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ORIGINAL ARTICLE

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On the pathogenesis of rectocele: the concept of the rectovaginal pressure gradient

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Abstract Coughing or straining evokes reflex bulbocavernosus (BCM) and puborectalis (PRM) muscle contraction, which apparently transforms the vagina into a closed high-pressure cavity [13]. This elevated vaginal pressure counteracts the increased intra-abdominal pressure and the tendency of the uterus to prolapse, and also supports the rectovaginal septum against the high straining-induced intrarectal pressure and possible consequent rectocele (posterior vaginal prolapse) formation. We investigated the hypothesis that a weak BCM and PRM share in the genesis of rectocele by changing the rectovaginal pressure gradient. Twenty-three women with rectocele (mean age 43.2 ± 6.6 years) and 12 healthy women volunteers (mean age 41.6 ± 6.2 years) were studied. The response of the intrarectal (intra-abdominal) and intravaginal pressure, as well as the EMG activity of the BCM and PRM to straining or coughing, was recorded. In the healthy volunteers the rectal and vaginal pressures showed a significant increase on coughing or straining, with no significant difference between the rectal or vaginal pressures. Also, the BCM and PRM EMG activity exhibited a significant increase. Rectocele patients showed a significantly low resting vaginal pressure. The increase in rectal and vaginal pressure, as well as of the EMG activity of the BCM and PRM on straining or coughing, was significantly lower and the latency of the EMG response was significantly longer than those of the healthy volunteers. A difference in the rectovaginal pressure gradient showing a significant increase in the rectal against the vaginal pressure, particularly on coughing or straining, is suggested to be the basic factor in the genesis of rectocele. This pressure difference appears to be caused by diminished BCM and PRM contractile activity. A disrupted rectovaginal septum is not a prerequisite for rectocele formation, as the septum appears normal in obstructed defecation despite the common occurrence of rectocele. A histopathologic study of the septum in rectocele seems necessary.

Keywords Bulbocavernosus muscle · Episiotomy · Obstructed defecation · Puborectalis muscle · Rectovaginal septum · Uterine prolapse

Abbreviations *BCM* Bulbocavernosus muscle · *PRM* Puborectalis muscle · *EMG* Electromyogram

Introduction

Rectocele (posterior vaginal prolapse) is a common pathologic condition. It is defined as an outpouching of the anterior rectal and posterior vaginal wall into the lumen of the vagina. Rectocele is associated with defecation disorders [1, 2], but may be asymptomatic [3, 4]. The common clinical presentation of rectocele is constipation in the form of straining and difficult evacuation [5, 6]. The patient has to push the rectocele back through the vagina to effect evacuation [5, 6]. Defecography is the method used to diagnose rectocele [3, 7, 8, 9]. Asymptomatic rectoceles have been found in up to 77% of women who underwent defecography and in 29%–41% of patients with constipation [3, 4]. Rectocele is associated with a high incidence of perineal descent [1, 10] and paradoxical puborectalis contraction [11]: 71% of rectocele patients had concomitant paradoxical puborectalis contraction. It is common in women but may also occur in men [12].

Results of a recent study suggested that the bulbocavernosus muscle (BCM) may have a role in the

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pathogenesis of rectocele [13]. On straining, the straining bulbocavernosus (BC) reflex is evoked and effects BCM contraction. Meanwhile, straining produces puborectalis muscle (PRM) contraction, mediated through the straining puborectalis reflex [14]. The vaginal introitus is presumably closed on straining as a result of BCM and PRM contraction. The vagina is transformed into a closed cavity with a high pressure that counteracts the increased intra-abdominal pressure on straining and the tendency of the uterus to prolapse [13]. Furthermore, the high pressure in the closed vaginal cavity appears to support the rectovaginal septum against the high rectal pressure evoked during straining, and is postulated to share in the prevention of rectocele formation [13].

We hypothesized that a weak PRM and BCM do not – or only poorly – respond to increased straining-induced intra-abdominal pressure, and the vaginal introitus remains open or incompletely closed, thereby allowing the intravaginal pressure to remain atmospheric; meanwhile, the intrarectal pressure is elevated in response to straining, resulting in a rectovaginal pressure gradient that predisposes to rectocele formation. This hypothesis was investigated in the present study.

Materials and methods

The study comprised 23 female patients with rectocele (mean age 43.2 ± 6.6 SD years, range 35–48) who complained mainly of constipation. The women were selected randomly from a large population presenting for consultation at the outpatient clinic of the Cairo University Hospital, which receives hundreds of patients daily. They were fully informed about the nature of the study, the tests to be done, and their role in the study. Their stool frequency was less than twice weekly and associated with excessive straining. All the patients were multiparous, with 4–6 (mean 5.2 ± 1.1) full-term deliveries and posterolateral episiotomy. Thirteen patients had had a prolonged second stage during one of the deliveries; another 4 patients gave a history of forceps delivery. The rectocele had a mean duration of 5.2 ± 2.6 SD years (range 3–10) and was anterior with a mean diameter of 4.1 ± 1.2 SD cm (range 3–5) in all patients, as shown by defecography. Defecography did not show associated intussusception. The patients managed their constipation with laxatives, enemas and/or digitation.

Twelve healthy women volunteers were also enrolled in the study. They matched the patients in age (mean age 41.6 ± 6.2 SD years, range 34–46) and were multiparous with 4–6 (mean 5.3 ± 1.1) full-term deliveries. Their deliveries had been normal, with no prolonged second stage or forceps deliveries. Posterolateral episiotomy was performed in all of them. The volunteers had no urogenital or anorectal complaints, either in the past or at the time of enrolment. The mean stool frequency was 9.2 ± 1.2/week (range 8–11), which is in accord with that of normal volunteers in our laboratory. Defecography showed no rectoceles.

All the patients and volunteers were sexually active. Physical examination and neurologic assessment were normal. Bulbocavernosus and anal reflexes were also normal. The laboratory work-up, comprising blood picture, hepatic and renal function tests as well as electrocardiography, was unremarkable. The subjects gave informed consent and our Faculty Review Board and Ethics Committee approved the study.

The women fasted for 8 h prior to the performance of the tests for our study and the bowel was emptied by defecation or saline enema. The rectal pressure was measured by means of a thin polyethylene infinitely compliant balloon of 1 cm in diameter, which had been attached to the end of a 10 Fr catheter (London Rubber Industries Ltd, London, UK). The pressure within the balloon was measured as being representative of the rectal pressure. The balloon was introduced via the anus for 10–12 cm into the rectum. The catheter was attached to a strain gauge pressure transducer (Statham 230B, Oxnard, CA) and connected via amplifiers to a chart recorder (Hewlett Packard 7798A, Waltham, MA, USA). The intravaginal pressure was measured by a similar manometric catheter introduced into the vagina for 3–4 cm from the introitus and connected to a Statham pressure transducer. The pressure transducers were zeroed to the atmospheric pressure, and the external transducers were at the level of the rectum or vagina.

The EMG recording of the BCM and PRM has been previously described [15, 16]. Briefly, a concentric needle electromyographic electrode (type 13L 49, Disa, Copenhagen), 45 mm in length and 0.65 mm in diameter, was used. The bulb of the clitoris was palpated and the needle electrode introduced into the BCM overlying it. For the PRM recording, the needle electrode was introduced into the perianal skin 1–1.5 cm lateral to the anal orifice and the needle pushed cephalad and medially to a depth of approximately 2–2.5 cm. A ground electrode was applied to the thigh.

A standard electromyographic (EMG) apparatus (type MES, Medelec, Woking, UK) was used to amplify and display the potentials recorded. The amplifier (type AA6 MK IIM, Medelec, Woking, UK) was set with a low-frequency filter at 16 Hz and a high-frequency filter at 3200 Hz. Films of the potentials were taken on light-sensitive paper (Linagraph type 1895, Kodak), from which measurements of motor unit potential duration were made. The EMG signals were also stored on an FM tape recorder (type 7758A, Hewlett Packard, Waltham, MA, USA) for further analysis as required.

The correct position of the needle electrode was monitored by the burst of activity heard on the loudspeaker and visualized on the oscilloscopic screen of the electromyograph. We had verified the normality of the EMG activity of the BCM and PRM in all the healthy volunteers before performing the tests. This was done by individual stimulation of the aforementioned muscles with a needle electrode and recording the action potentials by the needle electrode already inserted. All the healthy volunteers had normal EMG activity of the examined muscles.

The subject was asked to strain by coughing and by performing a Valsalva maneuver, and the intra-abdominal (intrarectal) and intravaginal pressures, as well as the EMG response of the BCM and PRM, were recorded. Readings were taken during the two types of straining: coughing, which represents sudden momentary straining, and Valsalva maneuver, which simulates the slow sustained straining that may be experienced with defecation or urination.

The needle electrode was withdrawn and introduced into the BCM and PRM on the contralateral side and the response of each muscle to straining was recorded as previously mentioned.

Reproducibility of the results was assessed by repeating the test at least twice in the individual subject and calculating the mean value. The results were analyzed statistically using analysis of variance (ANOVA). An intergroup analysis, as well as a comparison between the two groups (those with rectocele and those without) with regard to rectal pressure and EMG activity, was performed. Differences assumed significance at $P < 0.05$ and values were given as the mean ± standard deviation (SD).

Results

The study was completed in all the subjects with no adverse side effects during or after performing the tests, and all the subjects were evaluated.

Table 1 The rectal and vaginal pressures at rest and on coughing or straining in healthy volunteers and rectocele patients[+]

Pressure (cmH_2O)	Volunteers				Patients			
	Rectal		Vaginal		Rectal		Vaginal	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
At rest (basal)	7.2 ± 1.1	6–8	7.3 ± 1.1	6–8	7.5 ± 1.1º	6–8	1.1 ± 0.2**	0–2
On coughing or straining	167.3 ± 14.2	146–188	158.2 ± 13.7	150–175	106.4 ± 9.3*	86–116	29.6 ± 3.2**	26–35

[+] Values are given as the mean ± standard deviation
º $P > 0.05$
* $P < 0.05$
** $P < 0.01$
P values of the patients were compared to those of the volunteers

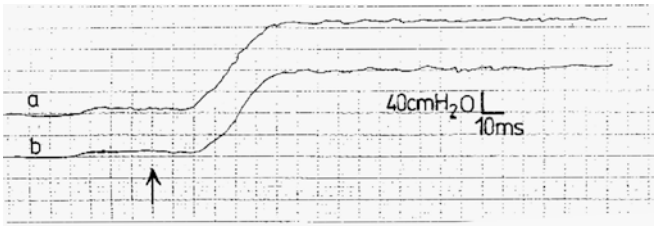

Fig. 1 Pressure tracing in a healthy volunteer showing increase in the rectal **a** and vaginal **b** pressure on coughing. ↑ = coughing

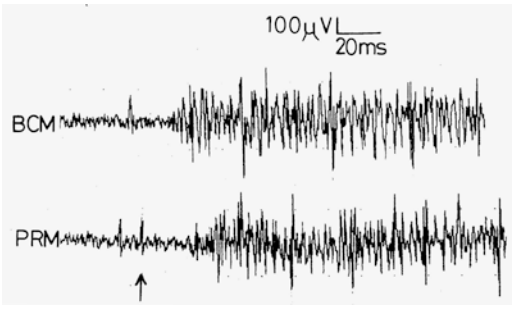

Fig. 2 EMG activity of the bulbocavernosus (BCM) and puborectalis (PRM) muscles on coughing in a healthy volunteer. ↑ = coughing

Healthy volunteers

The rectal and vaginal pressures at rest and on coughing or Valsalva maneuver are given in Table 1. On coughing or straining they showed a significant increase ($P < 0.0001$, $P < 0.0001$, respectively), with no significant difference between the rectal and vaginal pressures (Fig. 1). The EMG activity of the BCM also exhibited a significant increase on coughing or straining; it recorded a mean basal value of 108.6 ± 14.8 μV and on coughing or straining a mean of 486.4 ± 36.6 μV ($P < 0.01$, Fig. 2, Table 2). The mean latency was 20.6 ± 2.1 ms (range 17–24, Fig. 2). The PRM exhibited a significant increase of EMG activity on coughing or Valsalva ($P < 0.01$); it recorded mean motor unit action potentials of 118.4 ± 15.2 μV at rest and 536.8 ± 42.3 μV on coughing ($P < 0.01$, Fig. 2, Table 2). The latency recorded a mean of 21.6 ± 2.2 ms (range 18–26, Fig. 2). When the electrodes were transferred to the opposite side of the BCM and PRM, the potentials recorded from each muscle at rest and on coughing or straining did not differ significantly from those on the other side ($P > 0.05$).

Rectocele patients

The rectal and vaginal pressures at rest and on coughing or straining are given in Table 1. At rest, the rectal pressure did not differ significantly from that of the healthy volunteers ($P > 0.05$), whereas the vaginal pres-

Table 2 The EMG activity of the bulbocavernosus (BCM) and puborectalis (PRM) muscles at rest and on coughing or straining in healthy volunteers and rectocele patients[+]

Potentials (μV)	Volunteers				Patients			
	BCM		PRM		BCM		PRM	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
At rest (basal)	108.6 ± 14.8	96–122	118.4 ± 15.2	98–134	29.4 ± 6.6*	22–43	33.3 ± 9.6*	24–43
On coughing or straining	486.4 ± 36.6	406–520	536.8 ± 42.3	455–580	42.2 ± 7.3**	36–52	59.6 ± 11.3**	39–68

[+] Values were given as the mean ± standard deviation
* $P < 0.01$
** $P < 0.0001$
P values of the patients were compared to those of the volunteers

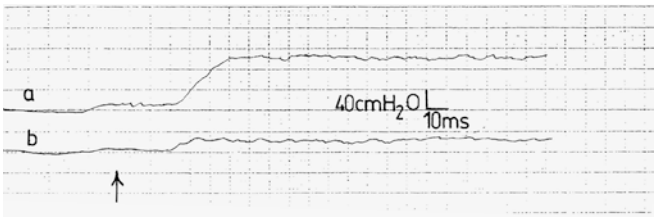

Fig. 3 Pressure tracing of a rectocele patient showing **a** rectal and **b** vaginal pressure response to coughing. ↑ = coughing

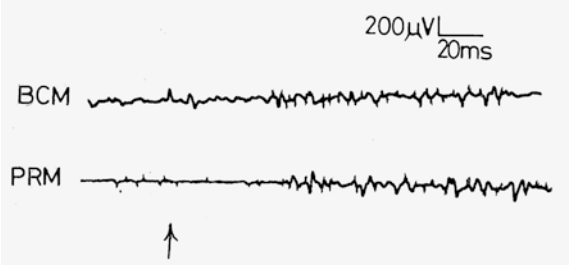

Fig. 4 EMG activity of the bulbocavernosus (BCM) and puborectalis (PRM) muscles on coughing in a rectocele patient. ↑ = coughing

sure exhibited a significant decrease ($P < 0.01$, Table 1). On coughing or straining, vaginal and rectal pressures showed a significant increase compared to their basal values ($P < 0.001$, $P < 0.001$, respectively; Table 1, Fig. 3), but were significantly lower than the vaginal and rectal pressures of the volunteers ($P < 0.01$, $P < 0.05$, respectively; Table 1, Fig. 3). However, the rectal pressure on coughing or straining was significantly higher than the vaginal pressure ($P < 0.01$, Table 1). The EMG of the BCM and PRM at rest and on coughing or straining showed a significantly reduced activity compared to that of the healthy volunteers (Table 2, Fig. 4). The mean latency of the BCM was 56.6 ± 10.8 ms (range 42–86) and of the PRM 59.2 ± 11.2 ms (range 48–90, Fig. 4); the two latencies were significantly prolonged compared to those of the healthy volunteers ($P < 0.05$, $P < 0.05$, respectively).

The above-mentioned results were reproducible with no significant difference when the measurements and recordings were repeated in the individual subject. There was no significant difference between the results obtained on coughing or straining (Valsalva maneuver).

Discussion

The current study may shed some light on the pathogenesis of rectocele. The significantly low basal vaginal pressure of rectocele patients seems to be related to the diminished resting activity of both the BCM and the PRM. In the healthy volunteers, the basal vaginal pressure represents the intra-abdominal pressure and is maintained at this level by the BCM which, under normal physiologic conditions, apparently occludes the vaginal introitus. The PRM is directly related to the lower third of the vagina (Fig. 5) and its tone shares also in the generation of the intravaginal pressure [17]. The basal intravaginal pressure in rectocele patients was either atmospheric (0) or close to atmospheric. Intravaginal pressure of 0 indicates that the vagina is connected directly to the atmosphere, owing probably to the weak BCM and PRM, as demonstrated in the current study, which leave the vaginal orifice gaping. This gaping vaginal introitus in rectocele has been reported by other investigators [18].

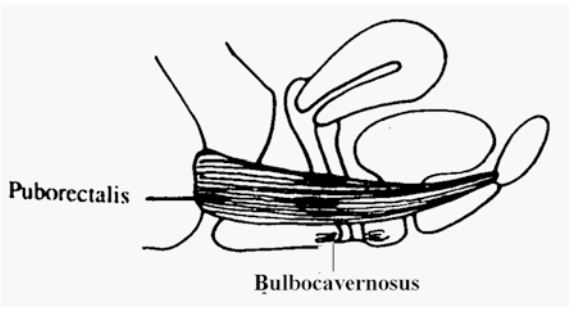

Fig. 5 Diagram illustrating the relation of the bulbocavernosus (BCM) and puborectalis (PRM) muscles to the vagina and rectum (from Shafik [17])

The motor unit action potentials on contraction of both the BCM and PRM in rectocele patients were significantly below those of the healthy volunteers. These findings supposedly indicate a weaker contractile activity of both muscles, and appear to explain the significantly low response of the intravaginal pressure on coughing or straining in rectocele patients compared to that of the healthy volunteers. The diminished contractile activity of the BCM and PRM is suggested to result from their traumatization by repeated deliveries, especially those associated with prolonged second stage or forceps application; this point needs to be investigated further.

The rise of the vaginal and rectal pressures on coughing in the healthy volunteers seems to be due in part to the elevated intra-abdominal pressure and in part to the BCM and PRM contractions, which are mediated through the straining bulbocavernosus and straining puborectalis reflexes, respectively [13, 14]. The lower vaginal and rectal pressure elevation in the rectocele patients as opposed to the healthy volunteers seems to indicate a disorder of these two reflexes; this is evidenced by the significantly prolonged latency of the reflexes and the low motor unit action potentials at rest and on contraction of the BCM and PRM compared to those in the healthy volunteers.

The physiologic mechanism of vaginouterine support on coughing or straining

Under normal physiologic conditions, coughing or straining elevates both intra-abdominal and vaginal

pressures. The vaginal pressure rise seems to be partly due to closure of the vaginal introitus, which is presumably produced by BCM contraction as mediated by the straining bulbocavernosus reflex [13]. Furthermore, coughing or straining evokes the straining puborectalis reflex, with a resulting PRM contraction [14]. As the PRM extends alongside the lower third of the vagina (Fig. 5) its contraction on coughing seems to support the vagina and probably shares in vaginal pressure elevation. Therefore, on coughing or straining, the increased intra-abdominal pressure is associated with BCM and PRM contraction, which closes the vaginal introitus and supports the vaginal wall. The vagina is thus transformed into a closed cavity with a high pressure. The high intravaginal pressure is suggested to counteract: a) the high rectal pressure induced by coughing or straining and its tendency to push the rectovaginal septum into the vagina, resulting in rectocele formation; and b) the increased intra-abdominal pressure and its tendency to push the uterus down, eventually resulting in uterine prolapse. Thus, the high intravaginal pressure induced by contraction of the BCM and PRM constitutes a mechanism that guards the uterus and rectovaginal septum against prolapse and rectocele formation, respectively.

On the pathogenesis of rectocele

The rectovaginal septum lies between two zones, the vagina and rectum, which under normal physiologic conditions, as in the healthy volunteers, exhibited equal pressure at rest. On straining or coughing, the pressure increased equally on both sides of the septum, apparently keeping the septum in its place. Thus a septum, when traumatized by repeated deliveries, would presumably not bulge into the vagina and form a rectocele if the pressure on both sides was equal. Accordingly, we suggest that the basic factor in the genesis of rectocele is the difference in the pressure on the two sides of the septum, with a significant increase of rectal over vaginal pressure.

The rectovaginal pressure gradient difference demonstrated in the current study is probably the result of the weak BCM and PRM, as is evident from their diminished EMG activity and prolonged latency. The diminished contractile activity of the BCM and PRM appears to be caused by their traumatization during delivery or by episiotomy. On contraction, the weak PRM and BSM most probably fail to support the vagina or to close the vaginal introitus, which is left gaping. The vaginal pressure becomes atmospheric or nearly atmospheric, and is significantly lower than the rectal pressure. The difference in the rectovaginal pressure gradient was more manifest on coughing or straining, as demonstrated in the current study; the rectal pressure was significantly higher than the vaginal pressure. This exposes the rectovaginal septum to unequal pressure on its two sides. Continuous exposure to this difference in the pressure gradient may eventually push the septum into the vagina and form a rectocele.

Role of episiotomy in the pathogenesis of rectocele

Episiotomy, whether posterolateral as in the current study or posterior, presumably divides the BCM; posterolateral episiotomy may, in addition, partially cut the PRM as the muscle stretches alongside the vagina to its insertion into the pubic bone (Fig. 5). The traumatic injury of the BCM and PRM, produced partly by the episiotomy and partly by a difficult delivery, appears to have a role in the genesis of rectocele through the above-mentioned mechanism.

Predisposing factors for rectocele formation may exist, including disruption of the rectovaginal septum during childbirth [18, 19]. However, the traumatized rectovaginal septum apparently does not constitute the prime factor in the genesis of rectocele. This is evident from the common occurrence of rectocele in obstructed defecation [20, 21, 22] despite the presence of a normal rectovaginal septum; disruption of the septum may occur as a secondary event after rectocele formation. Bowel habits may be an important factor in the development of rectocele [20, 21, 22, 23, 24]. Chronic constipation with straining at stool because of outlet obstruction may disturb the rectovaginal pressure gradient, with a resulting rectocele.

In conclusion, we suggest that the basic factor in the genesis of rectocele is a difference in the rectovaginal pressure gradient, with a significant increase of the rectal over the vaginal pressure, particularly on coughing or straining. The cause of this pressure difference appears to be the diminished contractile activity of the BCM and PRM affecting the support of the vaginal introitus and the lower third of the vagina. A disrupted rectovaginal septum, such as may occur in repeated deliveries, is not a prerequisite for the development of rectocele, as the septum in obstructed defecation is apparently normal. A histopathologic study of the rectovaginal septum in rectocele seems to be necessary.

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References

1. Mellgren A, Bremmer S, Johansson C et al. (1994) Defecography: Results of investigations in 2816 patients. Dis Colon Rectum 37:1133–1141
2. Bartolo DC, Bartram CI, Ekberg O et al. (1988) Proctography (symposium). Int J Colorectal Dis 3:67–69
3. Shorvon PJ, McHugh S, Somers S et al. (1987) Defecography findings in young healthy volunteers: results and implications. Gut 28:A1361–A1362
4. Agachan F, Pfeifer J, Wexner SD (1996) Defecography and proctography: results of 744 patients. Dis Colon Rectum 39:899–905

5. Siproudhis L, Dautreme S, Ropert A et al. (1993) Dyschezia and rectocele – a marriage of convenience? Physiologic evaluation of rectocele in a group of 52 women complaining of difficulty in evacuation. Dis Colon Rectum 36:1030–1036
6. Karlbom U, Graf W, Nilsson S et al. (1996) Does surgical repair of a rectocele improve rectal emptying? Dis Colon Rectum 39:1296–1302
7. Mahieu P, Pringot J, Bodart P (1984) Defecography: II. Contribution to the diagnosis of defecation disorders. Gastrointest Radiol 9:253–261
8. Ekberg O, Nylander G, Fort FT (1985) Defecography. Radiology 155:45–48
9. Shorvon PJ, Mc Hugh S, Diamant NE et al. (1989) Defecography in normal volunteers: results and implications. Gut 30:1737–1749
10. Yoshioka K, Matsui Y, Yamada O et al. (1991) Physiologic and anatomic assessment of patients with rectocele. Dis Colon Rectum 34:704–708
11. Johansson C, Nilsson BY, Holmstrom B et al. (1992) Association between rectocele and paradoxical sphincter response. Dis Colon Rectum 35:503–509
12. Cavallo G, Salzano A, Grassi R et al. (1991) Rectocele in males: clinical, defecographic, and CT study of singular cases. Dis Colon Rectum 34:964–966
13. Shafik A, Mostafa RM, Shafik AA et al. (2002) Study of the effect of straining on the bulbocavernosus muscle with evidence of a straining-bulbocavernosus reflex and its clinical significance. Int Urogynecol J 13:294–298
14. Shafik A (1991) Straining puborectalis reflex: Description and significance of a ''new'' reflex. Anat Rec 229:281–284
15. Shafik A (1995) The peno-motor reflex. Study of the response of the puborectalis and levator ani muscles to glans penis stimulation. Int J Impot Res 7:239–246
16. Shafik A (1995) Response of the urethral and intra-corporeal pressure to cavernosus muscle stimulation: Role of the muscles in erection and ejaculation. Urology 46:85–88
17. Shafik A (1995) The ''clitoromotor'' reflex. Int Urogynecol J 6:329–336
18. Symmonds RE (1991) Relaxation of pelvic support. In: Pernoll ML, Benson RC, eds. Current obstetric and gynecologic diagnosis and treatment, 7th edn. New Jersey, Prentice-Hall International Inc., 830–851
19. Kuhn RJ, Hollyock VE (1982) Observations on the anatomy of the rectovaginal pouch and septum. Obstet Gynecol 59:445–601
20. Johansson C, Nilsson BY, Mellgren A et al. (1992) Paradoxical sphincter reaction and associated colorectal disorder Int J Colorect Dis 7:89–92
21. Miller R, Duthie GS, Bartolo DC et al. (1991) Anismus in patients with normal and slow transit constipation. Br J Surg 78:690–692
22. Halligan S, Bartram CI, Park HJ et al. (1995) Proctographic features of anismus. Radiology 197:679–682
23. Van Dam JH, Schouten WR, Ginai AZ et al. (1996) The impact of anismus on the rectal clinical outcome of rectocele repair. Int J Colorect Dis 11:238–242
24. Tjandra JJ, Ooi BS, Tang CL et al. (1999) Transanal repair of rectocele corrects obstructed defecation if it is not associated with anismus. Dis Colon Rectum 42:1544–1550

Editorial comment

The investigation demonstrated decreased EMG activity and vaginal pressure in the women with rectoceles, especially during increased intra-abdominal pressure, compared to normal controls. Based on these data, the authors theorize that the decreased vaginal pressure results from poor tone and blunted reflex contraction of the BCM and PRMS during increases in intra-abdominal pressure, which in normal women closes the vaginal hiatus causing an equilibration of increased intra-abdominal pressure on the rectal and vaginal sides of the rectovaginal septum. This is a novel theory for the pathogenesis of rectocele and is supported by these preliminary data. The fact that the subjects had a stool frequency of less than twice weekly is more consistent with defecatory dysfunction secondary to a motility disorder rather than outlet obstruction. This raises the question of whether the rectocele is a result of the defecatory dysfunction rather than causative, and affects the external validity of the study population. Additionally, the theory fails to explain the association of paradox with rectocele. Nevertheless, this theory merits further investigation as one of several potential etiologies of rectocele.