Original article

A pilot study: The anal sphincter support procedure for the treatment of anal incontinence

MAXWELL E. HAVERFIELD

The Northern Hospital, Epping, Victoria, Australia

Abstract: Faecal incontinence is a significant and a very debilitating condition. The true prevalence of which is unknown but up to 3% of the general population have stool incontinence which increases with age. Faecal incontinence is much more common in women, increases with parity, and up to 30% have associated urinary incontinence and/or pelvic organ prolapse. Patients who have an identifiable external sphincter defect who fail conservative treatment options are best managed by a primary repair of the defect. This study presents a novel way of supporting the external anal sphincter with a circlage tape prosthesis. Based on the success of tension-free suburethral tapes used to treat stress urinary incontinence a pilot study was devised to assess Anal Sphincter Support System (ASSP) in patients presenting with mild faecal incontinence. Fourteen patients were followed up for a mean period of eighteen months. Outcomes were very encouraging with resolution of symptoms between 70 and 100%. There were no observed complications. This pilot study demonstrates that the ASSP provides a safe and effective method of restoring anal continence and can be used successfully with concomitant procedures which may be necessary for restoration of pelvic floor anatomy and function.

Key words: Faecal incontinence; Anal Sphincter Support Procedure; Hammock hypothesis.

INTRODUCTION

Faecal incontinence is rarely due to one single factor. Vaginal childbirth and obstetric trauma are widely accepted as significant causes of faecal and flatal incontinence in women. Approximately 10-15% report symptoms following vaginal delivery culminating in 2-5 % of women having faecal incontinence.[1] The most common contributing cause is anal sphincter injury occurring at the time of the second stage of labour. Other significant factors are instrumental delivery, midline episiotomy and prolonged second stage of labour.[2, 3] Also thought to be of significance is traction or entrapment of the pudendal nerve and its branches of the perineal nerve fibres, associated with injury to the levator ani plate (iliococcygeous, pubococcygeous and puborectalis muscles).

Women with symptoms of faecal incontinence following their first pregnancy will deteriorate after their second pregnancy.[4] In one study 26.9% of women had an identifiable occult anal sphincter injury on endoanal ultrasound. With each increase in parity the chance of a new defect increased by 8.5%, while 75-83% of women presenting with faecal incontinence will have an identifiable anal sphincter defect.[5] Follow up of women who underwent primary repair of a third degree vaginal tear has shown that 85% will have persistent structural defects and over 50% of this group will develop faecal incontinence.[6-10]

A wide range of causes may contribute to anorectal dysfunction and anal incontinence.[7] Some iatrogenic causes that may result in injury to the anal sphincter complex include anorectal procedures such as fistulotomy, sphincterotomy and haemorrhoidectomy. Medical etiological factors include psychiatric conditions, chronic constipation, malabsorption syndromes, laxative overuse, diabetes, thyroid disease, gastrointestinal inflammatory conditions, neuropathies and spinal cord disease.

Anatomical distortion associated with chronic straining is commonly known as pelvic floor prolapse syndrome and may also be associated with progressive denervation of the colon, rectum and anus at the pelvic floor, in conjunction with obstructive defaecation and intussusception. Lateral, central and posterior rectal defects including rectal diverticulum may also contribute to anorectal dysfunction with incontinence.

It is difficult to know the true incidence of faecal incontinence due to variations in definitions, age distribution, underreporting due to reluctance to broach the condition, coexistence with other pelvic symptoms such as voiding dysfunction and pelvic organ prolapse.[11] A complex, interactive psychological process is necessary for faecal/flatal continence including rectal motility and compliance with neurophysiology of sensation. Hyposensitivity associated with low resting pressure may indicate internal and external sphincter damage resulting in passive incontinence, whereas hypersensitivity with urge incontinence and the inability to postpone defaecation may be associated with low voluntary squeeze pressure thus indicating external anal sphincter malfunction.

The aim of this study was to explore the possibility that a circumferential mesh support for the anal sphincter would reduce symptoms in patients with faecal or flatal incontinence. This hypothesis arose from consideration of the need for additional anchoring support of the Apogee™ (American Medical Systems, Inc, Minnetonka, MN, USA) mesh used for the repair of posterior vaginal wall prolapse (rectocele and associated perineal repair). It was found that a percentage of these patients reported a significant improvement in bowel function and in particular a reduction in severity of their previously underreported anal incontinence.[12]

The use of mid urethral slings to treat Stress Urinary Incontinence (SUI) is based on the Hammock Hypothesis of DeLancey.[13] Since the anatomy [14, 15] and pathophysiology of defects arising in the anterior pelvic compartment have been successfully corrected using a mid urethral sling to treat SUI it was postulated that a mesh sling prosthesis placed around the anal sphincter may improve the anal sphincter mechanism of action and address the problem of faecal and flatal incontinence. It was decided to embark on a pilot study of patients with mild faecal incontinence using such a mesh prosthesis and to monitor the outcomes of this group.

ANORECTAL ANATOMY

The anal canal extends from the anorectal junction which lies just above the level of the puborectalis sling and sphincters (internal and external) to the anus below. It is surrounded by an external sphincter of voluntary muscle fibres and an internal sphincter of involuntary muscle fibres.[16] Between the two sphincters lies the longitudinal anal muscle of the rectum (LAM) [17] composed of muscle fibres which receive contributions from puborectalis, pubococcygeous

and iliococcygeous.[18] Recently Petros described that the LAM fibres contributed by the levator plate are incorporated into the sphincteric action as described by the integral theory.[19] This is probably important in closure and relaxation of the anal canal and that of the urethra. Individuals show significant variations in detail of the sphincter anatomy.[16]

The external anal sphincter is a voluntary muscle, cylindrical in form being approximately 2 cm deep placed around the anal canal for approximately 5-7 cm. It has three parts. The subcutaneous part is slender and encircles the anal orifice. The superficial part supports the anus in an ovoid circular fashion and extends from the tip of the coccyx and anorectal raphe to the perineal body in the median plane. The deep part encircles the anal canal with some fibres joining the superficial transverse perineal muscle and blends with the puborectalis muscle of the levator ani. The internal sphincter surrounds the superior two thirds of the anal canal and is supported by the levator ani. The inferior two thirds are surrounded by the external sphincter as described above. The longitudinal muscle is comprised of three layers and lies between the internal and external sphincters.[17]

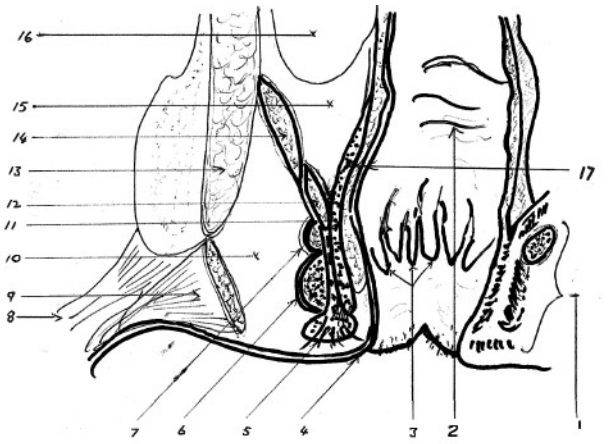

Fig. 1. – Anorectal Anatomy. 1. External anal sphincter. 2. Transverse rectal folds; rectal ampulla. 3. Anal sinuses. 4. Skin. 5. Subcutaneous part of external anal sphincter. 6. Superficial part of external anal sphincter. 7. Deep part of external anal sphincter and pubo rectalis. 8. Semi tendinosus. 9. Gluteous maximus. 10. Ischio rectal fossa. 11. The longitudinal muscle coat. 12. Pubo coccygeous. 13. Obturator internus. 14. Iliococcygeous. 15. Pelvi-rectal space. 16. Para rectal fossa. 17. Internal circular muscle coat/ internal ano rectal sphincter.

PATIENTS AND METHODS

The ASSP was performed on 14 patients between June 2005 and March 2007 with a total follow up time of 20.3 months. Mean follow up time was 18 months. Inclusion criteria for the study were symptoms of mild to moderate faecal soiling and flatal incontinence. Ten patients had a Wexner grading symptom score of 5-8 and four patients had a Wexner grading symptom score of 8-10. Past gynaecological and colorectal surgeries were as follows: Four patients had a past history of vaginal hysterectomy and anterior pelvic organ prolapse repair, six had abdominal hysterectomy (two also Burch colposuspension), four anterior and posterior vaginal repair, two had a haemorrhoidectomy, one an partial sphincterotomy for anal fissure, one an anal stretch procedure. The ages of patients ranged from 45 to 78 years, mean age 64.7 years. Obstetric history: all patients were parous. Five patients were parous 1-3, nine patients 3-5. Two patients had caesarian sections only. Interestingly both these patients had a past history of anorectal surgery. Of the 12 patients who had vaginal births more than 33% had instrumental deliveries. Mean foetal size was 3,700 g, ranging between 4,300 to 2,800 g.

All patients were studied using Magnetic Resonance Imaging (MRI) of the anal sphincter. At imaging 5 patients had evidence of external sphincter defects mainly right, lateral or central. Defects were considered small varying from 2-3 mm. Two patients aged 75 and 78 years had evidence of mild atrophy of the external anal sphincter while 50% of patients had no obvious anal sphincter defects on MRI. All patients were assessed pre and post operatively with resting anorectal pressures and maximal squeeze pressure. Six patients had a concomitant posterior organ prolapse procedure using an Apogee™ (American Medical Systems, Inc, Minnetonka MN, USA) mesh. Patients were assessed using the POPQ system.[20]

Operative technique: All patients had a 24 hour preoperative bowel preparation. Systemic triple antibiotics were given intraoperatively and this combination was continued for 24 hours postoperatively. Clindamycin cream was inserted vaginally as a single dose approximately 1-2 hours preoperatively. The operation was performed in the lithotomy position. Aqueous Betadine antiseptic solution (10% w/v Povidone/Iodine) was used as skin preparation and liberally applied to inner thighs, intravaginally and to the perineal and suprapubic areas. The patient was draped in the usual fashion facilitating good exposure of the operative site with the buttocks extending just below the end of the table. Infiltration of the perianal area and perineal body including the posterior vaginal mucosa was performed using a hydrodissection technique with dilute local anaesthetic. A stab incision of approximately 1 cm using an eleven blade was made 3 cm from the anal verge in the 5 o'clock and 7 o'clock position or a single incision was made at 6 o'clock (Fig. 4, 5).

At the vestibule of the vaginal orifice a transverse incision of approximately 4-5 cm was performed with dissection supramedially and laterally to expose the superior aspect of the perineal body. In the case of an adjuvant posterior prolapse procedure, blunt and minimal sharp tunneling dissection is continued proximally to create a pathway and identify the ischial spine and tendinous arch of the lateral pelvic wall.[21] This dissection is performed bilaterally (Fig. 2, 3).

Gentamycin solution 1 mg per 1cc of saline is used to liberally irrigate the operative site throughout the procedure.

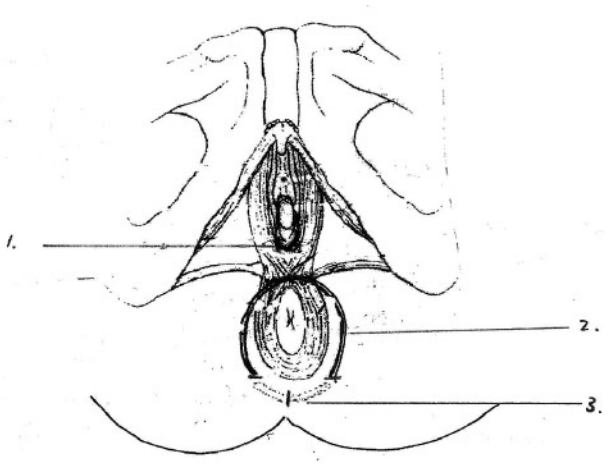

Fig. 2. – Placement of ASSP Monarc™ tape. 1. Incision over the perineal body at the hymenal ridge to expose the perineal body. 2. Anal sphincter support prosthesis, Monarc ™ in situ / surrounding the superficial part of the external anal sphincter. 3. Alternative incision site for placement of the Monarc ™ tape.

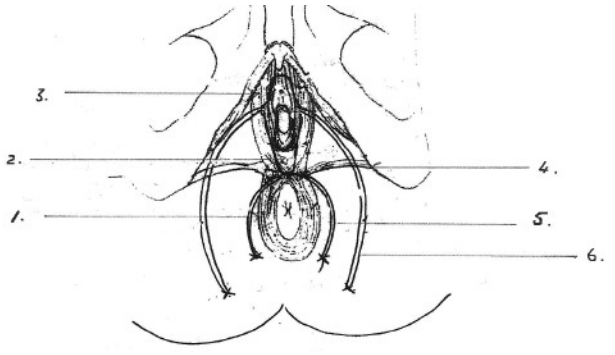

Fig. 3. – Position of Monarc™ and Apogee™ prosthesis when ASSP is performed together with a prolapse repair using the Apogee™ system. 1. External anal sphincter. 2. Perineal body. 3. Pubo rectalis and pubo vaginalis. 4. Superficial transverse perineal muscle. 5. Anal sphincter support tape (Monarc™) in situ. 6. Posterior pelvic compartment support prosthesis (Apogee™) in situ.

The recommended entry site for the Apogee™ (American Medical Systems, Inc, Minnetonka MN, USA) needles is 5-6 cm posterolateral to the anal verge which is 3 cm posterolateral to the incisions used for the ASSP. The posterior compartment mesh repair Apogee™ should be performed before the perianal support procedure. The Apogee™ mesh must be in position and the excess inferior cape trimmed approximately 1cm above the transverse perineal incision before the positioning of the ASSP is performed. The stab incisions are performed as explained above and with the index finger of a double gloved left hand the helical needle of a Monarc™ (American Medical Systems, Inc, Minnetonka MN, USA) prosthesis is inserted into the incision and directed approximately 2-3 cm in depth before rotation of the needle through its natural arc creates a track around the external anal sphincter to emerge laterally through the perineal body enabling visualization of the needle tip at the perineal incision. The same technique is performed on the contra lateral side of the external anal sphincter. With the tips of both needles at the perineal body, the Monarc™ sling is attached to both needle tips firmly with an audible click. The mesh is then gently positioned posteriorly through the track made by the helical needles emerging through the perianal incisions posterior to the anal verge. Gentle traction is used on the mesh contralaterally until it is seen and felt to sit undistorted and without tension over the perineal body. The needles are then detached. The plastic sheaths are removed and the mesh trimmed at the buttock incisions (Fig. 6). All incisions are then closed with 2-0 Vicryl interrupted mattress sutures.

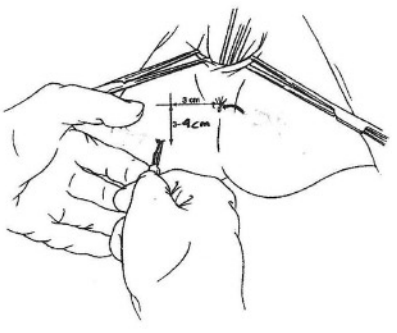

Fig. 4.

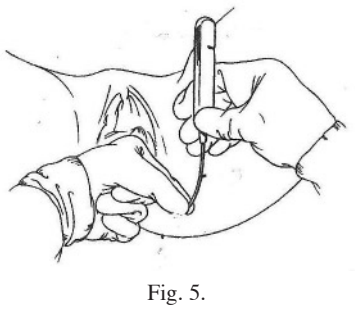

Fig. 5.

RESULTS

All 14 patients were studied over a minimum period of 6 months. Mean average time of follow up was 18 months. During that time there were no perioperative or postoperative complications. In particular there were no cases of mesh erosion or rejection and no cases of rectal perforation or trauma. Four cases had evidence of perianal bruising which resolved spontaneously with the judicious use of ice packs applied to the perianal area for approximately 24 hours. There were no cases of overt haematoma formation, infection or abscess formation. Ten patients whose initial Wexner score [22] was between 5 and 8 were reassessed to have between 90 and 100% improvement in symptoms. The other 4 patients with initial Wexner Score of 8-10 noted an improvement of 70% or better resulting in a Wexner score between 2 and 3.

CONCLUSIONS

The purpose of this study was primarily to evaluate the safety and efficacy of the ASSP and its impact on quality of life with respect to anorectal dysfunction and faecal incontinence. The ASSP was found to be a simple procedure with reproducible results and not associated with any significant peri or post operative complications within a mean follow up period of 18 months. In particular there was no evidence of mesh erosion, rejection or anorectal trauma. Subjective improvement of symptoms was noted using the Wexner grading profile and quality of life was maintained in all patients and improved in more than 90%. As a consequence of this pilot study it will be important to consider longer term prospective randomized controlled studies, comparing outcomes of other techniques for treating faecal incontinence with ASSP. The use of ASSP in conjunction with other concomitant procedures for posterior pelvic organ prolapse and dysfunction is efficacious and safe. This study suggests that there is a significant enhancement when used in conjunction with a posterior vaginal mesh procedure using the Apogee™. Further studies in mesh design and in particular a wider dimension in the mesh prosthesis may be desirable. Further research into the role of pelvic floor imaging will increase our knowledge into the pathogenesis and management of functional pelvic floor disorders.[23] There is also a need for standardized terminology and diagnostic criteria for defaecatory dysfunction and for a comprehensive classification of site specific anatomical variations of the posterior pelvic compartment. This will also need to be addressed from a multidisciplinary perspective as recently proposed by Farnsworth and Dodi.[24]

REFERENCES

1. Arramorowitz L, Sobhani I, Ganansia R, et al. Are sphincter defects the cause of anal incontinence after vaginal delivery? Results of a prospective study. Diseases of the colon and rectum 2000; 43: 590-596.
2. Donnelly V, Fynes M, Campbell D, et al. Obstetric events leading to anal sphincter damage. Obstetrics & Gynaecology 1998; 92: 955-961.

3. Kamm MA. Obstetric damage and faecal incontinence. Lancet 1994; 344: 730-733.
4. Fynes M, Donnelly V, Beham M, et al. Effects of second vaginal delivery on ano-rectal physiology and faecal incontinence: Prospective study. Lancet. 1999; 354: 983-986.
5. Varma A, Gunn J, Gardinera A, et al. Obstetric anal sphincter injury: prospective evaluation of incidence. Diseases of colon and rectum. 1999; 42: 1537-1542.
6. Faltin DC, Sangalli MR, Curtin F, et al. Prevalence of anal incontinence and other ano rectal symptoms in women. International Urogynaecology Journal 2001; 12: 117-120.
7. Kiff ES, Barnes PR, Swash M. Evidence of pudendal neuropathy in patients with perineal descent and chronic straining at stool. GUT 1984; 25: 1279-1282.
8. Oberwalder M, Conner J, Wexner SD. Meta analysis to determine the incidence of obstetric anal sphincter damage. British Journal of Surgery 2003; 90: 1333-1337.
9. Poen AL, Felt-Bersma RJ, Strijers RL, et al. Third degree obstetric perineal tear: long term clinical and functional results after primary repair. British Journal of Surgery. 1998; 85: 1433-1438.
10. Gjessing H, Racke B, Sahlin Y. Third degree obstetric tears: outcome after primary repair. Acta Obstetricia and Gynecologica Scandanavia 1998; 77: 736-740.
11. Ench P, Bielefeldt K, Raschmann W, et al. Epidemiology of faecal incontinence in selected patient groups. Int Urogyn J 1991; 6: 143-146.
12. Reid R. Recto-enterocele repair - past problems and new horizons (review). Pelviperineology 2007; 26: 9-16.
13. De Lancey JOL. Structural supports of the urethra as it related to stress urinary incontinence: The Hammock Hypothesis. Am J Obstet Gynec 1994; 170: 1713-1723.
14. Versi E, Cardozo LD, Studd JWW, Brincatt M, O'Dowell DJ. Internal urinary sphincter in maintenance of urinary continence. BMJ 1986; 292: 166-167.
15. Zaccharin RF. The anatomical supports of the female urethra. Obstets Gynaecology. 1969; 33: 754-759.
16. Last's Anatomy. Regional and applied. McMinn RMH. Churchill and Livingstone. Edinburgh, London, Melbourne, New York. 1990; 401-406.
17. Macchi V, Porzionato A, Stecco C, Benettazzo F, Stecco A, Portenti A, Dodi G, De Caro R. Histo-topographic study of the longitudinal anal muscle. Pelviperineology 2007; 26: 30-32.
18. Shafik A. A new concept of the anatomy of the anal sphincter mechanism and physiology of defaecation. XL11. Rectopuborectalis reflex. Coloproctology 1990; 12: 170-172.
19. Petros P. The internal continence society and integral theory systems for the management of the incontinent female - a comparative analysis. Pelviperineology 2007; 26: 25-29.
20. Bump RC, Matthiasson A, Bo K, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol 1996; 175: 10-17.
21. Davila GW, Beyer R, Moore R, DeRio S. Restoration of the vaginal apical and posterior support with Apogee system (Abstract). Int Urologynecology 2005; 16 (Supplement 2): 5118.
22. Jorge JM, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum 1993; 36: 77-97.
23. Stoker J, Halligan S, Bartram C. State of the art colon pelvic floor imaging. Radiology 2001; 218: 621-641.
24. Farnsworth B, Dodi G. Short IPGH system for assessment of pelvic floor disease. Pelviperineology 2007; 26: 73-77.

Competing Interests: None

Acknowledgements:
The author acknowledges Greg Gurr for the line drawings.

Correspondence to:
Dr. MAXWELL E. HAVERFIELD
Senior Consultant,
Department of Obstetrics and Gynaecology
The Northern Hospital - 185 Cooper Street,
Epping, Victoria. Australia 3076.
Email: max.haverfield@nh.org.au

Pelvic Floor Digest

continued from page 107

Prolapse repair by vaginal route using a new protected low-weight polypropylene mesh: 1-year functional and anatomical outcome in a prospective multicentre study. *de Tayrac R, Devoldere G, Renaudie J et al. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:251.* A low-weight polypropylene mesh coated with a hydrophilic absorbable film for vaginal repair of genital prolapse (anterior, posterior and anterior-posterior repair) seems to decrease local morbidity (vaginal erosions 6.3%, de novo dyspareunia 12.8%) while maintaining low recurrence rates (6.8% for cystocele and 2.6% for rectocele). The report is based on the analysis of the first 143 patients of a multicentre study evaluated after at least 10 months follow-up. The improvement of Pelvic Floor Distress Inventory and Pelvic Floor Impact Questionnaire were highly significant.

Changes in the extracellular matrix in the anterior vagina of women with or without prolapse. *Lin SY, Tee YT, Ng SC et al. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:43.* To investigate the changes in the connective tissues (collagen type I, III, IV, V, VI, elastin, and glycoproteins) located in the upper portion of the anterior vaginal wall associated with prolapse, in 23 women with prolapse an immunohistochemical study demonstrated that collagen III is significantly less than in a control group with a positive correlations with ageing.

Vaginal mesh erosion after transvaginal repair of cystocele using Gynemesh or Gynemesh-Soft in 138 women: a comparative study. *Deffieux X, de Tayrac R, Huel C et al. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:73.* In 138 women follow-up for 7-60 months cystocele repair was performed according to the technique of tension-free polypropylene mesh. Anatomically, the success rate was 95%. Vaginal erosion was reported in 20% of the patients with no statistically significant difference between Gynemesh and Gynemesh-Soft meshes. Cystocele stage >2 HWS is a protective factor against vaginal erosion. A partial excision of the mesh was necessary in 13/27 symptomatic patients (48%), associated with a vaginal mucosal closure, 2/27 underwent a complete excision. The incidence of de novo dyspareunia was 9% in patients with vaginal erosion and 11% in patient without erosion.

A prospective, randomised, controlled trial comparing 3 hour and 24 hour postoperative removal of bladder catheter and vaginal pack following vaginal prolapse surgery. *Glavind K, Morup L, Madsen H, Glavind J. Acta Obstet Gynecol Scand. 2007;86:1122.* The aim of this prospective randomised study was to determine whether or not there was a higher incidence of bleeding, reoperation, urinary retention or bacterial count in the urine depending on whether urinary catheter and vaginal pack were removed 3 or 24 h after prolapse surgery. Pack and catheter removal after 3 h is recommended with careful monitoring of the patient's voiding.

Robot-assisted vs. conventional laparoscopic rectopexy for rectal prolapse: a comparative study on costs and time. *Heemskerk J, de Hoog DE, van Gemert WG et al. Dis Colon Rectum. 2007 Aug 10; epub.* Robot-assisted laparoscopic rectopexy is a safe and feasible procedure, but results in increased time and higher costs than conventional laparoscopy.

Stapled hemorrhoidopexy and Milligan Morgan hemorrhoidectomy in the cure of fourth-degree hemorrhoids: long-term evaluation and clinical results. *Mattana C, Coco C, Manno A et al. Dis Colon Rectum. 2007 Aug 16; epub.* Long follow-up seems to indicate more favorable results in Milligan-Morgan procedure compared to stapled hemorrhoidopexy (mean follow-up 92 and 54 months respectively) in terms of resumption of symptoms and risk of recurrence.

The PFD continues on page 112