Anchoring the midurethra restores bladder-neck anatomy and continence

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Dynamic perineal ultrasound studies show that midurethral anchoring of vagina prevents bladder neck descent, funnelling, and urine loss on effort. Appearances are consistent with continence control by a musculoelastic mechanism.

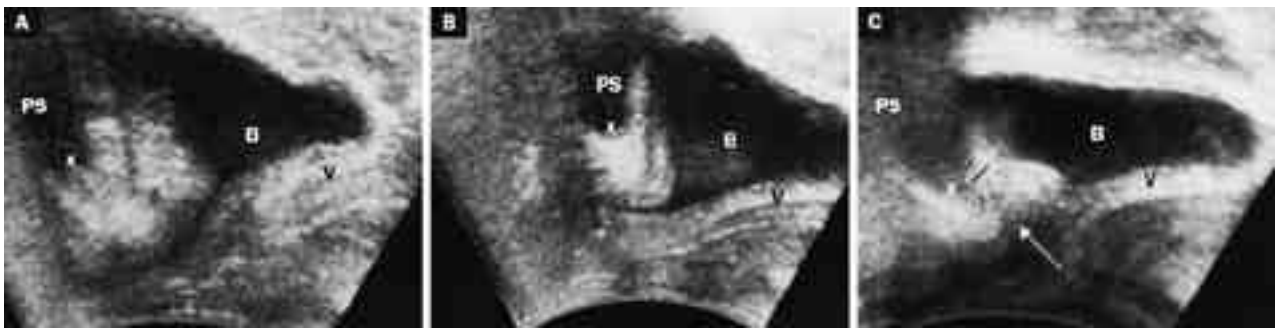
The mechanism for maintaining continence is not well understood. Bladder-neck fixation to the pubic bone, based on restoration of intra-abdominal pressure transmission,[1] is considered the most efficacious surgical treatment. Urinary retention, post-operative pain, and prolonged hospital stay characterise bladder-neck elevation operations. Cure of urinary-stress incontinence (without such complications) in almost 90% of women can be achieved by anchoring the midurethra, not bladder neck, with a 6 mm tension-free vaginal tape, plus paraurethral tightening of the hammock.[2]

20 patients with urodynamically proven genuine-stress incontinence were studied (figure, A). All had demonstrable urine loss in the supine position on coughing and straining, and excessive bladder-base displacement (>12 mm) and funnelling on perineal ultrasound examination (figure, B). The mean age of patients in the study was 50 years (range 32–68), with mean parity 3 (range 1–5).

Under perineal ultrasound control (Toshiba 3·75 MHz curvilinear probe) we did a "virtual operation". That is, we applied a Howard-Kelly haemostat to one side of the midurethra, and observed the effect on urethral and bladder-neck anatomy during straining. The aim was to mimic a competent pubourethral ligament. Supine urine loss on coughing was completely controlled in 14 patients, and substantially decreased in the other six. Subsequent tightening of the vaginal hammock by taking up a fold of vaginal skin ("pinch test") completed restoration of continence in the latter.

The musculoelastic closure mechanism, and the significance of the midurethral anchoring point, can only be conveyed with real-time dynamic data. It cannot be conveyed in a static image. Recent advances in internet technology have facilitated transmission of such data to a mass readership. This data is available on the *Lancet* website (www.thelancet.com/newlancet/sub/issues/vol354no9183/research997.html). Our studies indicate that both vagina and urethra are stretched backwards against the firmly anchored Howard-Kelly forceps, to their elastic limit, much like an elastic rubber glove. This restores bladder neck anatomy from the open configuration on straining (figure, B), to the closed configuration (figure, C). The urethra was closed at its distal end by a forward muscle force acting below the anchoring point. The bladder neck was closed by being stretched backwards and rotated downwards against the anchoring point. In the absence of this anchoring point, the posterior urethral wall was pulled open (funnelled) by the posterior muscle forces, causing urine loss (figure, B).

Cut away from a fresh cadaver, the vagina and urethra have no form and no strength. Like a sailcloth, the vagina and urethra must be stretched to a semirigid membrane not only to assume their familiar shape, but also, to function. In this analogy, the sail (urethra) needs to be stretched to its limit of stiffness against its ropes (pubourethral ligament), and, in turn, the mast (pelvic girdle), before the force of the wind (three directional muscle forces) can drive the boat forwards (close bladder neck and urethra). The strength of pubourethral ligament derives from its connective-tissue components.[3] Connective tissue degenerates with age and childbirth. Just as a sail with loose attachments flaps in the breeze, so pubourethral ligament and hammock laxity may prevent the muscle forces from effecting urethral closure. Of the two components, pubourethral ligament is the most important, as it anchors the muscle forces that stretch and narrow the urethra during effort. An adequately tight hammock helps achieve watertight closure. As pressure equals force divided by area, a smaller urethral area is reflected by a higher urethral pressure.[4] It is possible to explain restoration of "pressure cough transmission ratio" after surgical cure of urinary stress incontinence[5] entirely by the effect of these muscle forces on vaginal biomechanics.[4]



Ultrasound in sagittal section of a patient with genuine-stress incontinence

A: At rest—the bladder neck is closed, and is sited above the lower border ("x") of pubic symphysis (PS); B: On straining—backward/downward acting muscle forces pull vagina and bladder base down below "x", and open out the posterior urethral wall; C: One-sided upward pressure at midurethra (arrow) anchors the backward/downward muscle forces. Urine loss is controlled. Urethrovesical angle is restored. Bladder base is held above "x". B=bladder, V=anterior vaginal wall.

In addition to showing that elevation of the bladder neck is not a prerequisite for restoration of continence, this study challenges the long-held belief that raised intra-abdominal pressure causes urine loss. This cannot be so as such, because the pressure generated on straining on opening (figure, B) and closure (figure, C) was fairly equivalent. A more likely mechanism for stress incontinence may be active opening of urethra by muscle forces due to inadequate anchoring of their midurethral insertion point (figure, B).

1 Enhorning G. Simultaneous recording of intravesical and intraurethral pressure. *Acta Chir Scand* 1961; **176:** 1–68.

2 Petros PE. The intravaginal slingplasty operation, a minimally invasive technique for cure of urinary incontinence in the female. *Aust NZ J Obst Gyn* 1996; **36:** 463–61.

3 Nordin M. Biomechanics of collagenous tissues. In: Frankel VH, Nordin M, eds. Basic biomechanics of the skeletal system. Philadelphia: Lee and Febiger, 1980: 87–110.

4 Petros PE, Ulmsten U. The biomechanics of vaginal tissue: an integral theory and its method for the diagnosis and management of female urinary incontinence. *Scand J Urol Nephrol* 1993; **27:** 29–45.

5 Faysal MH, Constaninou CE, Rother LF, Govan DE. The impact of bladder neck suspension on the resting and stress urethral pressure profile: a prospective study comparing controls with incontinent patients preoperatively and postoperatively. *J Urol* 1981; **125:** 55–60.

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Has advice on periconceptional folate supplementation reduced neural-tube defects?

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Periconceptional folate supplementation has been encouraged in the UK since the early 1990s, but no concurrent decline in neural-tube-defect pregnancies has been observed by regional congenital anomaly registers.

Studies published in the early 1980s suggested that periconceptional folic-acid supplementation reduced the risk of recurrence of neural-tube defects (NTDs)[1] and this was confirmed by the results of the Medical Research Council (MRC) Vitamin Study.[2] Further studies supported the hypothesis that there would be a similar protective effect in low-risk pregnancies.[3] In many countries, including the UK, attention turned to developing a strategy for increasing the folic-acid intake of women planning a pregnancy. Guidance advising periconceptional folic-acid supplementation went out to UK National Health Service staff on Aug 12, 1991, and, in 1992, the UK Expert Advisory group stated that first occurrences of NTDs could be reduced by means of increased periconceptional folic-acid consumption. The UK Health Education Authority awareness campaign was launched early in 1996. One measure of the effectiveness of the campaign is the rate of NTD pregnancies before and after publication of the results of the MRC Vitamin Study in 1991 and again before and after the launch of the Health Education Authority initiative. The need to look at time trends in NTD rates coincided with the establishment, in 1998, of the British Isles Network of Congential Anomaly Registers. Before the inaugural meeting, a letter was sent to each regional congenital anomaly register in England, Scotland, and Wales asking for the number of pregnancies affected by anencephaly, spina bifida, and encephalocele during each year for which that register had data. They were asked to indicate whether the figures included each of the following: live births, still births, terminations, miscarriages, aneuploid fetuses or babies, and multiply malformed fetuses

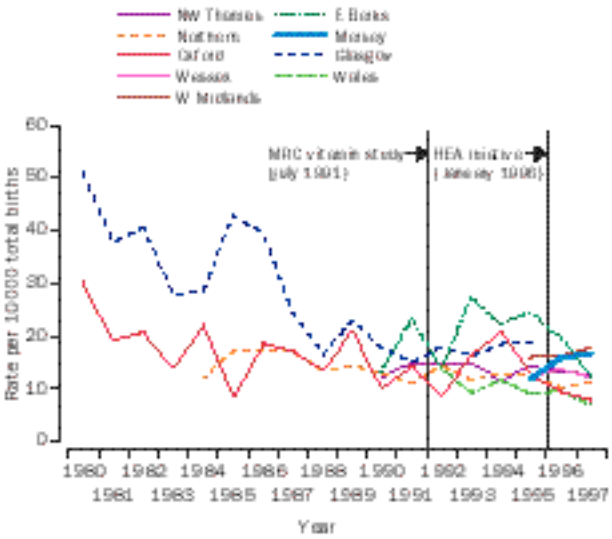

Figure 1: **NTD rates of all regions 1980–97**

or babies. Each register was also asked to supply the number of live and still births in the denominator population for each year.

All registers replied, and all registers that had been operational during the relevant time supplied data. Data from all registers included live births, still births, and terminations of pregnancy for fetal abnormality. Only three registers (Glasgow, Oxford, and NorCas) collected information before 1990; these reported a decline in the NTD rate during the 1980s. No decline was seen in the rate reported by registers after publication of the results of the MRC Vitamin Study in 1991 or after the launch of the Health Education Authority initiative in 1996 (figures 1 and 2).

The decline in NTD rates before the 1990s has been noted before.[4] The fact that those registers in our study operating in the 1980s showed a decline in NTD rates is important, because it shows that they are capable of detecting such a trend when it exists. It is discouraging to note that the decline has not continued into the 1990s. All the registers collect data on terminations. All registers except Oxford and Wessex reported NTD cases known to be aneuploid. Our data show that the decline in NTD rates in the 1980s is not due to affected pregnancies being identified prenatally, terminated, and thus not reported; nor is it due to affected fetuses or babies being found to have an abnormal karyotype and registered only as aneuploid. There has been speculation about why NTD rates came down before the 1990s, and improved diet has been suggested.[5] Some authors have suggested that the decline was due to a return to more usual

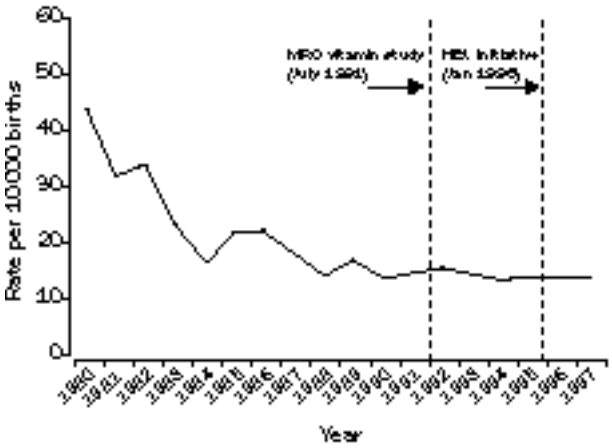

Figure 2: **Combined NTD rates 1980–1997**
Without Wessex 1994 data.