Table 2: Outcomes

	Intervention Group N=12 (Mean Reduction)	Control Group N=14 (Mean Reduction)	F	Sig
Reduction in caffeine intake	40%	-18%	8.74	0.007*
Change in voids per day	28%	18%	0.80	0.381
Change in episodes urgency per day	61%	16%	2.8	0.109
Change in episodes leakage per day	74%	32%	7.0	0.015*

*P<0.05

Conclusion: This trial generates new evidence in relation to the effect of caffeine upon urinary urgency, frequency and urge incontinence. The change in caffeine intake result is heartening as it establishes that the caffeine reduction intervention was successful in achieving actual caffeine reduction. Outcome comparisons between the two groups are suggestive of a beneficial effect among those who received the intervention. Significant improvement in occasions of leakage per day are established (P=0.015) in this early analysis. Trends are evident in voids/day and occasions of urge/day, however significance is not established at this stage. The results address a gap in the research literature and provide an empirical basis for continence practitioners advice to patients in relation to caffeine intake.

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3. Management of urinary incontinence in the elderly 1997 Gerontology 43:151-157
4. Symptoms of urinary incontinence among older community-dwelling men 1996 Journal of Wound, Ostomy and Continence Nurses 23(6):314-321

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Author(s):
P TOOZS-HOBSON, D ROBINSON, L CARDOZO, J BIDMEAD, K ANDERS.
Institution, city, country:
KINGS COLLEGE HOSPITAL, LONDON, UK.

Title (type in CAPITAL LETTERS, leave one blank line before the text):

CAN BLADDER NECK MOBILITY AND URETHRAL SPHINCTER VOLUME BE USED TO PREDICT PUDENDAL NERVE DAMAGE?

Aims of Study. Antenatal bladder neck hypermobility has been associated with the development of stress incontinence[i] in the postpartum period although the exact mechanism has not been elucidated. Possibilities include a loss of pressure transmission to the urethra or nerve disruption resulting in sphincter muscle atrophy. Nerve damage is known to be associated with stretching injury and the relationship between pudendal neuropathy and perineal descent has been demonstrated for anal incontinence[ii]. A stretching injury would be associated with a lower motor neurone deficit causing an increased pudendal nerve terminal motor latency and atrophy of the urethral sphincter. To investigate this hypothesis we examined the association between bladder neck mobility and urethral sphincter volume with pudendal nerve terminal motor latency.

Methods. Primiparous women were recruited from the antenatal clinic between 32 and 42 weeks of pregnancy. All completed a symptom questionnaire prior to undergoing a transperineal ultrasound of the bladder neck. Scans were performed using a Kretz Technik 360 Combison ultrasound machine with two and three dimensional imaging facilities. Images of the bladder neck were taken using the cine loop at rest, maximum excursion of the bladder neck during valsalva and maximum incursion during squeeze. Further scans were taken of the levator hiatus. A three-dimensional transvaginal scan of the urethra was then performed. Images were analysed blinded to symptoms. Measurements of absolute position were made using an X Y co-ordinate system with the axis of the pubis as the zero to 180^0 line. A perpendicular line was dropped through from the bladder neck to mark the intersect of this line. The X represented the displacement along the zero axis from the inferior border of the pubis and the Y co-ordinate the displacement along the perpendicular line. The angle was calculated using Pythagoras' theorem as the angle between the plane of the pubic symphysis and the intersection of the bladder neck with the inferior border of the pubic symphysis. Pudendal nerve terminal motor latency was measured using a Dantec machine with a St.. Marks electrode and the transvaginal approach. Correct positioning was confirmed by noting

the characteristic waveform produced and by documenting patient response. This was repeated on the contralateral side. Analysis was then performed using Chi Squared tests and scatter plots.

Results. 114 women were recruited to the study. We were unable to demonstrate an association between urethral sphincter volume (SPHSIZE) and pudendal nerve terminal latency (ANPNR) in the antenatal period **(Figure 1)** and there being no correlation found; Chi-Squared=0.640. Equally, there was no evidence of an assocation between bladder neck mobility (ANMOVT) and pudendal nerve terminal latency (ANPNR) **(Figure 2).** Again there was no evidence of correlation; Chi Squared=0.317.

Conclusion. These findings would suggest that there is no association between pudendal nerve damage, bladder neck mobility and urethral sphincter volume. This implies that stretching of the pudendal nerve is not associated with urethral sphincter deficiency and that any reduction in sphincter volume is unlikely to be due to a lower motor neurone type lesion. These findings would confirm previous reports that suggest sphincter damage is due to direct obstetric trauma rather than neurological causes. Because of this pudendal nerve terminal latency is not likely to be of prognostic value when assessing the risk of developing stress incontinence in the postnatal period.

Figure 1: Scatter Plot of Urethral sphincter size and Pudendal Nerve Terminal Latency.

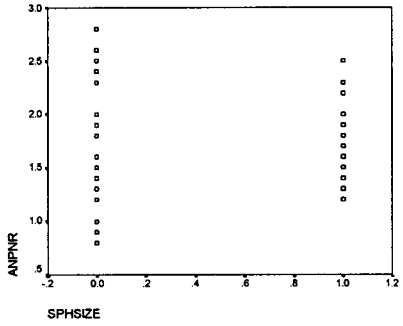
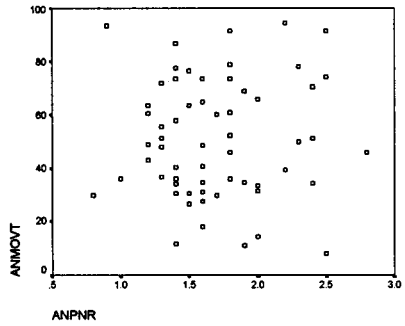


Figure 2: Scatter Plot of Bladder neck movement and Pudendal Nerve Terminal Latency.



[1] BJOG 105:1300-1307
[2] Int J of Colorectal surgery 1995; 10(2):107-11

97B

Author(s): P. Toozs-Hobson, J. Bidmead, L. Cardozo, D.Robinson, V. Khullar

Institution, city, country: Kings College Hospital, London, UK

Title (type in CAPITAL LETTERS, leave one blank line before the text):

DO BLADDER NECK MOBILITY, URETHRAL SPHINCTER VOLUME AND LEVATOR HIATUS CORRELATE IN LATE PREGNANCY?

Introduction Nerve damage may result from stretching. The association between pudendal

neuropathy and perineal descent has been demonstrated for anal incontinence[i]. To date an association of nerve damage through bladder neck hypermobility has not been researched.

King and Freeman[ii]. demonstrated an association between antenatal bladder neck hypermobility and the risk of developing urinary incontinence postnatally The mechanism by which bladder neck hypermobility results in urinary incontinence has yet to be elucidated. Possibilities include a loss of pressure transmission to the urethra or nerve disruption resulting in sphincter muscle atrophy. We therefore decided to investigate the relationship between bladder neck movement and urethral sphincter volume measured by three dimensional ultrasound, to see whether increased mobility of the bladder neck was associated with a decrease in the urethral sphincter volume. The aim of this study was to investigate the relationship between bladder neck hypermobility, urethral sphincter volume and the size of the levator hiatus.

Methods Women were recruited from the antenatal clinic. All completed a symptom questionnaire prior to undergoing a transperineal ultrasound of the bladder neck. Scans were performed using a Kretz Technik 360 Combison ultrasound machine with two and three dimensional imaging facilities. Images of the bladder neck were taken using the cine loop at rest, maximum excursion of the bladder neck during valsalva and maximum incursion during squeeze. Further scans were taken of the levator hiatus. A three dimensional transvaginal scan of the urethra was then performed. Images were analyzed blinded to symptoms. Measurements of absolute position were made using an X Y co-ordinate system using the axis of the pubis as the zero to 180^0 line. A perpedicular line was then dropped through from the bladder neck to mark the intersect of this line. The X represented the displacement along the zero axis from the inferior border of the pubis and the Y co-ordinate the displacement along the perpendicular line. The angle was calculated using Pythagorous' theorum as the angle between the plane of the pubic symphysis and the intersection of the bladder neck with the inferior border of the pubic symphysis. Analysis was performed using Spearman's correlation and independent t-tests. A cut off for urethral sphincter volume of less than 2.25 cm^3 has previously been shown to be 86% sensitive and 59% specific for predicting postnatal urinary incontinence and this figure was therefore adopted to differentiate between high risk and low risk women for postnatal urinary incontinence.

Results 114 women participated in this study. Results were available on 106 women. We were unable to demonstrate any association between bladder neck movement and urethral sphincter volume other than an increased y measurement at rest and squeeze in women with larger sphincters (greater distance from the pubic bone). There was no increased movement in degrees rotation or by movement on co-ordinates. Urethral sphincter volume did not correlate with the levator hiatus or assessment of the levator hiatus.

There was however a significant correlation between the bladder neck movement and the levator hiatus at rest and at maximum excursion, showing an increase in the bladder neck movement with an increased levator hiatus.

Correlations

		X R	X V	X S	Y R	Y V	Y S	angle R	angle V	angle S	ANMOVT
LHV	Correl'n	0.17	0.36	0.11	-0.1	-0.25	-0.12	-0.18	-0.36	-0.09	0.30
	Sig.	***0.09***	***0.00***	0.26	.309	***0.01***	0.22	0.06	***0.00***	0.37	***0.00***
	N	105	103	106	105	104	106	105	103	106	103
VOL	Correl'n	-0.01	0.01	-0.06	.188	-0.03	0.20	0.02	-0.00	0.03	0.03
	Sig.	0.89	0.93	0.55	0.05	0.76	***0.03***	0.85	0.98	0.77	0.73
	N	105	103	106	105	104	106	105	103	106	103

Conclusion Urethral sphincter volume does not change as a result or in association with increased bladder neck mobility, however bladder neck movement is greater with increasing levator hiatus size.

These data suggest that urethral hypermobility and urethral sphincter insufficiency are separate

factors in the genesis of stress incontinence. These data also suggest that the degree of bladder neck movement is not enough to contribute significantly to nerve damage and that nerve disruption, if significant occurs through mechanisms.

[1] Int J of Colorectal surgery 1995; 10(2):107-11
[2] BJOG 105:1300-1307

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Author(s): Chaliha C*°Khullar V, °°Sultan AH,*Stanton SLS •
Institution, city, country:
* St George's Hospital Medical School, London, °Imperial College School of Medicine °°Mayday University Hospital, Surrey

Title (type in CAPITAL LETTERS, leave one blank line before the text):
POSTPARTUM STRESS INCONTINENCE: A GENUINE SYMPTOM?

Aims of study: Postpartum incontinence is common with a reported incidence of up to 35%[1,2]. However is not clear whether this reflects the true incidence of genuine stress incontinence as most data is based on self reporting of symptoms and is often retrospective and unvalidated by objective studies. The aetiology of postpartum incontinence is unclear and commonly stress incontinence is thought to be secondary to genuine stress incontinence secondary to pelvic floor trauma at vaginal delivery [1,2,3].
The aims of this study were to undertake an alternative analysis of our subjective and objective epidemiological data to analyse postpartum urinary symptoms and their relationship to the underlying urodynamic diagnosis.
Methods: 161 nulliparous women were assessed at 12 weeks postpartum. A urinary questionnaire was completed asking questions regarding urinary frequency, nocturia, hesitancy, dribbling, incomplete emptying, staining to void, urgency, urge incontinence and stress incontinence. All women then underwent subtracted cystometry in the sit and stand fill position. Exclusion criteria included a history of urinary tract infection, anatomical urinary tract abnormality, diabetes or neurological abnormalities. All definitions conform to the ICS classifications.
Results: The incidence of reported urgency, urge incontinence and stress incontinence were 14%, 5% and 19%, respectively. The incidence of genuine stress incontinence, detrusor instability and voiding disorder were 5%, 6.8%, and 3.1% respectively. Normal urodynamic findings were found in 85.1% of women.
Symptom analysis of these women compared to urodynamic findings is shown in Table 1.
The urodynamic findings in those who were asymptomatic were compared with those who reported stress incontinence, or irritative urinary symptoms (urgency and/or urge incontinence), and are shown in Table 2.
Stress incontinence was reported by all women with a urodynamic diagnosis of genuine stress incontinence but also by those who had detrusor instability. Irritative urinary symptoms were also as common in both groups.
In those women who had a vaginal delivery urgency, urge incontinence and stress incontinence were reported by 15%, 5% and 21% respectively and 10%, 3% and 13% respectively of those who had a caesarean section. The incidence of detrusor instability and genuine stress incontinence on urodynamics was 7% and 5% respectively in those who underwent a vaginal delivery, and 6% and 3% in those who underwent a caesarean section.

Table One. Urinary symptoms and urodynamic diagnosis

Urinary symptoms	Urodynamic diagnosis			
	Normal (n=136)	Genuine stress Incontinence (n=8)	Detrusor instability (n=12)	Voiding disorder (n=5)
Stress incontinence	15%	100%	17%	-
Urgency	12%	38%	33%	-
Urge incontinece	5%	13%	-	-
Nocturia	32%	38%	33%	40%
Frequency	26%	25%	25%	40%
Hesitancy	0.7%	-	-	-
Poor stream	0.7%	-	-	-
Incomplete emptying	5%	-%	-	20%
Dribbling post voiding	1.4%	-	8%	20%