World Journal of Medical Sciences 16 (2): 79-85, 2019
ISSN 1817-3055

DOI: 10.5829/idosi.wjms.2019.79.85

Pelvic Floor Electrophysiological Changes Associated with Female Pelvic Organ Prolapse

Emmanuel Kamal Aziz Saba and Mervat Sheta Elsawy

Physical Medicine, Rheumatology and Rehabilitation Department,
Faculty of Medicine, Alexandria University, Alexandria, Egypt

Abstract: The study aimed to assess and determine different pelvic floor electrophysiological changes and patterns associated with female pelvic organ prolapse. The current study included 30 non-virgin female patients with pelvic organ prolapse and 11 apparently healthy female subjects as control group. Anal manometry and pelvic floor electrophysiological studies were done. The electrophysiological assessment included pudendal nerve terminal motor latency assessment, pudendo-anal reflex and needle electromyography of the external anal sphincter, external urethral sphincter and puborectalis muscles. Results displayed that most common pelvic floor electrophysiological changes in female pelvic organ prolapse were bilateral pudendal neuropathy, abnormal pudendo-anal reflex, denervation, reinnervation and incomplete interference pattern of external anal sphincter, external urethral sphincter and puborectalis muscles; anismus in the external anal sphincter and puborectalis muscles; and localized defect in external anal sphincter muscle. There were three different pelvic floor electrophysiological patterns among the studied patients depending on the results of pudendal nerve terminal motor latency assessment. Conclusions: There were various different pelvic floor electrophysiological changes and patterns associated with female pelvic organ prolapse. These electrophysiological changes represent a spectrum of changes which extend from minimal to severe neuromuscular abnormalities.

Key words: Electromyography · Female Pelvic Organ Prolapse · Pelvic Floor Muscles · Pudendal Nerve Terminal Motor Latency · Pudendo-anal Reflex

INTRODUCTION

Female pelvic organ prolapse (POP) is the downward descent of one or more of female pelvic organs whether urinary bladder, uterus, vaginal wall and intestine from their normal anatomical sites towards or through the vaginal opening [1-3]. POP is considered a form of pelvic floor dysfunction [4]. POP is a common condition among women [5, 6]. No age is immune. However, POP is a common condition in older women [2]. There is no single etiology for the development of POP. There are usually multiple factors combined together in the women resulting in the initiation and perpetuation of POP. These include vaginal delivery, obesity, menopause, advance aging, perineal trauma, chronic increase in the intra-abdominal pressure from chronic straining with constipation, chronic cough, repeated heavy lifting due to occupational causes, abnormalities in the pelvic floor connective tissue and ethnicity. All of them lead to the loss of the pelvic floor support with the resultant of POP [2-4].

There are a lot of evidence about the role of pelvic floor neuromuscular damage in the development of POP [1, 7, 8]. There are a variety of pelvic floor electrophysiological tests that assess different neuromuscular structures of the pelvic floor as electromyography and electrophysiological reflex studies [7, 9, 10]. There are scanty studies that assessed different pelvic floor electrophysiological changes associated with female patients with POP [7, 11]. The aim of the current study was to assess and determine different pelvic floor electrophysiological changes and patterns in female patients with POP.

Corresponding Author: Emmanuel Kamal Aziz Saba, Department of Physical Medicine, Rheumatology and Rehabilitation Department, Faculty of Medicine, Alexandria University, Alexandria, Egypt.
E-mail: emadaziz55@yahoo.com.

MATERIALS AND METHODS

The current cross-sectional study included 30 non-virgin female patients with POP. They were recruited randomly from those attending the Pelvic Floor Rehabilitation outpatient clinic of Physical Medicine, Rheumatology and Rehabilitation Department, Main University Hospital, Faculty of Medicine, Alexandria University, Egypt. The study included a control group of 11 apparently healthy non-virgin females free from any symptoms and signs of POP. Exclusion criteria included pregnancy, diabetes mellitus, metabolic disorders, endocrine disorders, neurological disorders including upper motor neuron lesions, cauda equine lesions, lumbosacral plexopathy and peripheral neuropathy; and malignancies and previous surgery for malignancies in the pelvic region. The study was explained to the participants and an informed consent was given by each. The local Institutional Ethics Committee of the Faculty of Medicine, Alexandria University, Egypt had approved the study.

The patients were subjected to the following: demographic data collection, anthropometric measurement and history taking stressing on the risk factors for POP and different symptoms of POP [1, 2, 12].

Clinical examination was done. Badden and Walker clinical grading of POP was done [13] while the patient was in lithotomy position and performing maximal valsalva. Badden and Walker clinical grading has 5 grades as the following: Grade (0) there is no prolapse; grade (1) the prolapse is halfway to the hymenal ring; grade (2) the prolapse reaches the hymenal ring; grade (3) the prolapse exceeds the hymenal ring; and grade (4) maximum descent of the prolapse past the hymenal ring [4, 13]. Anorectal examination was done to assess pelvic floor muscle strength using Modified Oxford Scale (MOS) [14]. The muscle strength was quantified from zero to five according to the MOS. Zero indicates absence of muscle contraction while five indicates a completely normal muscle strength.

Anal manometry measurements were performed using the manometric biofeedback device (Myomed 632-equipment, Enraf Nonius, Delft, Netherlands). Maximum resting and maximum squeeze anal pressures were measured [15].

Pelvic floor electrophysiological studies were conducted on a NIHON KOHDEN Neuropack MEB-7102 mobile unit with a two channel evoked potential/EMG measuring system (Nihon Kohden Corp., Tokyo, Japan). This included the following: (1) Pudendal nerve terminal motor latency (PNTML) assessment using St. Mark electrodes [16]. (2) Pudendo-anal reflex (PAR) assessment [16]. (3) Needle electromyography (EMG) for the external anal sphincter (EAS) muscle, external urethral sphincter (EUS) muscle and puborectalis (PR) muscle [16].

Statistical analysis of data was done by using the Statistical Package of Social Science (SPSS version 17) software [17]. Descriptive measures included count, frequency, minimum, maximum, median, mean and standard deviation (SD). Analytic measures included Mann Whitney test, Chi-square test or Fisher's Exact test (when required) and Spearman correlation test. Statistical significance was assigned to any P value at <0.05.

RESULTS

The current study included 30 non-virgin female patients with POP. Their mean age was 40.33±10.53 years (ranged from 24 to 64 years). The control group consisted of 11 apparently healthy non-virgin females and their mean age was 42.36±8.30 years (ranged from 28 to 55 years). There were no statistical significant differences between patients and control group regarding age (Z=-0.810, P=0.418). The demographic, anthropometric, clinical and manometric characteristics of the patients group and control group are tabulated in Table 1.

The most common risk factors for POP were the presence of chronic straining in 24 patients (80%), obesity in 13 patients (43.3%), followed by menopause in 7 patients (23.3%) (Table 1). There were 26 patients (86.66%) had vaginal delivery. There were 13 patients (43.3%) with history of obstetric trauma. Past history of hysterectomy operation was present in 3 patients (10%) and haemorrhoidectomy in 5 patients (16.66%).

The most common POP symptoms were bowel symptoms mainly obstructed defecation in 18 patients (60%), urinary symptoms in the form of urinary incontinence in 15 patients (50%) and pelvic organ bulge in 9 patients (30%). Rectocele was the most common finding and it was present in 29 patients (96.7%) followed by increased perineal descent in 28 patients (93.34%) (Table 1). The maximum resting and maximum squeeze anal pressures were significantly lower among POP patients in comparison to healthy control group (Table 1).

The PNTML and PAR latencies were significantly delayed among POP patients in comparison to the control group (Table 2).

There was a statistical significant negative correlation between PNTML and MOS (r=-0.475, P=0.016) and maximum squeeze anal pressure (r=-0.660, P<0.0001). There was a statistical significant negative correlation between PAR latency and MOS (r=-0.519, P=0.013) and maximum squeeze anal pressure (r=-0.761, P<0.0001).

Table 1: Demographic, anthropometric, clinical and manometric characteristics of the patients group and control group

Demographic, anthropometric, clinical and manometric characteristics of the participants	Patients group (n=30) Median(mean±SD)	Control group (n=11) Median(mean±SD)	Test of significant	*P*
Age (year)	38.00(40.33±10.53)	42.00(42.36±8.30)	Z=-0.810	0.418
Parity†	2(0-5)	2(0-4)	Z=-0.045	0.965
Menopause‡	7(23.3)	5(45.5)	X^2=1.903	0.247§
Duration of the symptoms (months)	8(9.86±6.98)	NA	NA	NA
Anthropometric measures				
Height (cm)	162.00(161.80±8.52)	161.00(160.54±7.47)	Z=-0.413	0.695
Weight (kg)	78.00(75.56±14.52)	72.00(74.54±14.20)	Z=-0.383	0.717
BMI (kg/m^2)	28.85(28.87±5.34)	28.30(28.97±5.37)	Z=-0.059	0.965
Clinical findings				
Rectocele‡	29(96.7)	NA	NA	NA
Rectocele grade		NA	NA	NA
Grade 1‡	21(70.0)	NA	NA	NA
Grade 2‡	7(23.3)	NA	NA	NA
Grade 3‡	1(3.3)	NA	NA	NA
Cystocele‡	17(56.7)			
Cystocele grade		NA	NA	NA
Grade 1‡	16(53.3)	NA	NA	NA
Grade 2‡	1(3.3)	NA	NA	NA
Uterine prolapse‡	11(36.7)	NA	NA	NA
Uterine prolapse grade		NA	NA	NA
Grade 1‡	4(13.3)	NA	NA	NA
Grade 2‡	3(10.0)	NA	NA	NA
Grade 3‡	4(13.3)	NA	NA	NA
Presence of increased perineal descent‡	28(93.34)	NA	NA	NA
MOS†	3(2-4)	5(4-5)	Z=-4.732	<0.0001*
Anal manometric assessment				
Maximum resting anal pressure (hpa)	20.50(20.76±8.15)	33.00(34.00±5.54)	Z=-4.228	<0.0001*
Maximum squeeze anal pressure (hpa)	64.50(74.26±27.30)	91.00(93.36±17.04)	Z=-2.296	0.021*

cm, centimeter; kg, kilogram; BMI, body mass index; VAS, visual analogue scale; POP, pelvic organ prolapse; MOS, Modified Oxford Scale; hpa, hectopascal (it is the unit of pressure and it is equal to 100 pascals); n, number of subjects; SD, standard deviation; Z, value of Mann Whitney test; X^2, value of Chi-square test; NA, not applicable.

**P* is significant at <0.05.

†Data are reported as median (minimum-maximum).

‡Data are reported as number (percentage).

§ Fisher Exact test.

Table 2: Comparison of pudendal nerve terminal motor latency and pudendo-anal reflex latency between patients group and control group

Electrophysiological studies	Patients group (n=30) Median (mean±SD)	Control group (n=11) Median (mean±SD)	Test of significant†	*P*
Rt side PNTML (ms)	2.30(2.97±1.24)	1.92(1.99±0.30)	-3.720	<0.0001*
Lt side PNTML (ms)	2.32(3.11±1.35)	2.00(1.98±0.23)	-3.980	<0.0001*
Rt PAR latency (ms)	47.60(49.00±9.37)	39.50(39.74±3.20)	-3.000	0.002*
Lt PAR latency (ms)	42.95(48.39±9.43)	39.40(39.91±3.16)	-2.636	0.007*

Rt, right; PNTML, pudendal nerve terminal motor latency; Lt, left; PAR, pudendo-anal reflex; n, number of subjects; SD, standard deviation.

**P* is significant at <0.05.

† Value of Mann Whitney test

The frequency of different pelvic floor electrophysiological abnormalities and patterns among patients group are tabulated in Table 3. There were three different pelvic floor electrophysiological patterns among POP patients depending on the results of PNTML assessment. The most common pattern was pattern III.

Table 3: Frequency of pelvic floor electrophysiological abnormalities and patterns among patients group

		Pelvic floor electrophysiological patterns according to pudendal nerve terminal motor latency status		
Pelvic floor electrophysiological abnormalities	Patient group (n=30) n(%)*	Pattern I (normal PNTML) n(%)*	Pattern II (unilateral PNTML delay) n(%)*	Pattern III (bilateral PNTML delay) n(%)*
Pudendal nerve terminal motor latency status (normal/ unilateral delay /bilateral delay)	11/4/15 (36.7/13.3/50)	11(36.7)	4(13.3)	15(50)
Abnormal PAR latency (unilateral /bilateral)	16(3/13) [53.3(10/43.3)]	0(0)	2(1/1) [6.7(3.35/3.35)]	14(2/12) [46.6(6.65/39.95)]
EMG of EAS				
EAS denervation	20(66.7)	4(13.3)	2(6.7)	14(46.7)
EAS reinnervation	30(100)	11(36.7)	4(13.3)	15(50)
EAS incomplete interference	21(70.0)	4(13.3)	3(10)	14(46.7)
EAS anismus	16(53.3)	3(10)	2(6.6)	11(36.7)
EAS localized defect	7(23.3)	2(6.7)	0(0)	5(16.6)
Abnormal EAS EMG	30(100)	11(36.7)	4(13.3)	15(50)
EMG of EUS				
EUS denervation	18(60.0)	4(13.3)	2(6.7)	12(40)
EUS reinnervation	23(76.7)	8(26.7)	2(6.7)	13(43.3)
EUS incomplete interference	11(36.7)	1(3.3)	2(6.7)	8(26.7)
Abnormal EUS EMG	23(76.7)	8(26.7)	2(6.7)	13(43.3)
EMG of PR				
PR denervation	20(66.6)	4(13.3)	1(3.3)	15(50)
PR reinnervation	30(100)	11(36.7)	4(13.3)	15(50)
PR incomplete interference	27(90)	9(30)	4(13.3)	14(46.7)
PR anismus	23(76.7)	8(26.7)	1(3.3)	14(46.7)
Abnormal PR EMG	30(100)	11(36.7)	4(13.3)	15(50)
Total number of patients	30(100)	11(36.7)	4(13.3)	15(50)
Forms of POP	R=11(36.7) C=1(3.3) R+C=7(23.3) R+U=2(6.7) R+C+U=9(30)	R=7(23.4) R+C=3(10) R+U=1(3.3)	R=2(6.7) C=1(3.3) R+C+U=1 (3.3)	R=2(6.7) R+C=4(13.3) R+U=1(3.3) R+C+U=8 (26.7)

PAR, pudendo-anal reflex; EMG, electromyography; EAS, external anal sphincter muscle; EUS, external urethral sphincter muscle; PR, puborectalis muscle; POP, pelvic organ prolapse; n(%), number (percentage) of patients; PNTML, pudendal nerve terminal motor latency; R, rectocele; C, cystocele, U, uterine prolapse.

* Data are reported as number (percentage) of patients with abnormalities

DISCUSSION

Pelvic floor electrophysiology is important in the assessment of pelvic floor muscles and nerves in female patients with POP [18]. In the current study, there was 63.3% of the patients had pudendal neuropathy. The pudendal neuropathy could be due to stretch injury of the pudendal nerve during vaginal delivery (especially prolonged and difficult deliveries) and/or it is a sort of traction neuropathy due to increased perineal descent that takes place with chronic straining. The down movement of the pelvic floor structures stretches the pudendal nerve from its fixation point at the ischial spine [3, 19]. Detection of pudendal neuropathy is essential for proper management of POP [20]. The presence of pudendal neuropathy especially bilateral pudendal neuropathy is associated with poor surgical outcome [21]. This study is in accordance with previous studies [7, 22]. Lacima *et al.* [7] reported that pudendal neuropathy was present in 43% of female patients with combined faecal and urinary incontinence.

In the present study, delayed PAR latency was found in 53.3% of the patients. This could be explained by the presence of an abnormal reflex arc due to the presence of pudendal neuropathy. This percentage was less than the percentage of pudendal neuropathy among the studied patients. It was reported that PAR latency lack sensitivity in detecting minimal to moderate pudendal neuropathy. The PAR is not sensitive to incomplete nerve lesions (whether demyelinating or axonal lesions) because its

assessment is based on conduction (and not compound muscle action potential amplitude) [23]. This could be because PAR latency has a wide physiological range of latencies. Minimal delay in the PAR latency could takes place and still the PAR latency is within the accepted limit [9]. Subsequently, a normal PAR latency dose not exclude a lesion [16, 23]. The current study is in agreement with other studies [9, 24].

In the current work, EMG abnormalities in the EAS were present in all patients (100%). The EAS muscle had ongoing neurogenic injury as indicated by the presence of denervation potentials in EMG. This could be due to pudendal neuropathy as a consequence of perineal descent during delivery or due to chronic straining [25, 26]. The presence of neuropathic motor unit action potentials in the EAS muscle indicates a process of chronic reinnervation following denervation [9]. The presence of denervation with reinnervation indicates the presence of incomplete recovery of the axonopathic injury of the pudendal nerve [9]. This is in agreement with previous studies [9, 27]. There were seven patients (23.3%) had a localized defect in the EAS muscle detected by EMG mapping. The localized defect could be a contributing factor for weakness of the pelvic floor muscles with subsequent POP development [3, 4, 28].

In the present research, EUS muscle had EMG abnormalities in 23 patients (76.7%). These abnormalities were similar to that found in the EAS muscle, in spite that EUS muscle is supplied by the perineal branch of the pudendal nerve while EAS muscle is supplied by the inferior rectal branch of pudendal nerve. The current study is in agreement with previous studies [6, 29, 30]. The lower percentage of EMG abnormalities in the EUS muscle in comparison to EAS muscle, indicated that stretch pudendal neuropathy affecting the inferior rectal branch of the pudendal nerve was more than that affecting the perineal branch of the pudendal nerve. This could be due to the longer anatomical course of the perineal branch of the pudendal nerve [31].

In the current study, EMG abnormalities in the PR muscle were present in all patients (100%). These abnormalities were similar to those present in the EAS muscle. The explanation of these abnormalities is similar to those of the EAS muscle, in spite of the difference in the motor nerve supply between PR and EAS muscles. This is in agreement with previous studies [9, 27, 32, 33].

The presence of incomplete interference pattern at squeezing in the pelvic floor muscles including EAS, EUS and PR muscles reflect the defect in their role in protection against POP and continence control. These were reflected by the presence of statistical significant negative correlations between PNTML, as well as, PAR latency with MOS and maximum squeeze anal pressure. This means that the more the severity of pudendal neuropathy the more is the weakness of the pelvic floor muscles mainly EAS muscle [3, 8, 9, 16].

In the current research, there was electrophysiological evidence of anismus in the EAS and PR muscles. This could be a predisposing factor for POP in the form of obstructed defecation that was reported in 18 patients (60%). Obstructed defecation is a common cause of chronic straining [34-36].

The current study is in agreement with other studies that assessed the electromyographic changes among POP patients [37, 38]. Weidner *et al*. [37] and Aanestad *et al.* [38] reported that there were neuropathic changes in EAS and levator ani muscles in the form of denervaion with reinnervaiton among women with POP.

The electrophysiological assessment of the pudendal nerves and pelvic floor muscles showed that all female patients with POP had different degrees of neuromuscular defects. This is essential in taking the decision regarding the effective treatment of patients with POP which include conservative and surgical treatment [39].

In the present work, pelvic floor electrophysiological changes detected in POP patients were categorized into three different patterns depending on the PNTML results. The most common pattern was pattern III. This pattern was present in 15 patients (50%). This pattern represents bilateral pudendal neuropathy in the form of delayed PNTML and delayed PAR latency with denervation and reinnervation in the EAS and EUS muscles. The pathology of pudendal neuropathy is mixture of axonopahtic lesion with demyelinating lesion. The axonopahtic lesion leads to denervation with subsequent reinnervation in the EAS and EUS muscles. The demyelinating lesion leads to delayed PNTML and PAR latency. This was associated with denervation with subsequent reinnervation in the PR muscle [9].

The second most common pattern was pattern I. It was present in 36.7% of the patients. In this pattern, the right and left PNTMLs were not delayed. However, the presence of EMG abnormalities in the EAS and EUS muscles indicate the presence pudendal neuropathy of mild degree. The EMG changes in the PR muscle could be due to stretch neuropathy of its motor nerve supply that originate directly from the sacral plexus [9, 24].

In the present study, these three different electrophysiological patterns represent a wide spectrum of neuromuscular changes that take place in the pelvic

floor muscles in POP. They range from mild to severe changes. They indicate the presence of variable degree of neurogenic lesions affecting the pelvic floor muscles and pudendal nerves.

The present study had some limitations. First limitation was the relatively small number of studied patients. This was due to the wide range of exclusion criteria in the current study, the preference of patients with POP to seek gynecological consultation for their condition and the presence of some patients who refused the participation in the study. Second limitation, dynamic magnetic resonance imaging for assessment of POP was not done. It was reported that there was a good correlation between imaging and physical examination in POP [40].

CONCLUSIONS

There were various different pelvic floor electrophysiological changes and patterns associated with female POP. These included pudendal neuropathy, abnormal PAR, denervation, reinnervation and incomplete interference pattern of the EAS, EUS and PR muscles; and anismus in the EAS and PR muscles. These electrophysiological changes represent a spectrum of changes which extend from minimal to severe neuromuscular abnormalities.

REFERENCES

1. Jelovsek, J.E., C. Maher and M.D. Barber, 2007. Pelvic organ prolapse. Lancet, 369: 1027-1038.
2. Kuncharapu, I., B.A. Majeroni and D.W. Johnson, 2010. Pelvic organ prolapse. Am. Fam. Physician, 81: 1111-1117.
3. Torre, F.I., F. Pucciani, G. Dodi, G. Giuliani, A. Frasson, D. Coletta and P. Petros, 2017. Pelvic organ prolapse working group: doubts and evidence for practical guide. J. Inflam. Bowel Dis. and Disord., 2: 115.
4. Chen, G.D. and S.C. Ng, 2007. Updated definition of female pelvic organ prolapse. Incont. Pelvic Floor Dysfunct., 1: 121-124.
5. Thakar, R. and S. Stanton, 2002. Management of genital prolapse. B.M.J., 324: 1258-1262.
6. Bozkurt, M., A.E. Yumru and L. Sahin, 2014. Pelvic floor dysfunction and effects of pregnancy and mode of delivery on pelvic floor. Taiwanese Journal of Obstetrics and Gynecology, 53: 452-458.
7. Lacima, G., M. Pera, J. Valls-Sole, X. Gonzalez-Argente, M. Puig-Clota and M. Espuna, 2006. Electrophysiologic studies and clinical findings in females with combined fecal and urinary incontinence: a prospective study. Dis. Colon Rectum, 49: 353-359.
8. Kapoor, D.S., R. Thakar and A.H. Sultan, 2005. Combined urinary and faecal incontinence. Int. Urogynecol., 16: 321-328.
9. Sultan, H.A., M.H. Zahran, I.K. Ibrahim, M.A. Shehata, G.A. El-Tantawi and E.K. Saba, 2013. Pelvic floor electrophysiology patterns associated with faecal incontinence. Alex. J. Med., 49: 111-117.
10. Kenton, K., 2005. Pelvic floor neurophysiology. Rochester: American Association of Neuromuscular and Electodiagnostic Medicine.
11. Ibrahim, I.K., M.M. Abdel Hameed, E.M. Taher, E.M. Shaheen and M.S.A.G. Elsawy, 2015. Efficacy of biofeedback-assisted pelvic floor muscle training in females with pelvic floor dysfunction. Alex. J. Med., 51: 137-142.
12. Shek, K.L. and H.P. Dietz, 2016. Assessment of pelvic organ prolapse: a review [Editorial]. Ultrasound Obstet. Gynecol., 48: 681-692.
13. Baden, W.F. and T.A. Walker, 1972. Genesis of the vaginal profile: a correlated classification of vaginal relaxation. Clin. Obstet. Gynecol., 15: 1048-1054.
14. Lang, J., H. Brown and E. Crombie, 2007. Assessment of the anal sphincter muscle: comparison of a digital and a manometric techniques. Physiotherapy, 93: 121-128.
15. Gad El-Hak, N., M. El-Hemaly, T. Salah, A. El-Raouf, M. Haleem and H. Hamed, 2007. Normal variation of anorectal manometry among the Egyptian population. Arab. J. Gastrooenterol., 8: 53-56.
16. Roberts, M., 2008. Neurophysiology in neurourology. Muscle Nerve, 38: 815-836.
17. SPSS, 2007. Statistical package for the social sciences, version 17. London: University of Cambridge Computing Service.
18. Vodusek, D., 2002. The role of electrophysiology in the evaluation of incontinence and prolapse. Curr. Opin. Obstet. Gynecol., 14: 509-514.
19. Snooks, S.J., M. Swash, M.M. Henry and M. Setcheill, 1986. Risk factors in childbirth causing damage to the pelvic floor innervation. Int. J. Colorect. Dis., 1: 20-24.
20. Weber, A.M. and H.E. Richter, 2005. Pelvic organ prolapse. Obstet. Gynecol., 106: 615-634.

21. Phillips, R. and T. Brown, 2009. Surgical management of anal incontinence. Part A. Secondary anal sphincter repair. In: Perineal and anal sphincter trauma. Eds., Sultan, A., R. Thakar and D. Fenner. London: Springer-Verlag, pp: 144-153.
22. Beevors, M.A., D.Z. Lubowski, D.W. King and M.A. Carlton, 1991. Pudendal nerve function in women with symptomatic utero-vaginal prolapse. Int. J. Colorect. Dis., 6: 24-28.
23. Fowler, C., 1995. Pelvic floor neurophysiology. In: Clinical neurophysiology: electromyography, nerve conduction and evoked potentials. Eds., Binnie, C., R. Cooper, C. Fowler, F. Mauguiere and P. Prior. Oxford: Butterworth-Heinemann, pp: 233-252.
24. Fitzpatrick, M., C. Brien, P. Connell and C. Herlihy, 2003. Patterns of abnormal pudendal nerve function that are associated with postpartum faecal incontinence. Am. J. Obstet. Gynaecol., 189: 730-735.
25. Benson, J.T., 1990. Clinical application of electrodiagnostic studies of female pelvic floor neuropathy. Int. Urogynecol. J., 1: 164-167.
26. Allen, R.E., G.L. Hosker, A.R. Smith and D.W. Warrell, 1990. Pelvic floor damage and childbirth: a neurophysiological study. Br. J. Obstet. Gynaecol., 97: 770-779.
27. Snooks, S., M. Henry and M. Swash, 1985. Anorectal incontinence and rectal prolapse: differential assessment of the innervation to puborectalis and external anal sphincter muscles. Gut, 26: 470-476.
28. Lindsey, I., O. Jones, M. Humpherys, C. Cunningham and N. Mortensen, 2004. Patterns of faecal incontinence after anal surgery. Dis. Colon Rectum, 47: 1643-1649.
29. Eardley, I. and C.J. Fowler, 1993. Urethral sphincter electromyography. Int. Urogynecol., 4: 282-286.
30. Fowler, C.J., R.S. Kirby and M.J.G. Harrison, 1984. Individual motor unit analysis in the diagnosis of disorders of urethral sphincter innervation. J. Neurol. Neurosurg. Psych., 47: 637-641.
31. Thakar, R. and D. Fenner, 2009. Anatomy of the perineum and the anal sphincter. In: Perineal and anal sphincter trauma: diagnosis and clinical management. Eds., Sultan, A., R. Thakar and D. Fenner. London: Springer-Verlag, pp: 1-12.
32. Dietz, H.P. and J.M. Simpson, 2008. Levator trauma is associated with pelvic organ prolapse. B.J.O.G., 115: 979-984.
33. Lien, K.C., B. Mooney, J.O. De Lancey and J.A. Ashton-Miller, 2004. Levator ani muscle stretch induced by simulated vaginal birth. Obstet. Gynecol., 103: 31-40.
34. Duthie, G. and D. Bartolo, 1992. Anismus: the cause of constipation? Results of investigation and treatment. World J. Surg., 16: 831-835.
35. Jelovsek, J.E., M.D. Barber, M.F.R. Paraiso and M.D. Walters, 2005. Functional bowel and anorectal disorders in patients with pelvic organ prolapse and incontinence. Am. J. Obstet. Gynecol., 193: 2105-2111.
36. Saba, E.K.A., G.A.Y. El-Tantawi, M.H. Zahran, I.K. Ibrahim, M.A. Shehata, H.A. Sultan and M. Anwar, 2015. Pelvic floor electrophysiology patterns associated with obstructed defecation [abstract]. International Journal of Medical and Health Sciences, 2: 118.
37. Weidner, A.C., M.D. Barber, A.G. Visco, R.C. Bump and D.B. Sanders, 2000. Pelvic muscle electromyography of levator ani and external anal sphincter in nulliparous women and women with pelvic floor dysfunction. Am. J. Obstet. Gynecol., 183: 1390-1399.
38. Aanestad, O. and R. Flink, 1999. Urinary stress incontinence. A urodynamic and quantitative electromyographic study of the perineal muscles. Acta. Obstet. Gynecol. Scand., 78: 245-253.
39. Giarenis, I. and D. Robinson, 2014. Prevention and management of pelvic organ prolapse. F1000Prime Reports, 6: 77 (doi:10.12703/P6-77).
40. Azab, I.A., M.A. Nasef and A.M. Ibrahim, 2014. Dynamic magnetic resonance imaging; reliability of assessment and correlation with clinical findings of pelvic organ prolapse. Egypt. J. Radio. Nucl. Med., 45: 1003-1010.