

anesthetized and conscious rats. NMB has been shown to contract bladder smooth muscle via an action on one of the receptors in the bombesin group (now designated as BB_1). Therefore, it was hypothesized that NMB might selectively contract the bladder *in vivo* and thereby influence micturition.

Methods

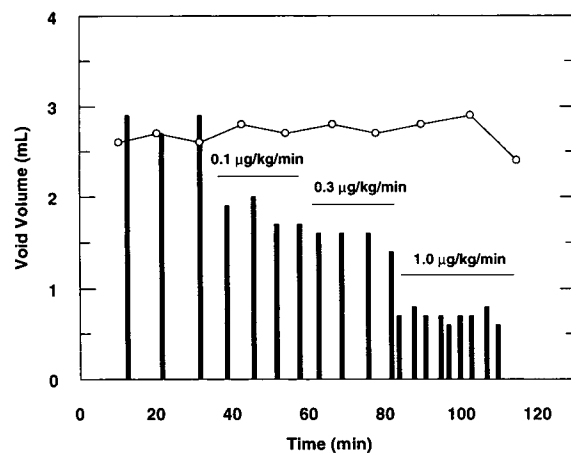
Bladder tone was evaluated in anesthetized female rats (200-250 g; urethane, 1.5 g/kg, IP) by measuring pressure under isovolemic conditions. The ureters were tied, and a cannula inserted into the urethra. The bladder was partially filled with physiological saline (0.25 ml), and NMB was administered via a cannulated jugular vein.

Effects on micturition in conscious animals were evaluated either in rats with chronically implanted gastric, venous and arterial cannula or in rats with these cannulae plus a bladder cannula. Animals were allowed at least two weeks recovery prior to an experiment. Rats were placed in a restraining cage to allow collection of urine. Physiological saline was administered via bladder cannula, and micturition interval and void volume were measured. NMB was administered via intravenous infusion.

Results

In the anesthetized rat, a dose-related increase in bladder pressure was observed, with doses as low as 1 $\mu\text{g}/\text{kg}$ producing a measurable response, and a maximum pressure of 72 ± 10 cm H_2O obtained at 34 $\mu\text{g}/\text{kg}$. The ED_{50} for NMB in this assay was approximately 3 $\mu\text{g}/\text{kg}$.

As shown in the figure, intravenous infusion of NMB to a conscious rat during bladder perfusion produced a decrease in the interval between spontaneous micturitions, and a decrease in void volume. Summary data from 3-4 animals showed NMB to decrease void volume from a control value of 2.3 ± 0.4 ml to 1.98 ± 0.4 , 1.49 ± 0.1 and 0.78 ± 0.1 ml at infusion rates of 0.1, 0.3 and 1.0 $\mu\text{g}/\text{kg}/\text{min}$, respectively. No effect on blood pressure or heart rate was observed at any of these doses, and no changes in the behavior of the rat were apparent. Similar effects were observed in either WKY or SH rats without bladder cannulae, where diuresis was induced by an oral water load (3% body weight). In these animals, NMB at 0.3 $\mu\text{g}/\text{kg}/\text{min}$ decreased urine volume per void from 3.2 ± 0.7 to 1.9 ± 0.4 ml (WKY) or 1.5 ± 0.2 to 0.6 ± 0.2 ml (SHR).



Effect of Intravenous Infusion of NMB on micturition in a conscious rat. Bladder perfused with saline at 0.18 ml/min. Each bar denotes a spontaneous micturition. Void volumes /interval in a control saline-perfused rat shown by open circles.

Conclusions

In vitro data, both from the literature and our laboratory, show that functional BB_1 receptors are present on rat bladder. In the anesthetized rat, systemic administration of NMB will contract the bladder, with a maximum response equivalent to that produced by other bladder spasmogens such as the tachykinins. These *in vitro* and *in vivo* data suggest that the decrease in void volume produced by NMB in the conscious rat is a consequence of contraction of bladder smooth muscle, which would reduce the volume required to trigger spontaneous micturition. As previously reported [1] void volumes of SHR were lower than WKY; NMB produced a similar reduction in the two strains. Effects on bladder smooth muscle appear to be selective, since blood pressure was unaffected by a dose of NMB having dramatic effects on micturition parameters. NMB is present in spinal cord, and has been postulated to be involved in the control of micturition [2]. The ability of exogenous NMB to decrease bladder capacity suggests a

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potential role for the endogenous peptide, and the possibility that a selective NMB antagonist may have therapeutic activity in conditions where bladder capacity is inappropriately low.

References:

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

THE INFLUENCE OF INTRATHECAL BACLOFEN ON DETRUSOR FUNCTION – A URODYNAMIC STUDY

Aims of Study: The effect of Baclofen (B.) on the striated muscle is well known, however, its effect on the detrusor resp. its clinical relevance, especially when administered intrathecally, is still under discussion. Urodynamic studies were undertaken before and after the implantation of a B.pump to objectify the influence of B. on the detrusor.

Methods: between IV/99 und XII/99 a B.pump was implanted in 6 pat.s, 23-40 yrs., mean age 25 yrs., because of severe cerebral spasticity. Preoperatively (I), between 24 and 48 hours postop. (II) and at least 10 days postop., after achieving the therapeutic drug release dosage (III), urodynamics, according to ICS-criteria, were performed: the volume at the first hyperreflexic contraction, the max. bladder capacity, the max. detrusor pressure and the residual urine were evaluated.

Results:

Volume at first contraction: (I) 143.6 cc, (II) 248.3 cc, (III) 486.2 cc, the differences between I and III, $p < 0.001$, and between I and II, $p < 0.05$, stat.significant

Max.cystometric bladder capacity: (I) 169.4 cc, (II) 270.2 cc, (III) 483 cc, the differences between I and III, $p < 0.001$, stat.significant

Max. detrusor pressure: (I) 89.0 H₂O, (II) 73.2 H₂O, (III) 31.0 H₂O, the differences between I and III, $p < 0.005$, stat.significant

Residual urine: (I) 18.2 cc, (II) 26.2 cc, (III) 69.5 cc, the differences between I and III, $p < 0.05$ stat.significant

Conclusions: B. administered intrathecally by pump delivery increases the volume at the first hyperreflexic contraction, the max. cystometric bladder capacity and the residual urine, the max. detrusor pressure decreased considerably. Therefore after implantation of a B.pump possible effects on micturition must be checked, because unbalanced voiding could occur and could cause secondary damage to the lower urinary tract. In our 6 pat.s however, despite a considerable decrease of the detrusor pressure and an increase in the max. cystometric bladder capacity the uninhibited micturition remained balanced and no further measurements were necessary. It may well be that the relaxation of the striated sphincter by B. neutralizes the weakening of the detrusor, therefore micturition may remain balanced.

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Variation in the Apparent Diameter of the Female Urethra: An Example of Anisotropism

Introduction: The term anisotropism is derived from the Greek words *aniso* which is to be unequal or different and the verb *tropos* meaning to turn or direct. In the context of ultrasound this term implies that

the reflective pattern may be altered by changing the angle of insonation. Sonographic anisotropism was first described by Fornage in 1987 in relation to ultrasound evaluation of normal tendons [1]. The author recognised this phenomenon as a potential pitfall encountered in the evaluation of patients presenting with suspected tendon injury [1,2].

Transvaginal and perineal ultrasound has been used to demonstrate the female urethra in women presenting with urinary incontinence [3-5]. Previous reports have noted that the female urethra and bladder neck appear patent on perineal and endovaginal [EV] sonography performed in women with stress urinary incontinence [SUI] [4-7]. Urethral ultrasound however, has not gained widespread acceptance in the evaluation of SUI as there is no clear distinction between continent and incontinent subjects [4,8,9,10].

Aim: The aim of this study was to evaluate the effect of angle of incidence of ultrasound beam on the apparent width and patency of the female urethra and bladder neck.

Methods: Seven women referred for routine pelvic ultrasound assessment from a Gynaecology Clinic (four nulliparous and three multiparous) were prospectively recruited. Each woman was questioned regarding symptoms of altered urinary continence. Translabial and EV ultrasound was performed in all cases using a linear array 6-10 MHz transducer and a 6-8 MHz EV transducer in the supine position. The appearance of the urethra was then re-assessed in all seven cases, varying the angle of the incident beam to the urethra. All women were asked to strain to assess the effect of mechanically altering the angle of the urethra in relation to the incident beam.

Results: The mean patient age was 24 years (range 22 - 43years). None of the women reported any symptoms of altered urinary continence. Translabial and EV ultrasound images obtained in the sagittal and coronal planes demonstrated the urethra as a widely patent hypoechoic funnel tapering toward the perineal surface. The bladder neck and urethral walls appeared open and unopposed in sagittal and coronal planes. The mean urethral lumen diameter (measured 1cm inferior to the bladder neck) was 5.6mm in the sagittal plane (range 4.2 - 6.5mm). Increasing the transmit power and receiver gain and reducing the median frequency produced apparent narrowing of the urethra. Altering the angle of incidence of the ultrasound beam resulted in echogenic tissue appearing within what originally appeared as an empty lumen. The bladder neck and urethral lumen appeared narrower as the angle of incidence of the beam approached 90 degrees to the long axis of the urethra. Voluntary straining resulting in alteration of the bladder neck and urethra relative to the incident beam produced a similar effect to angling the ultrasound beam. Apparent narrowing of the bladder neck and urethra was observed as the mechanical act of straining moved the urethra into a position perpendicular to the ultrasound beam.

Conclusion: Our study suggests that the appearance of a patent bladder neck may be an ultrasound artefact due to *anisotropism* rather than true bladder neck patency. Previous studies reporting bladder neck and urethral closure on straining may have been inaccurate as apparent urethral closure can be an artefact due to the urethral axis rotating on straining. We suggest that this effect should be taken into consideration when performing ultrasound evaluation of the female bladder neck and urethra particularly with regard to stress urinary incontinence.

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