

Stress urinary incontinence: current understanding

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Many new concepts were introduced in epidemiology, a etiopathology and treatment of stress urinary incontinence. This review gives a short account of these concepts and compares the results of commonly used treatment options with new ones recently introduced.

The International Continence Society (ICS) defines urinary incontinence as the complaint of any involuntary leakage of urine and stress incontinence as a complaint of involuntary leakage on effort or exertion, or on sneezing or coughing (Abrams et al, 2002). Stress urinary incontinence (SUI) is generally more common in women than men; its presence has great social, physical, economical and psychological impact.

Prevalence between studied groups has varied widely, mostly because of previously inconsistent definitions of incontinence. Modern definitions from the ICS have, however, clarified the situation. A recent European study conducted in four countries showed that overall 35% of women reported incontinence. Stress incontinence was the most common form (46%), followed by mixed (28%) and urge (26%) (Hunskar et al, 2004). The true prevalence of stress incontinence is probably higher than this, as actual urodynamic evaluation reveals that mixed incontinence may be over diagnosed at the expense of SUI (Sandvik et al, 1995).

These figures highlight the potential workload for both primary and secondary care facilities. However, despite the high prevalence of incontinence, symptoms in patients in the community may not necessarily be bothersome and disabling. It has therefore been suggested that treatment should generally be directed at those with the most disabling of symptoms (Perry et al, 2000).

RISK FACTORS

There is common agreement that pelvic floor dysfunction, usually secondary to childbirth, ageing or surgery, is the main factor associated with the development of SUI. Recent diet and

lifestyle analyses have confirmed the causal association between SUI and obesity, and implicated dietary factors such as carbonated drink consumption. The link between smoking and SUI remains contentious, and this factor appears to be implicated more in urge incontinence (Dallosso et al, 2003).

SOCIO-ECONOMIC IMPACT

Urinary incontinence is not only a cause of considerable personal distress, but also a heavy burden on the economy of the health service. The lifetime costs of women with SUI have been shown to be almost twice that those without SUI, and this figure is comparable to the costs of higher profile conditions, e.g. diabetes (Birnbaum et al, 2003).

Regarding quality of life issues, SUI may lead to social embarrassment, and the exclusion of individuals from certain leisure or work activities. Particular concerns in the expanding ageing population also include skin breakdown and the problems associated with pressure sores.

PATHOGENESIS OF STRESS INCONTINENCE

Stress incontinence is a multi-factorial condition, and may be a result of anatomical, neurological and muscular abnormalities. An understanding of the condition is dependent on a clear appreciation of the anatomy and physiology of the lower urinary tract. In women the main causes of SUI are failure of urethral support, nerve injury or intrinsic sphincter deficiency.

Urinary incontinence during increases in intra-abdominal pressure, may be caused by either a failure of the urinary sphincter mechanism or disruption to the structures supporting this. The

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sphincteric mechanism consists of detrusor smooth muscle looping around the proximal urethra, the circular striated muscle of the external sphincter and both longitudinal and circular smooth muscle layers within the urethra. The watertight seal attributable to the prominent vascular plexus surrounding the lumen of the urethra ultimately provides coaptation.

In order for rises in intra-abdominal pressure to be effectively transmitted to the urethra (thus enhancing urethral closure), a 'backstop' of supporting structures must be functional. The support to the urethra is derived from the endopelvic fascia of the anterior vaginal wall through connections to the arcus tendineus and levator ani.

Ageing in itself results in a reduction of the number and density of urethral striated muscle fibres (Perucchini et al, 2002), which will have a bearing on urethral function. Injury to the innervation of the sphincter, such as lower motor neurone-type lesions (e.g. cauda equina syndrome or thoraco-lumbar spinal cord injury), radiation or pelvic surgery may also lead to sphincteric weakness and deficiency.

The neuromuscular sequelae of vaginal delivery clearly have a role in the pathophysiology of SUI. Recent MRI studies have demonstrated the high incidence of levator ani defects following vaginal delivery (DeLancey et al, 2003), and neurophysiological studies have described the associated denervation (and subsequent reinnervation) of the pelvic floor following first delivery, using pudendal nerve conduction tests and concentric needle electromyography (Allen et al, 1990).

A further study on this same cohort of patients concluded that when stress incontinence occurred following the first pregnancy, this led to a doubling of the risk of SUI at 15 years (Snooks et al, 1990). Multiple deliveries have been shown to be associated with a higher risk of persistent SUI, with the delivery of a child greater than 4 kg being a predominant factor (Groutz et al, 1999). The role of instrumented delivery and SUI is contentious; comparisons are difficult as these births are inherently more likely to be associated with a longer second stage of labour.

CURRENT TREATMENT OPTIONS

Current treatment options for SUI may be usefully divided into conservative, pharmacotherapy (medical), or surgical methods

Conservative treatment

Conservative treatments of SUI should be adopted in all cases as a primary intervention.

Lifestyle changes are important and goals, e.g. weight loss, stopping smoking (for those with chronic cough), fluid intake reduction along with advice on incontinence pad usage, can help dramatically. Active interventions can take several forms, which include:

- Pelvic floor exercises (PFE)
- Electrical stimulation
- Vaginal weighted cones
- Bladder neck supports
- Urethral plugs.

Well-controlled evidence concerning the relative efficacy of these modalities is lacking, although it is generally accepted that they are all useful techniques.

Pelvic floor muscle exercises, first described by Arnold Kegel in 1948, consist of the repeated contraction of the muscles of the pelvic floor (not the abdominal wall or buttocks) several times a day as an ongoing process. The suggested mechanisms of action include aiming to build up the muscle strength around the bladder outlet to help the maintain continence, or training women to precontract the pelvic floor before rises in abdominal pressure (Bo, 2004).

Exercises can be combined with aides that provide biofeedback (visual or tactile) to improve efficiency of contractions and outcome (Glavind et al, 1998). Therapists can help by providing intensive training, education and encouragement. Indeed, Bump et al (1991) suggest that written or verbal instruction is insufficient in isolation for women about to undertake PFE. Improvements in up to 58–70% of patients have been reported in the short term, as assessed by pad weighing and frequency of incontinent episodes (Bo and Talseth, 1996; Cammu and Van Nylen, 1995).

Long-term follow-up suggests that, if initially successful, benefits can be maintained in 66% of individuals over 10 years (Cammu et al, 2000). PFE are sometimes combined with vaginal weighted cones and electrical stimulation, yet these additional interventions do not appear to add to improved continence as assessed in one study comparing the relative benefits of these interventions (Bo et al, 1999). Another study directly comparing vaginal cones to PFE suggests that one intervention is no better than the other (Cammu and Van Nylen, 1998).

Vaginal weights of various varieties exist. The most common being vaginal cones. As stated these show no increased efficacy over PFE. They have been cited as having a lower rate of compliance but are cheaper (not requiring the level of therapist intervention) and afford biofeedback. They also allow demonstrable progress as