Microinvasive, Watertown, MA.), which releases monopolar current through a probe. After positioning of a proctoscope, the tip of the probe is placed in contact with the rectal mucosa at the apex of the haemorrhoidal plexus. The current, the intensity of which is set to the maximum tolerated by the patient, is applied for about 10 min [63–66]. Generally only one application is made per session, also because of the rather long duration of each application.

Results

Norman et al. [67] concludes from the analysis of results of a study conducted on 120 patients, that, although in over 20 % of patients further applications were necessary, this technique guarantees complete success, without complications, even in patients with third- and fourth-degree haemorrhoids.

Dennison et al. [68] pointed out the excessive length of this procedure. This author reports similar results to those obtained with monopolar diathermy and other outpatient procedures but denounces the considerable duration of each single treatment, particularly unpleasant for the patients and even the physicians themselves; this

aspect probably contributed to the scarce spreading of this technique.

Bipolar Diathermy

Bipolar diathermy (BICAP; Circon ACMI; Stamford, CT) is another technique that uses heat to cause destruction of haemorrhoidal tissue with subsequent ulceration, fibrosis and fixation of mucosal tissue to the underlying layers. This methodology of application of electrical current to tissues is supposed to limit the depth of damage in contrast to what happens with monopolar current, photocoagulation and laser therapy [62, 63].

Technique

This technique, originally developed to treat upper GI ulcers, is based on the use of dedicated forceps connected to a generator and operated by a pedal command. The heat is produced by the passage of electric current through adjacent electrodes situated at the tip of the forceps. A few instants after the application, a whitish clot forms that extends in depth for about 3 mm. All the haemorrhoids can be treated in a single session and generally no anaesthesia is necessary.

Results

Different studies have been conducted that compare bipolar diathermy with other outpatient treatments. Randall et al. [63] and Hilton et al. [64] have compared bipolar diathermy with treatment by Ultroid (monopolar diathermy): both procedures turned out to be equally effective, but due to the shorter duration of the procedure involving the use of bipolar diathermy, the latter was better accepted by the patients.

Advantages as a result of less deep penetration of the heat, and hence of less collateral damage, as well as with regard to the possibility of treating several plexuses in a single session, were reported by Hinton [69] and Dennison et al. [70] in a comparison between bipolar diathermy and photocoagulation.

Looking at bipolar diathermy in comparison with rubber band ligation has been the focus of a study by Griffith et al. [71], which did not point out any particular advantage of either technique over the other.

Finally, less postoperative pain was highlighted by Yang, in a study that compared the bipolar technique and Ultroid (monopolar diathermy) [72].

Comment on Outpatient Treatment of Haemorrhoids

Destroying haemorrhoidal tissue means accepting a theory of pathogenesis which is based on the assumption that the haemorrhoidal tissue itself is affected by primary alterations causing symptoms. Almost always the only symptom taken into account is bleeding. What emerges from the vast literature on the topic is that symptoms like soiling and itching are almost never taken into consideration, as they are considered to be independent of haemorrhoidal disease. Hence, the efficacy of these techniques is almost always judged in relation to the resolution of bleeding. When this symptom is solved, a treatment is considered effective.

In the best of scenarios, destruction of haemorrhoidal tissue implies fixation of the residual tissue and of anal mucosa, certainly not correction of the prolapse. Therefore, these techniques should not be indicated in haemorrhoidal prolapse, and in fact they are often used for bleeding internal and non-prolapsed haemorrhoids, especially cryotherapy and sclerotherapy.

We know that bleeding originates from the submucosa and that in rare cases of mucosal ulceration haemorrhoidal tissue may bleed, too. So destroying the haemorrhoidal cushions is not rational. This type of therapy is believed to be indicated in swollen haemorrhoids protruding into the lumen! It has to be noted that haemorrhoids always protrude into the anal canal lumen, and their supposed increase in volume is obviously a completely arbitrary and illogical judgement lacking any reference parameter whatsoever, given the physiological ability of the haemorrhoids to adapt their size.

The only acceptable hypothesis is a bulge which is always temporary and caused by

impeded extra-haemorrhoidal venous outflow, but also in this case it would be a therapy aimed at the effect and not the cause of a symptom.

Finally, a consideration which is certainly not shared by those who consider the destruction of haemorrhoidal tissue an appropriate treatment: what conceptual and technical revolution is that supposed to be, compared to what has been around since the life and times of Hippocrates? From using a hot iron to thermal ablation, cryotherapy, etc.? Of course Hippocrates used to treat prolapsed haemorrhoids so at least the indication was correct! In terms of its biological outcome, the effect caused by the use of the red-hot iron hook is comparable to diathermy, cryotherapy, etc. So the only revolution and evolution so far has probably taken place in the fields of anaesthesiology and analgesics and not with regard to the chemical or physical means used to destroy haemorrhoidal tissue. Given the indications and sometimes serious complications of these procedures, a critical review of their use may be necessary.

Some critical observations need to be made with regard to rubber band ligation (RBL). Blaisdell [19] and Barron [20, 21] indicated RBL as an ideal outpatient procedure to destroy internal prolapsed haemorrhoids by means of necrosis. The elastic band positioned above the dentate line needed to include internal haemorrhoid. If we agree that prolapsed haemorrhoids are not longer than usual, on the contrary, they are often shorter as they are no longer stretched by longitudinal support fibres, it is inevitable that the positioning of a rubber band causes dragging of rectal mucosa towards the dentate line. If the procedure is repeated on all of the three cushions, the anal canal will eventually be lined almost completely with mucosa. In other terms, this would be equivalent to creating an ultra-low anastomosis that in turn causes wet anus, soiling and impairment of the discrimination capacity, not to mention a certain degree of stenosis. However, quite strangely such complications are almost never mentioned and the positive results reported in recent years in the literature but above all in congresses and by American speakers reach the amazing rate of 90–95 %. In truth, without overtly admitting it, many colleagues have converted to the theory of the muco-haemorrhoidal prolapse, and instead of applying the rubber band on the haemorrhoid, they position it across the mucosa lying above, thus performing – in a much less effective manner – a haemorrhoidopexy. Far from wanting to make any accusations, it turns out that the reported success rate for RBL is much higher in those countries where insurance reimbursement for this outpatient procedure is almost equivalent to that paid for a haemorrhoidectomy for which the patient is admitted to hospital.

Haemorrhoidal Artery Ligation: HAL, DG-HAL, THD, and HAL-RAR

This technique known under the acronym of HAL (haemorrhoidal artery ligation) is a non-invasive surgical procedure for the treatment of haemorrhoid pathology developed and proposed in 1995 by the Japanese surgeon Morinaga et al. [73]. As we already had a chance to point out in the introduction to this chapter, the theoretical starting point for this approach is based on the assumption that the aetiopathology of the haemorrhoidal pathology lies in an excessive influx of blood into these structures. The procedure therefore implies the precise identification, under Doppler guidance, of the terminal branches of the superior haemorrhoidal arteries and their ligation. Various centres in Europe and in America have adopted this technique with some minimal variations and using different names: Doppler-guided haemorrhoidal artery ligation (DG-HAL) and transanal haemorrhoidal dearterialisation (THD).

Technique

With the patient in gynaecological position, local anaesthesia is performed with perianal infiltration in the four quadrants. A dedicated fenestrated anoscope which, at its tip, holds an 8.2 Mhz Doppler microprobe is inserted. The function of this microprobe is to allow identification of the terminal branches of the superior haemorrhoidal arteries. The fenestration on the anoscope, situated just below the Doppler microprobe, allows

putting a stitch (rounded needle and absorbable material) and thus tying the arterial branch found with the Doppler signal. Each arterial branch is ligated about 2–3 cm above the dentate line. The disappearance of Doppler signal documents the correct execution of the manoeuvre. Ligation of terminal branches of the superior haemorrhoidal artery determines a reduction of blood pressure inside the plexuses with subsequent reduction of bleeding and swelling of the haemorrhoidal tissue. In general, the procedure ends after performing 6 ligatures at 11, 12, 2, 5, 7 and 9 o'clock; duration is about 20–30 min.

Results

The first encouraging results were published by Morinaga et al. [73] himself, who reports that 1 month after the procedure of the 116 patients operated, 96 % did not have bleedings anymore and 95 % did not feel any pain; and in 78 % of cases, he reported an improvement of prolapse-related symptoms.

Between 2004 and 2007 also Scheyer et al. [74], Dal Monte et al. [75] and Lienert and Ulrich [76] reported very positive results in their studies: the technique is considered to be well tolerated by patients and relatively painless and can be performed in an outpatient setting under local anaesthesia/sedation. Moreover, in a randomised study in which he compares the technique in question to conventional haemorrhoidectomy, Bursics et al. [77] finds that, although the two techniques are equally efficient, arterial ligation is less painful and allows a quicker return to social life.

It has to be said, however, that in the quoted studies, the majority of patients treated by arterial ligation were affected by second- to third-degree haemorrhoids. In fact, although this technique seems able to provide satisfactory results in haemorrhoids mostly affected by bleeding, similar results are not achievable for haemorrhoids with symptoms of prolapse. The aforementioned Scheyer et al. [74] himself remarks how 60 % of patients with fourth-degree haemorrhoids complained of a residual prolapse and how, on the other hand, only 6.7 % of patients with second-degree haemorrhoids presented with a similar affliction in the postoperative period. The problem seems related to the impossibility of achieving a satisfactory correction of the prolapse in the advanced stages of the disease.

With the aim of overcoming this limit, Morinaga's technique was modified at the end of 2005 with the addition of a "rectoanal-plasty": rectoanal repair (RAR) – hence, the acronym HAL-RAR. This procedure adds to the simple HAL a plication of the redundant haemorrhoidal tissue that is suspended and fastened to the rectal walls as an effect of scar formation. This technical precaution leads to a correction of prolapse-related symptoms (mucorrhea, pruritus, occasional soiling) frequently seen in patients with third- and fourth-degree haemorrhoids. Middleton et al. [78] reports that the addition of this further technical step improves outcome and does not translate into greater pain to the patient.

Comment on Haemorrhoidal Artery Ligation

This method initially proposed by Morinaga has been the source of much perplexity. The therapeutic foundation of the method is that bleeding and haemorrhoidal prolapse are caused by an arterial hyperafflux. Therefore, a reduction in blood flow would cure this. As mentioned before, blood hyperflow to haemorrhoidal cushions is a normal anatomical and physiological condition that allows haemorrhoids to rapidly increase or decrease in size and with that modifies the volume and closure of the anal canal, thus improving continence.

The haematic flow to the cushions is variable and influenced by a number of factors; for example, the middle haemorrhoidal arteries and veins are intrasphincteric, and therefore, blood inflow and outflow are affected by sphincter tone. So blood afflux is variable and cannot be considered an absolute and measurable rate.

The diagnostic instruments currently available are not sensitive enough to detect a difference in blood flow in the haemorrhoidal arteries of two different study groups. Theoretically, in order to confirm the theory of hyperafflux, we would have

to be able to measure, and differentiate, blood inflow in thousands of patients and, in a prospective trial, check and compare the incidence of haemorrhoidal disease between the normal-flow and the hyperflow group. Even if we did establish that patients with haemorrhoidal disease have an increased haemorrhoidal blood flow, we still would not know whether this is a cause or an effect of the disease. The incidence of haemorrhoidal disease is so high that assuming such an odd and rare alteration would be among the pathogenetic factors is at least highly improbable.

Therefore, I think that some of the papers appearing in the literature [79] should be considered unreliable. It is biological nonsense arguing that the supporting tissue of the anal mucosa and haemorrhoids that consists of smooth muscle and elastic fibres, when hypervascularised and hence hyperoxygenated, transforms into frail fibrotic fibres that in turn break and then cause prolapse. It would mean jettisoning what has been confirmed in histochemical findings, i.e. that it is a deficit in blood flow that transforms muscle fibres into fibrotic tissue. It is even more difficult to imagine how haemorrhoids are pulled back up into the anal canal when blood flow is reduced.

However, despite the aforementioned reservations on the therapeutic principles of haemorrhoidal dearterialisation, I have had chance to establish that this method can be efficient in some patients with modest second-degree prolapse, although with a high incidence of recurrences and postoperative internal and external haemorrhoidal thrombosis. From the beginning it has always been my conviction that the efficacy of this method was due to the numerous stitches applied at full thickness to the rectum. These stitches fix the rectal mucosal prolapse, thus keeping it from sliding into the anal canal. Therefore, whether you close the arteries (or not) does not have any influence on the outcome, nor does using the Doppler ultrasound.

Confirmation of these assumptions has nonetheless arrived from the owners of the THD patent. The two surgeons at first had only performed simple ultrasound-guided ligation, but when they determined the procedure's scarce efficacy on the prolapse, they associated a continuous suture to fix haemorrhoids. Later they showed that when performing only the fixing suture, the results were the same. Consequently they abandoned the Doppler.

I know of similar studies that were sent to renowned coloproctology journals and, for inexplicable reasons, have always been rejected.

Operative Treatment

Open Haemorrhoidectomy (Milligan-Morgan)

The so-called "open" haemorrhoidectomy, the first description of which dates back to as long ago as 1937, is still frequently performed in the United Kingdom. Although the operation is based on the procedure originally proposed by Milligan only, it is known always and anyway as Milligan-Morgan haemorrhoidectomy [80].

Technique

The procedure can be performed under general or subarachnoid anaesthesia or by pudendal nerve blockade associated with intravenous sedation. The patient is positioned in lithotomy with the buttocks protruding beyond the edge of the operative table and spread apart. Some surgeons find it useful to infiltrate the perineal region with saline solution to which adrenaline is added, with the double advantage of reducing bleeding and facilitating dissection of the haemorrhoidal pedicles.

For each of the three main haemorrhoidal piles, the overlying skin is grasped with a robust forceps and is retracted outwards. This manoeuvre allows exposure of the inferior margin of the mucosa covering the haemorrhoidal pile. The latter is grasped with another forceps so that three pairs of forceps can be seen in the operative field, one for each haemorrhoidal plexus.

Usually, the first haemorrhoidal pile to be treated is the left lateral one. By holding with the left hand both forceps, that – as already said – grasp skin and mucosa of that pile, and by pulling them together towards the centre of the

anus, the surgeon performs a "V"-shaped incision with the scissors in his right hand. The two margins of this incision run from the mucocutaneous junction to the two sides of the pile and meet at the apex of the "V", about 2–3 cm from the dentate line. He then proceeds cautiously to dissect the haemorrhoidal plexus from the underlying muscle, being careful to preserve the internal sphincter fibres. Progressively, while dissection proceeds upwards, the mucosa is sectioned on both sides with incisions that converge towards the pedicle. This is extremely important in order to leave wide mucosal bridges behind at the end of the procedure. When a pedicle is well isolated, it is transfixed and tied. The haemorrhoidal tissue is then sectioned from its pedicle and removed. The procedure is repeated exactly with the same modalities for the other haemorrhoidal piles. At the end of the operation, three open wounds are visible, similar in shape to three pears whose stalks converge towards the anus. The region is dressed with paraffin-soaked gauze positioned in the anal canal.

Other versions of the operative technique just described are haemorrhoidectomy with LigaSure and laser haemorrhoidectomy.

Haemorrhoidectomy with LigaSure

The technical procedure is extremely simple. In this way to perform an haemorrhoidectomy, haemorrhoidal plexuses are excised by applying electrothermal bipolar energy is delivered by a forceps device. The first results were published by Jayne et al. [81], Palazzo [82], Chung and Wu [83] and by Franklin et al. [84]. One of these publications, by Palazzo, compare this technique with haemorrhoidal dissection by diathermy and with open haemorrhoidectomy, respectively. The author find advantages in terms of operative time, post-operative pain, need for medications and return to social and work activities. The cost of the instrument, however, needs to be considered.

Laser Haemorrhoidectomy

Among the various types of lasers with the widest applications in medicine, carbon dioxide laser and neodymium-based laser (neodymium:yttrium-aluminium garnet, Nd-YAG) proved the most adequate types for surgical proctology. This is due to the specific wavelength of these types of laser that, when in contact with venous tissue, have shown a good coagulation potential with scarce collateral tissue damage. Apart from the obvious set of special instruments, the use of a laser device requires a specific preparation of the medical and paramedical staff.

Having said this, the technique itself is relatively easy: after local anaesthesia is induced, with the patient in prone position and with the aid of a Hill-Ferguson anal retractor, the laser beam is aimed directly at the haemorrhoidal plexus.

The laser light, applied with a dedicated handle, is slowly moved onto the area to be treated until total tissue destruction becomes evident by the appearance of a uniform whitish membrane. The entire procedure requires about 30 min.

Nicholson et al. [85] report that postoperative results regarding early and late complications, need for pain killers and wound healing time are not different from those reported after conventional haemorrhoidectomy.

Wang and colleagues are not of the same opinion [86]. In 1991, they compared haemorrhoidectomy with neodymium laser to closed haemorrhoidectomy (Ferguson technique) in a randomised trial. From their results it emerges that patients after laser haemorrhoidectomy needed less analgesics and had less urinary retention, and the operative time was shorter.

The opinion held by Senagore et al. [87] is different as emerges from one of his publications in 1993. In this randomised trial the author compares haemorrhoidectomy with neodymium laser to the one performed with a "cold" scalpel. Both the procedures were performed by standard closed Ferguson haemorrhoidectomy: there were no statistically significant differences between the two procedures, although a higher incidence of suture dehiscence was recorded in the group of patients treated by laser. Therefore, Senagore concluded that, also because of the higher costs, the use of laser haemorrhoidectomy was not justified.

Closed Haemorrhoidectomy (Ferguson Procedure)

This surgical technique developed by Ferguson and Heaton in 1959 (Rosa) was conceived with the aim of overcoming the common disadvantages attributed to open haemorrhoidectomy [88]. The authors had the aim of removing most of the haemorrhoidal tissue while sparing the anoderm, reducing postoperative serous secretions and preventing stenosis following healing by second intention. To date, this is still the haemorrhoidectomy technique most frequently used in the United States.

Technique

Closed haemorrhoidectomy can be performed under any type of anaesthesia. In case of local anaesthesia, the drug of choice is bupivacaine with adrenaline. Regional anaesthesia with caudal block and subarachnoid anaesthesia are also among the possible options.

Although the procedure is performed by some surgeons with the patient in gynaecological position and although Ferguson himself, in describing his original technique, suggested to position the patient in Sims position, the genupectoral position (jackknife) is currently the one which is most widely used. The buttocks are spread apart with adhesive tape to better expose the anal region. The area is disinfected with iodine solution, and there is no need for hair removal. The anal canal is explored with a Pratt bivalve anoscope in order to allow the surgeon to evaluate in which and in how many quadrants haemorrhoidectomy is necessary. In fact, although the three-quadrant haemorrhoidectomy including the anterior, right posterior and left lateral regions is the most common one, it is not the rule at all. Generally, after exploration with the Pratt anoscope, a Fansler operative anoscope will be inserted; thanks to a fenestration running over the whole length of the anoscope, this device allows avoiding an excessive excision of the anal epithelium, helping to prevent stenosis. The anoscope is positioned in line with the haemorrhoidal plexus to be resected.

The haemorrhoidal plexus and relative anal tag which may be adjacent to it are grasped with a forceps and drawn upwards and towards the centre of the anal canal. With Metzenbaum scissors the tissue below the forceps is incised, from the skin towards the centre of the anus, paying attention to keep the internal sphincter fibres away. Usually the most voluminous haemorrhoidal plexus is excised first. After separating the haemorrhoidal plexus from the internal sphincter over its whole extension, the surgeon proceeds to the ligation/section or to the diathermy coagulation of the vascular pedicle. An absorbable suture is used to close the surgical wound completely.

In order to keep the risk of stenosis as low as possible, the surgeon generally performs wound closure with the anoscope in situ.

Submucosal Haemorrhoidectomy (Parks Procedure)

Parks [89] describes this procedure in 1956. It is substantially an excision of haemorrhoidal tissue performed after incision of the mucosa inside the anal canal and the rectum, followed by mucosal suture: the aim is to reduce healing time and the rate of stenosis.

Technique

Any anal retractor can be used. Also for this procedure, it can be useful to infiltrate the area to be treated with a solution containing adrenaline. The incisions are performed starting from the skin just outside the anal verge, and exerting traction on the skin just beyond the external margin of the wound, the incision is then extended upwards including the rectal mucosa above the haemorrhoid up to 4 cm from the mucocutaneous junction. The haemorrhoidal tissue is exposed and separated from the mucosal folds and from the underlying muscular plane and then removed after being transfixed and tied in proximity of the vascular pedicle. The mucosal folds within the anal canal are reapproximated. The margins of the anoderm below and the rectal mucosa above next to the pedicle ligatures are left open.

Circular Haemorrhoidectomy (Whitehead Procedure)

Described for the first time by Whitehead 130 years ago [90], circular haemorrhoidectomy was initially met with keen interest mainly in the United Kingdom but has now been abandoned almost completely, mostly because of its difficult technical execution.

Technique

The procedure originally described by the author implies a circular incision at the level of the dentate line for each plexus to treat. Through this incision, the surgeon proceeds, moving upwards, to the dissection of the haemorrhoidal tissue. After completing ligation of the vascular pedicle, the haemorrhoidal plexus is removed, and the surgeon proceeds to suture the mucosa to the anal canal at the level of the dentate line.

In a wrong interpretation of the descriptions given by Whitehead, many surgeons sutured the mucosa to the skin at the external anal margin. Apart from leading to frequent dehiscences of the sutures caused by excessive tension, this technical error often resulted in a deformity of the anus characterised by the protrusion of the anal mucosa out of the external anal margin. This complication, consisting of a mucosal ectropion, has become known as "Whitehead deformity", making the illustrious colleague more notorious for a complication for which he was not responsible than for the procedure first described by him.

Results

Whenever the technique is performed following the original indications given by Whitehead the reported results are similar to those of other haemorrhoidectomies. Bonello [91], Wolff and Culp [92], Barrios and Khubchandani [93] agree with this.

Comment on Haemorrhoidectomy

The purpose of surgical haemorrhoidectomy has always been that of removing haemorrhoids as radically as possible, with the minimum possible postoperative pain and limited complications. The Whitehead procedure certainly is the one that allows the most complete excision of haemorrhoidal tissue, but it has never been well accepted because of the anal deformity of the same name. In reality, this is an iatrogenic mucosal ectropion due to the suture placed between the anal mucosa and the dentate line or, by mistake, the anal skin. An almost constant complication of this procedure is anal incontinence, varying from moist anus to faecal incontinence. For those who have no direct experience of the Whitehead procedure, it is sufficient to remember that these sequelae are comparable to those of ultra-low coloanal anastomosis. These complications give us the chance to comment on something I suggest we should keep in mind: removal of haemorrhoids and of anal mucosa implies that the anal canal becomes lined with rectal mucosa; rectal mucosa directly secretes mucus outwards, causing soiling; the rectal contents can no longer be adequately discerned by the receptors of the anal mucosa, and the haemorrhoidal cushions can no longer play their role in improving the closure of the anus so that continence ends up being weakened.

Two banal conclusions can be drawn from the considerations above: in order to adequately perform a retaining function, the muscular anal canal must be covered with anal skin and mucosa, while the haemorrhoids improve this function. In grade IV haemorrhoidal prolapse, the anal canal is covered, entirely or in part, by rectal mucosa prolapsed into the anus which frequently causes soiling. Soiling consists of mucus with a high bacterial load, variable PH and a number of dissolved substances. For this reason mucus can cause bacterial, chemical and also fungal dermatitis. As soiling and dermatitis are caused by haemorrhoidal prolapse, they should be considered correlated symptoms. Hence, surgical treatment of haemorrhoidal disease should also aim at healing soiling and pruritus, and the efficiency of a technique should also be evaluated in relation to the resolution of these symptoms. Instead, in the literature these symptoms are rarely taken into consideration pre- and postoperatively.

Much confusion clearly exists about haemorrhoidal disease when it comes to determining its specific symptoms.

All the longitudinal haemorrhoidectomies – Milligan-Morgan, Fergusson, etc. – imply the removal of longitudinal folds of anal mucosa covering the haemorrhoids. These mucosal folds are the ones that mainly allow the anus to dilate, because they are not directly adherent to the sphincters. Therefore, haemorrhoidectomy inevitably entails a reduction of anal dilatability.

In a certain percentage of cases, which varies considerably in the literature, this deficit is pathological. The tight anal stenoses are due to the almost complete excision of the anal skin and mucosa. The procedures for rotation and sliding of perineal cutaneous flaps restore the anal lumen more or less satisfactorily. But because this type of tissue is not specialised in sensory discrimination and these flaps have no receptors connected to the anal sphincters, the continence function, including the anal opening and closing reflex, results severely and definitely compromised and so is the patients' quality of life.

Severe anal stenosis is reported in the literature at very variable rates. It certainly is an uncommon complication, although there is a feeling that this problem is underestimated. In fact if every proctologist compared the cases of stenosis (obviously occurring in patients operated on elsewhere!) that he had to manage with the overall number of haemorrhoidectomies performed, he would come to the conclusion that his data are not consistent with data reported in the literature.

The historical problem with haemorrhoidectomies is postoperative pain and the long healing time. A very high proportion of patients refuse haemorrhoidectomy even though they have significant symptoms because they know that postoperative recovery is extremely painful. And there is no doubt that this is the popular opinion about this procedure. This leads patients to try whichever remedy in the hope of avoiding an operation. Therefore, tons of rectal ointments and drugs are being used all over the world every day. The result of this is an incalculable waste of money, with frequent iatrogenic damages which are often worse than the disease itself. Very probably this widespread opinion on haemorrhoidectomy has not changed substantially despite our efforts to reassure patients and allay their fears. Certainly postoperative recovery has become significantly more tolerable. It still remains to be clarified whether this is owed to more effective antalgic therapies or to the innovative techniques and technologies proposed. In order to understand and control post-haemorrhoidectomy pain, it is imperative to understand what postoperative incidents are causing it and if these traumas can be modified by adopting different forms of energy for tissue dissection.

Understanding the origin of post-haemorrhoidectomy pain is not easy at all, and there is a risk of being sidetracked by studies that in the last decades have created many expectations and just as many disillusions. The ordinary post-haemorrhoidectomy wound is made up of a muscular base consisting of corrugator ani and smooth sphincter and of two severed margins consisting of anoderm, mucosa and submucosa. The smooth sphincter contains only pressure receptors and is not traversed by sensory fibres directed to the anal mucosa, as these come from the rectum. This means that the only pain sensation perceivable by the smooth sphincter is either caused by specific stimulation of the pressure receptors or by an aspecific stimulus to the sectioned and exposed nerve terminations. The same considerations apply to the mucosa of the anal canal which has few pain and temperature receptors but is endowed with pressure receptors. In fact the only form of pain perceivable in the anal mucosa and haemorrhoids is tensive pain caused by tissue distension, as, for example, in case of thrombosis, oedema or strangulation following RBL. This is confirmed by the fact that any procedure performed above the anoderm is almost painless. Thus the site of post-haemorrhoidectomy pain is in all likelihood the dermal-anodermal region. This region with its very dense somatic innervation contains numerous receptors specialised towards both physical stimuli, like temperature, stretching and pressure, and chemical stimuli, mainly PH.

The idea that the energy adopted for tissue excision (monopolar diathermy, laser, Ultracision, LigaSure) can modify postoperative pain has been and is still being advocated with much emphasis. This conviction can be rebutted with a few obvious considerations. If the use of a type of energy that develops little heat improves pain, it would be ideal to incise and excise the anal skin with a cold scalpel blade to avoid heat-related tissue damage. Clinical experience tells us otherwise. The thermal receptors are stimulated by temperatures different from those typical of the body, both hot and cold, and only when this difference becomes remarkable, the stimulus is perceived and transmitted as painful. There is no doubt that, whichever temperature the tissue reaches during the procedure, the patient does not perceive pain thanks to anaesthesia; after a few minutes, this tissue resumes normal body temperature and so the patient cannot feel pain generated from heat.

It could be objected that there is also significant biological damage to the residual anoderm and the perianal skin, but here too the consideration that the use of a cold blade, with selective coagulation or ligation of the vessels, should cause less damage and less pain, is valid. It should be remembered that tissue repair, both by first intention (closed haemorrhoidectomies) and by second intention (open haemorrhoidectomy) happens thanks to fibrin, fibroblasts and other repair factors exuding from arterial, venous and lymphatic capillaries and deposited on the injured surfaces. This might cause a delay in scarring, and I do think that this is not an unfounded assumption. This adverse event, which has been described in closed haemorrhoidectomies (references), strangely is not reported in the case of Milligan-Morgan procedures.

Experience and data from the literature confirm that the type of energy used do not influence pain. For many years countless papers – whether entirely objective or not – have been reporting significantly better results with the use of a laser. These results have since been irrefutable disproved by subsequent more reliable studies.

I am convinced that all the other forms of energy currently proposed for haemorrhoidectomy will be facing the same fate as laser.

Unitary Theory of Rectoanal Prolapse

In order to introduce the principles of stapled haemorrhoidopexy (SH) and stapled transanal rectal resection (STARR), I think it is useful to briefly present the considerations, clinical observations and original studies which form the rational basis of these techniques.

At the beginning of the 1990s, after performing hundreds of Milligan and Morgan, Ferguson, Whitehead and other procedures, I realised that the postoperative period was a very painful experience for patients and that the sad notoriety of these procedures induced many persons to exclude surgery.

Initially I developed an inferiority complex, thinking I was not able to perform the technique correctly, but later on, after having visited renowned coloproctology centres, I understood that I was obtaining the same results they did and that there was – and is even more so today – an enormous discrepancy between clinical reality and publication of results. I became convinced that a haemorrhoidectomy, whether closed, open, semi-closed or other, did not represent an adequate therapeutic response to the problem. Through the simple analysis of and extensive reflections on the literature, I sensed that the very essence of haemorrhoidal pathology had still not been captured. I came across some incoherences and contradictions that represented a stimulus for me to study of the problem more in depth. Thomson [1] thought that the disruption of the haemorrhoids' supportive tissue caused prolapse. Haas et al. [2], in 1984, demonstrated that haemorrhoidal supportive tissue disintegrates in all subjects after the age of 30, but not everyone has symptomatic haemorrhoids or prolapse.

This information matches the clinical observation that haemorrhoidal prolapse is always associated to rectal mucosal prolapse, that can protrude into the anal canal and then outside the anus. The external prolapse of rectal mucosa is permanent in the so-called fourth-degree prolapse, and so it is clinically comparable to an ectropion.

Non-reducible rectal mucosa prolapse is thus certainly the cause of soiling which in turn causes

perianal dermatitis and hence pruritus. Where and how does rectal mucosa, which prolapses together with the haemorrhoids, return to within the rectum at the end of evacuation in the case of second- and third-degree prolapses?

To answer this question, we performed a defecography in all patients with haemorrhoidal prolapse of grades II, III and IV in addition to routine proctoscopy. The result was that all patients with any degree of haemorrhoidal prolapse presented a rectoanal invagination whose size could not be correlated with the size and grade of the external prolapse.

Moreover, in women a rectocele was almost always associated to rectoanal invagination. Rectoanal invagination and rectocele associated to haemorrhoidal prolapse explain the previously unclear correlation between haemorrhoidal disease, straining and obstructed defecation (OD). If we consider the consistent association of haemorrhoidal prolapse and rectal prolapse, we can exclude that this is just an occasional concomitance of two distinct pathologies as has always have been thought and written. Clearly this must be a clinical and pathological picture of its own. What still had to be determined was a cause-effect relationship: is it the haemorrhoids that, when prolapsed, draw the rectal mucosa down, or is it the rectal prolapse that pushes the haemorrhoids out of the anus? There was no doubt for us that the second hypothesis had to be correct. In fact it is impossible to find a haemorrhoidal prolapse without rectal prolapse, while the opposite is frequently observed. We came up with the theory that rectoanal invagination causes a kink in the superior haemorrhoidal veins which hampers haemorrhoidal venous outflow and leads to dilatation of the haemorrhoids. This dilatation and stretching of the haemorrhoids can cause increased friction and mechanical trauma to the overlying mucosa during the passage of faeces, with subsequent de-epithelisation and bleeding. We think that this clinical condition constitutes what it is defined rather fuzzily as first-degree haemorrhoidal prolapse.

Subsequently rectal invagination extends to the anal canal during evacuation, causes its obstruction and induces increased straining. It is easy to imagine but also demonstrable in dynamic cinedefecography how the faecal bolus pushes both rectal prolapse and haemorrhoids out of the anal canal with force. In fact, only once the prolapse is expelled, the anal canal is cleared and evacuation can start.

The above-mentioned clinical studies have led us to a revolutionary conclusion that is in conflict with all the traditional ideas on the pathogenesis of haemorrhoidal disease: haemorrhoidal prolapse and all its related symptoms constitute a pathology secondary to the internal rectal prolapse; the rupture of the supportive tissue of the haemorrhoids is a necessary, but not sufficient, precondition for a prolapse to occur. In fact, rupture of supporting fibres is a physiological phenomenon typical of aging (Haas [2]) and it does not necessarily imply haemorrhoidal prolapse. Seemingly, in young subjects the rupture of the Treitz fibres is caused when the rectal prolapse repeatedly pushes against the haemorrhoidal cushions. Haemorrhoidal prolapse is therefore only one of a number of possible clinical manifestations of an internal rectal prolapse.

This new theory explains the correlation between haemorrhoidal disease and obstructed defecation. In fact, rectoanal invagination is also the main cause of the obstructed defecation syndrome.

The limited scope of this chapter does not allow us to go into detail about all the clinical aspects related to rectoanal prolapse and the studies that led us to some conclusions. It is however necessary to give at least a short summary in order to explain the rational basis of the techniques for the therapy of haemorrhoidal prolapse that we are about to describe. Internal rectal prolapse, whether associated or not to anal, mucosal and haemorrhoidal prolapse, can be a mucosal rectal prolapse (about 10 % of our cases) or a full-thickness rectal prolapse.

When performing a baseline 2-view X-ray of the empty rectum with barium contrast, we can see that some patients present with a rectum the shape of which, especially distally, indicates a detachment from the sacral-coccygeal plane; this type of rectum is usually folded on itself, with unnatural loops lying on the perineum, and is longer than usual. We have called this conformation “rectal redundancy”.

When performing a varied dynamic rectal videodefecography in patients with rectal redundancy, i.e. with only the sigmoid filled with barium and potato starch, we note that such a redundancy always causes an obstacle to the transit of barium into the rectal lumen, causing more intense straining and repeated attempts at evacuating.

During straining it is possible to observe how this rectal redundancy can assume different morphological aspects including various combinations: simple or multiple invagination, rectocele caused by rectal dilatation or by formation of a loop and partial or total outward expulsion of the rectum.

In patients with haemorrhoidal prolapse, a good impregnation of the anal canal allows to visualise how a rectoanal invagination pushes haemorrhoids and anal mucosa outside. It is also interesting to observe that in some patients a descent of the Douglas pouch or formation of an enterocele can be seen during straining; by compressing the rectum from above and pushing it towards the sacrum, this facilitates emptying of the rectal contents.

Enterocele and Douglas pouch dislocation are always associated to an excessive perineum lowering. These pelvic alterations can disappear completely or partially and can persist at the end of straining. With regard to their behaviour, we have divided these pelvic alterations into stable and dynamic ones. They are indeed caused by excessive straining and in our opinion should be considered supporting mechanisms compensating for the incapacity of a prolapsed rectum to empty physiologically.

With regard to the nature of the rectocele, I would like to point out that cadaveric studies and ultrasonographic mapping of the rectum in patients with a similar clinical and defecographic picture have clearly demonstrated that the only perceivable alteration is the thinning or disappearance of the muscular layer of the rectum. This type of defect begins just above the anal canal and extends variably upwards. As the rectal ampulla is no longer effectively supported by the muscular layer, it can expand anteriorly and, after occupying the perineal body, push onto the posterior vaginal wall, causing a colpocele. Large rectoceles can dilate the vaginal wall abnormally causing secondary structural damage. In any case, a rupture of the famous rectovaginal septum – the definition, function and existence of which have always been controversial – cannot be considered a primary cause of rectocele. In numerous cadaveric dissections, we have actually never been able to detect such a septum, and we therefore think that this is probably a wrong definition given by gynaecologists (out of self-interest?). We have recently received a clear confirmation of this theory by pathologists who never detected such a "septum" in specimens sent in by gynaecologists. We thought that these – unfortunately rather detailed – preliminary remarks were necessary to make the following conclusion understandable: haemorrhoidal prolapse is a pathology secondary to and consequent upon rectal prolapse, be it only mucosal or full thickness.

We therefore consider the clinical and pathological distinction between haemorrhoidal disease, rectal prolapse and rectocele an artificial one. Although the haemorrhoidal prolapse is the pathological alteration that causes the typical symptoms of haemorrhoidal disease, it must be considered simply an external manifestation of a prolapse of – initially – the rectum that subsequently and progressively can cause a prolapse of the anal mucosa, the haemorrhoidal cushions and the anoderm. Not always does an internal rectal prolapse cause a mucous and haemorrhoidal prolapse associated to it, but all haemorrhoidal prolapses are invariably associated to rectal prolapse. For this reason a more correct definition would be that of a rectoanal prolapse which would provide a more correct description of the anatomical and pathological condition. An in-depth revision of these pathologies and a clinical reclassification based on a new theoretical foundation is therefore necessary. We have proposed a single combined classification of these pathologies termed "unitary theory of rectoanal prolapse".

Based on the results of our studies and our observations, we came to the conclusion that a treatment consisting of the correction of internal rectal prolapse could also represent a rational treatment for haemorrhoidal prolapse, as it would

cure the cause that determines it. Moreover, the resection of the internal rectal prolapse would also resolve obstructed defecation if present, both because it would eliminate the mechanical obstacle and because resection of the distal rectum would include removing any rectoceles present, leading to improved of rectal compliance. Given the focus of this chapter, we will limit ourselves to stressing the fact that bleeding, thrombosis and haemorrhoidal oedema are only a few of the possible symptoms of the prolapses defined as recto-anal, and therefore obstructed defecation and continence disorders have to be taken into consideration when taking the history of these patients.

The rectal prolapse associated to a haemorrhoidal prolapse can present with different sizes, and there is no correlation in terms of size between haemorrhoidal and rectal prolapse. For this reason the simple clinical evaluation of an external haemorrhoidal prolapse is not predictive of the size of the rectal prolapse that has to be removed, and so it does not allow us to determine the technique that needs to be chosen. Histologically, a simple mucosal prolapse is a detachment of mucosa and submucosa from the muscular layer of the rectum, and given their increased length, it presents as a redundancy. Full-thickness prolapse is generally characterised by a lengthening of the rectum because of structural alterations of the muscular layers. The rectum tends to form multiple loops that fold on themselves. In other cases rectal prolapse can be due to slippage of the whole rectum-sigmoid. In this case the natural evolution is a complete external rectal prolapse. Mucous rectal prolapses can be resected with the stapled anopexy technique. STARR is reserved to large mucosal prolapses or to full-thickness prolapses. STARR can be performed by means of two PPH devices or with the more recently introduced curved stapler that goes by the (unfortunate) name of TRANSTAR.

Stapled Haemorrhoidopexy (SH)

Technique

Also known as the PPH procedure, Longo procedure, stapled anopexy and circumferential mucosectomy, PPH is a technique developed in 1993 that reduces the prolapse of haemorrhoidal tissue by excising a doughnut-like ring of the prolapsed rectal mucosa with a circular stapling device: with this the haemorrhoidal cushions, anal mucosa and anoderma are lifted and permanently fixed in their anatomical position [94], and a haemorrhoidal prolapse during defecation is prevented.

The procedure can be performed under subarachnoid anaesthesia; the patient is placed in lithotomic position. A PPH-01 or PPH-03 kit (Ethicon Endo-Surgery, Cincinnati, Ohio) is necessary. The introduction of the circular anal dilator (CAD) causes the reduction of the anal prolapse into the rectum. After removing the obturator, the prolapsed rectal mucosa falls into the lumen of the dilator. The purse-string anoscope (PSA) is then introduced through the dilator. This anoscope will push the prolapsing mucosa back against the rectal wall along a 270° circumference, while the mucous membrane that protrudes through the anoscope window can be easily captured with a stitch (Prolene TM 00, Ethicon). By rotating the anoscope, it will be possible to complete a purse-string suture around the entire rectal circumference, 2–3 cm above the haemorrhoidal apex.

A PPH-01/PPH-03 stapler is opened to its maximum position. Its head is introduced until crossing the purse-string which is then tied with a knot. The ends of the suture are knotted externally. Then the stapler is partially tightened while keeping the casing outside. Once half the casing is inserted into the CAD, it is pushed against the purse-string, and while exerting moderate traction on the ends of the suture, the instrument is tightened. Keeping the stapling device in the maximum closed position, for approximately 30 s, may improve the haemostasis. Firing the stapler releases a double staggered row of titanium staples through the tissue. The circular stapler knife excises the tissue. A circumferential column of mucosa is removed. Finally, the staple line is examined using the anoscope. Additional haemostasis can be achieved by stitches (Vicryl TM 2-0, Ethicon).

Results

A recent systematic review [95] has allowed us to document the fact that a huge number of scientific publications are available in the literature about the PPH procedure: there are 29 publications [96–124] on 25 randomised clinical trials comparing PPH stapled haemorrhoidopexy with conventional haemorrhoidectomy. They included a total of 1,918 patients, of whom 971 underwent stapled haemorrhoidopexy (PPH procedure) and 947 had surgical haemorrhoidectomy. The main results are reported here.

Procedure Time

In the 23 trials [96, 98, 100, 102, 103, 105–124] in which it was possible to calculate, it was found that the PPH procedure stood out for its significantly shorter operating time compared with conventional haemorrhoidectomy [mean operating time, 17.55 vs. 28.90 min; weighted mean difference (WMD) – 11.35 min; $P=0.006$].

Pain

The PPH procedure caused significantly less postoperative pain than conventional surgery. Twenty-three trials [96, 98, 100, 102–108, 110–112, 114, 115, 117–124], reported significantly less pain after PPH as evidenced by reduction of the pain scores at rest and on defecation by 42.3 %.

Recovery

There was a faster surgical and functional recovery after stapled haemorrhoidopexy. The PPH haemorrhoidopexy allowed a faster functional recovery with shorter time off work (WMD – 8.45 days; $P<0.00001$) and earlier return to normal activities (WMD – 15.85 days; $P=0.03$).

Patient Satisfaction

Significantly more patients in the PPH than in the conventional haemorrhoidectomy group rated the procedure as satisfactory [93.3 vs. 86.4 %; odds ratio (OR) 2.33; $P=0.003$] [98, 102, 105, 106, 112, 119, 123].

Re-intervention

The PPH procedure did not increase the overall need of surgical (OR, 1.27; $P=0.4$) and nonsurgical (OR, 1.07; $P=0.82$) re-intervention compared with conventional haemorrhoidectomy [96, 98, 100, 103–105, 107–110, 112, 114, 116–119, 121–123].

Bleeding

There was no significant difference in the amount of intraoperative bleeding ($P=0.26$) or the incidence of early postoperative bleeding (bleeding within 24 h of surgery; $P=0.11$). At more than 1 day after surgery, the PPH procedure was associated with significantly less risk of bleeding (9.8 vs. 15.1 %; OR, 0.52 $P=0.001$) [96, 100, 102–106, 108–111, 114–116, 118, 124]. There was no difference between the groups regarding the need for readmission as a result of bleeding (OR, 0.63; $P=0.67$), blood transfusion (OR, 0.64; $P=0.54$), or further nonoperative (OR, 4.06, $P=0.08$) or operative interventions for bleeding (OR, 1.02; $P=0.95$).

Perianal Complications

There was no significant difference between the two procedures regarding early (OR, 1.82; $P=0.52$) [21, 24, 28, 30, 36] or late anal stenosis (OR, 0.69; $P=0.33$) [96, 98, 100, 103, 105, 107, 108, 111, 112, 115, 116, 124], anal fissure (OR, 0.93; $P=0.88$) [96, 105, 112, 114, 116, 117, 123, 124] or perianal fistula (OR, 0.25; $P=0.23$) [107, 110, 112, 116, 117].

Early Recurrence

There was no significant difference between the two groups with regard to early postoperative recurrence (within 6 months) or persistence of symptoms from haemorrhoids: 24.8 and 31.7 % after PPH procedure and conventional haemorrhoidectomy, respectively (OR, 0.68; $P=0.08$) [98, 108, 111, 114, 117, 122, 124]. There was no difference in the need for further operation for early recurrent haemorrhoids (OR, 0.71; $P=0.69$).

Late Recurrence

The incidence of recurrent haemorrhoids at 1 year or more after surgery was higher after stapled haemorrhoidopexy (5.7 vs. 1 %; OR, 3.48, $P=0.02$). However, the overall incidence of

recurrent or persistent symptoms from haemorrhoids was similar in the groups (PPH vs. conventional: 25.3 vs. 18.7 %; OR, 1.57; $P=0.07$) [98, 106–108, 111, 114, 117].

Quality of Life

Three trials [96, 100, 109] addressed the quality of life after surgery. There was no significant difference in quality of life after either surgical procedure, as both the Short-Form 36 Quality of Life questionnaires [97, 109] and the Eypasch Gastrointestinal Quality of Life instrument [100] showed. However, there was a tendency towards higher median physical and mental scores after PPH procedures.

Cost-Effectiveness

Four trials [100, 103, 109, 120] investigated the cost-effectiveness of stapled haemorrhoidopexy compared with conventional surgery. When both the operating cost and hospital stay charges were taken into account, conventional haemorrhoidectomy was more expensive than the PPH procedure, although the differences were not statistically significant [103, 109]. Thus, the cost of the disposable stapler was offset by a shorter hospital stay. In an Asian study [100], where hospital charges are less expensive than in the West, the total medical cost was higher after the PPH procedure (US $1,283.09 ± T 31.59 vs. US $921.17 ± 16.85).

In light of all the above-mentioned considerations, PPH stapled haemorrhoidopexy is safe with many short-term benefits, and long-term results are similar to the conventional procedure.

Stapled Transanal Rectal Resection (STARR)

Technique

STARR was proposed by Longo in 1998. We suggest performing the procedure under subarachnoid anaesthesia with the patient in lithotomic position.

Two PPH-01 or PPH-03 kits (Ethicon Endo-Surgery, Cincinnati, Ohio) are necessary. The anal canal is checked digitally, in order to ensure optimal relaxation. Any haemorrhoidal prolapse or procidentia is reduced using a gauze pack inserted into the anal canal. Four skin stitches are applied to fix the circular anal dilator (CAD) which is lubricated with Vaseline before being inserted. A swab is inserted and slowly retracted to assess the extent of the invagination. A spatula is passed through the posterior window of the CAD to protect the posterior rectal wall. A purse-string suture anoscope (PSA) is used to place 2–3 anterior semicircular sutures (Prolene TM 00, Ethicon) between 9 and 3 o'clock, the most caudal one 2–3 cm above the haemorrhoidal apex, the following ones in 2 cm steps. The homolateral threads are tied under moderate tension. A lubricated maximally opened stapler is introduced with the head passing beyond the sutures. The bundled threads are pulled through the holes of the casing and clamped. Keeping the stapler head in a stable position while the threads are pulled tight, the stapler is partially closed until the resistance of the first suture is felt. By that it is ensured that the edge of the casing is placed above the haemorrhoids and anal ring. Prior to full stapler closure, a vaginal spatula is inserted, and using two fingers the integrity of the vaginal wall is checked. The stapler is closed, fired and removed. Lateral "dog ears" joined by a small bridge which has to be cut are observed frequently. In case of relevant bleeding, haemostatic stitches (Vicryl TM 2-0, Ethicon) are applied. The spatula is now repositioned to protect the anterior rectal wall. The first posterior semicircular suture is placed starting from the base of the left dog ear and moving on to the right dog ear, the second one from the left dog ear's apex to the right one. The posterior prolapse is then resected as described above. Haemostasis is achieved using haemostatic stitches and checked carefully.

Please note: The number of anterior sutures is related to the extension of the invagination. The semicircular sutures on the anterior and posterior wall of the rectum can be substituted by a short running suture. Generally, this option ensures a more homogeneous traction on the rectal walls and, at present, is preferred by colorectal surgeons in Italy.

We suggest to oversew residual dog ears and to apply four to five stitches to reinforce the anterior and posterior staple line. A strip of Vaseline gauze tied to a suture should be introduced to prevent the formation of submucosal haematomas and to facilitate diagnosis of postoperative bleeding. A urinary catheter was inserted in all cases.

Results

Recently, in an attempt to prevent the incidence of failures after SH caused by incomplete resection of the prolapsed tissue (due to the limited volume of the stapler casing), the STARR procedure was adopted successfully for those patients in which a large prolapse was associated with the haemorrhoidal disease. Boccasanta et al. [125] stated that, in patients with an association of prolapsed haemorrhoids and large rectal prolapsed, STARR results in a more complete resection of the prolapsed tissue than SH, with equal morbidity and a significantly lower incidence of residual disease and skin tags. The author used the circular anal dilator, CAD, in order to determine the appropriate surgical technique.

Furthermore, as reported in a recent randomised multicentre trial involving more than 400 patients [126], even if both the PPH-01 and PPH-03 kit can be used, the use of the PPH-03 stapler instead of the PPH-01 ensures a statistically significant reduction of intraoperative bleeding and a significant decrease of operative time.

Comment on SH and STARR

What emerges clearly from a review of the available literature on stapled haemorrhoidopexy and STARR is that many surgeons consider stapled haemorrhoidopexy a procedure indicated for haemorrhoidal prolapse and STARR an operation exclusively indicated in cases of obstructed defecation due to internal rectal prolapse and rectocele. Others consider that in case of haemorrhoidal prolapse, indications for STARR should be limited to patients with associated OD [127]. It is fundamental to revise and adjust these ideas regarding indications for SH and STARR in order to obtain optimal results. As said before, haemorrhoidal prolapse is always a consequence of an internal rectal prolapse of variable size and not correlated to the degree and dimensions of muco-haemorrhoidal prolapse. This implies that a modest external prolapse can be associated to a significant internal prolapse. If we perform SH in these kinds of patients, we will certainly leave a residual internal rectal prolapse behind. We believe that this may predispose for a higher rate of recurrences and may also be the reason why a possibly associated OD is not cured or, worse, even aggravated as the residual prolapse can be jammed inside the anastomosis, especially if a fibrotic ring forms.

It is therefore important to state clearly that for haemorrhoidal prolapses, whether symptoms of obstructed defecation are associated or not, STARR is the procedure of choice whenever an important mucosal prolapse is detected and in all the cases in which there is a full-thickness prolapse or a rectocele. I hope not to shock anyone by confessing that in the last few years, I have myself performed STARR in about 95 % of patients with muco-haemorrhoidal prolapse. Thanks to this decision the rate of recurrences has dropped, at 3 years of follow-up, from 4.9 to 0.4 %. Also with regard to curing OD as one of the complications of this procedure, the results are much more satisfactory. Paradoxically postoperative pain and bleeding have also decreased. In any case, the key aspect is that after so many years the theory has been proven that haemorrhoidal prolapse is secondary to rectal prolapse and that it can be effectively cured by rectal prolapsectomy sparing the haemorrhoids.

Generally, with regard to the advantages of SH and STARR, reduced postoperative pain and faster return to work are frequently highlighted. In my personal opinion the main advantages are resolution of OD (which is often associated), efficient outcome regarding soiling and continence, and the rare incidence of stenosis and, if they occur, the relatively easy treatment of stenosed anastomoses. Now that initial scepticism about SH and STARR as a cure for haemorrhoids has been overcome, the usual detractors insist on a

supposedly higher rate of recurrences following these procedures, which has been proven absolutely incorrect, and on supposed severe complications. Obviously complications can occur, but after three million procedures, only very few cases have been reported and overemphasised with the support of some compliant journal that has published a number of articles without the necessary verifications on the trustworthiness of results. In fact, if some of the articles that report on severe and frequent complications were reliable [128], one would have to suspect a sadomasochistic tendency among thousands of surgeons performing this technique and an inclination to economical failure on the part of the five new companies that have copied the original PPH.

This whole chapter can be summarised by saying that prolapsed and hence symptomatic haemorrhoids are only an epiphenomenon of an internal rectal prolapse which is the primary pathology. Therefore, by adequately treating the internal rectal prolapse, haemorrhoidal disease and all the other symptoms caused by rectoanal prolapse are cured.

References

1. Thomson WHF. The nature of haemorrhoids. Br J Surg. 1975;62:542.
2. Haas PA, Fox Jr TA, Haas GP. The pathogenesis of hemorrhoids. Gut. 1977;18:651.
3. Gass OC, Adams J. Haemorrhoids: aetiology and pathology. Am J Surg. 1950;79:40–3.
4. Goligher J, Duthie H, Nixon H. Surgery of the anus, rectum and colon. London: Balliere Tindall; 1984. p. 5.
5. Andrews E. The treatment of haemorrhoids by injection. Med Rec. 1879;15:451.
6. Edwards FS. The treatment of piles by injection. BMJ. 1888;2:815.
7. Kelsey CB. How to treat haemorrhoids by injection of carbolic acid. N Y Med J. 1885;42:545.
8. Terrell EH. The treatment of haemorrhoids by new method. Trans Am Proctol Soc. 1916;5:65.
9. Anderson HG. The "injection" method for the treatment of haemorrhoids. Practitioner. 1924;113:339.
10. Khoury GA, Lake SP, Lewis MCA, et al. A randomized trial to compare single with multiple phenol injection treatment of haemorrhoids. Br J Surg. 1985;72:741.
11. Dencker H, Hjorth N, Norryd C, et al. Comparison of results with different methods of treatment of internal haemorrhoids. Acta Chir Scand. 1973;139:742.
12. Santos G, Novell JR, Chir M, et al. Long term results of large-dose, single-session phenol injection sclerotherapy for hemorrhoids. Dis Colon Rectum. 1993; 36:958.
13. Ambrose NS, Morris D, Alexander-Williams J, et al. A randomized trial of photocoagulation or injection sclerotherapy for the treatment of first- and second-degree hemorrhoids. Dis Colon Rectum. 1985;28: 238.
14. Gartell PC, Sheridan RJ, McGinn FP. Outpatient treatment of hemorrhoids: a randomized clinical trial to compare rubber band ligation with phenol injection. Br J Surg. 1985;72:478.
15. Rosser C. Chemical rectal stricture. JAMA. 1931;96: 1762.
16. Adami B, Eckardt VF, Suermann RB, et al. Bacteremia after proctoscopy and hemorrhoid injection sclerotherapy. Dis Colon Rectum. 1981;24:373.
17. Ribbans WJ, Radcliffe AG. Retroperitoneal abscess following sclerotherapy for hemorrhoids. Dis Colon Rectum. 1985;28:188.
18. Guy RJ, Seow-Choen F. Septic complications after treatment of haemorrhoids. Br J Surg. 2003;90:147.
19. Blaisdell PC. Prevention of massive hemorrhage secondary to hemorrhoidectomy. Surg Gynecol Obstet. 1985;106:485.
20. Barron J. Office ligation of internal hemorrhoids. Am J Surg. 1963;105:563.
21. Barron J. Office ligation treatment of hemorrhoids. Dis Colon Rectum. 1963;6:109.
22. Corman ML. Hemorrhoids. In: Corman ML, editor. Colon and rectal surgery. Philadelphia: Lippincott Williams & Wilkins; 2005.
23. Mattana C, Maria G, Pescatori M. Rubber band ligation of hemorrhoids and rectal mucosal prolapse in constipated patients. Dis Colon Rectum. 1989;32:372–5.
24. Bartizal J, Slosberg P. An alternative to haemorrhoidectomy. Arch Surg. 1977;112:534.
25. Lee HH, Spencer RJ, Beart Jr RW. Multiple hemorrhoidal bandings in a single session. Dis Colon Rectum. 1994;37:37–41.
26. Walker AJ, Leicester RJ, Nicholls RJ, Mann CV. A prospective study of infrared coagulation, injection and rubber band ligation in the treatment of haemorrhoids. Int J Colorectal Dis. 1990;5:113–6.
27. Marshman D, Huber Jr PJ, Timmerman W, Simonton CT, Odom FC, Kaplan ER. Hemorrhoidal ligation. A review of efficacy. Dis Colon Rectum. 1989;32(5): 369–71.
28. Wrobleski DE, Corman ML, Veidenheimer MC, Coller JA. Long-term evaluation of rubber ring ligation in hemorrhoidal disease. Dis Colon Rectum. 1980;23:478–82.
29. Savioz D, Roche B, Glauser T, Dobrinov A, Ludwig C, Marti MC. Rubber band ligation of hemorrhoids: relapse as a function of time. Int J Colorectal Dis. 1998;13(4):154–6.
30. Bayer I, Myslovaty B, Picovsky BM. Rubber band ligation of hemorrhoids. Convenient and economic treatment. J Clin Gastroenterol. 1996;23:50–2.

31. Armstrong DN. Multiple hemorrhoidal ligation: a prospective randomized trial evaluation a new technique. Dis Colon Rectum. 2003;46:179–86.
32. O'Regan PJ. Disposable device and minimally invasive technique for rubber band ligation of hemorrhoids. Dis Colon Rectum. 1999;42:683–5.
33. Poon GP, Chu KW, Lau WY. Conventional vs. triple rubber band ligand for hemorrhoids. A prospective, randomized trial. Dis Colon Rectum. 1986;29:836–8.
34. Khubchandani IT. A randomized comparison of single and multiple rubber band ligations. Dis Colon Rectum. 1983;26:705–8.
35. Lau WY, Chow HP, Poon GP, Wong SH. Rubber band ligation of three primary hemorrhoids in a single session. A safe and effective procedure. Dis Colon Rectum. 1982;25:336–9.
36. Templeton JL, Spence RA, Kennedy TL, Parks TG, Mackenzie G, Hanna WA. Comparison of infrared coagulation and rubber band ligation for first and second degree haemorrhoids: a randomized prospective clinical trial. Br Med J (Clin Res Ed). 1983;286:1387–9.
37. Ambrose NS, Hares MM, Alexander-Williams J, Keighley MR. Prospective randomized comparison of photocoagulation and rubber band ligation in treatment of haemorrhoids. Br Med J (Clin Res Ed). 1983;286:1389–91.
38. O'Hara VS. Fatal clostridial infection following hemorroidal banding. Dis Colon Rectum. 1980;23:570.
39. Clay III LD, White Jr JJ, Davidson JT, et al. Early recognition and successful management of pelvic cellulitis following hemorrhoidal banding. Dis Colon Rectum. 1986;29:579.
40. Quevado-Bonilla G, Farkas AM, Abcarian H, et al. Septic complication of hemorrhoidal banding. Arch Surg. 1988;123:650.
41. Russell TR, Donohue JH. Hemorrhoidal banding: a warning. Dis Colon Rectum. 1985;28:291.
42. Scarpa FJ, Hills W, Sabetta JR. Pelvic cellulitis: a life-threatening complication of hemorrhoidal banding. Surgery. 1988;103:383.
43. Sim AJW, Murie JA, Mackenzie I. Three-years follow-up study on the treatment of first- and second-degree hemorrhoids by sclerosant injection or rubber band ligation. Surg Gynecol Obstet. 1983;157:534.
44. Wechter DG, Luna GK. An unusual complication of rubber band ligation. Dis Colon Rectum. 1987;30:137.
45. Bat L, Malzer E, Koler M, et al. Complications of rubber band ligation of symptomatic internal hemorrhoids. Dis Colon Rectum. 1993;36:287.
46. Cooper IS, Lee AS. Cryostatic congelation: a system for producing a limited, controlled region of cooling or freezing of biologic tissues. J Nerv Ment Dis. 1961;133:259.
47. Gill W, Fraser J, da Costa J, et al. The cryosurgical lesion. Am Surg. 1970;36:437.
48. Lewis MI. Cryosurgical hemorrhoidectomy: a follow-up report. Dis Colon Rectum. 1972;15:128.
49. White AC. Liquid air: its application in medicine and surgery. Med Rec. 1899;56:109.
50. Williams KL, Haq IU, Elem B. Cryodestruction of haemorrhoids. BMJ. 1973;1:666.
51. Kaufman HD. Haemorrhoids: an 'out-patient package. J R Coll Surg Edinb. 1978;23:40–3.
52. Southam JA. Haemorrhoids treated by cryotherapy: a critical analysis. Ann R Coll Surg Engl. 1983;65:237–9.
53. Berry AR, D'Acosta EFD. The treatment of haemorrhoids by cryosurgery. J R Coll Surg Edinb. 1978;23:37–9.
54. Smith LE, Goodreau JJ, Fouty WJ. Operative hemorrhoidectomy versus cryodestruction. Dis Colon Rectum. 1979;22:10.
55. O'Callaghan JD, Matheson TS, Hall R. Inpatient treatment of prolapsing piles: cryosurgery versus Milligan-Morgan haemorrhoidectomy. Br J Surg. 1982;69:157–9.
56. Goligher JC. Cryosurgery for hemorrhoids. Dis Colon Rectum. 1976;1976:213–8.
57. Keighley RB, Williams NS. Haemorrhoidal disease. In: Keighley RB, Williams NS, editors. Surgery of the anus, rectum and colon. Saunders Co. Lts; 2000.
58. Nath G, Kreitmaier A, Kiefhaber P, et al. Neue Infrarotkoagulationsmethode. Verhaundlungsband des 3 Kongresses der Deutscher Gesellschaft fur Gastroenterologie, Munich. Erlangen: Perimed; 1976.
59. Nieger A. Haemorrhoid in everyday practice. Proc Natl Acad Sci U S A. 1979;2:22.
60. Marques CF, Nahas SC, Nahas CS, et al. Early results of the treatment of internal hemorrhoid disease by infrared coagulation and elastic banding: a prospective randomized cross-over trial. Tech Coloproctol. 2006;10:312–7.
61. Weintein SJ, Rypins EB, Houck J, et al. Single-session treatment for bleeding hemorrhoids. Surg Gynecol Obstet. 1987;165:479.
62. Jensen DM, Jutabha R, Machicado GA, et al. Prospective randomized comparative study of bipolar electrocoagulation versus heater probe for treatment of chronically bleeding internal hemorrhoids. Gastrointest Endosc. 1997;46:435–43.
63. Randall GM, Jensen DM, Machicado GA, et al. Prospective randomized comparative study of bipolar versus direct current electrocoagulation for treatment of bleeding internal hemorrhoids. Gastrointest Endosc. 1994;40:403–10.
64. Hilton CP, Morris DL. A randomized trial comparing direct current therapy and bipolar diathermy in the outpatient treatment of third-degree hemorrhoids. Dis Colon Rectum. 1990;33:931–2.
65. Zinberg SS, Stern DH, Furman DS, Wittles JM. A personal experience in comparing three nonoperative techniques for treating internal hemorrhoids. Am J Gastroenterol. 1989;84:488–92.
66. Varma JS, Chung SC, Li AK. Prospective randomized comparison of current coagulation and injection sclerotherapy for the outpatient treatment of hemorrhoids. Int J Colorectal Dis. 1991;6:42–5.
67. Norman DA, Newton R, Nicholas GV. Direct current electrotherapy of internal hemorrhoids: an effective,

safe and painless out-patient approach. Am J Gastroenterol. 1989;84:482–7.
68. Dennison AR, Whiston RJ, Rooney S, et al. The management of hemorrhoids. Am J Gastroenterol. 1989;84:475.
69. Hinton CP, Morris DL. A randomized trial comparing direct current therapy and bipolar diathermy in the outpatient treatment of third-degree hemorrhoids. Dis Colon Rectum. 1990;22:931.
70. Dennison AR, Whiston RJ, Rooney S, et al. A randomized comparison of infrared photocoagulation with bipolar diathermy for the outpatient treatment of hemorrhoids. Dis Colon Rectum. 1990;33:32.
71. Griffith CDM, Morris DL, Wherry DC, et al. Outpatient treatment of hemorrhoids: a randomized trial comparing contact bipolar diathermy with rubber ring ligation. Coloproctology. 1987;9:332.
72. Yang R, Migikovsky B, Peicher J, et al. Randomized, prospective trial of direct current versus bipolar electrocoagulation for bleeding internal hemorrhoids. Gastrointest Endosc. 1993;39:766.
73. Morinaga K, Hasuda K, Ikeda T. A novel therapy for internal hemorrhoids: ligation of the hemorrhoidal artery with a newly devised instrument (Moricon) in conjunction with a Doppler flow meter. Am J Gastroenterol. 1995;990:610–3.
74. Scheyer M, Antonietti E, Rollinger G, Mall H, Arnold S. Doppler-guided hemorrhoidal artery ligation. Am J Surg. 2006;191:89–93.
75. Dal Monte PP, Tagariello C, Sarago M, Giordano P, Shafi A, Cudazzo E, et al. Transanal haemorrhoidal dearterialisation: nonexcisional surgery for the treatment of haemorrhoidal disease. Tech Coloproctol. 2007;11:333–8.
76. Lienert M, Ulrich B. Doppler-guided ligation of the hemorrhoidal arteries. Report of experiences with 248 patients. Dtsch Med Wochenschr. 2004;129:947–50.
77. 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;19:176–80.
78. Middleton SB, Lovegrove RE, Reece-Smith H. Management of haemorrhoids: symptoms govern treatment. BMJ. 2008;336:461.
79. Ratto C, Donisi L, Parello A, Litta F, Doglietto GB. Evaluation of transanal hemorrhoidal dearterialization as a minimally invasive therapeutic approach to hemorrhoids. Dis Colon Rectum. 2010;53: 803–11.
80. Milligan ETC, Morgan CN, Jones LE, et al. Surgical anatomy of the anal canal, and operative treatment of haemorrhoids. Lancet. 1937;2:1119.
81. Jayne DG, Botterill I, Ambrose NS, et al. Randomized clinical trial of Ligasure versus conventional diathermy for day case haemorrhoidectomy. Br J Surg. 2002;89:428.
82. Palazzo FF, Francis DL, Clifton MA. Randomized clinical trial of Ligasure versus open haemorrhoidectomy. Br J Surg. 2002;89:154–7.
83. Chung YC, Wu HJ. Clinical experience of sutureless closed hemorrhoidectomy with LigaSure. Dis Colon Rectum. 2003;46:87–92.
84. Franklin EJ, Seetharam S, Lowney J, Horgan PG. Randomized, clinical trial of Ligasure vs conventional diathermy in hemorrhoidectomy. Dis Colon Rectum. 2003;46:1380–3.
85. Nicholson JD, Halleran DR, Trivisonno DP, et al. The efficacy of the contact sapphire tip Nd:YAG laser hemorrhoidectomy. Poster presented at the 89th annual meeting of the America Society of Colon and Rectal Surgeons, St. Louis, April 29–May 4, 1990.
86. Wang JY, Chang-Chien CR, Chen J-S, et al. The role of laser in hemorrhoidectomy. Dis Colon Rectum. 1991;34:78.
87. Senagore A, Mazier WP, Luchtefeld MA, et al. Treatment of advanced hemorrhoidal disease: a prospective, randomized comparison of cold scalpel versus contact Nd:YAG laser. Dis Colon Rectum. 1993;36:1042.
88. Ferguson JA, Heaton JR. Closed hemorrhoidectomy. Dis Colon Rectum. 1959;2:176.
89. Parks AG. Surgical treatment of haemorrhoids. Br J Surg. 1956;43:337.
90. Whitehead W. The Surgical treatment of haemorrhoids. BMJ. 1882;1:148.
91. Bonello JC. Who's afraid of the dentate line? The whitehead hemorrhoidectomy. Am J Surg. 1988;156: 182.
92. Wolff BG, Culp CE. The Whitehead hemorrhoidectomy: an unjustly maligned procedure. Dis Colon Rectum. 1988;31:587.
93. Barrios G, Khubchandani M. Whitehead operation revisited. Dis Colon Rectum. 1979;22:330.
94. Longo A. Treatment of hemorrhoidal disease by reduction of mucosal and hemorrhoidal prolapse with circular suturing device: a new procedure. In: Proceedings of the 6th world congress of Endoscopic Surgery. Bologna: Monduzzi Editore; 1998. p. 777–84.
95. Tjandra JJ, Chan MK. Systematic review on the procedure for prolapse and hemorrhoids (stapled hemorrhoidopexy). Dis Colon Rectum. 2007;50:878–92.
96. Mehigan BJ, Monson JR, Hartley JE. Stapling procedure for haemorrhoids versus Milligan-Morgan Haemorrhoidectomy: randomized controlled trial. Lancet. 2000;355:782–5.
97. Smyth EF, Baker RP, Wilken BJ, Hartley JE, White TJ, Monson JR. Stapled versus excision hemorrhoidectomy: long term follow up of a randomized controlled trial. Lancet. 2003;361:1437–8.
98. Rowsell M, Bello M, Hemingway DM. Circumferential mucosectomy (stapled haemorrhoidectomy) versus conventional haemorrhoidectomy: randomized controlled trial. Lancet. 2000;355: 779–81.
99. Au-Yong M, Rowsell M, Hemingway DM. Randomised controlled clinical trial of stapled haemorrhoidectomy vs. conventional haemorrhoidectomy; a three and a half year follow up. Colorectal Dis. 2004;6:37–8.

100. Ho YH, Cheong WK, Tsang C, et al. 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;43:1666–75.
101. Ooi BS, Ho YH, Tang CL, Eu KW, Seow-Choen F. Results of stapling and conventional hemorrhoidectomy. Tech Coloproctol. 2002;6:59–60.
102. Helmy MA. Stapling procedure for hemorrhoids versus conventional hemorrhoidectomy. J Egypt Soc Parasitol. 2000;30:951–8.
103. Boccasanta P, Capretti PG, Venturi M, et al. Randomised controlled trial between stapled circumferential mucosectomy and conventional circular hemorrhoidectomy in advanced hemorrhoids with external mucosal prolapse. Am J Surg. 2001;182:64–8.
104. Ganio E, Altomare DF, Gabrielli F, Milito G, Canuti S. Prospective randomized multicentre trial comparing stapled with open haemorrhoidectomy. Br J Surg. 2001;88:669–74.
105. Shalaby R, Desoky A. A randomized clinical trial of stapled versus Milligan-Morgan haemorrhoidectomy. Br J Surg. 2001;88:1049–53.
106. Pavlidis T, Papaziogas B, Souparis A, Patsas A, Koutelidakis I, Papaziogas T. Modern stapled Longo procedure vs. conventional Milligan-Morgan hemorrhoidectomy: a randomized controlled trial. Int J Colorectal Dis. 2002;17:50–3.
107. Hetzer FH, Demartines N, Handschin AE, Clavien PA. Stapled vs excision hemorrhoidectomy: long-term results of a prospective randomized trial. Arch Surg. 2002;137:337–40.
108. Correa-Rovelo JM, Tellez O, Obregon L, Miranda-Gomez A, Moran S. Stapled rectal mucosectomy vs. closed hemorrhoidectomy: a randomized, clinical trial. Dis Colon Rectum. 2002;45:1367–74.
109. Wilson MS, Pope V, Doran HE, Fearn SJ, Brough WA. Objective comparison of stapled anopexy and open hemorrhoidectomy: a randomized, controlled trial. Dis Colon Rectum. 2002;45: 1437–44.
110. Ortiz H, Mazo J, Armendariz P. Randomized clinical trial of stapled hemorrhoidopexy versus conventional diathermy haemorrhoidectomy. Br J Surg. 2002;89:1376–81.
111. Kairaluoma M, Nuorva K, Kellokumpu I. Day-case stapled (circular) vs. diathermy hemorrhoidectomy: a randomized, controlled trial evaluating surgical and functional outcome. Dis Colon Rectum. 2003; 46:93–9.
112. Palimento D, Picchio M, Attanasio U, Lombardi A, Bambini C, Renda A. Stapled and open hemorrhoidectomy: randomized controlled trial of early results. World J Surg. 2003;27:203–7.
113. Picchio M, Palimento D, Attanasio U, Renda A. Stapled vs open hemorrhoidectomy: long-term outcome of a randomized controlled trial. Int J Colorectal Dis. 2006;21:668–9.
114. Cheetham MJ, Cohen CR, Kamm MA, Phillips RK. A randomized, controlled trial of diathermy hemorrhoidectomy vs. stapled hemorrhoidectomy in an intended day-care setting with longer-term follow-up. Dis Colon Rectum. 2003;46:491–7.
115. Krska Z, Kvasnieka J, Faltyn J, et al. Surgical treatment of haemorrhoids according to Longo and Milligan-Morgan: an evaluation of postoperative tissue response. Colorectal Dis. 2003;5:573–6.
116. Racalbuto A, Aliotta I, Corsaro G, Lanteri R, Di Cataldo A, Licata A. Hemorrhoidal stapler prolapsectomy vs Milligan-Morgan hemorrhoidectomy: a long-term randomized trial. Int J Colorectal Dis. 2004;19:239–44.
117. Senagore AJ, Singer M, Abcarian H, Procedure for Prolapse and Hemorrhoids (PPH) Multicenter Study Group, et al. A prospective, randomized, controlled multi-center trial comparing stapled hemorrhoidopexy and Ferguson hemorrhoidectomy: perioperative and one-year results. Dis Colon Rectum. 2004;47:1824–36.
118. Lau PY, Meng WC, Yip AW. Stapled haemorrhoidectomy in Chinese patients: a prospective randomized controlled study. Hong Kong Med J. 2004; 10:373–7.
119. Bikhchandani J, Agarwal PN, Kant R, Malik VK. Randomized controlled trial to compare the early and mid-term results of stapled versus open hemorrhoidectomy. Am J Surg. 2005;189:56–60.
120. Basdanis G, Papadopoulos VN, Michalopoulos A, Apostolidis S, Harlaftis N. Randomized clinical trial of stapled hemorrhoidectomy vs. open with Ligasure for prolapsed piles. Surg Endosc. 2005; 19:235–9.
121. Ortiz H, Marzo J, Armendariz P, De Miguel M. Stapled hemorrhoidopexy vs. diathermy excision for fourth- degree hemorrhoids: a randomized, clinical trial and review of the literature. Dis Colon Rectum. 2005;48:809–15.
122. Chung CC, Cheung HY, Chan ES, Kwok SY, Li MK. Stapled hemorrhoidopexy vs. harmonic scalpel hemorrhoidectomy: a randomized trial. Dis Colon Rectum. 2005;48:1213–9.
123. Gravie JF, Lehur PA, Huten N, et al. Stapled hemorrhoidopexy versus Milligan-Morgan hemorrhoidectomy: a prospective, randomized, multicenter trial with 2-year postoperative follow up. Ann Surg. 2005;242:29–35.
124. Kraemer M, Parulava T, Roblick M, Duschka L, Muller- Lobeck H. Prospective, randomized study: proximate PPH stapler vs. ligasure for hemorrhoidal surgery. Dis Colon Rectum. 2005;48: 1517–22.
125. Boccasanta P, Venturi M, Roviaro G. Stapled transanal rectal resection versus stapled anopexy in the cure of hemorrhoids associated with rectal prolapse. A randomized controlled trial. Int J Colorectal Dis. 2007;22:245–51.
126. Renzi A, Brillantino A, Di Sarno G, et al. PPH-01 versus PPH-03 to perform STARR for the treatment

of hemorrhoids associated with large internal rectal prolapse: a prospective multicenter randomized trial. Surg Innov. 2011;18:241–7.
127. Corman ML, Carriero A, Hager T, et al. Consensus conference on the stapled transanal rectal resection (STARR) for disordered defaecation. Colorectal Dis. 2006;8:98–101.
128. Cheetham MJ, et al. Persistent pain and fecal urgency after stapled hemorrhoidectomy. Lancet. 2000;356:730–3.