ground electrode was sited over the lateral chest wall. Accurate localisation was determined by evaluating the appropriate motor and sensory responses to electrical stimulation. Once accurate placement was achieved the electrode leads were passed down the foramen needles and coupled to a hand-held pulse generator.

Lateral sacral X-rays were performed following insertion and prior to lead removal. The distance between the lead-tip and the ventral surface of the S3 foramen was measured on each radiograph by two independent assessors. Lead migration was evaluated by comparison of the distance from the S3 foramen to the lead-tip immediately following lead insertion and prior to lead removal. The efficacy of continuous sacral nerve root stimulation was assessed over a seven-day period using the new lead. Subjective improvement in symptoms was assessed by analysis of the urinary diaries before and during therapy. A reduction of 50% or more in the mean number of incontinent episodes and or urinary frequency per day was interpreted as a positive response.

Results: The mean patient age was 49 years (range 23-79 years). The mean duration of symptoms was 42 months (30-120 months). 7(58%) had a diagnosis of detrusor instability and 5(42%) interstitial cystitis. One connector pin lead on the PNE 3057 model became detached at the time of insertion and required replacement. There were no complications recorded during the trial period. 10(83%) patients were positive responders. There was good correlation between the distances measured by both assessors between the lead-tip and ventral surface of the S3 foramen on X ray after insertion ($r = 0.88$) and prior to removal ($r=0.95$, Pearson's correlation test). The mean distance migrated by the new lead was 4mm (range 2-11mm) compared to a mean migration distance of 12mm(range 10-45mm) for the old lead ($p = 0.02$, Wilcoxon rank sum test).

Conclusion: The new PNE electrode described in this study is associated with a high positive response rate during trial stimulation and reduced lead migration. This new lead may help overcome the problem of determining whether a negative trial stimulation is due to lead migration or true non-response and reduce the need for bilateral lead placement or repeat test stimulation.

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

The Role of Angiotensin II in Detrusor Overactivity

AIMS OF STUDY: Detrusor overactivity is a major cause of bladder dysfunction and has a dramatic impact on health care costs and quality of life. Despite the considerable prevalence of detrusor overactivity, the pathophysiology of this problem is poorly understood.

Angiotensin II (AngII) plays an important role in the development and progression of cardiovascular and renal diseases [1]. An overproduction of locally generated Ang II can be induced by mechanical stretch and is observed in experimental animals models of hypertension such as

spontaneously hypertensive rats (SHR). In the kidney and cardiovascular system of SHR, increased autocrine/paracrine regulators, increased isolated smooth muscle oscillations, sympathetic hyperinnervation, smooth muscle hypertrophy, and accumulation of extracellular matrix proteins have been well described. Furthermore, alterations in the local renin-angiotensin system have been linked to each of these processes [2,3]. Inhibition of the renin-angiotensin system in SHRs has been shown to improve the functional properties of vascular smooth muscle [4], normalize the sympathetic hyper-reactivity [5], and restore levels of paracrine regulators. Recent studies have shown that micturition in SHR is characterized by decreased bladder capacity and increased voiding frequency [6]. This type of voiding pattern is typical of detrusor overactivity. The bladders of SHRs have increased catecholamine and decreased acetylcholine concentrations, elevated nerve growth factor secretion and noradrenergic hyperinnervation compared with normal rats [7,8]. These data parallel the cellular processes occuring in the renal and cardiovascular systems of SHRs that have been attributed to an over production of AngII. Since AngII is also produced in bladder smooth muscle, these similarities point to the possibility that increased local production of AngII may play a role in detrusor overactivity. Therefore, the purpose of this study was to determine the effect of AngII receptor inhibition on bladder function in an animal model of detrusor overactivity.

METHODS: SHRs and normotensive control (WKY) rats were anesthetized and a double lumen catheter was inserted per urethra into their bladders for filling and measurement of bladder pressure. The bladder was filled at 0.1ml/min to a cystometric volume of 1ml. Bladder compliance and the frequency and amplitude of spontaneous contractions were measured. At cystometric capacity, the pelvic nerve was stimulated (2-8volts) and the isovolumetric pressure change was measured to generate a pressure-voltage response curve. At least 3 minutes elapsed between stimulations to ensure reproducibility of the response to nerve stimulation. Bladder compliance, spontaneous activity and stimulation responses were measured before and after intravenous administration of an Ang II receptor antagonist (eprosartan).

RESULTS: Bladders of SHRs demonstrate increased spontaneous contractions of significantly greater amplitude compared with normotensive rats (figure 1). These findings indicate a pattern of marked detrusor overactivity in this animal model. Bladder compliance in SHR was significantly lower (0.054 ± 0.038 ml/cmH_2O) than in WKY (0.13 ± 0.081 ml/cmH_2O).

The frequency and amplitude of spontaneous bladder contractions during filling cystometry were suppressed by administration of eprosartan in SHRs in a dose dependent manner (figure 1). In addition, eprosartan significantly improved bladder compliance in SHRs (figure 2). Pelvic nerve stimulation (PNS) produced a voltage-dependent increase in isovolumetric bladder pressure that was attenuated after intravenous administration of eprosartan (figure 3). Systemic blood pressure was unchanged during eprosartan administration.

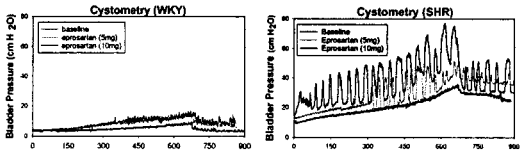

Figure 1: Spontaneous contractions during cystometry were more apparent in SHRs (right panel). The frequency and amplitude of spontaneous activity were significantly higher in SHR compared to WKY rats. Administration of eprosartan had a moderate effect on spontaneous activity in WKY at higher concentrations (left panel). In SHRs, eprosartan significantly decreased spontanous activity at lower doses.

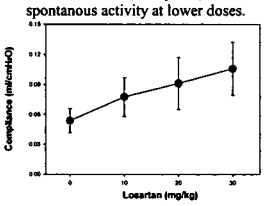

Figure 2: In SHRs, eprosartan administration produced a significant dose dependent improvement in bladder compliance.

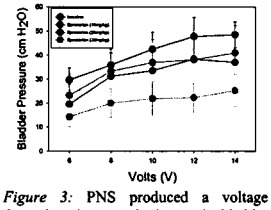

Figure 3: PNS produced a voltage dependent increase in isometric bladder pressure. This response was attenuated after eprosartan administration in SHRs.

CONCLUSION: These results suggest that Ang II may play a role in detrusor overactivity. The finding that spontaneous contractions can be diminished with administration of Ang II receptor antagonists supports the concept that inhibition of the renin-angiotensin system in the bladder may alleviate the symptoms or prevent the development of bladder overactivity.

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

IS THERE AN ASSOCIATION BETWEEN ANTERIOR VAGINAL WALL RELAXATION AND DETRUSOR INSTABILITY?

Aims of Study

About a decade ago, Ulf Ulmsten and Peter Papa Petros first proposed their "Intregral Theory of Pelvic Floor Dysfunction" (1) which has subsequently been put forward and developed further in a large number of publications. It has also resulted in the development of surgical procedures designed to cure stress and urge incontinence (2,3,4), prolapse, voiding dysfunction (5), symptoms of bowel dysfunction and chronic pelvic pain (6). One of the main tenets of the Integral Theory is that urgency, frequency, nocturia and urge incontinence are caused by anterior vaginal wall relaxation.
So far there has been no systematic attempt to prove or disprove this hypothesis which, if true, would allow us to surgically cure a condition that, so far, has been regarded as virtually incurable (7). This study attempts to correlate symptoms and urodynamic signs of bladder irritability (frequency, urge incontinence, nocturia, sensory urgency and detrusor instability) with ultrasonic evidence of anterior vaginal wall relaxation.

Methods

275 consecutive patients with symptoms of lower urinary tract dysfunction underwent urodynamic evaluation. After completing a detailed history, multichannel urodynamics using microtransducer catheters were performed. Multiple challenge manoeuvres such as fast fill at 50ml/ min., tilting to the upright position, coughing and handwashing in cold water, were undertaken to optimise detection of detrusor instability. Imaging was performed both with fluoroscopy and with translabial ultrasound, the latter after bladder emptying and in the supine position. A variety of ultrasound scanners were used with 3.5- 7 MHz curved array transducers. Findings were documented on videotape and/or printers and evaluated subsequently by the first author who was blinded regarding the urodynamic data. 272 datasets were complete and used for the analysis. Translabial imaging data was collected as previously described (8) with the addition of quantification of maximal cystocele descent on Valsalva manoeuvre. Descriptive statistics were obtained with Microsoft Excel on a PC system. Comparative statistics were performed using minitab ™ (v 12).

Results

Table 1 shows correlations between indices of anterior vaginal wall descent and lower urinary tract symptoms. There were the expected positive relationships with stress incontinence but none of the other symptoms correlated positively. Opening of the retrovesical angle was negatively associated with nocturia and urge incontinence. The former was also negatively associated with bladder neck descent and descent of a cystocele.

Symptoms	Stress Incontinence	Frequency	Nocturia	Urge Incontinence
US parameters				
RVA-S	p=0.0002*	p=0.091	p=0.005#	p=0.0036#
Rotation	p=0.0088*	p=0.96	p=0.077	p=0.46
BND	p<0.0001*	p=0.41	p=0.002#	p=0.17
Cystocele	p=0.0063*	p=0.61	p=0.0043#	p=0.26

Table 1: Correlations between indices of anterior vaginal wall descent and symptoms of lower urinary tract dysfunction (* positive, # negative relationship).