Urol Res (1998) 26:331–336

ORIGINAL PAPER

Ahmed Shafik

Effect of renal pelvic and ureteral distension on the striated urethral sphincter with recognition of the "reno-vesico-sphincteric reflex"

Received: 2 December 1997 / Accepted: 31 March 1998

Abstract Renal pelvic and ureteral distension occurs in physiologic (diuresis) and pathologic (calculus) conditions. Its effect on the vesical and posterior urethral pressures as well as on the electromyographic (EMG) activity of the striated urethral sphincter (SUS) was investigated. The renal pelvis of 10 healthy volunteers (7 men, 3 women; mean age 35.8 ± 8.6 years) was distended by means of a 4-F balloon-tipped catheter in increments of 2 ml of saline up to 10 ml and the response of the vesical and posterior urethral pressures and SUS EMG activity was recorded. The test was repeated with ureteral distension in increments of 0.25 ml up to 1 ml. The response of the aforementioned parameters was also registered after anesthetization of the renal pelvis, ureter and SUS. Two rates of renal pelvic and ureteral distension were tested: rapid (1 ml/s) and slow (1 ml/min). Renal pelvic distension with large volumes effected an increase of the renal pelvic and urethral pressures ($P < 0.05$, $P < 0.05$, respectively), a vesical pressure drop ($P < 0.05$) and increased EMG activity of the SUS. Ureteral distension caused a rise of ureteral and urethral pressures as well as of SUS EMG activity. With rapid distension, the aforementioned parameters responded at smaller volumes than with slow distension. Renal pelvic, ureteral or SUS anesthetization effected no urethral or SUS EMG response. It is suggested that the reaction of above parameters to distension indicates a mechanism regulating the urine flow so as to protect the renal pelvis and the ureter from being overloaded. The vesical pressure drop with increased SUS EMG activity on renal pelvis distension postulates a reflex relationship that we call the "reno-vesico-sphincteric reflex". The role of this reflex in urine transport requires further study.

Key words Micturition · Urination · Urethra · Pressure · Urinary bladder

A. Shafik
Department of Surgery and Experimental Research,
Faculty of Medicine, Cairo University,
Cairo, Egypt

A. Shafik (✉)
2, Talaat Harb Street, Cairo, Egypt
Tel./Fax: +20-2-349 8851

Introduction

Distension of the renal pelvis or ureter occurs in physiologic and pathologic conditions as for example in diuresis or in obstruction by a calculus [4, 6, 9, 10]. Recent studies have shown that renal pelvic distension evoked the renal pelvivesical reflex which effected a pressure drop within the ureteropelvic junction (UPJ), in the ureterovesical junction (UVJ) and in the urinary bladder [12]. The opening of both the UPJ and UVJ as well as vesical relaxation seem to assist the delivery of urine from the renal pelvis to the urinary bladder thus protecting both the renal pelvis and the ureter against overloading and consequent dilatation [12].

The striated urethral sphincter (SUS) is a striated muscle which consists of fast twitch fibers; it contains also slow twitch fibers [3, 5, 13, 16]. It acts to oppose, interrupt or terminate the act of micturition and this is conducted through the fast twitch fibers [2, 8, 14, 15]. However, it has a tonus induced by the slow twitch fibers. The role of the SUS during renal pelvic or ureteral distension by an obstructive lesion in the urinary passages could not be traced in the literature.

The purpose of this communication is to study the effect of renal pelvic or ureteral distension on the striated urethral sphincter. The study was approved by our Faculty Review Board.

Materials and methods

Subjects

Ten healthy volunteers were enroled in the study after giving informed consent. Seven were men and three women. The mean age

was 35.8 ± 8.6 SD years (range 26–46), and the mean weight 67.2 ± 5.3 (range 61–72). The subjects had no genitourinary manifestations in the past or at the time of presentation. Physical examination, including neurologic assessment, was normal. Urinalysis and sonogram of the urinary tract showed unremarkable findings.

Methods

With the subject lying supine, a 4-F balloon-tipped ureteral catheter with a metallic clip at its distal end for fluoroscopic control was introduced cystoscopically into the renal pelvis. The balloon measured 0.5 cm in diameter and was made of latex (London Rubber Industries, London, UK). The catheter was connected to a strain gauge pressure transducer (Statham, 230 B, Oxnard, Calif.). The balloon was filled with saline in increments of 2 ml. Renal pelvic and ureteral distension was done twice: rapidly and slowly. The balloon was filled at a rate of 1 ml/s in the rapid and 1 ml/min in the slow distension. Pressure measurements started with the gauge at zero level. The balloon response to filling when it was not in the renal pelvis (i.e. in air) showed no transient pressure rise similar to the one obtained when the balloon was filled in the renal pelvis; this indicates that the response was from the renal pelvis and not from the balloon.

The pressures in the urinary bladder and posterior urethra were simultaneously measured by means of a two-channel microtip catheter (Wiest, Urocompact, Calif.). The catheter was introduced so that the distal transducer lay in the urinary bladder while the proximal one was adjusted to lie in the posterior urethra. The microtip transducer faced laterally in the urethra. The positioning of the catheter was controlled fluoroscopically. The bladder was emptied before the measurements began.

The response of the SUS EMG activity to rapid and slow renal pelvic and ureteral distension was evaluated by inserting a concentric needle electromyographic electrode into the SUS using the technique previously described [11]. The normality of the SUS EMG activity had been proved before starting the experiment.

With the balloon in the renal pelvis, the response of the SUS EMG to rapid and slow balloon distension in increments of 2 up to 10 ml was determined. The balloon was then withdrawn from the pelvis to the ureter using an automatic catheter withdrawal device (902, Disa, Copenhagen, Denmark); the ureter was distended by inflating the balloon in increments of 0.25 ml up to 1 ml. The catheter positioning was fluoroscopically controlled when it was found necessary. The effect of rapid and slow ureteral distension of the upper, middle and lower third of the ureter on the ureteral, vesical and posterior urethral pressures as well as on the EMG activity of the SUS was recorded.

Anesthetization of the renal pelvis, ureter and SUS

The vesical and urethral pressure and SUS response to distension of the anesthetized renal pelvis or ureter was evaluated. The renal pelvis and ureter were anesthetized by administration of 5 ml of 2% xylocaine (Astra, Södertälje, Sweden) diluted with 10 ml of saline into the renal pelvis and the ureter by means of a 3-F ureteral catheter. The vesical and urethral pressure and EMG response to separate renal pelvic and ureteric distension as aforementioned was determined after 20 minutes of xylocaine administration and 2 hours later when the anesthetic effect had worn off. The test was repeated after saline administration in the renal pelvis and ureter.

On another day, the SUS was anesthetized by injecting 1 ml of 2% xylocaine into the substance of the sphincter at different points using the technique previously described [11]. The response of the vesical and urethral pressures as well as of the SUS EMG activity to renal pelvic and ureteral distension was assessed 20 minutes and 2 hours after anesthetic administration. The test was repeated after saline injection into the SUS.

To assure reproducibility, the aforementioned measurements were repeated at least twice and the mean value was calculated. The results were analyzed statistically using the Student's *t*-test. Differences assumed significance at $P < 0.05$ and values were given as mean ± SD.

Results

The aforementioned tests were completed in all the volunteers with no adverse effect. The mean basal pressure in the renal pelvis was 6.6 ± 1.4 cm H_2O (range 4–9), in the ureter 5.6 ± 1.6 cm H_2O (range 3–8), in the urinary bladder 5.2 ± 1.3 cm H_2O (range 3–7) and posterior urethra 80.9 ± 11.2 cm H_2O (range 71–89).

Effect of renal pelvic distension on vesical and urethral pressures

The effect of rapid and slow renal pelvic distension on the renal pelvic, vesical and posterior urethral pressures is shown in Figs. 1–4. Renal pelvic distension, rapid and slow, produced no significant pressure changes with small volume distension (2 and 4 ml, $P > 0.05$). Large volume distension (6–10 ml) effected elevation of the pressure in the renal pelvis and posterior urethra ($P < 0.05$, and $P < 0.05$, respectively) associated with a drop of the vesical pressure ($P < 0.05$; Figs. 1–4). However, rapid renal pelvic distension produced significant changes in the aforementioned parameters with volumes smaller than in the slow distension (Figs. 1–4). Furthermore, while the renal pelvic pressure continued to rise as the renal pelvic distension increased, the vesical and urethral pressure response showed no significant change on renal pelvic distension with various volumes whether performed slowly or rapidly (Figs. 1, 3).

The pressure response of the renal pelvis, bladder and urethra was momentary, i.e., it remained for a mean period of 6.6 ± 1.2 seconds (range 4–8) and returned to the basal level even though the renal pelvis was still distended by the balloon. However, the same response was obtained when, after distension release, the renal pelvis was re-distended.

Effect of ureteral distension on vesical and urethral pressures

The effect of rapid and slow ureteral distension on the pressure in the ureter, urinary bladder and posterior urethra is shown in Figs. 5–8. Rapid distension effected a significant rise of the ureteric and urethral pressures at 0.50, 0.75 and 1 ml ureteric distension; the vesical pressure showed no significant change (Figs. 5, 6). Slow ureteric distension effected pressure responses similar to those of the rapid distension but only at 1 ml distension (Figs. 7, 8); no response was obtained with 0.25, 0.5 and 0.75 ml distension (Fig. 7). These results were achieved with no significant difference ($P > 0.05$) whether the ureter was distended in its upper, middle or lower third and whether distended slowly or rapidly.

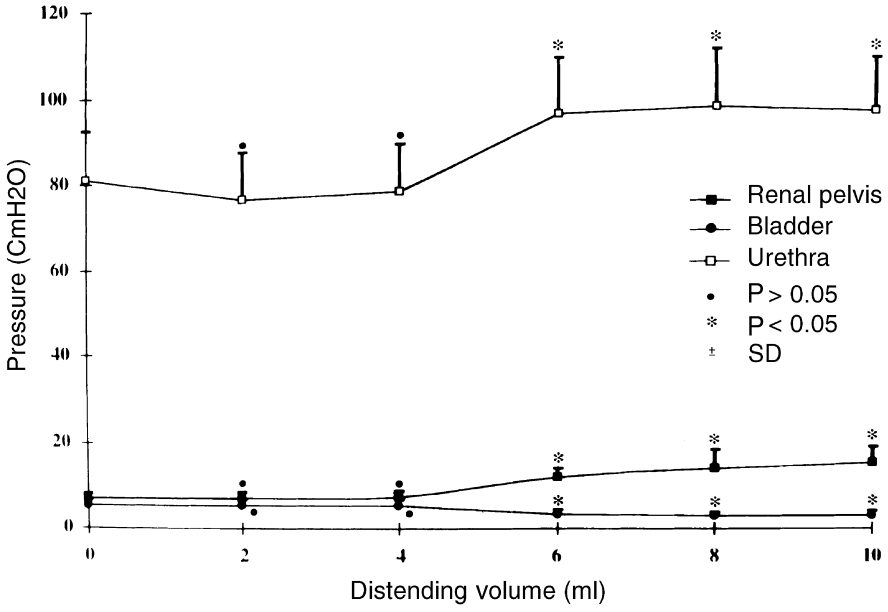


Fig. 1 The effect of rapid renal pelvic distension on the pressure in the renal pelvis, urinary bladder and posterior urethra

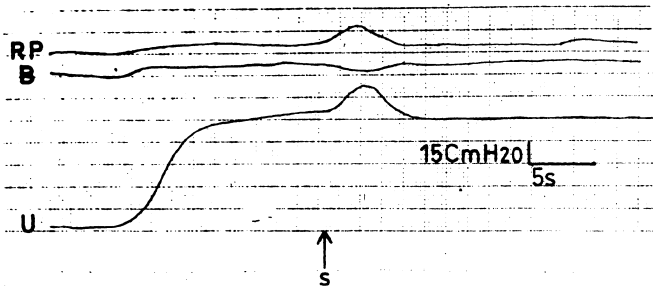


Fig. 2 Pressure tracing showing the effect of rapid renal distension with 8 ml of saline on the pressure in the renal pelvis (*RP*), urinary bladder (*B*) and posterior urethra (*U*); *s* stimulus

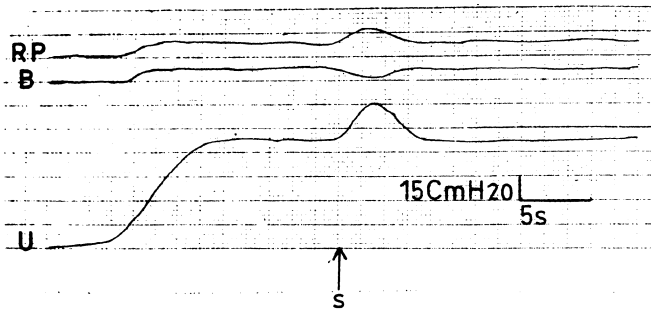
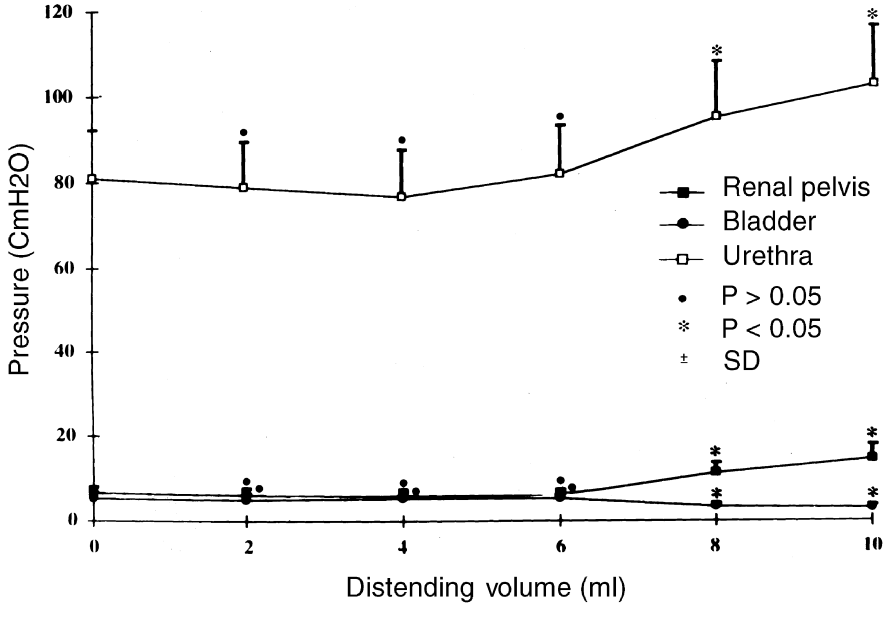


Fig. 4 Pressure tracing showing the effect of slow renal distension with 8 ml of saline on the pressure in the renal pelvis (*RP*), urinary bladder (*B*) and posterior urethra (*U*); *s* stimulus

Fig. 3 The effect of slow renal pelvic distension on the pressure in the renal pelvis, urinary bladder and posterior urethra



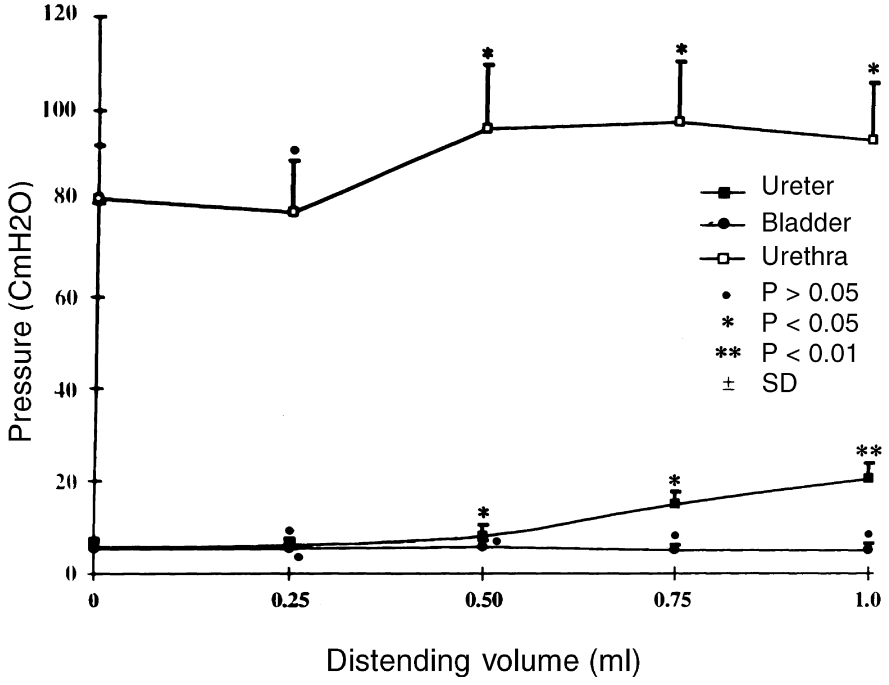


Fig. 5 The effect of rapid ureteral distension on the pressure in the ureter, bladder and posterior urethra

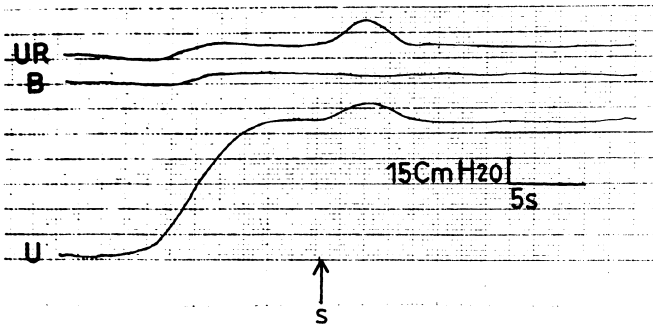


Fig. 6 Pressure tracing showing the effect of rapid ureteral distension with 1 ml of saline on the pressure in the ureter (*UR*), bladder (*B*) and posterior urethra (*U*); *s* stimulus

Response of the SUS EMG activity to renal pelvic and ureteric distension

The SUS showed, at rest, a mean motor unit action potential of 78.6 ± 18.4 μV (range 56–90; Fig. 9). Upon rapid renal pelvic distension with 2 and 4 ml, no SUS response was obtained. The sphincter showed increased EMG activity at 6, 8 and 10 ml renal pelvic distension with a mean motor unit action potential of 546.2 ± 96.3 μV (range 408–712; $P < 0.001$; Fig. 9). The amplitude of increased EMG activity of the SUS showed no significant difference between the 6, 8 and 10 ml renal pelvic distension ($P > 0.05$). Slow renal pelvic distension effected an increased SUS EMG activity at 8 and 10 ml with no significant difference between the two distending volumes ($P > 0.05$).

Rapid ureteral distension produced increased EMG activity at 0.50, 0.75 and 1 ml distension, with no significant difference between the distending volumes ($P > 0.05$; Fig. 10). The sphincter recorded mean motor unit action potentials of 492.8 ± 82.2 μV (range 396–678). Slow ureteric distension effected increased SUS EMG activity only with 1 ml distension. The amplitude of motor unit action potentials was similar to that of rapid ureteral distension ($P > 0.05$).

Urethral and vesical pressures and EMG responses to anesthetized renal pelvis, ureter or SUS

Distension of the anesthetized renal pelvis or ureter 20 minutes after anesthetic administration produced no significant change ($P > 0.05$) in the vesical and urethral pressures nor in the SUS EMG activity. Two hours later when the anesthetic effect had disappeared, the pressure and EMG responses were similar to those before anesthetic administration ($P > 0.05$). On the other hand, distension of the renal pelvis or ureter while the SUS was anesthetized produced no urethral or SUS EMG responses, whereas the urinary bladder responded as aforementioned. Saline administration into the renal pelvis or ureter or saline injection into the SUS did not affect the vesical and urethral pressure or SUS EMG responses to renal pelvic or ureteric distension; the response was similar to that without saline administration or injection ($P > 0.05$).

The aforementioned results were reproducible with no significant difference ($P > 0.05$) when the tests were repeated in the same subject.

Discussion

The current study has demonstrated that filling of the renal pelvis or ureter with small volumes was not asso-

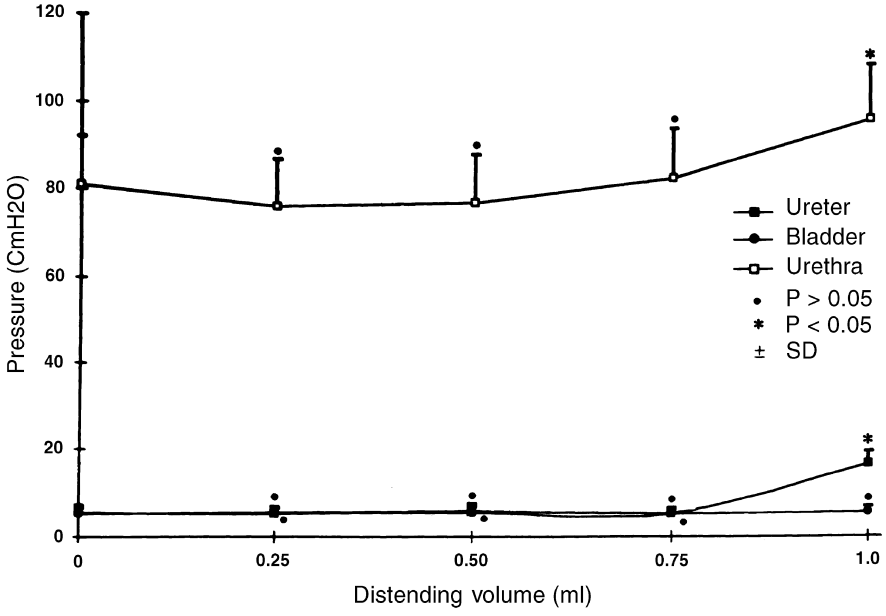


Fig. 7 The effect of slow ureteral distension on the pressure in the ureter, bladder and posterior urethra

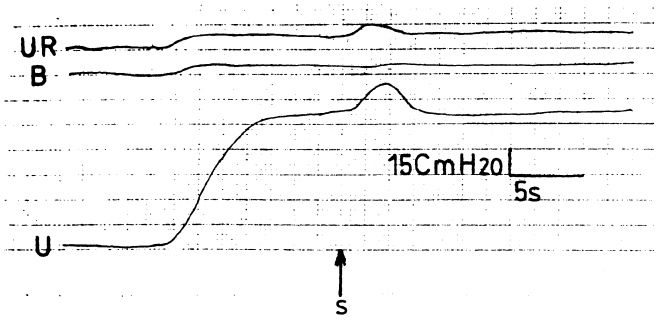


Fig. 8 Pressure tracing showing the effect of slow ureteral distension with 1 ml of saline on the pressure in the ureter (*UR*), bladder (*B*) and posterior urethra (*U*); *s* stimulus

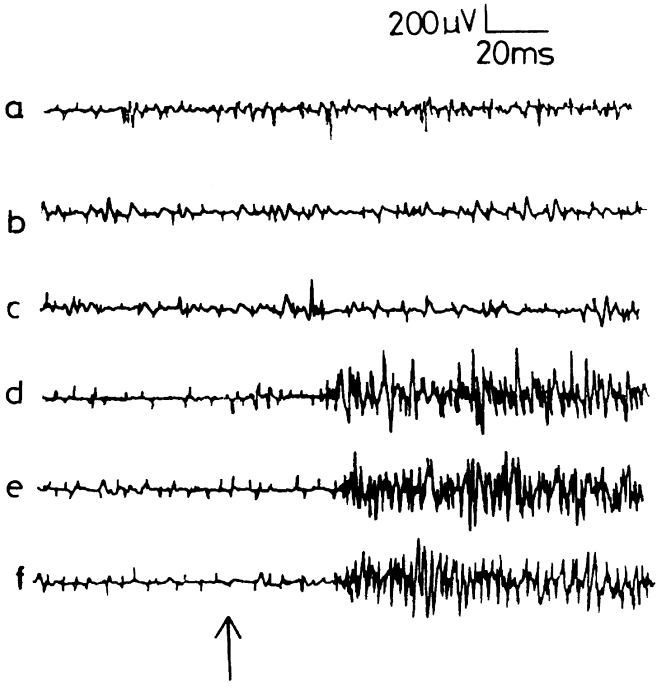


Fig. 9 EMG activity of the striated urethral sphincter on rapid renal pelvic distension. *a* Basal activity, *b*, *c*, *d*, *e* and *f* activity on 2, 4, 6, 8 and 10 ml distension, respectively (*arrow* points to start of distension)

ciated with significant changes in the renal pelvic, ureteric or vesical pressures nor in the EMG activity of the SUS. These volumes probably represent the volume which fills the renal pelvis or ureter under normal physiologic conditions. Distension of the renal pelvis or ureter with large volumes was associated with responses that are believed to assist urine transport through the pelvi-uretero-vesical passages with no adverse effect. Elevated pressure of the renal pelvis or ureter presumably indicates an increase of their contractile activity, which appears to enhance the urine flow when big volumes of urine collect in the renal pelvis or ureter as happens in case of obstruction or diuresis. The vesical pressure drop seems to denote relaxation of the urinary bladder preparatory to receiving the excess urine passing from the kidney. These responses are believed collectively to protect the renal pelvis and ureter from becoming overloaded and distended by large volumes of urine that enter the renal pelvis. The urethral pressure rise is believed to be due to an increase of the SUS EMG activity which probably acts to secure the continent mechanism under such abnormal conditions of renal pelvic and ureteric distension.

The current study also demonstrated that the responses of the aforementioned parameters differ according to the rate of renal pelvic or ureteral fillings. The response occurred earlier and with smaller volumes on rapid rather than on slow distension. The mechanoreceptors in the renal pelvic or ureteral wall appear to be stimulated earlier with rapid rather than with slow distension.

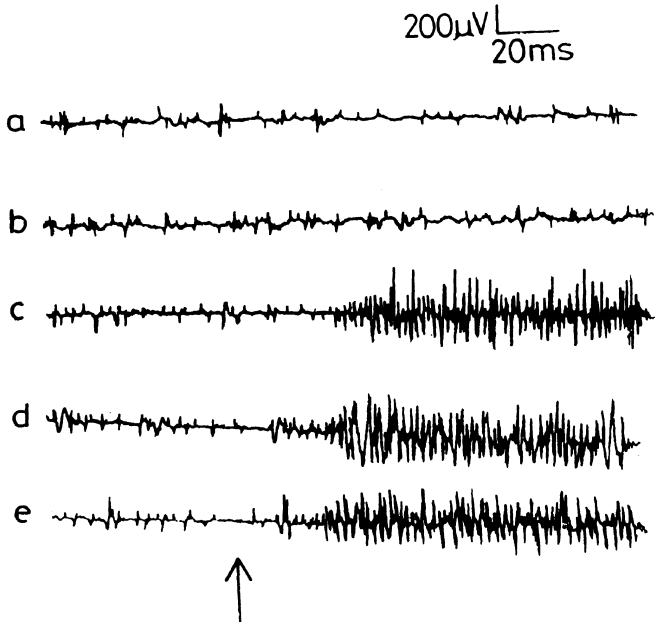


Fig. 10 EMG activity of the striated urethral sphincter on rapid ureteral distension. *a* Basal activity, *b*, *c*, *d*, and *e* activity on 0.25, 0.50, 0.75 and 1.0 ml distension, respectively (*arrow* points to start of distension)

Reno-vesico-sphincteric reflex

The vesical and SUS responses to renal pelvic distension seem to be reflex in nature. Renal pelvic distension when reaching a certain level appears to stimulate the mechanoreceptors in the renal pelvic wall [1, 7]. Impulses are suggested to be transmitted to the urinary bladder and SUS either through the intramural plexus of nerves or extramurally. Neuroregulation probably coordinates renal pelvic distension with vesical and SUS response. This neural action might be mediated through sympathetic–parasympathetic innervation, a point that needs to be studied.

The vesical pressure drop and increased SUS EMG activity on renal pelvic distension postulates a reflex relationship between them. This reflex relationship was reproducible and confirmed by its disappearance upon anesthetizing the renal pelvis or SUS, both probably representing the two arms of the reflex arc. The reflex which we call "reno-vesico-sphincteric reflex" is suggested to function to relax the urinary bladder and contract the SUS in regulation of the urine flow.

Clinical significance of the reno-vesico-sphincteric reflex

Renal pelvic and ureteric distension is a common urologic finding that occurs in obstructive lesions of the urinary passages [4, 6, 9, 10]. It presents clinically with loin pain. Patients with such lesions commonly complain also of difficulty in, or interruption of, micturition which may be associated with dysuria, despite the fact that the obstructive lesions occur in the upper urinary passages (ureter or renal pelvis) and that the urinalysis shows normal findings. We suggest that the reflex increased EMG activity of the SUS, which is evoked on renal pelvic or ureteric distension, might explain the difficulty of urination during an attack of loin pain from calcular ureteral or renal pelvic obstruction. On the other hand we do not know the role of the reno-vesico-sphincteric reflex that might be evoked in obstructive lesions of the lower urinary passages associated with ureteral and renal pelvic distension. We propose that in such lesions the activation of this reflex with consequent SUS contraction might result in augmentation of their obstructive manifestations. However, these points have to be further studied.

Acknowledgment Waltraut Reichelt and Margot Yehia assisted in preparing the manuscript.

References

1. Ammons WS (1992) Renal afferent inputs to ascending spinal pathways. Am J Physiol 262:165
2. Andreson JT, Bradley WE (1978) Urethral pressure profilometry: assessment of urethral function by combined intraurethral pressure and EMG recording. Urol Int 33:40
3. Booth FW, Thomason DB (1991) Molecular and cellular adaptation of muscle in response to exercise: perspectives of various models. Physiol Rev 71:541
4. Bude RO, DiPietro MA, Platt JF, Rubin JM (1994) Effect of furosemide and intravenous normal saline fluid load upon the renal resistive index in nonobstructed kidney in children. J Urol 151:438
5. Close R (1964) Dynamic properties of fast and slow skeletal muscles of the rat during development. J Physiol (Lond) 173:74
6. Comiter CV, Sullivan MP, Schacterle RS, Cohen LH, Yalla SV (1997) Urodynamic risk factors for renal dysfunction in men with obstructive and non-obstructive voiding dysfunction. J Urol 158:181
7. Frokiaer J, Nielsen AS, Knudsen L, Djurhuus JC, Petersen PE (1993) The effect of indomethasin infusion on renal hemodynamics and on the renin-angiotensin system during unilateral obstruction of the pig. J Urol 150:1557
8. Henriksson L, Anderson K-E, Ulmsten U (1979) The urethral pressure profiles in continence and stress-incontinent women. Scand J Urol Nephrol 13:5
9. Koff SA (1990) Pathophysiology of the ureteropelvic junction obstruction: clinical and experimental observations. Urol Clin North Am 17:263
10. Palmer JM, DiSandro M (1995) Diuretic enhanced duplex Doppler sonography in 33 children presenting with hydronephrosis: a study of test sensitivity, specificity and precision. J Urol 154:1885
11. Shafik A (1991) Straining-urethral reflex. Description of a reflex and its clinical significance. Preliminary report. Acta Anat 140:104
12. Shafik A (1998) Effect of renal pelvic distension on the ureteropelvic and ureterovesical junctions and the urinary bladder: the renal pelvivesical reflex. World J Urol 16:219
13. Tanagho EA (1986) Anatomy of the lower urinary track. In: Walsh PC, Gittes RF, Perlmutter AD, Stamey TA (eds) Campbell's urology. 5th edn. Saunders, Philadelphia, pp 46–74
14. Tanagho EA, Schmidt RA (1992) Neuropathic bladder disorders. In: Tanagho EA, McAninch JW (eds) Smith's general urology. 13th edn. Appleton and Lange, Norwalk, Conn., pp 454–472
15. Thuroff JW (1982) Mechanism of urinary constancy. Animal model to study urethral response to stress conditions. J Urol 127:1202
16. Windhorst U, Mommaerts WFHM (1996) Physiology of skeletal muscle. In: Greger R, Windhorst U (eds) Comprehensive human physiology. Springer, Berlin Heidelberg New York, pp 911–934