We are IntechOpen,
the world's leading publisher of
Open Access books
Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS
BOOK CITATION INDEX
INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Anal Incontinence as a Complication of Vaginal Delivery and Anorectal Surgery

Małgorzata Kołodziejczak and
Iwona Sudoł-Szopińska

Additional information is available at the end of the chapter

1. Introduction

Incontinence is a complex term and encompasses a broad spectrum of disorders with various intensities. They range from gas incontinence, through incontinence of liquid stools (soiling), to incontinence of solid stools.

The factors determining the function of faecal continence comprise: structural elements such as the anal sphincter muscles and pelvic floor muscles, including the Parks angle (between the long axis of the rectum and that of the anal canal), as well as functional elements such as central nervous system, anorectal sensation, volume and compliance of the rectum, stool consistency, high-pressure zone and abdominal pressure.

Faecal incontinence affects 2-7% of the adult population. The range of the problem is not exactly known due to its embarrassing nature and reluctance to report stool and/or gas incontinence to physicians [1, 2, 3]. The majority of patients are women. It has been reported that 1 on 10 women suffers from various types of incontinence [4], the most common cause being obstetric trauma (60%). The second most common cause is iatrogenic injury to the anal sphincter muscles sustained during anorectal surgeries (approximately 16%) [5, 6]. More and more often, incontinence is caused by injuries that occur after inserting foreign bodies to the rectum and after traffic injuries. The number of patients with inflammatory bowel diseases is rising as well. Neurogenic incontinence is also a contributor [7]. This chapter discusses two groups of causes: obstetric and those occurring after anorectal surgery.

2. Obstetric anal sphincter defects

Obstetric trauma is the most common cause of faecal incontinence in women. Symptoms of incontinence that persist for more than 6 months after the delivery are reported by ¼ of women. The number is even higher in primiparas [8].

Risk factors for post-partum faecal incontinence may generally be divided into those dependent on the mother and the course of the labour itself, and those related to the child. The most significant risk factors related to the mother and the course of labour are: occurrence of a third- or fourth-degree perineal tear, instrumental delivery, primiparity, age of the mother, number of previous deliveries, duration of the second stage of labour, application of epidural anaesthesia, oxytocin in the second stage of labour and breech delivery [9, 10]. The most commonly identified child-related risk factors include an excessively heavy infant and large head circumference. A strong correlation between fetal macrosomia and anal sphincter defects of the mother as well as injuries during labour was confirmed by Gumundson et al. [11]. Amongst all these aforementioned risk factors, the most significant is the occurrence of either a third- or fourth-degree perineal tear.

There are several classifications of obstetric anal sphincter injuries. One of them distinguishes evident and occult anal sphincter defects on clinical examination. Evident obstetric injuries of the anal sphincters are diagnosed by an obstetrician during suturing the perineum. They encompass third- and fourth-degree perineal tears, i.e. those which involve the anal sphincters (third-degree defects) or in addition, encompass the rectal mucus membrane (four-degree defects).

An additional division of third-degree perineal tears [12] includes the size of sphincteric defects i.e.:

- First degree. The rupture encompasses less than 50% of the thickness of the external anal sphincter.

- Second degree. The rupture encompasses more than 50% of the thickness of the external anal sphincter.

- Third degree. The rupture encompasses the external and internal anal sphincters.

Occult obstetric defects constitute cases that are not diagnosed directly after the delivery when the perineum is sutured. These defects are not usually accompanied by changes in the perineal region, which is the main reason for the failure to diagnose them by an obstetrician. In our own research, the percentage of detected defects after natural delivery reached 3.9% [3].The symptomatic presentation of sphincteric insufficiency connected with occult defects is usually delayed, sometimes up to a dozen years after the delivery. This is probably because such injuries are less extensive than in evident cases.

Another classification of obstetric defects is their division into structural (or mechanical) injuries and neurogenic incontinence.

3. Obstetric mechanical (structural) defects

Mechanical injuries of the anal sphincters encompass the complete or incomplete disruption of the external or internal anal sphincters (Figure 1 a, b).
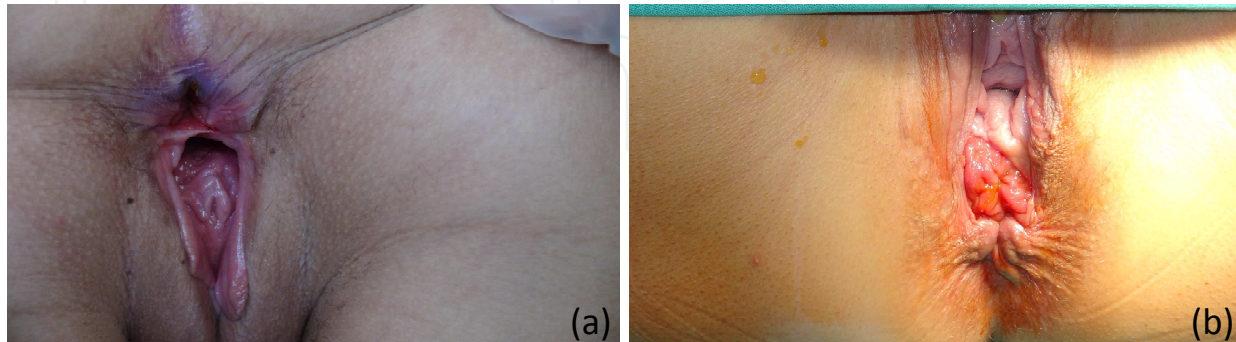
Figure 1. a) An obstetric tear of anal sphincters; b) an obstetric tear of anal sphincters with a rupture of the rectovaginal septum

The anatomy of the anal sphincters in women predisposes them to such injuries. In men the entire length of the anal anterior wall in made up of two muscle layers corresponding to internal and external sphincters. In contrast, in the majority of women, the external sphincter is not present in the superior aspect of the anal canal on the anterior wall. Traumatic vaginal deliveries occur particularly frequently in women in whom the distance between the fourchette and the anal canal is short.

Mechanical injuries of the sphincters are not always accompanied by incontinence. In younger women, obstetric injuries may be compensated by the function of the remaining, retained mass of the sphincters, mainly by the puborectalis muscle. By creating the, so-called, Parks angle between the anus and rectum, this is a crucial muscle responsible for continence [13]. Symptoms of sphincteric insufficiency may eventually develop in the menopausal period when the pelvic floor muscles weaken, pudendal nerve neuropathy occurs.

4. Obstetric neurogenic incontinence

Neurogenic injuries are primarily occur during delivery of a large fetus. A prolonged second stage of labour lasting more than 60 minutes results in prolonged compression of the pudendal nerves by the fetal head (the nerve is pressed against the ischial spine). The nerve may also become irreversibly damaged when it is exposed to pressures of more than 80 mmHg for more than 8 hours [14]. Prolonged bearing down may stretch the pelvic floor structures, including the pudendal nerves. This disruption may involve branches of the spinal roots in the sacral spine S3-4 which run on the internal aspect of the pelvic floor and innervate, among others, the puborectalis muscle.

The second stage of labour, lasting from full cervical dilation to the delivery, is considered the most traumatic for the pelvic floor muscles, including the sphincters [14]. In this period, the muscles and tissues of the pelvic floor are subject to the greatest pressure, perineal descent is observed and directly after the delivery, transitory changes in the muscle tone may occur [15]. As many as 80% of women develop transient denervation of the pelvic floor [16,17]. Muscle fibres become mechanically damaged when they are stretched by more than 50% and the perineal nerve fibres undergo damage as a result of their stretching by 6-22% [18].

In prolonged labour, the pudendal nerve, which is compressed by the fetal head against the ischial spine, is the most susceptible to injuries. Snooks et al. [19] found evidence of prolonged latency of the pudendal nerve in as many as 48% of women within 48-72 hours following delivery. This was interpreted as a direct proof of injury. Damage to the pudendal nerve may cause not only faecal incontinence, but also urinary incontinence and sexual dysfunction. Obstetric injuries may also be mixed with both structural defects features of pudendal nerve neuropathy.

5. Anal sphincter defects following anorectal surgeries

Faecal incontinence may appear even after the simplest anal surgery. It may occur after haemorrhoidectomy as well as surgeries for anal fissures or fistulae.

5.1. Incontinence after haemorrhoidectomy

Classical haemorrhoidectomy may result in the inability to discriminate between the contents of the rectum with resulting incontinence. More rarely sphincteric defects can occur. Inability to discriminate between the nature of the rectal contents (anal sensation failure) results from too extensive excision of the mucus membrane that is rich in sensory receptors.

The receptors that differentiate between solid and liquid stools as well as gases are localised in the transitional zone which constitutes an area of approximately 1.5 cm in length situated over the dental line.

Pudendal nerve damage can also result in faecal urgency. This can occur after too extensive excision of haemorrhoids in the commonly employed Milligan-Morgan procedure. It may also occur following Longo's stapled haemorrhoidectomy if the stapler is placed too low, inadvertently resecting the entire transitional zone.

Haemorrhoidectomy may also result in mechanical injury of the sphincters, particularly the internal sphincter. "Post PPH syndrome" has been reported when the staplers are placed slightly low, possibly causing the internal sphincter muscle to become inflamed.

5.2. Incontinence after anal fissure surgery

One of the main reasons for the appearance of anal fissure is increased tension with subsequent hypertrophy of the internal anal sphincter. Anorectal manometry has identified a considerably

higher mean maximum resting pressure in the anal canal (reflecting mainly internal sphincter function) in patients with anal fissures as compared with controls [20].

Anal fissure surgery always requires cutting the sphincter responsible for the continence of gases and liquid stools. It has been estimated that faecal incontinence occurs after anal fissure surgery in 3-38% patients [21]. The surgical treatment of anal fissures includes the following methods: open and closed lateral sphincterotomy, open posterior sphincterotomy without excision of the fissure and open posterior sphincterotomy with excision of the fissure. Open lateral sphincterotomy is the gold standard for the management of chronic anal fissures [22]. The proportion developing postoperative incontinence when using this technique ranges from 0-16% [23, 24]. The surgical treatment of chronic or recurring fissures may involve techniques based on mucosal advancement flap anoplasty, anal advancement flap or anodermal flap [25, 26].

The most recent modification of lateral sphincterotomy is segmental internal sphincterotomy [27]. It appears that this type of surgery does not carry the risk of incontinence [27]. Another surgical treatment involves anal dilation with the use of either a Parks' anal retractor that is spread to 4.8 cm, or a rectosigmoid balloon 40 mm in diameter and 60 mm in length [28]. The effectiveness of this method is comparable to classical lateral sphincterotomy (83.3% and 92% respectively). Following 24 months of observation, no complications in the form of incontinence were observed in a group treated by means of the dilator [29].

Open posterior sphincterotomy, apart from the consequences resulting from cutting the internal sphincter, may also cause a "keyhole deformity" of the anal canal, resulting in anal leakage and soiling of the underwear.

Patients identified with increased pressure in the anal canal have been treated with botulinum toxin administered intersphincterically. Worsening of continence was not observed over a 24 month observation period, and only 8% of patients suffered a relapse [26].

5.3. Incontinence after anal fistula surgeries

Anal fistula surgery nearly always results in the controlled severing of anal sphincter muscles. The risk of incontinence increases with so-called height of the fistula (the length of the external anal sphincter involved by the fistula), its localisation in relation to the wall of the anal canal (anterior fistulae in women), recurring fistulae and coexistence of a neurogenic component in addition to the morphological defect.

The most common cause of incontinence after anal fistula surgery is from extensive damage of the sphincter muscles sustained during the course of the procedure (Figure 2).

Reasons for sphincteric damage include:

• overly aggressive surgical techniques in patients whose sphincteric function was impaired before surgery;

• incorrect intraoperative assessment of the type of fistula and inappropriate surgical method;

Figure 2. Iatrogenic anal sphincter injury following fistulectomy

- setons kept in the wound for too long, resulting in fibrosis of the sphincter muscles and worsening gas and stool continence;

- anal canal deformation (keyhole deformity), which often appears following the Hippocratic method of treatment and may lead to faecal soiling;- bilateral damage to the inferior anal nerves that innervate the external anal sphincter in the surgery of horseshoe abscesses or fistulae (a rare complication).

Patients with fistulae and abscesses accompanying non-specific inflammatory bowel disease can be particularly difficult cases.

The proportion developing postoperative incontinence depends on the surgical technique. Following fistulotomy, worsening of continence is observed in as many as 45% of patients [30, 31] and following fistulotomy with sphincter reconstruction - from 4 to 32% of cases [32, 33, 34]. Incontinence is hardly ever observed after direct closure of the internal fistula opening.

Treatment of the internal orifice of the fistula by endorectal advancement flap results in postoperative incontinence in 13.2% of cases [35]. Methods involving anal fistula plugs or fibrin glue introduced to the fistula canal do not cause gas and stool incontinence, but a high percentage of relapse is observed [36]. Cutting seton fistulotomy is not currently recommended since the rate of continence disorders after this procedure reaches 90% [37, 38].

The risk of incontinence resulting from surgical treatment of fistulae can be minimised by assessing sphincteric sufficiency by means of anorectal manometry. It is also important to identify the position of the fistula in relation to the sphincters by means of supplementary examinations such as anorectal endosonography or MRI.

6. Diagnosis and treatment of obstetric and postoperative anal sphincter defects

The general procedures performed in obstetric and postoperative incontinence are similar. All patients after anorectal surgeries and all women after third- or fourth-degree perineal tear

should undergo a rectal examination and endosonographic scan. Additionally, in the case of faecal incontinence symptoms, functional tests should also be performed. Repairing obstetric sphincter defects should be treated in the same way as repair surgeries of any other types of sphincteric damage since improper suturing of the muscles with ongoing functional failure by the sutured muscles may result in permanent disablement. The costs of treatment of remote complications are higher than the costs of preventive diagnostic examinations.

6.1. Diagnosis of obstetric and postoperative anal sphincter defects

Diagnosis of incontinence requires doctor-patient interview, anorectal examination, functional tests and imaging examinations particularly endosonography and magnetic resonance imaging.

Interview

The interview and physical rectal examinations are the basic methods for diagnosing and formulating treatment of incontinence.

Patients should be asked about the presence of underwear soiling, uncontrollable passage of gasses or stools as well as about the nature of such symptoms (temporary or permanent) and the frequency with which they occur. Female patients should be asked about their deliveries (instrumental delivery, episiotomy, fetal size, duration of labour etc.). It is important to find out whether incontinence is related to other factors (e.g. physical effort or stress), if there has been previous anorectal surgery or whether there are coexistant diseases that might affect continence such as neurological conditions, lumbosacral discopathies or inflammatory bowel diseases. An objective assessment of the symptoms can be provided through various scoring systems for the evaluation of anal sphincteric insufficiency which also include subjective elements (e.g. Wexner score) [39]. Quality of Life tests are also available (e.g. Fecal Incontinence Quality of Life) [40].

6.1.1. Anorectal examination

Visual examination of the anal region enables identification of post-surgical scarring, anal deformities and episiotomy scars.

During digital anorectal examination, one should assess passive and active tone of the sphincters and content of the anus. It is also possible to determine the site of the external sphincter defect by asking the patient to contract the sphincters. Nevertheless, some studies report that the percentage of sphincteric defects detectable by means of a clinical examination does not exceed 50% [41].

6.1.2. Functional test

From the point of view of possible surgical treatment, the most useful supplementary exami‐ nation is anorectal manometry. This allows determination of anorectal function by measuring resting and squeeze pressure in the anal canal as well as other significant parameters such as: cough reflex, manometric changes during attempted defecation, rectoanal inhibitory reflex,

rectal sensation, high pressure zone length, functional sphincter length, and basic rectosphincteric reflexes for instance, rectoanal inhibitory reflex (RAIR).

Anorectal manometry can be particularly useful when surgeries, e.g. of anal fistulae [42], are planned and before sphincter repair procedures. The effectiveness of sphincteric repair surgeries is not always satisfactory and functional documentation of the sphincters from before the procedure is crucial not only for medical but also legal reasons.

Another useful functional test is electromyography. This determines the myoelectrical activity of the sphincters when resting, during contraction and during faecal urgency. The examination can be performed invasively with the use of thin concentric needle electrodes placed in the lateral fragments of the sphincter around and parallel to the anal canal. It can also be performed in a less invasive manner with the use of superficial cutaneous electromyography and for instance a 16-channel probe [43]. Other functional examinations are of less importance.

Pelvic floor descent may be visualised by means of conventional defecography as well as in dynamic MRI. It provides a comprehensive evaluation of the lateral and anterior aspects of the pelvic floor while resting and during urgency and involves the introduction of water, gel or solution with contrast agent (depending on the experience of the centre) into the lumen of the bladder, vagina or rectum (dynamic MR colpocystorectography). This examination enables assessment of pelvic floor motility.

6.1.3. Imaging examinations

The imaging examination of choice in patients with incontinence symptoms is anal endosonography (AES). The primary aim of AES is to differentiate between the causes of incontinence: whether there is evidence of morphological injury of the sphincters as might be expected in a tear or whether the features are more consistent with a neurogenic cause. In the case of neurogenic incontinence, the sphincters may appear normal or atrophic, and during attempted sphincter contraction as part of a dynamic AES there is no evidence of contraction of the external sphincter and puborectalis muscle. This examination should be performed prior to planned anal and rectal surgeries in all patients who manifest symptoms of incontinence (in order to minimise further impairment of sphincteric function) as well as in patients who have already undergone anorectal surgeries but have postoperative incontinence, in order to explain its cause (Figure 3 a, b).

In the case of women who sustain labour-induced third- or fourth-degree injury to the perineum, AES is important to perform because of the asymptomatic character of numerous injuries which may be compensated by the undamaged retained portion of the sphincters (Figure 4 a, b).

If defects are detected, one should determine:

- which muscle has been damaged (internal, external anal sphincter or puborectalis muscle);
- the range of damage (circumference and length in relation to anal canal levels);
- the presentation of the retained sphincters (muscle stumps).
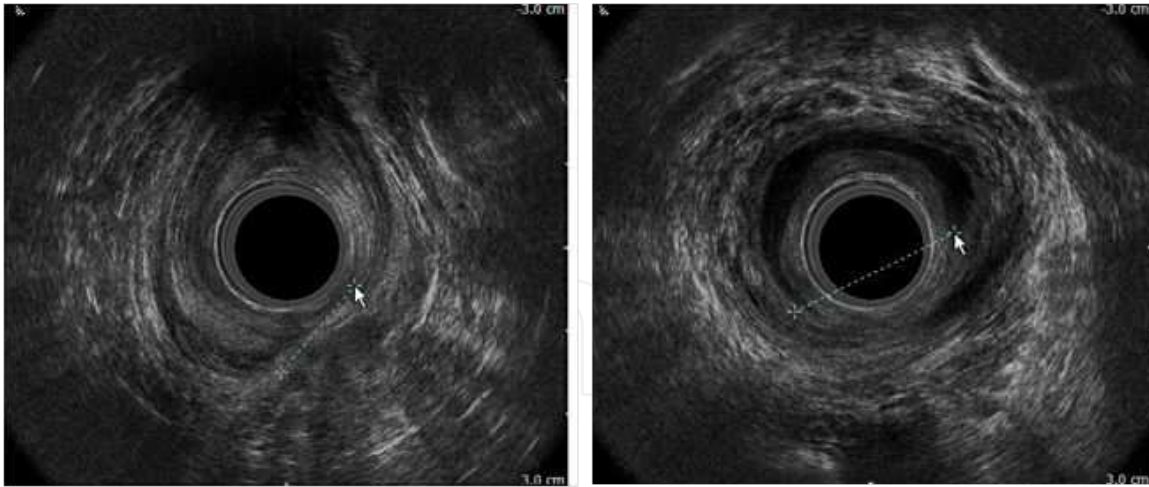
Figure 3. External and internal anal sphincter tears following fistulectomy due to posterior transsphincteric fistula surgery, affecting: a) in a distal part of the anal canal 30% of the posterior-right circumference of the external anal sphincter; b) in mid anal level 50% of the posterior aspect of the internal anal sphincters. Muscle stumps are normal
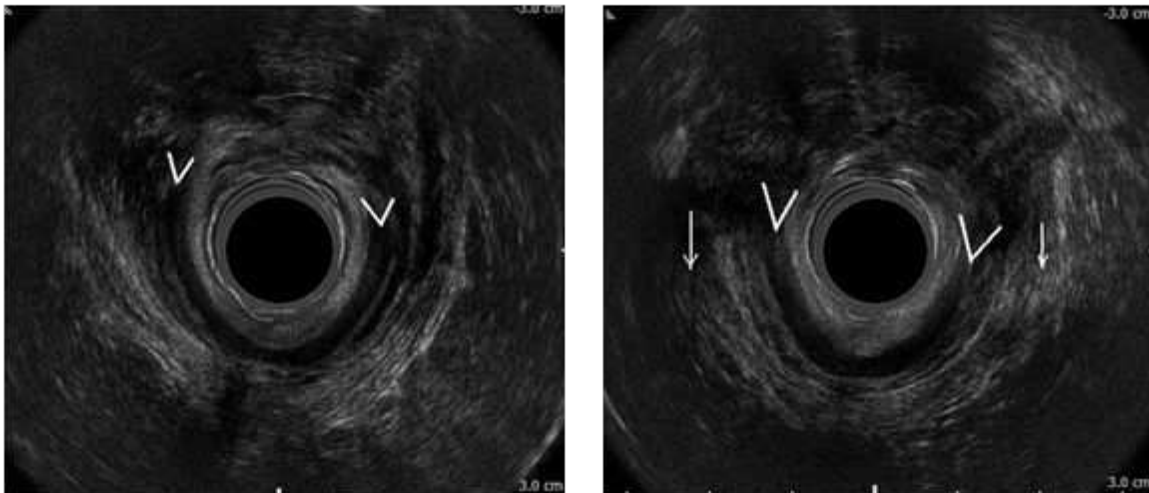

Figure 4. Obstetric structural defect of anal sphincters in AES: a) high anal canal level with an anterior tear of the 30% of the internal anal sphincter circumference (dashes); b) mid anal canal level with an anterior tear of the 50% of the internal and external anal sphincters circumference (arrows). Retained muscles are normal

Endosonographic examination is also performed to assist in the diagnosis of rectovaginal fistulae which frequently accompany obstetric sphincter defects (Figure 5).

Magnetic resonance imaging with the use of a torso/pelvic coil or endoanal coil is rarely used in the postoperative diagnosis of faecal incontinence because endosonography, which is cheaper and simpler, allows for good visualisation of the sphincters. However, the selection of diagnostic methods mainly depends on their accessibility, experience of the centre/examiner and costs of the examination (Figure 6).

Computed tomography, on the other hand, is not helpful in diagnosing anal sphincter defects because it does not allow for the discrimination between individual anal sphincters.
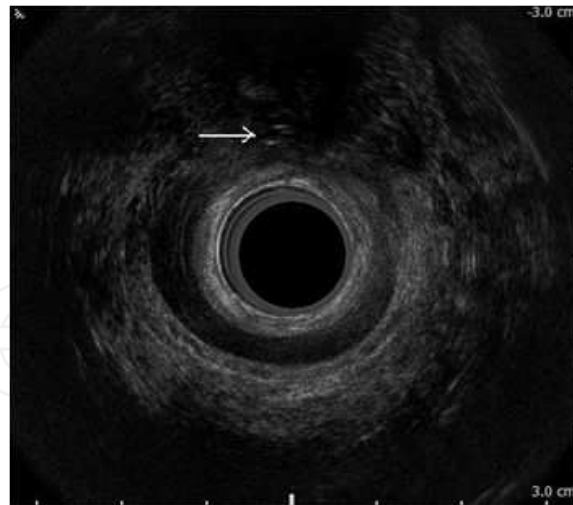
Figure 5. Obstetric ano-vaginal fistula in AES (arrow)
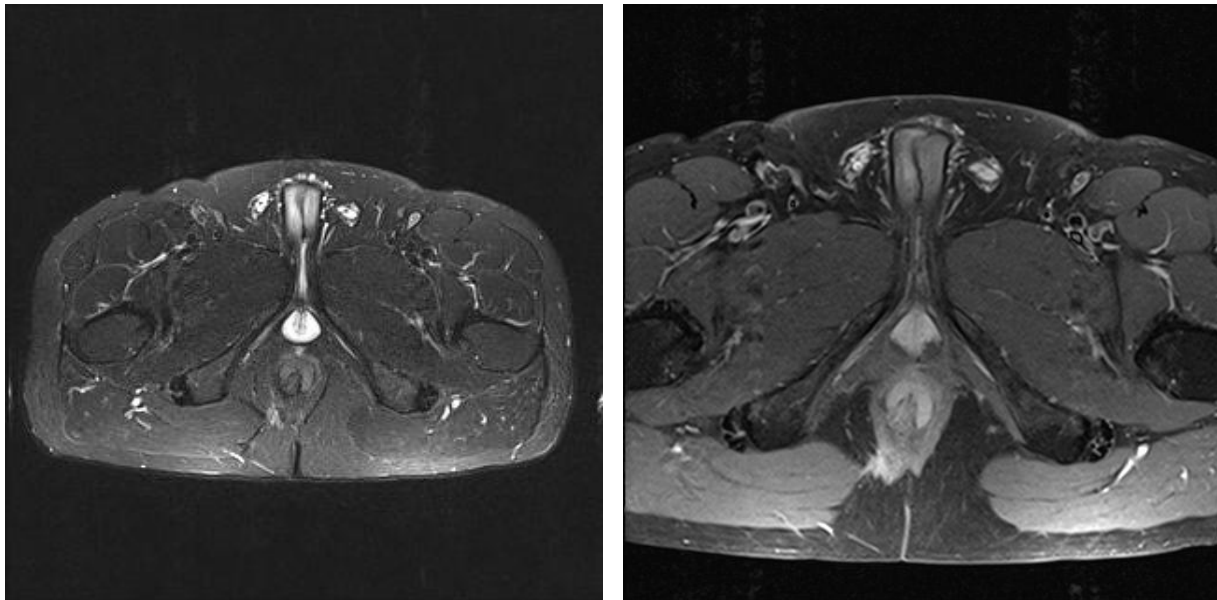
Figure 6. Posterior defect of the internal anal sphincter in: a) T2 TIRM and b) T1F2CE axial images following surgery for intersphincteric fistula

7. Treatment of obstetric and postoperative incontinence

The treatment of incontinence should be comprehensive and should take place in coloproctological referral centres.

7.1. Conservative treatment

In the majority of cases, conservative treatment is offered initially and encompasses a stool thickening diet, antidiarrheal medications, exercising sphincter muscles and pelvic floor

muscles as well as electrostimulation of the sphincters with biofeedback. Psychological supervision is also essential in numerous patients.

The biofeedback method uses biological feedback i.e. enables patients to monitor the progress of their therapy by means of special devices which allow for the visual (change of colour) or auditory (sound) registration of the therapy progress (e.g. increasing contraction strength of the sphincters while exercising).

Electrostimulation of sphincters is performed with devices using endoanal electrodes. The effectiveness of such procedures is assessed in various ways. This, among others, results from the lack of uniform qualification criteria for such therapies.

7.2. Surgical treatment

The best functional effects are brought about by immediate suturing of the muscles directly after the trauma. This particularly refers to obstetric damage to the anal sphincters. If immediate repair is not possible (due to inexperienced team or occult damage) the therapeutic decision should be postponed until the tissues have healed [44].

Scheduled reconstruction of anal sphincters is burdened with a high risk of failure even in specialised referral centres. Prior to such procedures, it is crucial to conduct imaging and functional examinations of the anal sphincters in order to qualify the patient for the procedure. If the defect involves more than 30% of the anal circumference, the operator is forced to consider protective stoma. If, however, the sphincteric defect exceeds 50%, the repair surgery is practically certain to fail. When the damage to the external sphincter is partial (not involving the entire thickness), the muscles may be sutured by interrupted sutures (end-to-end repair) without mobilising the ends. If, however, the muscle is completely severed, the stumps should be mobilised and sutured by the overlapping technique. This method brings about the best outcomes.

Surgical treatment should be complemented with conservative therapy i.e. exercises strengthening the muscles and anorectal sensation (biofeedback) as well as electrical stimulation of the anal sphincters.

8. Conclusion

Incontinence is a condition of a multifactorial etiology. It is difficult to treat and requires specialised diagnosis. What is more, patients with faecal incontinence should remain under the supervision of a multidisciplinary team of physicians including specialists in fields such as surgical coloproctology, radiologists as well as psychologists. Diagnosis and treatment should take place in the centres specialising in coloproctology. Finally, rehabilitation of patients following sphincteric surgeries is essential and may considerably improve the ultimate outcome of the procedure.

Author details

Małgorzata Kołodziejczak[1*] and Iwona Sudoł-Szopińska[2,3]

*Address all correspondence to: drkolodziejczak@o2.pl

1 Department of Proctology, Hospital at Solec, Warsaw, Poland

2 Department of Radiology, Institute of Rheumatology, Warsaw, Poland

3 Department of Diagnostic Imaging, Warsaw Medical University, Warsaw, Poland

References

[1] Nelson RL. Epidemiology of fecal incontinence. Gastroenterology 2004;126(1): 3-7.

[2] Madoff RD. Fecal incontinence in adults. Lancet 2004;364: 621-32.

[3] Kołodziejczak M, Sudoł-Szopińska I, Stefański R, Panorska AK, Gardyszewska A, Krasnodębski I. Anal endosonographic findings in women after vaginal delivery. European Journal of Radiology 2011;78(1): 157-9.

[4] Bharucha AE, Zinsmeister AR, Locke GR, Seide BM, McKeon K, Schlec CD, Melton J. Prevalence and burden of fecal incontinence: a population-based study in women. Gastroenterology 2005;129: 42-9.

[5] Barthet M, Bellon P, Abou E, Portier F, Berdah S, Lesavre N, Orsoni P, Bouvier M, Grimaud JC. The International Journal of Colorectal Disease 2002;17(2): 123.

[6] Morris C, Osterweil P, Hong LI, Rosenberg D, Greenlick MN. Incidence of fecal incontinence after childbirth. Obstetrics & Gynecology 2007;109(1): 281-8.

[7] Satish S.C Rao Pathophysiology of adult fecal incontinence 2004;126: 14–22.

[8] Fitzpatrick M, O'Herlihy C. The effects of labour and delivery on the pelvic floor Best Practice and Research Clinical Obstetrics & Gynecology 2001;15: 63-79.

[9] Leuw JW, Struijk PC, Vierhout ME, Wallenburg HCS. Risk factors for third degree perineal ruptures during delivery. British Journal of Obstetrics and Gynaecology 2001;108: 383-7.

[10] De Parades V, Etienney I, Thabut D, Beaulieu S, Tawk M, Assemekang B, Marie V, Toubia ML, Wehbe A, Mosnier H, Gadonneix P, Harvey T, Atienza P. Anal sphincter injury after forceps delivery: myth or reality? Diseases of the Colon & Rectum 2004;47: 24-34.

[11] Gudmundsson S, Henningsson AC, Lindqvist P. Correlation of birth injury with material height and birthweight. British Journal of Obstetrics and Gynaecology 2005;112(6): 764-7.

[12] Fernando R, Sultan AH, Kettle C, Thakar R, Radley S. Methods of repair for obstetric anal sphincter injury. Cochrane Database Systematic Reviews 2006;19: 3-CD002866.

[13] Choi JS, Wexner SD, Nam YS, Mavrantonis C, Salum M R, Yamaguchi T Weiss EG, Nogueras JJ, Yu CF. Intraobserver and interobserver measurements of the anorectal angle and perineal descent in defecography. Diseases of the Colon & Rectum 2000; vol 43,(8): 1121-6.

[14] Devine JB, Ostergard DR, Noblett KL. Long term complications of the second stage of labor. Obstetrics & Gynecology 1993;44(6): 119-21.

[15] Gregory WT, Lou JS, Stuyvesant A, Clark AL. Quantitative electromyography of anal sphincter after uncomplicated vaginal delivery. Obstetrics & Gynecology 2004;104: 327-35.

[16] Allen RE, Hosker GL, Smith ARB, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. British Journal of Obstetrics and Gynaecology 1990;97: 770-9.

[17] Casey BM, Schaffer JI, Steven L, Bloom SL, Heartwell SF, McIntire DD. Obstetric antecedents for postpartum pelvic floor dysfunction. American Journal of Obstetrics and Gynaecology 2005;192: 1655-62.

[18] Jünemann K, Thüroff J. Innervation. In: Schüssler B, Laycock J, Norton P, Stanton S (eds.) Pelvic floor re-education. Principles and practice. London, Berlin, Heidelberg, New York, Paris, Tokyo, Hong Kong, Barcelona, Budapest: Springer-Verlag, 1994. p22-7.

[19] Snooks SS, Swash M, Henry MM, Setchell M. Risk Factors in childbirth causing damage to pelvic floor innervations. British Journal of Surgery 1985;72: 15-7.

[20] Schouten WR, Briel JW, Auwerda JJA. Relationship between anal pressure and anodermal blood flow. The vascular pathogenesis of anal fissures. Diseases of the Colon & Rectum 1994;37: 664-9.

[21] Hill K, Fanning S, Fennerty MB, Faigel DO. Endoanal ultrasound compared to anorectalmanometry for the evaluation of fecal incontinence: a study of the effect these tests have on clinical outcome. Digestive Diseases & Sciences 2006;51(2): 235-40.

[22] Argov S, Levandovsky O. Open lateral sphincterotomy is still the best treatment for chronic anal fissure. American Journal of Surgery 2000;179(3): 201-2.

[23] Kastinelos P, Papaziogas B, Koutelidakis I, Paroutoglou G, Dimiropoulos S,Souparis A, Atmatzidis K. Topical 0,5% nifedipine vs lateral internal sphincterotomy for the

treatment of chronic anal fissurae: long- term follow-up. The International Journal of Colorectal Disease 2006;21: 179-83.

[24] Richard CS, Gregoire R, Plewes EA, Silverman R, Burul C, Buie D, Reznick R, Ross T, Burnstein M, O'Connor BI, Mukraj D, McLeod RS. Internal sphincterotomy is superi-or to nitroglycerin in the treatment of chronic anal fissurae: results of a randomized, controlled trial by the Canadian Colorectal Surgical Trials Group. Diseases of the Co-lon & Rectum 2000;43: 1048-57.

[25] Ouaïssi M, Giger U, Sielezneff I, Yawovi KA, Pamela A, Pirro N, Sastre B.Mucosal advancement flap anoplasty for chronic anal fissure resistant to conservative thera-py. World Journal of Surgery 2011;35(4): 900-4.

[26] Patel SD, Oxenham T, Praveen BV. Medium-term results of anal advancement flap compared with lateral sphincterotomy for the treatment of anal fissurae. The Interna-tional Journal of Colorectal Disease; 26(9): 1211-4.

[27] Lasheen AE, Morsy MM, Fiad AA. Segmental internal sphincterotomy - a new tech-nique for treatment of chronic anal fissure. Journal of Gastrointestinal Surgery 2011;15: 2271-4.

[28] Sohn N, Eisenberg MM, Weinstein MA, Lugo RN, Ader J. Precise anorectal sphincter dilatation - its role in the therapy of anal fissures. Diseases of the Colon & Rectum 1992;35 (4): 322-7.

[29] Renzi A. Clinical, manometric and ultrasonographic results of pneumatic balloon di-latation vs lateral internal sphincterotomy for chronic anal fissurae: a prospective, randomized controlled trial. Diseases of the Colon & Rectum 2008;51 (1): 121-7.

[30] van der Hagen SJ, Baeten CG, Soeters PB, van Gemert WG. Long-term outcome fol-lowing mucosal advancement flap for high perianal fistulas and fistulotomy for low perianal fistulas: recurrent perianal fistulas: failure of treatment or recurrent patient disease? The International Journal of Colorectal Disease 2006;21: 784–90.

[31] Bokhari S,Lindsey I. Incontinence following sphincter division for treatment of anal fistula. The International Journal of Colorectal Disease 2010;12: 135-9.

[32] Perez F, Arroyo A, Serrano P et al. Fistulotomy with primary sphincter reconstruc-tion in the management of complex fistula-in-ano: prospective study of clinical and manometric results. The Journal of the American College of Surgeons 2005;200: 897-903.

[33] Gemsenjager E. Results with a new therapy concept in anal fistula:suture of the anal sphincter. Schweizerische Medizinische Wochenschrift 1996;126: 2021-5.

[34] Jordan J, Roig JV, Garcia-Amengol J García-Granero E, Solana A, Lledó S.Risk Fac-tors for reccurence and incontinence after anal fistula surgery. The International Jour-nal of Colorectal Disease 2010;12: 254-60.

[35] Garg P, Song J, Bhatia A, Kalia H, Menon GR. The efficacy of anal fistula plug in fistula in ano. A systematic reviews of Colorectal Disease 2010;12: 965-70.

[36] Soltani A, Kaiser AM. Endorectal advancement flap for cryptoglandular or Crohn's fistula-in-ano. Diseases of the Colon & Rectum 2010;53: 486-95.

[37] Ritchie RD, Sackier JM, Hodde JP. Incontinence rates after cutting seton treatment for anal fistula. The International Journal of Colorectal Disease 2009;11: 564-71

[38] Vial M, Pares D, Pera M, Grande L. Fecal incontinence after seton treatment for anal fistulae with and without surgical division of internal anal sphincter: a systematic review. The International Journal of Colorectal Disease 2010;12: 172-8.

[39] Jorge JMN, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum 1993; 36, 77-97.

[40] Rockwood TH, Church JM, Fleshman JW, Kane RL, Mavrantonis C, Thorson AG, Wexner SD, Bliss D, Lowry AC. Fecal incontinence quality of life scale. Diseases of the Colon & Rectum 2000;vol 43(1): 9-16.

[41] Vaizey CJ, Kamm MA. Prospective assessment of the clinical value of anorectal investigations. Digestion 2000;61: 207-14.

[42] Pescatori M, Maria G, Anastazio G, Rinallo G. Anal manometry improves the outcome of surgery for fistula-in-ano. Diseases of the Colon & Rectum 1989;32(7): 588-592.

[43] Merletti R, Bottin A, Cescon C,Farina D, Gazzon M, Martina S,Mestin L, Pozzo M, Rainoldi A, Enck P. Multichannel surface EMG for the non-invasive assesment of the anal sphincter muscle. Digestion 2004;69 (2): 112-22.

[44] Fernardo RJ, Sultan AH. Risk factors and management of obstetric perineal injury. Current Obstetrics & Gynaecology 2004;14: 320-6.