

PELVIC FLOOR DYSFUNCTION IN AGING WOMEN

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SUMMARY

The occurrence of pelvic floor dysfunction may increase steadily during the aging process in women. Pelvic floor dysfunction may be associated with dysfunctions of micturition, defecation, prolapse, and sex. The natural history and mechanism of pelvic floor dysfunction in aged women are not well understood or explored. In this article, we review the effect of age on the prevalence of pelvic floor dysfunction and on the structural and functional changes of the lower urinary tract, anorectum and pelvic floor. Altogether, the aging process has a negative impact on either the function or structure of the lower urinary tract, anorectum and pelvic floor in women. [Taiwan J Obstet Gynecol 2007;46(4):374-378]

Key Words: aging, anal function, elderly, lower urinary tract dysfunction, pelvic floor dysfunction, pelvic floor

Introduction

Pelvic floor dysfunction impairs the lives (or quality of life) of a large number of women of all ages throughout the world. A higher life expectancy, owing to modern medical achievement, has added about 10 years (or even more) to a woman's previous life expectancy of 65 years. The occurrence of pelvic floor dysfunction may increase steadily during the aging process in women [1]. However, only a limited number of studies have assessed the effect of age on the structure and function of the pelvic floor in the absence of disease [2]. Estimates of the prevalence of pelvic floor dysfunction in this population vary widely because of the absence of standardized and well-validated definitions and measures. Interpretation of data derived from epidemiology surveys is also difficult, because levels of dependency may differ in different residential settings and different countries.

The natural history of pelvic floor dysfunction in aged women is not well understood. In the past, parity and mode of delivery [3] were included in epidemiologic studies; and later, menopausal estrogen deficiency,

higher body mass index, previous pelvic surgery, and comorbidity such as diabetes mellitus and hypertension history were also included in epidemiologic studies to evaluate the potential contributory factors for the occurrence of symptoms of pelvic floor dysfunction in women [4-7]. Some comorbidities may coexist in women with increasing age and frailty. For the purpose of examining the effect of aging on the pelvic floor function, these potential predisposing factors should be seen as confounding or add-on effects through the whole life time of women, which will require a longitudinal study to clarify this issue. In my opinion, the functional and structural changes of the pelvic floor in aging women are the carry-over (or so-called summative) effects of the above factors altogether.

Prevalence of Symptoms of Pelvic Floor Dysfunction in Aging Women

Pelvic floor symptoms, which include five types of dysfunction (i.e. dysfunctions of micturition, defecation, prolapse, sex and pain), are known to increase with age [1]. In previous studies, my colleagues and I found that lower urinary tract symptoms during filling and voiding phase were not only affected by the pre- to postmenopausal transition but were also closely associated with aging changes [8]. The odds ratio for the presence of an overactive bladder (OAB) in community women over 65 years old was 1.49 [5]. Another study

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conducted for hospital-based patients showed that female OAB syndrome was a highly prevalent condition in each birth cohort, with a statistically significant increase with advancing age (65 years or older) [7]. The results of this survey in Taiwan were similar to those in the US and Europe [9,10]. The US survey results of the National Overactive Bladder Evaluation Program [9] showed that age-specific prevalence of "OAB wet" (OAB with urge incontinence) continues to increase with increasing age. Moreover, at older ages, the transition rate from OAB without to OAB with urge incontinence may exceed the rate of occurrence of new cases of OAB without incontinence. The population-based prevalence of OAB symptoms among men and women aged 40 years or older from six European countries revealed that the overall prevalence of OAB in individuals aged 40 years or older was 16.6%. However, the prevalence of OAB elevates after the age of 65 years (65–69 years: 17.5%; 70–74 years: 22.1%; 75 years or older: 31.3%). Goepel et al [11] also showed that women aged 50–59 years had an odds ratio of frequency, urgency, urge incontinence and stress incontinence of greater than 5, and greater than 8 for the above symptoms for those aged 60–79 years, and greater than 20 for the above symptoms for women aged 80 years or older. A previous study by Lin et al [12] also demonstrated that the increasing occurrence of nocturia was age-related and not affected by the transition from pre- to postmenopause in community women.

In the past, voiding difficulty, defined as abnormally slow and/or incomplete micturition, has been relatively overlooked as a diagnosis in comparison with urodynamic stress incontinence, OAB, and uterovaginal prolapse [13]. Haylen et al [13] analyzed 592 women of a median age of 57 years (range, 16–98 years) with symptoms of lower urinary dysfunction and found that the prevalence of voiding difficulty was 39% for women who were referred for an initial urogynecologic assessment including urodynamics. The prevalence of voiding difficulty increased significantly with age and increasing grades of all types of uterovaginal prolapse. More than 60% of women with voiding difficulty had a maximal flow rate under the 10th percentile of the equivalent Liverpool nomogram [14] and/or a residual urine volume of more than 30 mL.

In addition to an increased presence of lower urinary tract dysfunction with age, a previous community survey in 1999 by Chen et al [6] found no significant difference of occurrence of anal incontinence among each birth cohorts (cohorts were divided into decades of age, beginning with 20 years old and finishing with women older than 65 years who were included in the same birth cohort), and the prevalence of anal incontinence

did not increase after the age of 65 years. However, the prevalence of constipation increased significantly in elderly women aged 65 years and over, and one-fourth of the women with constipation in that study needed to take laxatives for defecation. Data derived from the epidemiology of constipation in the United States showed that the prevalence of constipation increased in the sixth decade of life and that one-third of elderly patients complained of constipation [15].

A study in 2002 by Ng et al [4] revealed an inconsistent result compared with that found in community women. Three hundred and eight women with a mean age of 45.14 years were referred to our urogynecology outpatient clinic for urodynamic evaluation. We found that 31.5% of the women with lower urinary tract symptoms had constipation, a prevalence that was much higher than that of the general population [16]. However, constipation was significantly more prevalent among the younger cohort and also among women aged 70 years and older. The odds ratio for elderly women with anal incontinence was 1.77 (95% confidence interval, 0.33–9.41) and that for those with constipation was 2.18 (95% confidence interval, 0.35–8.87).

The first comprehensive population survey of pelvic floor dysfunction was conducted on South Australian adults (15 years and older) in 1998. All participants were interviewed by trained female interviewers. The results revealed that, overall, the prevalence of pelvic floor disorders, including urinary incontinence, anal incontinence, previously pelvic repairs, difficulty with defecation and hemorrhoids, was greater in women aged 65 years and older [3].

Changes of Pelvic Floor Function During Aging Process

A hospital-based study by Hung et al [7] showed that the prevalence of OAB syndrome increased significantly with advancing age (65 years or older). The percentage of patients with OAB wet were as high as 33.3% in women after the age of 65 years. However, the elderly have an increased tendency towards incomplete bladder emptying, and this frequently coexists with detrusor instability [17]. A series of studies by Griffiths et al [18] showed that elderly patients may have underperfusion of the frontal lobes of the cerebral cortex (reflecting regional impairment of function), which appears to be causally related to urge incontinence and reduced bladder sensation. They also demonstrated that factors, such as reduced sensation of bladder filling, previous bacteriuria, elevated fluid intake and infrequent voiding,

may contribute to severity of urine leakage in elderly patients [19]. Incomplete bladder emptying, which elevates post-void residual, may be caused by impaired detrusor contractility. The impaired detrusor contractility in elderly patients may have two aspects: elevated post-void residual urine volume and reduced detrusor contraction strength. Griffith et al [20] defined a patient with urge incontinence, poor detrusor contractility, and elevated residual urine without urethral obstruction as having detrusor hyperactivity with impaired contractile function. These kinds of functional impairments may be found in elderly patients and present as a challenging clinical problem for these patients. However, voiding disorders in the elderly may occur in the absence of symptoms. Therefore, it is not clear which patients with voiding disorders can be safely left unattended [21].

In addition to changes in the central nervous system, the function of peripheral nerves may also attenuate during the aging process. Gilpin et al [22] found that a significant linear reduction in the amount of acetylcholinesterase-positive nerves and reduction in the number of nerve axons in human detrusor muscle tissue occurred with increasing age. This phenomenon may be associated with a variety of functional bladder impairments in advanced age. Homma et al [23] conducted a survey and urodynamics on an elderly population who had no spontaneous complaints of symptoms and revealed that nearly all of the urinary symptoms increased with age. They also found that the detrusor function, such as maximal detrusor pressure and maximal flow rate, progressively deteriorated with age in women. Urethral pressure may also be affected by aging. Trowbridge et al [24] discovered that increasing age was associated with a decreasing maximal urethral closure pressure averaging a 15 cmH₂O drop per decade in nulliparous women.

Anorectal function may also deteriorate during the aging process. Ng and Chen [25] found that aging was closely associated with reduced anorectal pressure in anal-continent women with lower urinary tract symptoms. Aging was associated with lower anal resting and squeeze pressure, reduced rectal compliance, reduced rectal sensation, and perineal laxity, which were assessed by manometry, staircase balloon distension or a visual analog scale during phasic distensions. Pelvic magnetic resonance imaging also revealed that the location of the anorectal junction at rest, squeeze, and Valsalva maneuver were lower in elderly women, and these changes were associated with age [26]. Together, these changes may predispose elderly females to fecal incontinence.

Neural function and related pelvic floor muscle weakness have been measured with electromyography. Aukee et al [27], using a vaginal surface electromyography

probe to measure the electromyography activity, revealed through regression analysis that electromyography values were dependent on age whether in supine or standing position in both incontinent patients and asymptomatic women, but not on parity, body mass index or episiotomy. They found that the electromyography activity of pelvic floor muscles decreased during the aging process. The effect of age on denervation of the pelvic floor was reported by Allen and Warrell [28]. They found a gradual denervation with advancing years, which was more pronounced in parous women. Thus, it seems likely that neurologic damage from vaginal delivery, surgery, neuropathy, and aging has a role in the development of pelvic floor failure [29]. Olsen et al [30] conducted a study in 2003 that tried to establish data for normative distributions for pudendal and perineal nerve compound muscle action potential in healthy women across a wide age range and varied history of vaginal deliveries. They studied 42 continent women aged 20 to 67 years, including nulliparous women (29%), to evaluate their pudendal terminal latency and amplitude and perineal nerve responses. Their data showed that increasing age, more vaginal deliveries, and widening urogenital hiatus were associated with increased latency and decreased amplitude. However, it remains difficult to precisely quantify the specific contributions of these three variables. They suggested that the effects of age and parity must be recognized when interpreting pelvic nerve conduction study results. They also confirmed that advancing age has an important effect on the normal limits for pudendal and perineal latency and amplitude.

Structural Changes of Pelvic Floor During Aging Process

The age-related decrease in mean urethral closure pressure found in nulliparous women may corroborate and extend a similar finding in groups containing multiparous women. These functional changes most likely reflect histologic changes seen in urethral striated muscle, blood vessels, and connective tissue of the urethra [24]. Perucchini et al [31] found that, with age, there is a decrease in the muscle fiber to connective tissue ratio and muscle fiber diameter in the urethral sphincter. The muscle fiber to connective tissue ratio and strength of skeletal muscle deteriorate with age. Thus, these same age-related changes may occur in the pelvic floor, leading to poorer support in older women.

Perucchini collaborated with DeLancey's laboratory to study 25 female cadavers with a mean age of 52 years (range, 15–80 years) and found age effects on

urethral striated muscle [32,33]. They reported that the number and density of urethral striated muscle fibers, not fiber diameter, in the ventral wall of the striated urogenital sphincter declined significantly with age. They estimated that over a life span, an average of 364 fibers (2%) are lost per year and mean fiber density also decreases by 13 fibers/mm² per year [32]. Later, they did another study to measure the thickness of urethral muscle layers at specific locations and along the length of the urethra [33]. They also demonstrated that the striated muscle loss in the proximal ventral and dorsal urogenital sphincter muscle occurred with advancing age. Muscle loss in this portion of the urethra with advancing age might explain the progressive reduction of urethral closure pressure that occurs with age [34].

A study by Boreham et al [35] revealed that the fraction of smooth muscle in the muscularis of the anterior vaginal wall significantly decreased in women with pelvic organ prolapse compared with normal control subjects using an immunohistochemistry study and morphometric analysis. The fraction of smooth muscle in the anterior vaginal wall decreased significantly in women aged 60 years or older compared with that in women under 50 years old. Lin et al [36] investigated the changes in the connective tissues located in the upper portion of the anterior vaginal wall of women with or without prolapse and also found that quantitative immunoreactivity of collagen I and III had significant positive correlations with aging.

Similar findings of age-related changes may also be found in the anal sphincter. Rociu et al [37] performed high-spatial-resolution endoanal magnetic resonance imaging to assess sex- and age-related variations in the dimensions of the anal sphincter from 100 health volunteers (50 women and 50 men, evenly distributed between 20 and 85 years old). They found a significant decrease in the thickness of the longitudinal muscle and an increase in the thickness of the internal sphincter with advancing age in both sexes. There was also an age-related decrease in the thickness of the external sphincter, but it was not a significant change.

Conclusion

The natural history and mechanism of pelvic floor dysfunction in aged women are not well understood or explored. The aging process seems to play a negative role in either the function or structure of the pelvic floor in women. Aging may either add to the deterioration of preexisting pelvic floor dysfunction during the life span of a woman or interact with other potential

predisposing factors (such as parity and mode of delivery, menopausal estrogen deficiency, high body mass index, previous pelvic surgery, and comorbidity including diabetes mellitus, hypertension and poor cognitive function) to cause major pelvic floor failure.

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References

1. Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford Family Planning Association Study. *Br J Obstet Gynaecol* 1997;104:579-85.
2. Jundt K, Kiening M, Fischer P, et al. Is the histomorphological concept of the female pelvic floor and its changes due to age and vaginal delivery correct? *Neurourol Urodyn* 2005; 24:44-50.
3. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG* 2000; 107:1460-70.
4. Ng SC, Chen YC, Lin LY, Chen GD. Anorectal dysfunction in women with urinary incontinence or lower urinary tract symptoms. *Int J Gynaecol Obstet* 2002;77:139-45.
5. Chen GD, Lin TL, Hu SW, Chen YC, Lin LY. Prevalence and correlation of urinary incontinence and overactive bladder in Taiwanese women. *Neurourol Urodyn* 2003;22:109-17.
6. Chen GD, Hu SW, Chen YC, Lin TL, Lin LY. Prevalence and correlations of anal incontinence and constipation in Taiwanese women. *Neurourol Urodyn* 2003;22:664-9.
7. Hung MJ, Shen PS, Ho ESC, Sun MJ, Lin ATL, Chen GD. Urgency is the core symptom of female overactive bladder syndrome, as demonstrated by a statistical analysis. *J Urol* 2006;176:636-40.
8. Chen YC, Chen GD, Hu SW, Lin TL, Lin LY. Is the occurrence of storage and voiding dysfunction affected by menopausal transition or associated with the normal aging process? *Menopause* 2003;10:203-8.

9. Stewart WF, van Rooyen JB, Cundiff GW, et al. Prevalence and burden of overactive bladder in the United States. *World J Urol* 2003;20:327–36.
10. Milsom I, Abrams P, Cardozo L, Roberts RG, Thuroff J, Wein AJ. How widespread are the symptoms of an overactive bladder and how are they managed? A population-based prevalence study. *BJU Int* 2001;87:760–6.
11. Goepel M, Hoffmann JA, Piro M, Rubben H, Michel MC. Prevalence and physician awareness of symptoms of urinary bladder dysfunction. *Eur Urol* 2002;41:234–9.
12. Lin TL, Ng SC, Chen YC, Hu SW, Chen GD. What affects the occurrence of nocturia more: menopause or age? *Maturitas* 2005;50:71–7.
13. Haylen BT, Krishnan S, Schulz S, Verity L, Law M, Zhou J, Sutherst J. Has the true prevalence of voiding difficulty in urogynecology patients been underestimated? *Int Urogynecol J Pelvic Floor Dysfunct* 2007;18:53–6.
14. Haylen BT, Ashby D, Sutherst J, Frazer MI, West CR. Maximum and average urine flow rates in normal male and female populations—the Liverpool nomograms. *Br J Urol* 1989;64:30–8.
15. Whitehead WE, Drinkwater D, Cheskin LJ, Heller BR, Schuster MM. Constipation in the elderly living at home: definition, prevalence, and relationship to lifestyle and health status. *J Am Geriatr Soc* 1989;37:423–9.
16. Johanson JF, Sonnenberg A, Koch TR. Clinical epidemiology of chronic constipation. *J Clin Gastroenterol* 1989;11:525–36.
17. Malone-Lee JG, Wahedna I. Characterisation of detrusor contractile function in relation to old age. *Br J Urol* 1993;72:873–80.
18. Griffiths DJ, McCracken PN, Harrison GM, Gormley EA. Characteristics of urinary incontinence in elderly patients studied by 24-hour monitoring and urodynamic testing. *Age Ageing* 1992;21:195–201.
19. Griffiths DJ, McCracken PN, Harrison GM, Gormley EA. Cerebral aetiology of urinary urge incontinence in elderly people. *Age Ageing* 1994;23:246–50.
20. Griffiths DJ, McCracken PN, Harrison GM, Gormley EA, Moore KN. Urge incontinence and impaired detrusor contractility in the elderly. *Neurourol Urodyn* 2002;21:126–31.
21. Smith NKG, Morratt JD. Post-operative urinary retention in women: management by intermittent catheterization. *Age Ageing* 1900;19:337–40.
22. Gilpin SA, Gilpin CJ, Dixon JS, Gosling JA, Kirby RS. The effect of age on the autonomic innervation of the urinary bladder. *Br J Urol* 1986;58:378–81.
23. Homma Y, Imajo C, Takahashi S, Kawabe K, Aso Y. Urinary symptoms and urodynamics in a normal elderly population. *Scand J Urol Nephrol* 1994;157(Suppl):27–30.
24. Trowbridge ER, Wei JT, Fenner DE, Ashton-Miller JA, DeLancey JOL. Effects of aging on lower urinary tract and pelvic floor function in nulliparous women. *Obstet Gynecol* 2007;109:715–20.
25. Ng SC, Chen GD. Age effects on anorectal pressure in anal continent women with lower urinary tract dysfunction. *Int Urogynecol J Pelvic Floor Dysfunct* 2007;18:295–300.
26. Fox JC, Fletcher JG, Zinsmeister AR, Seide B, Riederer SJ, Bharucha AE. Effect of aging on anorectal and pelvic floor functions in females. *Dis Colon Rectum* 2006;49:1726–35.
27. Aukee P, Penttinen J, Airaksinen O. The effect of aging on the electromyographic activity of pelvic floor muscles: a comparative study among stress incontinent patients and asymptomatic women. *Maturitas* 2003;44:253–7.
28. Allen RE, Warrell DW. The role of pregnancy and childbirth in partial denervation of the pelvic floor. *Neurourol Urodyn* 1992;6:183–4.
29. Bidmead J, Cardozo LD. Pelvic floor changes in the older woman. *Br J Urol* 1998;82(Suppl 1):18–25.
30. Olsen AL, Ross M, Stansfield RB, Kreiter C. Pelvic floor nerve conduction studies: establishing clinically relevant normative data. *Am J Obstet Gynecol* 2003;189:1114–9.
31. Perucchini D, DeLancey JOL, Blaivas M. Evidence of major myopathic changes in the striated urethral sphincter muscle in the female. *Neurourol Urodyn* 1997;15:394–5.
32. Perucchini D, DeLancey JOL, Ashton-Miller JA, Peschers U, Kataria T. Age effects on urethral striated muscle. I. Changes in number and diameter of striated muscle fibers in the ventral urethra. *Am J Obstet Gynecol* 2002;186:351–5.
33. Perucchini D, DeLancey JOL, Ashton-Miller JA, Galecki A, Schaer GN. Age effects on urethral striated muscle. II. Anatomic location of muscle loss. *Am J Obstet Gynecol* 2002;186:356–60.
34. Rud T, Andersson KE, Asmussen M, Hunting A, Ulmsten U. Factors maintaining the intraurethral pressure in women. *Invest Urol* 1980;17:343–7.
35. Boreham MK, Wai CY, Miller RT, Schaffer JI, Word RA. Morphometric analysis of smooth muscle in the anterior vaginal wall of women with pelvic organ prolapse. *Am J Obstet Gynecol* 2002;187:56–63.
36. Lin SY, Tee YT, Ng SC, Chang H, Lin PP, Chen GD. Changes in the extracellular matrix in the anterior vagina of women with or without prolapse. *Int Urogynecol J Pelvic Floor Dysfunct* 2006;18:43–8.
37. Rociu E, Stoker J, Eijkemans MJ, Laméris JS. Normal anal sphincter anatomy and age- and sex-related variations at high-spatial-resolution endoanal MR imaging. *Radiology* 2000;217:395–401.