INTRODUCTION

Stress urinary incontinence and pelvic organ prolapse are common, often debilitating conditions, necessitating surgical correction in 11% of women by the age of 80 years. A common pathophysiology may be implied as aetiological and aggravating factors are similar and both arise from failure of pelvic floor support.

CLASSIFICATION

Stress urinary incontinence

Stress urinary incontinence is the complaint of involuntary leakage of urine on effort or exertion, or on sneezing or coughing. In urodynamic terms, stress continence is maintained when the maximum urethral pressure exceeds the intravesical pressure. In the McGuire classification system, type 1 and 2 stress incontinence occur because of urethral hypermobility and type 3 because of intrinsic sphincter deficiency (ISD). More recently it has been suggested that there is a spectrum of urethral characteristics in women with stress urinary incontinence and ‘delineation into categories may be simplistic and arbitrary and requires further research’.

Pelvic organ prolapse

Several descriptive systems have been developed to classify pelvic organ prolapse. However, few epidemiological studies have examined the distribution of pelvic floor support within a female population. The Pelvic Organ Prolapse Quantification system (POP-Q) has been formally accepted by three international societies, although not yet widely adopted into clinical practice in many areas.

The pathophysiology of both these conditions might be considered to represent a function of the mechanisms that maintain a low pressure urinary reservoir, a high pressure sphincter, and urethral and vaginal support.

NORMAL PELVIC FLOOR FUNCTION

The fascial and muscular components within the pelvic floor and its neural pathways create a dynamic support mechanism that facilitates storage and voiding of urine and faeces, and normal sexual function. Stress urinary incontinence and pelvic organ prolapse arise through abnormality in pelvic floor architecture, at cellular or gross morphological levels, leading to mechanical failure of this support.

Normal functioning of the lower urinary tract depends on the balance between the powers of urethral resistance and the forces of urinary expulsion. A low pressure reservoir and a high pressure sphincter are essential for maintenance of urinary continence. Mechanical support of the vagina has been proposed to be reliant on three mechanisms: closure of the vagina at its introitus (by pelvic floor contraction), vertical suspension of the vagina by the utero-sacral ligaments, and the flap valve effect created from the near horizontal position of the vagina on the pelvic floor.

The reservoir function of the bladder

Maintenance of a low pressure reservoir is dependent on hydrostatic pressure acting at the bladder neck and transmitted pressures from adjacent intra-abdominal viscera, and tension in the bladder wall (see Fig. 1). When there is adequate urethral support the first of these factors is insignificant. However, when urethral support is inadequate they may become relevant especially during stress provocation.

Tension in the bladder wall is in part a passive phenomenon related to the distensibility or visco-elastic properties of the bladder wall itself, and in part an active phenomenon due to the contractility of the detrusor muscle and its neurological control.

The neurological control of detrusor contractility is dependent on a sacral spinal reflex under the control of several higher centres. This basic reflex arc is best considered as a loop extending from sensory receptors within the bladder wall through the pelvic plexus and via visceral afferent fibres travelling with the pelvic splanchnic nerves; it enters the spinal cord in the S2–S4 levels synapsing with cell bodies in the intermedio-lateral grey area of the same sacral levels, and then via pelvic splanchnic nerves to the smooth muscle cells of the detrusor (see Fig. 2). Higher control over the basic reflex arc is mediated through descending pathways from the pontine reticular formation.
and is primarily inhibitory; the normal influence is therefore to prevent contraction of the detrusor and thus to encourage the maintenance of a low intravesical pressure during the filling phase of the micturition cycle.

Urethral closure mechanisms

The usual level of continence in the female is at the level of the bladder neck where passive elastic tension is the most important factor leading to urethral closure.\textsuperscript{10,11} In the mid-urethra at rest, closure is largely maintained through the intrinsic striated muscle of the rhabdosphincter; however, the extrinsic striated muscles of pubo-coccygeus and compressor urethrae may be significant in maintaining urethral closure with increased intra-abdominal pressure. Although an open bladder neck at rest does not distinguish stress continent from incontinent women, stress continent women who have an incompetent bladder neck on coughing might be at risk of developing urodynamic stress incontinence should the distal sphincter mechanism fail.\textsuperscript{12}

The plasticity of the urethra or its ability to act as a watertight seal is also important in maintaining continence. There has been much debate over the morphological components that contribute to the softness, compression and tension in the urethra. Urethral pressure studies have shown that approximately one-third of the resting urethral pressure is due to striated muscle effects, one-third to smooth muscle effects and one-third to the vascular supply.\textsuperscript{13} With increasing age both morphological and functional changes are observed, which may predispose to the development of stress incontinence.\textsuperscript{14} These include an increase in intravesical pressure and a reduction in both mid-urethral vascular pulsations and maximum urethral closure pressure;\textsuperscript{10,13} the latter starts early and becomes significant after 36 years.\textsuperscript{13}

Urethral and vaginal support

Support and suspension of the pelvic organs is dependent on pelvic floor striated muscle and connective tissue, and the attachments between muscle and connective tissue and the bony pelvis. The levator ani comprises diaphragmatic and pubovisceral components,\textsuperscript{15} and contains type 1 (slow-twitch) fibres that provide resting tone, and type 2 (fast-twitch) fibres that can respond to stress and thus maintain urethral closure and prevent stretching of the pelvic ligaments. Pelvic connective tissue facilitates organ displacement and volume changes and functions more as a mesentery, unlike connective tissue in other parts of the body where its function is to facilitate locomotion. Both of these structures are damaged after delivery with tears in the endopelvic fascia and thinning of the levators seen on MRI.\textsuperscript{16}

The common action of the muscular and the fascial components in providing pelvic floor support and their role in the development of pelvic organ prolapse has been recognised from the beginning of the last century. Parapmore used the analogy of a ‘boat in a dry dock’ to explain the loss of support provided by the levators, and the suspensory role of the ligaments and fascia.\textsuperscript{17} Using levator myography, Berglas and Rubin showed that in normal nulliparous women the vaginal axis is almost horizontal and is supported by the levator plate. In genital prolapse, however, the levator support is lost and as a consequence the vaginal axis inclines towards the vertical, the levator hiatus widens and fascial supports are placed under strain.\textsuperscript{18}

On the basis of cadaveric studies, DeLancey has described pelvic floor support at three levels.\textsuperscript{19} The cervix, and upper third of the vagina are suspended almost vertically by a sheet of connective tissue from the pelvic wall; this may be termed the paracolpium, and includes the uterosacral ligaments (level I). The middle third of the vagina is attached laterally to the pelvic side walls by fascial sheets extending transversely between the bladder and rectum, and compromising the pubo-cervical fascia and prerectal fascia attaching laterally to the arcus tendineus fasciae pelvis and...
superior fascia of the levator ani (level II). In the lower third of the vagina the wall is directly attached to surrounding structures. Anteriorly it fuses with the urethra, posteriorly with the perineal body and laterally with the levator ani muscles (level III). Loss of support at level I leads to uterine and vault prolapse whilst loss of level II support results in cystocele and rectocele. During rises in intra-abdominal pressure in continent women, the urethra is supported through its lateral attachments and is compressed by the downward force of intra-abdominal pressure against the resistance offered by the anterior vaginal wall and endopelvic fascia below. When compression is inadequate because of a failure in suburethral support arising from a defect in the endopelvic fascia or its attachments, stress urinary incontinence will occur.

**ABNORMAL PELVIC FLOOR FUNCTION**

**Neuromuscular injury**

The pudendal nerve, which arises from the anterior rami of S2, S3 and S4, provides somatic innervation to the striated muscle of levator ani and to the striated muscle within the external anal and urethral sphincters.

Neuromuscular injury has been proposed as an important factor in predisposing to pelvic floor dysfunction (see Table 1). Interest in this area was first stimulated when histochemical evidence of denervation was observed in the external anal sphincter in those with faecal incontinence. Since then several neurophysiological studies have used pudendal nerve latency or single fibre or concentric needle pelvic floor electromyography (EMG) to investigate the role of nerve injury in pelvic floor dysfunction. Reinnervation, which occurs in response to denervation, is taken as a marker for injury, and both prolapse and stress incontinence are found in association with denervation. Progressive pelvic floor denervation is thought to lead to sagging of the levators, widening of the levator hiatus with loss of urethral and vaginal support leading to stress urinary incontinence and genital prolapse.

Ageing and childbearing are often considered as the two major factors predisposing to pelvic floor denervation. Other factors include chronic straining at stool and congenital neurological conditions such as spina bifida and muscular dystrophy. A gradual increase in denervation of the pelvic floor has been observed with increasing age, as is observed in striated muscle elsewhere in the body. During childbirth, the pudendal nerve might be damaged by compression or by traction within Alcock’s canal. Snooks et al. using pudendal nerve latency and single fibre EMG of the external anal sphincter found evidence of denervation following vaginal delivery which persisted 2 months after delivery. Subsequent follow-up in a small group of women at 5 years showed denervation to have persisted or become worse. In a much larger prospective study, Allen et al. found that 80% of women had evidence of partial denervation to the pelvic floor after delivery. Longitudinal follow-up of this cohort 7 and 15 years later indicated that denervation had progressed and the numbers with stress incontinence had increased from the postnatal period. Although there is much evidence to suggest an association between these conditions and denervation injury, the relationship between neurogenic injury at childbirth and long-term risk of stress urinary incontinence remains unresolved.

**Connective tissue injury**

Connective tissue is composed of collagen, elastin, smooth muscle, fibroblasts and blood vessels. However, collagen is the main constituent of endopelvic fascia and abnormalities in the quantity, type and quality of collagen have been observed in both stress incontinence and in genitourinary prolapse. Several studies have reported a decrease in the total collagen content in women with stress urinary incontinence. A reduction in total collagen and increased turnover of collagen has been identified in women with stress urinary incontinence. Different sites of defect have been identified in genital prolapse. In stress urinary incontinence a defect in endopelvic fascia is also likely to be of functional significance given that the urethra is indirectly attached to the levators by endopelvic fascia and an intact arrangement assists in urethral positioning.
women with genitourinary prolapse. However, prolapse and stress incontinence may be acquired through conditions causing changes in connective tissue. Genitourinary prolapse is more common with increasing age. With increasing age connective tissue to muscle ratio is reduced, and although the formation of collagen cross-links stabilizes the molecule it prevents remodelling and flexibility. Stress incontinence and prolapse are more common in multiparous women. We have discussed that there is evidence to suggest that trauma during childbirth may cause neuromuscular injury. However, changes in connective tissue also occur during pregnancy. Fascia becomes more elastic and vulnerable and women who have antenatal stress incontinence might have a greater degree of fascial weakness compared with those who remain continent. Women who develop antenatal stress incontinence even when it resolves in the post natal period, are twice as likely to develop it again in the future compared with those without antenatal stress incontinence. The hormonal changes during pregnancy or abnormal remodelling of collagen may be important in the development of these conditions.

CONCLUSION

There would seem to be two major factors determining the proneness of the human female to problems of stress urinary incontinence and prolapse. Firstly, changes in pelvic floor anatomy resulting from the change from the pronograde to orthograde posture has meant that the pelvic floor has had to adapt from its largely muscular function (of moving the tail) in four legged animals, to a supportive role in the human. This has been associated with a decrease in muscle bulk and an increase in connective tissue. Secondly, the human reproductive process results in a relatively large fetus with a large bony cranium that has to pass through the pelvic floor during the processes of labour and delivery. As a consequence there is a certain inevitability to the problems of prolapse and incontinence as the increasingly tenuous pelvic floor muscles, and persistently vulnerable pelvic floor connective tissues are subjected to this degree of trauma.

The complex aetiology of stress urinary incontinence and pelvic organ suggests that greater attention should be given to studying the natural history of these conditions. Although there is much debate as to the exact site and mechanism of injury during childbirth, the intimate arrangement of connective tissue and striated muscle within the pelvic floor suggests that injury to one or other structure is likely to impact on their co-ordinated function. Further understanding of the biomechanics of the pelvic floor and dynamic imaging may improve knowledge of how and when structural abnormalities occur and perhaps ultimately guide more site-specific treatment. In future family studies may allow the identification of those women who may be genetically predetermined to develop these conditions, and allow timely intervention to prevent the development of symptoms.

References


