increases bladder compliance in spinal cord injured patients. This treatment modality linked to chronic and automatic monitoring of bladder activity, may become an alternative to augmentation procedures and the Brindley prosthesis.

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A RANDOMISED TRIAL OF THE EFFECTS OF CAFFEINE UPON FREQUENCY, URGENCY AND URGE INCONTINENCE

Aim of the Study: Recommending caffeine reduction is an internationally accepted practice among continence practitioners who treat the symptoms of urinary urgency, frequency and urge incontinence. Despite an apparent consensus that caffeine does impact on urinary symptomatology, little is known about the size/nature of its effect, and whether usage pattern/history and demographics are significant factors mediating any effect. Previous workers have demonstrated a detrusor pressure rise on bladder filling among a small sample of symptomatic women who received a 200mg oral caffeine bolus prior to urodynamics[(1)]. Others have investigated urinary symptomatology in healthy younger men and found that caffeine consumption was assocated with urinary symptoms in 2-13% of subjects. Despite a lack of evidence regarding the effects of caffeine upon urinary frequency/urgency, some published commentaries identify caffeine as an inducer of bladder hyperactivity[(5)] and recommend that caffeine decrease should reduce urinary symptoms[(2,3,4,)]. Therefore, we aimed to investigate the effect of caffeine reduction upon urinary frequency, urgency and urge incontinence.

Method: a prospective randomised trial was conducted among consecutive adult patients with symptoms of urinary, frequency and/or urge incontinence referred to two nurse continence advisers at community health continence clinics. Patients who routinely ingested 100mg or more of caffeine per day were invited to join the trial. Patients with significant cognitive impairment or symptoms of urinary tract infection were excluded. Ethics Committee approval was granted for the study and patients received no remuneration for their participation. Patients were allocated to an intervention or control group. The intervention group received bladder training and advice to reduce their caffeine intake to <100 mgs per day. The control group received bladder training and a request to continue their normal caffeine intake during the one month study period. All subjects completed a caffeine usage history and a three day time/volume chart at trial entry. Subjects were enrolled in the trial for 30 days and during that period completed time/volume charts (including leakage and caffeine intake) for three days of each of the four trial weeks. The target sample population is 90 subjects and the results displayed below reflect findings in relation to the first 36 patients.

Results: 10 of the 36 consented subjects (28%) were lost to follow up. The results displayed below summarise findings for the remaining 26 participants. The mean age of the sample was 59 year (range 31-79) and 88% were female. A comparison of key baseline measures is set out in Table 1:

Table 1: Baseline measures

	Intervention Group	Control Group
Daily caffeine intake mg (mean)	193	315
Frequency (voids/day) (mean)	10	10
Urgency (occasions/day) (mean)	4	5
Leakage (occasions/day) (mean)	4	4

Differences between the two groups regarding baseline caffeine intake were marked. However, this value displays high variability and numbers of subjects are still relatively low. Outcomes after 30 days were measured by calculating change in voids/day, occasions of urgency/day and occasions of leakage/day. Comparative results are set out in table 2.

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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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