Original article

Descending perineum syndrome: pathophysiology of fecal incontinence

FILIPPO PUCCIANI

Department of Surgery and Translational Medicine, University of Florence, Italy

Abstract: *Aim of the study:* Patients with descending perineum syndrome (DPS) may also experience fecal incontinence. This study was undertaken to understand the types and pathophysiology of fecal incontinence in patients affected by DPS. *Methods:* Two groups of DPS patients were investigated. Group 1 included 62 women who had fecal incontinence and descending perineum. Group 2 consisted of 70 female patients affected by descending perineum but without fecal incontinence. All 132 patients received a preliminary clinical evaluation and were studied using defecography, endoanal ultrasound and anorectal manometry. Their results were compared with data obtained from 20 healthy women with normal bowel habits (control group). *Results*: There was a significantly higher Fecal Incontinence Severity Index (FISI) score in Group 1 compared to Group 2 ($P < 0.001$). Urge incontinence was present in 56.4% of Group 1 patients, passive incontinence in 13 patients (20.9%), mixed incontinence in 16.1% and post-defecatory incontinence in only 4 patients (6.4%). Endoanal ultrasound revealed a significantly diffuse thinning of the external anal sphincter (EAS) in Group 1 patients ($P < 0.02$) with a linear relationship between signs of EAS atrophy and FISI score (p_s: 0.78; $P < 0.03$); EAS atrophy was also inversely correlated with anal pressure (p_s: 0.71; $P < 0.04$). Anal resting pressure (P_{max} and P_m) was significantly lower in Group 1 patients than Group 2 patients ($P < 0.04$). The pelvic floor descent values in Group 1 patients were significantly higher at rest and during evacuation ($P < 0.01$) than in Group 2 patients. *Conclusions*: Urge incontinence, related to external anal sphincter atrophy, is the predominant pattern of fecal incontinence.

Keywords: Descending perineum syndrome; Fecal incontinence; Pelvic organ prolapse; Rectoanal intussusception; Anorectal manometry.

INTRODUCTION

Descending perineum syndrome (DPS) is a complex syndrome where signs and symptoms are an expression of all the pelvic-perineal areas that are involved [1]Although it was first described by Parks *et al*. in 1966[2], as being characterized by the ballooning of the perineum several centimeters below the bony outlet of the pelvis during straining, many other attempts to define this syndrome and its symptoms have been published since that time. Descending perineum, according to Park's definition, refers only to the external perineal plane which is joined to the descent of the deep perineal plane, thus involving the whole pelvic floor and pelvic content. Straining at stool, a sensation of incomplete evacuation and sometimes of anorectal obstruction/blockage, manual maneuvers to facilitate defecation, and loss of solid or liquid stool are mixed with vaginal (sensation of a bulge, heaviness) and urological (urinary incontinence, urgency, hesitancy, feeling of incomplete emptying) symptoms[1]. The involvement of all visceral pelvic content is testified by dynamic magnetic resonance imaging which shows different grades of pelvic organ prolapse, including the rectum, combined with pelvic floor relaxation and functional evidence of impaired defecation[3]. Clinically, fecal incontinence appears late during the course of descending perineum syndrome and overlaps with obstructed defecation[1,4].

The aims of this study, conducted on patients affected by descending perineum syndrome, were 1) to describe the clinical profile of fecal incontinence and 2) to identify the main pathophysiological mechanisms of fecal incontinence.

MATERIALS AND METHODS

Between January 2010 and October 2015, 1261 patients affected by anorectal diseases were seen at the outpatient unit of the surgery clinic of the University of Florence. All patients were entered into a prospectively constructed database, which contained 2878 patients at the time of the study. This research was a retrospective, data-mining study investigating the differences between 2 groups of patients. Exclusion criteria were inflammatory bowel disease, proctitis, anal intercourse, rectal prolapse, previous pelvic and anal surgery, pelvic radiation, neurological disorders and cognitive impairment. One hundred thirty-two female patients [age range 47-78 years (median age 63.2 years)] affected by descending perineum syndrome identified according to Parks' and colleagues criteria[2] and negative colonoscopy were included in this study. All 132 patients received a preliminary clinical evaluation and were studied using defecography, endoanal ultrasound and anorectal manometry. Two groups of patients were identified and assigned to two study arms regarding the presence or absence of fecal incontinence. Group 1 was made up of 62 women [age range 55-79 years (median age 67.5 years)] who had fecal incontinence and descending perineum. Group 2 consisted of 70 female patients affected by descending perineum but without fecal incontinence [age range 43-73 years (median age 58.1 years)]. We compared their results with data obtained from 20 healthy women, age range 50-70 years (median age 60.4 years) with normal bowel habits (control group). They perceived their defecation behavior as normal and had never visited a physician for intestinal problems.

All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Clinical evaluation

Information regarding number of bowel movements/week, stool form according to Bristol scale[5], symptoms of obstructed defecation according to Bartolo[6] and pathological conditions were collected from previous outpatient charts. Obstructed defecation was classified according to the obstructed defecation syndrome (ODS) score[7]. Fecal incontinence was defined as the uncontrolled passage of fecal material recurring for > 3 months; number of fecal incontinence episodes for week was noted. Fecal incontinence was

classified according to the Fecal Incontinence Severity Index (FISI) score[8]. The pattern of incontinence was defined according to Engel's criteria[9]: fecal incontinence was classified as urge incontinence, passive incontinence, mixed incontinence, and post-defecatory incontinence. We reviewed chart data and recorded deliveries, noting obstetric tears and episiotomy, and degree of genital relaxation assessed with the Pelvic Organ Prolapse Quantification system (POP-Q) [10]. Inspection of the ano-perineal region and digital rectal examination were carried out to detect any signs of organic disease.

Defecography

All the patients and controls underwent defecography, according to the methods suggested by the Italian Working Team[11]. It was performed with the patient at rest, during squeeze, and during expulsion of the barium. All the X-rays showed latero-lateral views. The radiological measurements included the anorectal angle (measured and expressed in degrees between the longitudinal axis of the anal canal and the tangential line to the posterior rectal wall) and pelvic floor descent (defined as the vertical distance between the pubococcygeal line and the anorectal junction). The latter was expressed in millimeters. Qualitative evaluation was made by noting barium trapping, rectoanal intussusception, and the persistence of the puborectalis indentation during evacuation. Rectocele was also identified as a herniation of the anterior wall of the rectum into the vagina; the dimension was measured in millimeters and was defined as the distance between the tip of the rectocele and the longitudinal axis of the anal canal. Rectocele displacement with abnormal pelvic floor descent at evacuation was noted[12]. Rectoanal intussusception was also graded according to the Oxford Radiological grading of rectal prolapse[13].

Endoanal ultrasound

Endoanal ultrasound was performed using a three-dimensional multifrequency (9-16 MHz) probe

(Flex-Focus 1202; B-K Medical, Herlev, Denmark; endoprobe 360° rotating type 2050, proximal-to-distal length of 6 cm) with the patient in the left-lateral position. The probe was introduced into the anus to the level of the anorectal verge and slowly withdrawn. A defect of the internal (IAS) or external anal sphincter (EAS) was defined as a discontinuity of the muscle with an area of mixed echogenicity due to replacement of muscle cells by fibrous tissue. The sphincter defect was measured in degrees. Sphincter quality was described as homogeneous or heterogeneous if signs of sphincter atrophy were present. External anal sphincter atrophy was defined as diffuse thinning and/or replacement of muscle fibers by fat. Internal anal sphincter atrophy was identified as diffuse thinning of the sphincter.

Anorectal manometry

Computerized anorectal manometry was performed in all patients using previously published standard techniques[12]. Maximal anal pressure (Pmax) and mean pressure (Pm) of the anal canal expressed anal resting pressures (ARP). The computer quantified the amplitude (MVC-A) in millimeters of mercury (mmHg) and duration (MVC-T) in seconds of the maximal voluntary contraction (MVC). The rectoanal inhibitory reflex (RAIR) was elicited according to Martelli et al.[14]. The first distension volume at which internal sphincter relaxation occurred (RAIR threshold, RAIRT) and the distension volume for which an initial transient sensation occurred [conscious rectal sensitivity threshold, (CRST)] were determined in all patients. The maximal tolerated volume (MTV) was also measured in all patients and was considered an expression of rectal reservoir capacity. Compliance of the rectum (expression of the ratio mmHg/ml of inflated air) was detected by means of the pressure/volume curve. The manometric procedure was completed by measuring anal pressures when the patient was asked to attempt defecation (straining test). The straining test was considered positive if an inappropriate increase or < 20% relaxation of basal resting pressure occurred (respectively types I and III, describing dyssynergic defecation, according to Rao *et al*.)[15].

Statistical analysis

The sample size adequacy and statistical power of the study were calculated (DSS Research: statistical power calculator). The results are expressed as the mean ± standard deviation (SD). Student's t-test for paired and unpaired samples was used for statistical analyses. All correlations were evaluated using Spearman's rank correlation coefficient (rho: ρ_S), where full correlation is 1 and correlation < 0.50 is considered not significant. A $P > 0.05$ was chosen for rejection of the null hypothesis.

RESULTS

The sample size was adequate (adequacy: π=0.84) to achieve a statistical power of 100%. Patients' clinical characteristics are reported in Table 1. The number of bowel movements/week was not significantly higher in Group 1 patients than in Group 2 subjects and 45 Group 1 patients had > 3 fecal incontinence episodes/week. 17.7% of Group 1 patients had loose stool while 40% of Group 2 patients had hard stool ($P < 0.001$). There was a significantly higher FISI score in Group 1 compared to Group 2 ($P < 0.001$). Urge incontinence was present in 56.4% of Group 1 patients, while passive incontinence was predominant in 13 (20.9%), mixed incontinence in 16.1% and post-defecatory incontinence in only 4 patients (6.4%). A significantly higher ODS score was found in Group 2 patients compared to those of Group 1 ($P < 0.01$). Obstetric tears and/or episiotomy were recorded in 112 women (84.8 %) without significant differences between Group 1 and Group 2. Only 5 of all 132 study women had no uro-gynecological problems: 50 had cystoceles (37.8%) and 77 had uterine pro-

TABLE 1. Patients' clinical characteristics.

Symptom - Sign	Study cohort (n = 132)	Group 1 (n = 62)	Group 2 (n = 70)
Stool frequency (n/week)	7.7 ± 6.1	10.8 ± 2.9	6.3 ± 3.6
Fecal incontinence episodes (n/week)	2.4 ± 1.1	5.2 ± 1.6*	0
Hard stool (Bristol scale 1 or 2)	33	5	28°
Loose stool (Bristol scale 6 or 7)	13	11*	2
FISI score	11.7 ± 7.3	21.4 ± 8.7*	0
ODS score	6.9 ± 4.2	5.7 ± 2.3	11.8 ± 3.1°
Deliveries	1.3 ± 1.1	1.6 ± 0.7	1.2 ± 1.0
Obstetric tears	62/132	33/62	29/70
Episiotomy	50/132	23/62	27/70
Cystoceles	50/132	24/62	26/70
Uterine prolapse	77/132	47/62§	30/70
Urinary incontinence	42/132	19/62	23/70

*Group 1 patients vs Group 2 patients: $P < 0.001$
° Group 2 patients vs Group 1 patients: $P < 0.01$
§ Group 1 patients vs Group 2 patients: $P < 0.05$

TABLE 2. Results of endoanal sonography and anorectal manometry*Group 1 patients vs Group 2 patients and Controls: P < 0.02.

	Controls (n = 20)	**Group 1 (n = 62)**	**Group 2 (n = 70)**
IAS thickness (mm)	3.1 ± 0.5	2.4 ± 0.4	2.7 ± 0.8
EAS thickness (mm)	5.2 ± 0.7	2.5 ± 0.7*	4.3 ± 0.5
Pmax (mmHg)	88.2 ± 10.1	41.8 ± 18.0°	70.6 ± 21.9
Pm (mmHg)	47.8 ± 7.6	20.6 ± 8.2°	40.7 ± 13.4
MVC – A (mmHg)	200.0 ± 20.0	135.3 ± 25.8°°	158.2 ± 25.5
MVC – T (sec)	35.6 ± 4.2	14.1 ± 11.9°°	24.2 ± 10.2
Straining test (patients positive/ total patients)	1/20	15/62	39/70§
RAIRT (ml)	30.7 ± 10.8	25.0 ± 2.1	28.3 ± 8.1
CRST (ml)	40.7 ± 10.3	85.3 ± 24.4**	61.3 ± 27.7
MTV (ml)	205.1 ± 23.4	198.2 ± 51.1	183.3 ± 36.6

° Group 1 patients vs Group 2 patients and Controls: P < 0.04
°° Group 1 patients vs Group 2 patients and Controls: P < 0.01
§ Group 2 patients vs Group 1 patients: P < 0.01
**Group 1 patients vs Group 2 patients and Controls: P < 0.05

lapse (58.3 %). Urinary incontinence was present in 42 patients (31.8%): 29 (69.0%) had stress incontinence. Anorectal physical examinations revealed grade II hemorrhoids in 7 Group 1 and in 9 Group 2 patients. No anal fistulas or fissures were detected.

The results of endoanal ultrasound and anorectal manometry are reported in Table 2. Endoanal ultrasound revealed a significant diffuse thinning of EAS in Group 1 patients ($P < 0.02$) with a linear relationship between signs of EAS atrophy and FISI score (ρ_S: 0.78; $P < 0.03$); EAS atrophy was also inversely correlated with anal pressure (ρ_S: 0.71; $P < 0.04$). There was an internal anal sphincter disruption in 14 Group 1 patients (22.5%) and in 9 Group 2 patients (12.8%); EAS defects (width: 71.4 ± 18.5°) were detected in 22 Group 1 patients (35.4%) and in 13 Group 2 patients (18.5%). Anal resting pressure (P_{max} and P_m) was significantly lower in Group 1 patients when compared to Group 2 patients and controls ($P < 0.04$). MVC amplitude and duration in both groups were significantly different from controls ($P < 0.01$). The straining test was positive in 39 Group 2 patients (55.7%) and this was significantly different from that of Group 1 patients ($P < 0.01$). A significantly higher CRST was found in Group 1 patients ($P < 0.05$) in comparison to Group 2 patients and controls; CRST was also significantly higher than RAIRT in both groups when compared to those of controls ($P < 0.01$). There were no significant differences in maximal tolerated volume and rectal compliance in either patient group.

Defecographic data are reported in table 3. The anorectal angle was significantly greater in Group 1 when compared to group 2 and controls ($P < 0.01$). The pelvic floor descent values in Group 1 patients were significantly higher at rest and during evacuation ($P < 0.01$) than in Group 2 patients and controls. Eighty-four patients had a poor anorectal angle opening at evacuation and 72 patients (54.5%) had puborectalis indentation: Group 2 patients had a higher incidence of puborectalis indentation when compared to Group 1 ($p < 0.05$). Rectoanal intussusception, with Oxford Grade III and Grade IV, was noted in 102 (77.2%) of all patients and in 77 of these (75.4%) the rectoanal intussusception was combined with rectocele. Rectocele was present in 86 patients (65.1%) altogether and 9 patients showed a displacement rectocele alone that was > 3 cm. Six patients showed signs of enterocele, 4 had sigmoidocele.

TABLE 3. Defecographic data.

	Controls (n = 20)	**Group 1 (n = 62)**	**Group 2 (n = 70)**
Anorectal angle (degrees)			
Resting	94.5 ± 3.1	109.6 ± 1,5*	93.8 ± 3,8
Evacuation	110.1 ± 3.4	127.3 ± 6.6*	112.4 ± 5.1
Pelvic floor descent (mm)			
Resting	17.3 ± 7.2	43.3 ± 3.5*	32.1 ± 3.0
Evacuation	25.2 ± 2.5	54.3 ± 5.6*	38.7 ± 6.5
Rectocele			
Affected Patients	0	46/62	37/70
Size (mm)	0	27.0 ± 5.2	24.7 ± 9.2
Rectoanal intussusception			
Affected Patients	0	50/62	52/70
Puborectalis indentation			
Affected Patients	0	28/62	44/70°
Enterocele			
Affected Patients	0	2/62	4/70
Sigmoidocele			
Affected Patients	0	1/62	3/70

*Group 1 patients vs Group 2 patients and Controls: P < 0.01
° Group 2 patients vs Group 1 patients: P < 0.05

DISCUSSION

Descending perineum syndrome is characterized by the involvement of all visceral pelvic content. Many clues suggest that obstructed defecation slowly evolves into fecal incontinence. The initial phase of obstructed defecation sustained by the pelvic floor dyssynergia evolves, over some years, into organic changes associated with the appearance of mobile descent of the pelvic floor[1]. In our study, 54 patients (40.9%) had a positive straining test, and 84 patients (63.6%) had a poor anorectal angle opening at evacuation, both of which are diagnostic signs of dyssynergic defecation. Signs of impaired rectal sensations are also present: CRST was higher in both groups in comparison to that of controls. This report underlines the presence of obstructed defecation in DPS: rectal sensory perception is in fact blunted or absent in the majority of patients with obstructed defecation[16].

The progressive decline in normal levator ani tone, induced by defecatory overstretching, results in an open urogenital hiatus, weakening of the horizontal orientation of the levator plate, and a bowl-like configuration[17]. The consequence is impairment of manometric and defecographic signs of pelvic floor dyssynergia that no longer appear in long-term DPS forms. Such anatomical arrangements are also seen in women with pelvic organ prolapse: the ballooning of the levator hiatus and the increase in the levator plate angle are imaging signs of pelvic floor impairment in pelvic organ prolapse[18,19]. These anatomical changes help to explain the coexistence of uro-gynecological and proctologic pathologies in descending perineum syndrome, a condition also present in our patients, 96.7% of whom had urogynecological problems. The presence of episiotomy, POP, and urinary incontinence indicate the participation of the urinary and gynecological districts. Unfortunately they are not the object of our paper but it might be useful to further investigate this topic in the future.

In parallel, the natural history of descending perineum evolves thusly: the pelvic floor, upset by progressive pudendal neuropathy and pelvic-perineal muscle flabbiness, results in fecal incontinence, which at first is partial and eventually total, and is combined, in one way or another, with obstructed defecation[20]. Our incontinent patients had obstructed defecation, as testified by their ODS score, and

the close connection between impaired defecation and fecal incontinence illustrates the late evolution of the syndrome. Although our Group 1 patients were, in fact, older than Group 2 subjects (Group 1: median age 67.5 years; Group 2: median age 58.1 years), this difference was not significant, probably because the speed of DPS development was different between subjects. In our study, the FISI score in Group 1 was lower than that reported as clinically significant for impaired quality of life by Cavanaugh et al[21]. This is an indirect demonstration that even if a cut-off value is proposed the clinical reality is different: these Group 1 patients sought medical care even though their fecal incontinence was moderate. Urge incontinence, either alone (56.4%) or combined with passive incontinence (20.9%) was the predominant pattern of fecal incontinence. Thinning of the EAS, with impaired maximal voluntary contraction, was the main reason for this urge fecal incontinence[20] but several factors contemporaneously came into play: EAS defects, rectoanal intussusception, loose stool and high defecatory frequency were also all present, in various combination, in our Group 1 patients. Their pelvic floor descent was significantly higher at rest and during evacuation ($P < 0.01$) when compared to Group 2 patients, being a demonstration that DPS was evolving towards a worse stage marked by the clinical appearance of fecal incontinence. There was also a high incidence (80.6%) of rectoanal intussusception in both Group 1 and Group 2 patients. Rectoanal intussusception has a complex etiology: damaged pelvic floor muscles, mechanism of a sliding hernia, structural defects of pelvic fascia, ligaments and connective tissue, are all mixed with impaired defecation as a consequence of pelvic floor dyssynergia with vector force lines and high intra-abdominal pressures canalized into the Douglas cul-de-sac[22]. When rectoanal intussusception is combined with levator hiatal widening and levator plate descent, it can become the morphological pathology underlying DPS fecal incontinence[22]. It is difficult to provide a single pathophysiological framework for DPS fecal incontinence. Although a multifactorial etiology seems have an impact on a weak pelvic floor, it is very difficult to understand how much a single factor may destabilize the descending perineum. Surely a descending perineum possesses *per se* a pathological structure of pelviperineal muscles, perineal body and supportive elements of the endopelvic fascia that can lead to fecal incontinence. For example, lax suspensory ligaments that inactivate striated pelvic muscle forces[23], increased collagen breakdown such as a pathological etiology of urinary incontinence and pelvic organ prolapse[24], the observation that 45% of patients with joint hypermotility, stool evacuatory disorders and abnormal connective tissue also have fecal incontinence not due to sphincter dysfunction[25], are all evidence that an impaired pelvic floor may be associated with fecal incontinence.

The weakness of the study is the absence of follow-up regarding the appearance or absence of fecal incontinence but it must be considered that follow-up would have to be very long, lasting several decades, in order to show the complete evolution from obstructed defecation to fecal incontinence. Moreover, we decided not to introduce a second control group, i.e. those with fecal incontinence but without DPS, into the study because we felt that these patients would have had very diverse etiologies with pathophysiological profiles differing from case to case: this population would not be homogeneous, and therefore not useful for the purposes of comparison.

In conclusion, fecal incontinence inevitably will materialize with the passage of time in patients affected by descending perineum syndrome, characterized by hypotonia of the pelvic floor and associated pudendal neuropathy,. It is obvious that many factors may influence the evolution towards fecal incontinence and its velocity, such as childbirth[26], pelvic surgery[27], recto-anal surgery[28], anal sphincter lesions[29], radiotherapy[30] and neurological diseases[31], which all can have a negative impact on continence function, and may thus lead to the pathophysiological mechanisms of fecal incontinence. Knowledge of the physiopathology of fecal incontinence is the prerequisite for proper treatment of the patient.

REFERENCES

1. Pucciani F. Descending perineum syndrome: new perspectives. Tech Coloproctol 2015; 19: 443-448.
2. Parks AG, Porter NH, Hardcastle J. The syndrome of descending perineum. Proc R Soc Med 1966; 59: 477-482.
3. Comiter CV, Vasavada SP, Barbaric Z et al. Grading pelvic prolapse and pelvic floor relaxation using dynamic magnetic resonance imaging. Urology 1999; 54: 454-457.
4. Bartolo DC, Read NW, Jarratt JA, Read MG, Donnelly TC, Johnson AG Differences in anal sphincter function and clinical presentation in patients with pelvic floor descent. Gastroenterology 1983; 85: 68-75.
5. O'Donnell LJ, Virjee J, Heaton KW. Pseudo-diarrhoea in the irritable bowel syndrome: patients' records of stool form reflect transit time while stool frequency does not. Gut 1988; 29: A1455.
6. Bartolo D, Roe A. Obstructed defaecation. Br J Hosp Med 1986; 35: 228-236.
7. Altomare DF., Spazzafumo L, Rinaldi M et al. Set-up and statistical validation of a new scoring system for obstructed defaecation syndrome. Colorectal Dis 2008; 10: 84-88.
8. Rockwood TH, Church JM, Fleshman JW et al. Patient and surgeon ranking of the severity of symptoms associated with fecal incontinence: the fecal incontinence severity index. Dis Colon Rectum 1999; 42: 1525-1532.
9. Engel AF, Kamm MA, Bartram CI et al. Relationship of symptoms in faecal incontinence to specific sphincter abnormalities. Int J Colorect Dis 1995; 10: 152-155.
10. Bump RC, Mattiasson A, Bo K et al The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynaecol 1996; 175:10-17.
11. Piloni V, Genovese M, Grassi R et al. National working team report on defecography. Radiol Med (Torino) 1993; 85: 784-893.
12. Pucciani F, Rottoli Ml, Bologna A et al. Anterior rectocele and anorectal dysfunction. Int J Colorect Dis 1996; 11: 1-9.
13. Collinson R. Rectal intussusception and unexplained faecal incontinence: findings of a proctographic study. Colorectal Dis 2009; 11: 77-83.
14. Martelli H., Devroede G, Arhan P et al. Some parameters of large bowel motility in normal man. Gastroenterology 1978; 75: 612-618.
15. Rao SS, Mudipalli RS, Stessman M, et al. Investigation of the utility of colorectal function tests and Rome II criteria in dyssynergic defecation (Anismus). Neurogastroenterol Motil 2004; 16: 589-596.
16. Gosselink MJ, Schouten MR. Rectal sensory perception in females with obstructed defecation. Dis Colon Rectum 2001; 44: 1337-44.
17. Singh K, Jakab, M, Reid et al. Three-dimensional magnetic resonance imaging assessment of levator ani morphologic features in different grades of prolapse. Am J Obstet Gynecol 2003; 188: 910-15.
18. DeLancey JOL, Hurd WW. Size of the urogenital hiatus in the levator ani muscles in normal women and women with pelvic organ prolapse. Obstet Gynecol 1988; 91: 364-68.
19. Hsu Y, Summers A, Hussain HK et al. Levator plate angle in women with pelvic organ prolapse compared to women with normal support using dynamic MR imaging. Am J Obstet Gynecol 2006; 194: 1427-433.
20. Pucciani F. Editorial. A theory of progression from obstructed defecation to fecal incontinence. Tech Coloproctol 2015; 19: 713-715.

21. Cavanaugh M, Hyman N, Osler T. Fecal incontinence severity index after fistulotomy: a predictor of quality of life. Dis Colon Rectum. 2002 Mar; 45 (3): 349-53.
22. Pucciani F. Rectal prolapse: pathophysiology. In Altomare DF, Pucciani F (eds). Rectal Prolapse. Milan, Springer; 2008: 13-19.
23. Petros P. Diagnosis. In: Petros P (ed) The female pelvic floor. Function, dysfunction and management according to the integral theory. Heidelberg: 3rd Edn. Springer; 2010: 77-117.
24. Chen BH, Wen Y, Li H, Polan ML. Collagen metabolism and turnover in women with stress urinary incontinence and pelvic prolapse. Int Urogynecol J Pelvic Floor Dysfunct 2002; 13: 80-87.
25. Mohammed SD, Lunniss PJ, Zarate N et al. Joint hypermobility and rectal evacuatory dysfunction: an etiological link in abnormal connective tissue? Neurogastroenterol Motil 2010; 22: 1085-e283.
26. Murad-Regadas SM, Fernandes GO, Regadas FS. Assessment of pubovisceral muscle defects and levator hiatal dimensions in women with faecal incontinence after vaginal delivery: is there a correlation with severity of symptoms? Colorectal Dis 2014; 16: 1010-1018.
27. Pucciani F, Boni D, Perna F et al. Descending perineum syndrome: are abdominal hysterectomy and bowel habits linked? Dis Colon Rectum 2005; 48: 2094-2099.
28. Varpe P, Huhtinen H, Rantala A et al. Quality of life after surgery for rectal cancer with special reference to pelvic floor dysfunction. Colorectal Dis 2011; 13: 399-405.
29. Alavi K, Chan S, Wise P et al. Fecal Incontinence: Etiology, Diagnosis, and Management. J Gastrointest Surg 2015; 19: 1910-1921.
30. Sunesen KG, Nørgaard M, Lundby L et al. Long-term anorectal, urinary and sexual dysfunction causing distress after radiotherapy for anal cancer: a Danish multicentre cross-sectional questionnaire study. Colorectal Dis 2015; 17: O230-O239.
31. Townsend MK, Matthews CA, Whitehead WE et al. Risk factors for fecal incontinence in older women. Am J Gastroenterol 2013; 108: 113-119.

Correspondence to:
Filippo Pucciani
Largo Brambilla 3 - Florence 50134 - Italy
E-mail: pucciani@unifi.it

Multidisciplinary UroGyneProcto Editorial Comment

To improve the integration among the three segments of the pelvic floor, some of the articles published in *Pelviperineology* are commented on by **Urologists, Gynecologists, Proctologists/Colo Rectal Surgeons** or **other Specialists**, with their critical opinion and a teaching purpose. Differences, similarities and possible relationships between the data presented and what is known in the three fields of competence are stressed, or the absence of any analogy is indicated. The discussion is not a peer review, it concerns concepts, ideas, theories, not the methodology of the presentation.

Gynecologist. According to the work of Henry[1], confirmed by Beco[2], an abnormal perineal (anal margin) descent could be defined as more than 1.5 cm using a perineocaliper in lying position. In such a case, a descending perineum syndrome (DPS) can occur. Levator plate sagging, the descending perineum syndrome key feature, can occur because of long term dyschesia and/or puborectalis damage during delivery[3].

For many years, proctologist were the only physicians aware of this syndrome because gynecologist and urologist, even specialized in prolapse treatments, forgot to look at anal margin descent while straining. During their clinical examination, they searched for cystocele, rectocele, uterine prolapse and urinary incontinence while straining but they forgot to look at anal margin descent. It explains why most of the works about the descending perineum syndrome were done by colo-proctologist only looking at dyschesia and anal incontinence (induced by pudendal nerve stretching).

Abnormal levator plate sagging is a key feature in perineology. The absence of backpressure explains partly the difficulty to defecate[4]. Levator hiatus widening favors genital prolapse and stress urinary incontinence. Anal descent by itself explains the appearance of stretch pudendal neuropathy which disturbs anal and puborectalis innervation (vicious circle) and induces a real pudendal syndrome. Loss of anorectal angle, together with obstructive defecation, favors rectal intussusception with its side effects (vicious circle). Last but not least, the absence of support below uterus creates overstretching of the utero-sacral ligaments with posterior fornix symptoms[5].

In his paper, Pucciani showed the importance of descending perineum syndrome in perineology. As a proctologist, he underscored the link between DPS and obstructive defecation. Like Henry[1], he demonstrated changes in external sphincter muscle (with consecutive ano-rectal manometry anomalies), consistent with damage to its nerve supply and probably induced by pudendal nerve stretching. Almost all the patients had urogynecological problems (96.7%) thus showing that a global perineological approach is indispensable to treat correctly the patient.

REFERENCES

1. Henry MM, Parks AG, Swash M. The pelvic floor musculature in the descending perineum syndrome. Br J Surg 1982; 69 (8): 470-472.
2. Beco J. Interest of retro-anal levator plate myorrhaphy in selected cases of descending perineum syndrome with positive anti-sagging test. BMC Surg 2008; 8 (1): 13.
3. Roche B, Robert-Yap J, Skala K, Zufferey G: La désinsertion du muscle pubo-rectal est-elle la cause des rectocèles? In. Genêve (Switzerland): Société Suisse de Chirurgie. 2011.
4. Beco J, Antolak S: Posterior perineal support during defecation, descending perineum syndrome, pudendal neuropathy and anal fissures. Tech Coloproctol 2010; 14 (2): 193-194; author reply 195.
5. Goeschen K: Role of uterosacral ligaments in the causation and cure of chronic pelvic pain syndrome. Pelviperineology 2015; 34: 2-20.

JACQUES BECO
Department of Gynecology and Obstetrics, University Hospital of Liège, Liège, Belgium

Colorectal surgeon. In essence this paper can be regarded as a hypothesis. It is compiling a set of observations made under test conditions in order to construct an understanding of how abnormal pelvic anatomy and physiology may lead to the presenting symptoms. An analysis of this manuscript is better performed by starting with the conclusion and working backwards.

The conclusion states that, "*faecal incontinence inevitably will materialise with the passage of time in patients affected by descending perineum syndrome, characterised by hypotonia of the pelvic floor and associated pudendal neuropathy…*" It continues to state that many factors may influence the evolution towards faecal incontinence, however, these particular factors were all excluded from the study.

For a hypothesis to become established as a Theory it requires testing. Unfortunately, there was no testing of pudendal nerve function in the methodology and this conclusion cannot therefore be stated as part of the hypothesis. Furthermore, only external sphincter and internal sphincter muscle resting tone and contraction was tested. There was no attempt to look at the function or tone in the more major pelvic floor muscles, including puborectalis, pubococcygeus and ileococcygeus/levator plate. These muscles play a fundamentally important role in pelvic floor function in both emptying and continence and cannot be excluded from testing, if it is hypothesised that subsequent failure of muscle function is a causative factor behind the development of symptoms. Although the hypothesis stands, in this paper it remains untested and therefore un-validated.

Nonetheless, the paper contains important data. Most important is the almost global coexistence of urogynaecological symptoms and other structural abnormalities lending further weight to the importance of the Pescatori Iceberg in the assessment of these cases[1,2].

Over the last few years the surgical literature has contained increasing numbers of publications that demonstrate that pelvic ligament augmentation in the presence of prolapse and the associated symptom complexes produces cures in excess of 80% (symptoms and prolapse)[3-6]. Virtually all of these papers involve cases that are individual presentations based on a well-documented spectrum of pelvic floor dysfunction and structural abnormality, the vast majority of which conform to the observations, testing and publications in accordance with the Integral Theory. It should be noted that since its initial publication almost 30 years ago not a single observation has been published that moves toward invalidating the Integral Theory. The data presented in this manuscript does more to confer further validation of the Integral Theory than it does to support the alternative hypothesis that it presents[7].

REFERENCES

1. Pescatori, M., M. Spyrou, and A. Pulvirenti d'Urso, A prospective evaluation of occult disorders in obstructed defecation using the 'iceberg diagram'. Colorectal Dis, 2007, 9 (5), 452-6.
2. Goeschen, K. and Gold, D.M., Surgical cure of chronic pelvic pain, associated bladder & bowel symptoms by posterior sling in 198 patients validates the Pescatori Iceberg principle of pelvic symptom co-occurrence. Pelviperineology, 2017, p. 84.
3. Liedl, B. et al., Is overactive bladder in the female surgically curable by ligament repair? Cent European J Urol, 2017, 70 (1), 53-59.
4. Inoue, H. et al., Repair of damaged ligaments with tissue fixation system minisling is sufficient to cure major prolapse in all three compartments: 5-year data. J Obstet Gynaecol Res, 2017, 43 (10), 1570-1577.
5. Sekiguchi, Y., et al., Is Chronic Pelvic Pain in the female surgically curable by uterosacral/cardinal ligament repair? Pelviperineology, 2017, 74.
6. F. Wagenlehner, I.-A.M.-F., G. Perletti, B. Abendstein, K. Goeschen, H. Inoue, Y. Sekiguchi, A. Sivaslioglu, M. Haverfield, P. Richardson, Vaginal apical prolapse repair using two different slingtechniques improves chronic pelvic pain, urgency and nocturia – a multicentre study of 1420 patients. Pelviperineology, 2016, 35 (4), 99-104.
7. Liedl, B. et al., Update of the Integral Theory and System for Management of Pelvic Floor Dysfunction in Females. European Urology Supplements, 2017.

DARREN M. GOLD
Colorectal Surgeon, Senior Lecturer in Surgery,
St Vincent's Clinical School, UNSW, Sydney, Australia
dandjgold@gmail.com