The menopause: effects on the pelvic floor, symptoms and treatment options

Postmenopausal women are at risk of urogynaecological dysfunction. With better understanding of the pelvic floor much can be done to improve care.

AUTHOR Judith Lee, MCSP, is clinical lead women’s health physiotherapist, Nottingham University Hospitals Trust.


This article discusses the effects of age and the menopause on the pelvic floor, subsequent risks of urogynaecological dysfunction, symptoms of this dysfunction and treatment options.

INTRODUCTION

The physiological effects of the decline in circulating oestrogen, combined with the ageing process, put postmenopausal women at risk of urogynaecological dysfunction. Incontinence, prolapse and sexual dysfunction are common, and their symptoms can greatly affect quality of life.

The average age at which women have the menopause is 51 years. For most it occurs between 45 and 55, so they may expect some 30 years of postmenopausal life. Healthcare professionals have a duty to develop the skills and knowledge to provide or direct women towards the best possible care. To do this, they need an understanding of the anatomy of the pelvic floor, its function and the consequences of dysfunction.

ANATOMY OF THE PELVIC FLOOR

The pelvic floor is a fascial and muscular sheet forming the inferior boundary of the abdominopelvic cavity (Fig 1).

Its main muscular components are the puborectalis, pubococcygeus, iliococcygeus and ischiococcygeus, collectively termed the levator ani. It is a voluntary muscle supplied by the pudendal nerve which emerges from the second, third and fourth sacral foramina. A horizontal sheet with an anterior midline cleft through which the urethra, vagina and rectum pass, it is attached to the inner surface of one side of the lesser pelvis, and unites in midline with the opposite side, forming the greater part of the floor of the pelvic cavity.

The fascia within and covering the levator ani is the pelvic fascia. The endopelvic fascia is the connective tissue that supports the urethra, bladder, vagina and rectum. This fascia contains dense bands of connective tissue, termed ligaments, which sling the pelvic viscera to the pelvic side walls. A thick fascial band on either side of the pelvis spanning from the ischial spine to the pubis, the arcus tendineus fascia pelvis (ATFP), provides attachment to both the endopelvic fascia and ilio coccygeus. The fascia and ligaments are made up of collagen fibres interlaced with elastin, smooth muscle cells, fibroblasts and vascular structures.

FUNCTIONS OF THE PELVIC FLOOR

Ashton-Miller and Delancey (2007) described the functional anatomy of the pelvic floor as a supportive hammock under the urethra and bladder neck, providing a firm backstop against which the urethra is compressed during increases in abdominal pressure to maintain urethral closure and continence. The fascia and ligaments act like a cat’s cradle of support to the pelvic organs and limit their downward movement during rises in intra abdominal pressure but, alone, are not able to provide continual support.

Coordinated action of the levator ani muscle, in the presence of intact fascia and neural control, maintains pelvic organ support and, with urethral and anal sphincteric control, urinary and faecal continence. This normal function relies on these structures being intact. Damage or weakness to any part can produce symptoms.

PHYSIOLOGICAL CHANGES

Epidemiological studies have shown that older women are at increased risk of pelvic floor dysfunction, stress urinary incontinence, prolapse and faecal incontinence. The most significant aetiological factors for the development of prolapse are advancing age and increasing parity (MacLennan et al, 2000).

Evidence of damage to muscles

Generally, muscle mass is lost with ageing. Skeletal muscle strength is usually at its peak at 20–30 years and deteriorates by 5% per decade. The pelvic floor muscle is not immune to this ageing process. In a study of 25 dead people aged 15–80, Perucchini et al (2002) noted a 3% reduction in urethral striated muscle thickness per year in older people, showing that the rate of deterioration may indeed accelerate.

Evidence of damage to connective tissue

Several studies have investigated tissue metabolism and properties in postmenopausal women with symptoms of prolapse and/or stress urinary incontinence. Links have been identified between these symptoms and alterations in connective tissue and oestrogen levels.

Goepel et al (2003) took biopsies of perirectal tissue from 29 women undergoing anterior repair or sacrospinous fixation surgery for prolapse. The results showed altered metabolism in all the postmenopausal women: there was less of types 1, 3 and 4 collagen and vitronectin was absent or fragmented. Vitronectin is a glycoprotein that promotes cell adhesion and inhibits cell membrane damage. Similarly, Moalli et al (2003) took biopsies of the ATFP from 27 postmenopausal women during repair surgery for anterior vaginal wall prolapse, which showed a decrease in type 1 collagen. It is considered that type 1 is the main determinant of tensile strength within connective tissue, that is, the amount of load that can be exerted on it before it permanently deforms or fails. Resilience is another property of connective tissue; it is its ability to recover to its original length after stress. In Reay Jones et al’s (2003) study, the uterosacral ligament was measured for resilience in women who were undergoing hysterectomy. It was shown to be reduced in those women who were postmenopausal. Also, the ligament itself was thinner.
Both these studies demonstrated a weakness in the passive tissues, with the consequences of pelvic organ support being more reliant on the pelvic floor muscles.

Alperin and Moalli (2006) summarised the understanding of alterations in tissues in women with prolapse and concluded that metabolism of collagen and elastin is altered in prolapsed tissue. However, they said further studies were needed to fully understand physiological changes.

**Evidence of damage during childbirth**

A number of studies have suggested that pudendal nerve damage and/or anal sphincter injury may occur during childbirth (Sultan et al, 1994; 1993).

Allen et al (1990) showed pudendal nerve denervation in 80% of women following their first delivery and Snooks et al (1990) showed that this nerve damage may persist. The risk is that the denervated muscles undergo atrophy and pelvic organ support would be more reliant on the endopelvic fascia, which over time may become stretched.

Nicholls et al (2004) also looked at the consequences of childbirth in later life. One hundred women with symptoms of prolapse and/or urinary incontinence were asked to complete a questionnaire about faecal incontinence. Symptoms of current faecal incontinence were significantly associated with anal sphincter injury sustained during childbirth.

It may be that young parous women are not troubled by symptoms as they are able to compensate with strong muscles and intact connective tissue. However, with ageing, muscles weaken and the connective tissue may lengthen, stiffen and/or fail at the time of menopause. It is not until this point that neural or anal sphincter defects become clinically evident and symptoms of faecal urgency or incontinence develop.

The fascia itself may be damaged during childbirth – either stretched or detached from the pelvic side walls, as Dietz and Lanzarone (2005) demonstrated by transperineal ultrasound. It is not yet known how significant this is later in life as the tissues age. Looking to the future, it may be that ultrasound techniques postnatally may identify women at risk of symptomatic dysfunction in later years.

**The urethra**

There is evidence that the urethra itself may be hormonally sensitive. The urethral tissues age. Looking to the future, it may be how significant this is later in life as the tissues age. There is evidence that the urethra itself may be hormonally sensitive. The urethral tissues age. Looking to the future, it may be how significant this is later in life as the tissues age.

**Symptoms**

Stress urinary incontinence (SUI) is the most common type of incontinence and involves involuntary leakage on effort or exertion, or on sneezing or coughing. Incidence is difficult to measure as many women never seek help, but it is speculated that 50% of 48 year olds experience SUI (Kuh et al, 1999). Women often report that urinary incontinence is embarrassing and that they need to manually assist defecation.

**Treatment**

For women to gain access to treatment for these symptoms, practitioners must give them time and privacy to ask questions about problems of bladder, bowel and sexual function and perineal comfort.
acceleration of the urogenital structures during rises in intra abdominal pressure. In women with SUI, this brake is applied late. It is worth considering lumbopelvic stability as an important component of the continence system. Grewer and McLean (2008) advised correcting postural and movement dysfunction and treating coexisting back pain or breathing disorders as part of the management of SUI.

These studies direct us in the technique of teaching pelvic floor muscle exercises, where timing and direction of contraction is as important as strength. The optimum direction is a cranial (upwards) ventral (forwards) movement to support the bladder neck. When this is established, women are given an individualised exercise programme of pelvic floor contractions, of maximal strength, endurance and repetitions with breathing control in positions of comfort, but to challenge against gravity if possible.

The ideal way to teach and assess exercise technique is by digital vaginal examination by practitioners. This should be followed by practising the short contractions of “the knack” (Miller et al, 1996). This is learning the skill of performing a short, quick, pelvic muscle contraction simultaneously with an event known to trigger leakage, in order to stop that leakage, mimicking the anticipatory response in normal muscle.

Electromyogram (EMG) biofeedback should be considered as an assessment and motivational tool to aid the pelvic floor exercise programme. A course of electrical muscle stimulation (EMS) or weighted cones are treatment options for those women with weak muscles.

If conservative measures fail, practitioners should consider pharmacological therapy or surgery for SUI.

**Faecal incontinence**

The conservative approach to treating faecal incontinence is similar, in that, following rectal examination, a specifically designed exercise programme is taught, aided if necessary by anal EMG or EMS, with advice on healthy bowels (NICE, 2007).

A Cochrane review examined conservative management of pelvic organ prolapse and concluded that the evidence was not sufficient to guide practice (Hagen et al, 2006). However, in Hagen et al’s (2004) survey of UK physiotherapy practice, 92% of women’s health physiotherapists were generally treating pelvic organ prolapse with a combination of pelvic floor rehabilitation and tailored advice to reduce the rise in intra abdominal pressure and strain on the pelvic organs and pelvic floor. Advice may include avoiding prolonged standing, high impact exercise and constipation to reduce symptoms, as well as bracing pelvic floor muscles before lifting, sneezing and so on, to protect the pelvic floor.

Following a feasibility study by Hagen et al (2005), the Pelvic Organ Prolapse Physiotherapy (POPPY) trial is under way. Intended to produce guidance, this is a randomised controlled trial of conservative treatments for women who have prolapse.

In the absence of evidence based guidance, practitioners should continue to give advice to educate women with prolapse and instruct them in pelvic floor exercises to empower them in managing their symptoms. For prolapse that is symptomatic, and for which conservative treatment is not effective, consideration should be given to mechanical devices, support pessaries or repair surgery.

**CONCLUSION**

The pelvic floor is influenced by ageing and the menopause, but more research is needed to fully understand its pathophysiology, treatment selection and prevention. However, with knowledge and understanding, much can be done to provide women with pelvic floor dysfunction good care and improve their quality of life.

REFERENCES


