

# Descending perineum syndrome: pathophysiology of fecal incontinence

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**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 ( $p$ : 0.78;  $P < 0.03$ ); EAS atrophy was also inversely correlated with anal pressure ( $p$ : 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. Although it was first described by Parks *et al.* in 1966<sup>2</sup>, 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<sup>1</sup>. 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<sup>3</sup>. Clinically, fecal incontinence appears late during the course of descending perineum syndrome and overlaps with obstructed defecation<sup>1,4</sup>.

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<sup>2</sup> 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<sup>5</sup>, symptoms of obstructed defecation according to Bartolo<sup>6</sup> and pathological conditions were collected from previous outpatient charts. Obstructed defecation was classified according to the obstructed defecation syndrome (ODS) score<sup>7</sup>. 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<sup>8</sup>. The pattern of incontinence was defined according to Engel's criteria<sup>9</sup>: 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)<sup>10</sup>. 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<sup>11</sup>. 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<sup>12</sup>. Rectoanal intussusception was also graded according to the Oxford Radiological grading of rectal prolapse<sup>13</sup>.

#### Endoanal ultrasound

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

(Flex-Focus 1202; B-K Medical, Herlev, Denmark; endo-probe 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<sup>12</sup>. 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.<sup>14</sup>. 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.*)<sup>15</sup>.

#### 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 ( $\rho_s$ ), 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:  $\pi=0.84$ ) 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 ( $\rho_s$ : 0.78; P < 0.03); EAS atrophy was also inversely correlated with anal pressure ( $\rho_s$ : 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<sub>max</sub> and P<sub>m</sub>) 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.

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<sup>1</sup>. 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<sup>16</sup>.

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<sup>17</sup>. 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<sup>18,19</sup>. 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 uro-gynecological 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<sup>20</sup>. Our incontinent patients had obstructed defecation, as testified by their ODS score, and

TABLE 3. Defecographic data.

	Controls (n = 20)	Group 1 (n = 62)	Group 2 (n = 70)
<b>Anorectal angle (degrees)</b>			
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
<b>Pelvic floor descent (mm)</b>			
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
<b>Rectocele</b>			
Affected Patients	0	46/62	37/70
Size (mm)	0	27.0 ± 5.2	24.7 ± 9.2
<b>Rectoanal intussusception</b>			
Affected Patients	0	50/62	52/70
<b>Puborectalis indentation</b>			
Affected Patients	0	28/62	44/70°
<b>Enterocele</b>			
Affected Patients	0	2/62	4/70
<b>Sigmoidocele</b>			
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

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.<sup>21</sup>. 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<sup>20</sup> 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<sup>22</sup>. When rectoanal intussusception is combined with levator hiatus widening and levator plate descent, it can become the morphological pathology underlying DPS fecal incontinence<sup>22</sup>. It is difficult to provide a single pathophysiological framework for DPS fecal incontinence. Although a multifactorial etiology seems to 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<sup>23</sup>, increased collagen breakdown such as a pathological etiology of urinary incontinence and pelvic organ prolapse<sup>24</sup>, 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<sup>25</sup>, 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<sup>26</sup>, pelvic surgery<sup>27</sup>, recto-anal surgery<sup>28</sup>, anal sphincter lesions<sup>29</sup>, radiotherapy<sup>30</sup> and neurological diseases<sup>31</sup>, 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.

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### 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<sup>1</sup>, confirmed by Beco<sup>2</sup>, 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<sup>3</sup>.

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<sup>4</sup>. 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<sup>5</sup>.

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<sup>1</sup>, 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.

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