Urol Res (2007) 35:207–213
DOI 10.1007/s00240-007-0100-x

ORIGINAL PAPER

# Effect of smooth muscle relaxant drugs on proximal human ureteric activity in vivo: a pilot study

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Received: 14 January 2007 / Accepted: 26 April 2007 / Published online: 26 May 2007


**Abstract** Drugs are increasingly being used to promote stone passage in renal colic. Diclofenac, nifedipine and tamsulosin cause ureteric smooth muscle relaxation in vitro; however, in clinical trials nifedipine and tamsulosin promote stone passage whereas diclofenac has no apparent benefit. We adapted a ureteric pressure transducer catheter in an attempt to compare the human ureteric response to these drugs in vivo. The catheter was inserted into the contralateral ureter following ureteroscopy for stone disease. Contraction frequency, pressure and velocity measurements were recorded at 24 h. Each patient was randomly allocated to receive oral diclofenac, nifedipine or tamsulosin. Measurements were taken following drug administration. Eighteen patients (mean age 50 years) were recruited. Two patients were excluded intraoperatively and three required early removal of the catheter. Prior to drug administration, the mean number of contractions recorded was 0–4.1/min and the peak contraction pressure ranged from 11 to 35 mmHg. Conduction velocity ranged from 1.5 to 2.6 cm/s. Ureteric peristalsis persisted in all patients despite these drugs. Diclofenac and nifedipine produced inconsistent ureteric pressure responses but had little effect on contraction frequency. Tamsulosin significantly reduced ureteric pressure but had no effect on contraction frequency. There are many limitations associated with the use of ureteric catheters, however, they may provide some useful information when used to record the response to an intervention in the same patient. These preliminary results suggest a reduction in pressure generation may be the essential factor in the promotion of stone passage. More work is required but these drugs may work by preventing the increased, uncoordinated muscular activity seen in renal colic whilst maintaining peristalsis, thereby promoting stone passage.

**Keywords** Alpha antagonist · Calcium channel antagonist · Non-steroidal anti-inflammatory drugs · Peristalsis · Renal colic

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## Introduction

Clinical research suggests stone passage in renal colic may be promoted by ureteric relaxation using smooth muscle relaxant drugs such as nifedipine or tamsulosin [1, 2]. However diclofenac, a prostaglandin synthetase inhibitor, appears to reduce ureteric activity in vitro without promoting stone passage in clinical trials [3].

Designing a method to record human ureteric activity without causing interference is difficult. To date, various methods have been used combining ureteric catheters with strain gauges, strain gauge manometers or electromanometers [4–9], endoluminal ultrasound [10] or laparoscopic ultrasound [11]. A 4F silicone rubber ureteric catheter with two pressure transducers has been designed by Gaeltec Ltd, Dunvegan, Isle of Skye, UK and used to record the return of peristalsis post ureteroscopy [12]. Ureteric activity was shown to be highly variable within the first 24 h of ureteroscopy. Although effective at recording ureteric activity, there were problems with insertion due to its small size and absence of an internal lumen.

The aim of this study was to devise a method for monitoring human ureteric activity and the ureteric response to smooth muscle relaxant drugs without the need for anaesthesia during monitoring. Initial work aimed to determine the ease of insertion, tolerability and quality of recordings achieved using a modified version of the ureteric catheter designed by Young et al. [12]. The catheter was subsequently used in an attempt to compare the proximal ureteric response to diclofenac, nifedipine and tamsulosin. The aim was to provide further information regarding the use of these drugs in the promotion of stone passage.

## Methods

We increased the catheter diameter to 6F and incorporated a 4 cm distal hollow tip to allow insertion over a standard 0.035″ guidewire (Fig. 1). The data recorder connects directly to the catheter to record activity.

With Local Research Ethics committee approval, we approached patients admitted for unilateral ureteroscopy for stone disease for inclusion. Those patients on medications known to affect ureteric peristalsis in vitro were excluded ($\alpha$-adrenoceptor antagonists [13], $\beta$-adrenoceptor agonists [14, 15], calcium channel antagonists [16], nitrates [17, 18], angiotensin converting enzyme inhibitors [19], non-steroidal anti-inflammatory drugs [20–22] and cyclo-oxygenase II inhibitors [23, 24]). Patients with bilateral disease, previous contralateral disease or instrumentation of the contralateral ureter were also excluded.

Following informed consent, the ureteric catheter was inserted into the contralateral ureter over a guidewire and attached to a urethral catheter during the same anaesthetic. The radio-opaque pressure transducers allowed confirmation of position within the proximal ureter. All patients received antibiotic prophylaxis on induction (3 mg/kg intravenous gentamicin). Patients were allowed paracetamol or morphine based analgesia only in the postoperative period to prevent interference with results. None of the recordings were performed during the duration of action for morphine.

Post-operatively, the catheter was connected to the Gaeltec Nanologger™ recorder to record peristaltic activity. Measurements were made 24 h post insertion following recovery from general anaesthesia. Patients were asked to remain on their beds during recording. An initial recording of ureteric activity was made over 1 h and then patients were randomly allocated to receive one oral dose of diclofenac 50 mg, long acting (LA) nifedipine 10 mg or tamsulosin modified release (MR) 400 mcg. The time the drug was given was noted. The drug effect was presumed to occur at $T_{max}$ (time of maximal drug concentration) and therefore, data was obtained for 1 h following $T_{max}$ ($T_{max}$ values: diclofenac 60 min, nifedipine LA 80 min and tamsulosin MR 240 min). Due to the positioning of the ureteric catheter, only proximal ureteric activity was measured.

All patients were encouraged to maintain a fluid intake of 2.5–3 L/day whilst the catheter was in situ.

The ureteric and urethral catheters were removed on the ward by deflating the urethral catheter balloon and withdrawing both catheters simultaneously. The urethral catheter was discarded. The ureteric catheter was washed with soap and water and sterilised using Cidex OPA (0.55% ortho-phthalaldehyde) solution.

Eighteen patients were recruited into this study, ten females and eight males. The mean age was 50 (range 36–67) years. Two patients were excluded intraoperatively due to failed catheter insertion.

Since this was an initial pilot study, the sample size is small. The results do not have a normal distribution and therefore the data is presented in terms of median and interquartile range (IQR). Rank sum test is used to determine significance between pre and post drug values for contraction frequency and pressure generation.

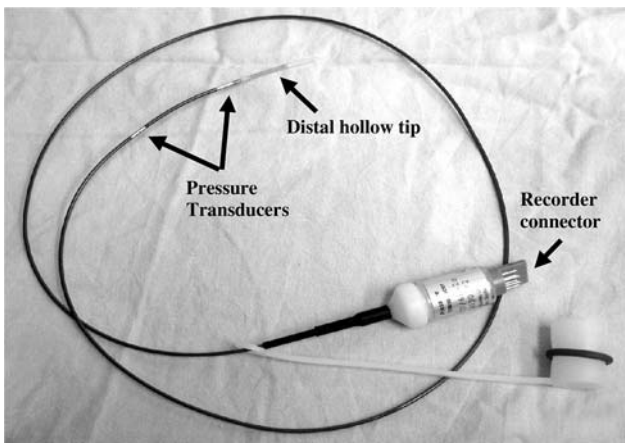


**Fig. 1** The 6F ureteric pressure transducer catheter used for these experiments

## Results

Sixteen patients (89%) had successful insertion of the ureteric pressure transducer catheter. Three of these sixteen patients (19%), two males and one female, required early removal of the catheter due to persistent discomfort at 5, 7 and 6 h post insertion (patients 3, 4 and 18). The discomfort resolved rapidly following catheter removal in the two male patients; however the discomfort persisted in patient 18 and was thought to be related to the ureteric stent in the contralateral ureter.

Pre drug data

Table 1 summarises the patient details, mean, median and IQRs for contraction frequency and pressure prior to administration of any drug.

Thirteen patients (81%) tolerated the catheter for the required 24 h. Unfortunately, one patient (patient 14) was given the test drug by a third party prior to obtaining a baseline recording of activity and therefore no pre drug data is available.

In the remaining 12 patients, the catheter provided sufficient tracings to allow calculation of contraction frequency and pressure. These values were obtained from 1-h recordings taken at 24 h post insertion. The mean number of contractions recorded ranged from 0 to 4.1/min and the mean peak pressure generated ranged from 11 to 35 mmHg.

Patient 17 had visible activity at 5 h post insertion, however by 24 h, only very occasional contractions were discernable.

Ureteric contractions have a typical appearance. Ten contractions were seen over 3 min in Fig. 2a, resulting in a mean contraction frequency of 3.3/min. Although in the first tracing, the contractions appear to be regular, ureteric activity was observed to vary in both frequency and pressure in all patients, as shown in the second tracing from the same patient 5 min later (Fig. 2b).

Although clear contractions were recorded in the majority, patients 5, 7 and 8 were seen to have erratic contrac-

**Table 1** Patient details, contraction frequency and pressure generation at 24 h post catheter insertion

| Patient no. | Age | Sex | Contraction frequency/min | | | Pressure generation/mmHg | | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | Median | IQR | Mean | Median | IQR |
| 1 | 47 | F | 1.53 | 1 | 1–2 | 14.4 | 15 | 11.5–17 |
| 2 | 55 | M | 0.76 | 0.5 | 0–1 | 17.4 | 13 | 9.3–23 |
| 5 | 63 | M | 0.58 | 0 | 0–1 | 10.5 | 9 | 6.5–13 |
| 7 | 64 | M | 0.4 | 0 | 0–1 | 12.7 | 11.5 | 9–16.3 |
| 8 | 58 | F | 0.4 | 0 | 0–1 | 12 | 10 | 9–14.5 |
| 9 | 44 | F | 4.1 | 4 | 3–5 | 21 | 21 | 19–23 |
| 10 | 57 | M | 1.58 | 1 | 0.8–3 | 34.7 | 35 | 32–39 |
| 11 | 48 | F | 0.77 | 1 | 0–1 | 14.2 | 13 | 10–17 |
| 13 | 60 | F | 2.58 | 3 | 2–3 | 25.1 | 25 | 22–28 |
| 15 | 54 | F | 0.18 | 0 | 0–0 | 15.6 | 15 | 13.5–18.5 |
| 16 | 53 | F | 1.03 | 2 | 2–2 | 27.4 | 28 | 23.3–32 |
| 17 | 48 | M | 0 | 0 | 0–0 | 23 | 23 | – |

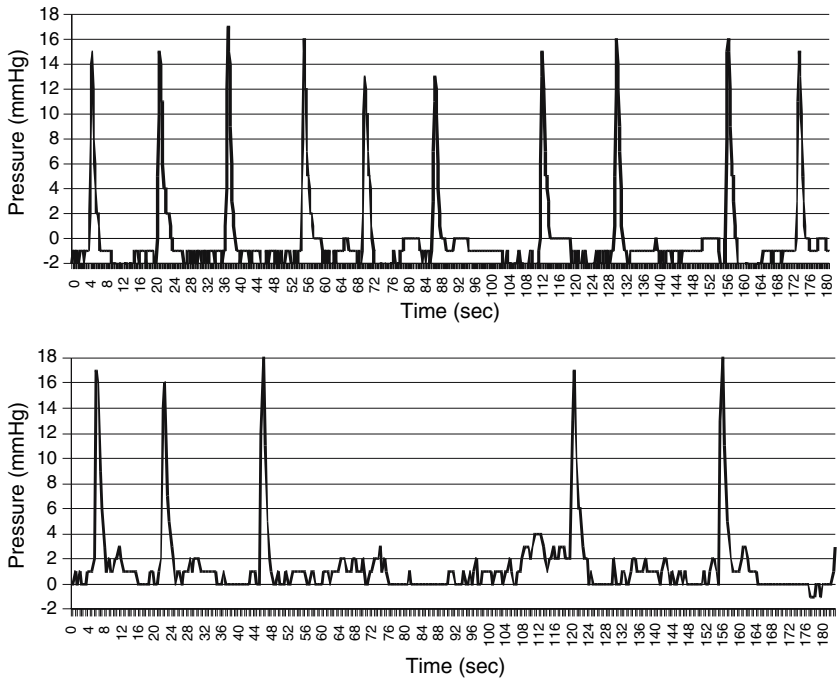


**Fig. 2** Two 3-min recordings obtained 24 h post-ureteroscopy from the same patient using the ureteric pressure transducer catheter to illustrate the variability in contraction frequency over time

tions, sometimes biphasic or triphasic in morphology (Fig. 3). These three also recorded the lowest contraction frequencies. It is likely that this disruption to peristalsis was due to the presence of the catheter.

The presence of two pressure transducers 5 cm apart allowed calculation of conduction velocity. Provided both transducers were sited within the ureter, conduction velocity could be calculated from the time the contraction peak is recorded by the first transducer (P1), to the time it is recorded by the second transducer (P2). In Fig. 4, peak 1 occurred at 0.9 s and peak 2 at 2.8 s. Therefore it took 1.9 s for the peristaltic wave to travel 5 cm, equivalent to a conduction velocity of 2.6 cm/s.

Clear contractions were recorded synchronously by both transducers in five patients. The results are summarised in Table 2. The median conduction velocity ranged from 1.5 to 2.6 cm/s. It is likely that proximal migration of the catheter prevented both transducers recording simultaneously in the other patients. However representative recordings were still obtained using the more distal transducer.

**Table 2** Conduction velocity data obtained using the ureteric pressure transducer catheter

| Patient no. | Conduction velocity (cm/s) | | |
|---|---|---|---|
| | Mean | Median | IQR |
| 11 | 2.68 | 2.6 | 2.6–2.8 |
| 13 | 2.18 | 2.1 | 2.1–2.3 |
| 15 | 2.48 | 2.5 | 2.4–2.5 |
| 16 | 1.5 | 1.5 | 1.4–1.5 |
| 17 | 1.85 | 1.85 | 1.8–1.9 |

Post drug data

*Diclofenac*

Three patients had complete data from before and 60 min following the administration of oral diclofenac 50 mg (see Table 3).

According to the results obtained for patient 1, diclofenac does not appear to have any effect on contraction frequency or pressure generation. However both patients 15 and 16 showed a significant increase in contraction frequency and pressure generation in the proximal ureter of these patients.

*Nifedipine*

Three patients had complete data from before and 80 min after the administration of oral nifedipine (Adalat) 10 mg.

Patient 5 showed a significant increase in contraction frequency, whereas patient 7 shows no difference and patient 10 demonstrated a significant reduction in contraction frequency (see Table 4).

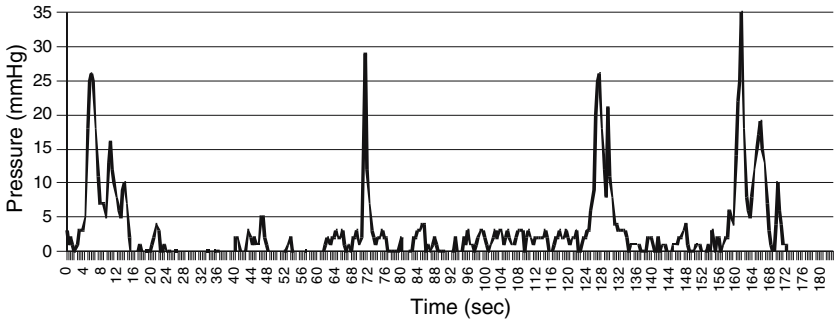


**Fig. 3** Three-minute recording demonstrating biphasic and triphasic waves thought to be related to the presence of the ureteric catheter

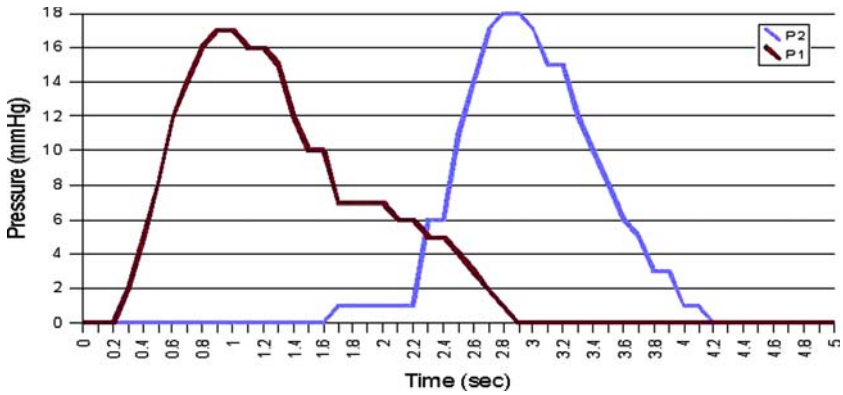


**Fig. 4** Calculation of conduction velocity using a single contraction recorded by both transducers

**Table 3** Contraction frequency and pressure data from patients before and after diclofenac

| Patient no. | Contraction frequency/min | | | | Pressure generation/mmHg | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | Median | IQR | *P* | Mean | Median | IQR | *P* |
| 1: Pre drug | 1.53 | 1 | 1–2 | 0.83 | 14.4 | 15 | 11.5–17 | 0.53 |
| Post drug | 1.48 | 1 | 1–2 | | 14 | 14 | 12–16 | |
| 15: Pre drug | 0.18 | 0 | 0–0 | 0.03 | 15.6 | 15 | 13.5–18.5 | 0.02 |
| Post drug | 0.49 | 0 | 0–1 | | 20.4 | 20 | 17.5–24 | |
| 16: Pre drug | 1.03 | 2 | 2–2 | 0.05 | 27.4 | 28 | 23.3–32 | <0.001 |
| Post drug | 1.58 | 2 | 1–2 | | 59 | 60 | 58–61 | |

**Table 4** Contraction frequency and pressure data from patients before and after nifedipine

| Patient no. | Contraction frequency/min | | | | Pressure generation/mmHg | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | Median | IQR | *P* | Mean | Median | IQR | *P* |
| 5: Pre drug | 0.58 | 0 | 0–1 | 0.04 | 10.5 | 9 | 6.5–13 | 0.08 |
| Post drug | 1.11 | 1 | 0–1.8 | | 9.1 | 8 | 5.5–10 | |
| 7: Pre drug | 0.4 | 0 | 0–1 | 0.52 | 12.7 | 11.5 | 9–16.3 | 0.92 |
| Post drug | 0.43 | 0 | 0–1 | | 13.3 | 12 | 9.3–14 | |
| 10: Pre drug | 1.58 | 1 | 0.8–3 | 0.01 | 34.7 | 35 | 32–39 | <0.001 |
| Post drug | 0.89 | 0 | 0–2 | | 19.4 | 22 | 11.5–25 | |

Patients 5 and 7 showed no change in pressure generation, however, patient 10 showed a significant reduction in pressure generation. Patients 5 and 7 have already been noted to have abnormal contraction patterns presumed to be related to the presence of the catheter and therefore their results may be less representative of their activity.

*Tamsulosin*

Two patients had complete data from before and 240 min following oral tamsulosin MR 400 mcg (see Table 5).

Patient 9 showed a significant reduction in contraction frequency ($P < 0.001$). Tamsulosin did not appear to affect the contraction frequency in patient 13 ($P = 0.95$). However, both patients showed a substantial reduction in pressure generation ($P < 0.001$ in both patients).

One patient developed symptoms resembling renal colic 90 minutes following the administration of tamsulosin (patient 2). This required removal of the catheter prior to the onset of drug action ($T_{max}$ 240 min) and so was considered to be related to the presence of the catheter not tamsulosin. The tracing obtained during this episode is shown in Fig. 5 As compared with the tracing in Fig. 2, the activity can be seen to be uncoordinated with abnormal contractions; both the contraction frequency and pressures are increased and the contractions no longer appear similar.

## Discussion

There are many published studies reporting human ureteric activity [4–7, 9–11]. There are far fewer studies reporting the ureteric response to drugs in vivo. None of those published to date report the in vivo response to diclofenac, nifedipine or tamsulosin.

Designing a method to record human ureteric activity with minimal interference to peristalsis is difficult. To date, various methods have been used combining ureteric catheters with strain gauges, strain gauge manometers or electromanometers [4–7, 9], endoluminal ultrasound [10] and

**Table 5** Contraction frequency and pressure data from patients before and after tamsulosin

| Patient no. | Contraction frequency/min | | | | Pressure generation/mmHg | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | Median | IQR | P | Mean | Median | IQR | *P* |
| 9: Pre drug | 4.1 | 4 | 3–5 | <0.001 | 21 | 21 | 19–23 | <0.001 |
| Post drug | 3.1 | 3 | 2–3.8 | | 11.9 | 12 | 9–14 | |
| 13: Pre drug | 2.58 | 3 | 2–3 | 0.95 | 25.1 | 25 | 22–28 | <0.001 |
| Post drug | 2.67 | 3 | 2–3 | | 16.1 | 16 | 14–18 | |

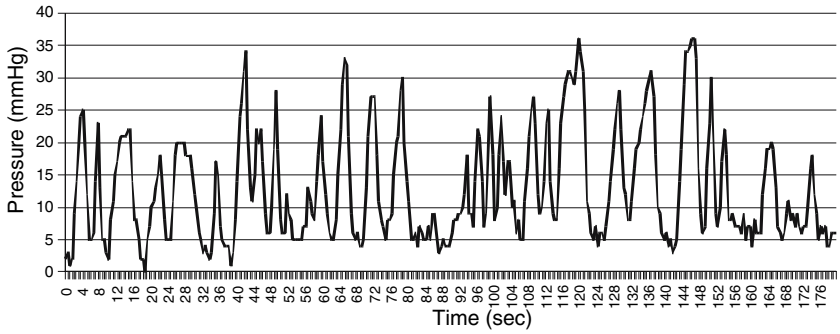


**Fig. 5** Three-minute tracing of ureteric activity during pain typical of acute renal colic

laparoscopic ultrasound [11]. The early ureteric catheters used fluid columns or external transducers with various complications. The more recent sophisticated methods have either required concurrent anaesthesia or fail to provide data on all the variables possible with this catheter. For the majority of patients involved, the catheter was well tolerated for over 24 h. This allowed excretion of the drugs used for anaesthesia prior to any measurements being recorded.

There are many problems associated with this study. Extraluminal methods for recording ureteric activity are generally preferred over intraluminal methods predominately due to the presumed effect the catheter presence can have on the ureter. 7F ureteric stents have been shown to reduce ureteric activity within 4 h of placement and completely ablate ureteric peristalsis by 1 week [25].

Three of the 13 patients (23%) were noted to have contraction abnormalities presumed to be due to the presence of the catheter. This has always been considered the main limitation with the use of intraluminal methods for monitoring ureteric activity. However, the contractions in these patients were clearly different, having biphasic and triphasic appearances. Conversely, the remaining ten patients (77%) had clear, reproducible contractions, which closely resembled those seen during episodes of spontaneous activity in human ureteric smooth muscle strips in vitro [13]. The contractions recorded have similar morphology to those reported previously using various other methods [6–9, 26]. It would therefore appear to be true that ureteric catheters may affect ureteric activity; however, this assumption does not appear to be true for the majority of patients and is certainly obvious when it does occur.

Extraluminal methods are inferior since no information regarding intraureteric pressures can be obtained. It is realised that intraluminal catheters most likely increase intraluminal pressures due to their presence, however, if the pressure values recorded are used to compare the effect of an intervention on the same patient, the changes in pressure, rather than the absolute values, may still provide important information.

Extraluminal methods are generally more invasive, requiring electrode placement or intra-abdominal ultrasound at laparotomy or laparoscopy. Although these frequency recordings are assumed to be more accurate, ureteric physiology is likely to be affected by operative dissection/exposure and the anaesthetic agents necessary. Butcher et al. [27] demonstrated that mobilisation of the canine ureter altered contraction amplitude and conduction velocity for as long as 20–30 min. Drugs used for general anaesthesia also have a relaxant effect on the ureter in vitro [28] and therefore these results may still be under representative.

Direct comparisons between intraluminal and extraluminal methods are difficult; however, studies suggest a slightly higher contraction frequency rate recorded by extraluminal methods [5–7, 9, 11, 29].

Despite limitations and complications, the ureteric catheter provided important information regarding the in vivo action of these three drugs. Contrary to results from animal studies, diclofenac does not cause complete ablation of human ureteric activity in vivo [21, 23]. In this study, ureteric contractions continued despite these drugs. Ureteric peristalsis has always been assumed to be essential for the spontaneous passage of stones and this work showed these drugs did not affect peristalsis.

Diclofenac appeared to increase both ureteric contraction frequency and pressure generation in the proximal ureter in vivo. Nifedipine had a variable effect; however, the most representative tracing (patient 10) showed a reduction in both contraction frequency and peak contraction pressure. Tamsulosin resulted in a significant reduction in the peak contraction pressure, with a variable effect on contraction frequency.

Finally problems with equipment failure and loss of data during transfer meant that the proportion of useable patient data was reduced. It must therefore be stressed that the ideal method for monitoring human ureteric activity remains unknown. However it would be worthwhile repeating these experiments using a 4F catheter of the same design to determine whether tolerability and recording quality improves. It would also be worthwhile using a shorter version to monitor distal ureteric activity since tamsulosin has its predominant effect on the distal ureter [13].

This study has demonstrated that peristalsis persists despite the use of these drugs. Smooth muscle relaxant drugs may reduce the ability to increase pressure during a contraction, which would have the advantage of reducing the ability to develop muscle spasm and the increased uncoordinated activity seen in renal colic. Greater numbers are required to determine the effect on contraction frequency. However further experiments would be preferred using the same catheter design but with a 4F catheter diameter to limit interference.

## Conclusion

This ureteric pressure transducer catheter remains less than ideal for monitoring ureteric activity; however, from these preliminary results diclofenac may increase pressure generation. Nifedipine had conflicting effects on contraction frequency but may decrease pressure generation. Tamsulosin significantly reduced pressure generation whilst maintaining contraction frequency in the proximal ureter. Most importantly, all three drugs allowed peristalsis to continue. The reduction in pressure generation seen with tamsulosin may be the essential factor in the promotion of stone passage.

## References


1. Porpiglia F, Ghignone G, Fiori C, Fontana D, Scarpa RM (2004) Nifedipine versus tamsulosin for the management of lower ureteral calculi. J Urol 172(2) 568–571
2. Dellabella M, Milanese G, Muzzonigro G (2005) Randomised trial of the efficacy of tamsulosin, nifedipine and phloroglucinol in medical expulsive therapy for distal ureteral calculi. J Urol 174(1):167–172
3. Laerum E, Omundsen OE, Gronseth JE, Christianson A, Fagertun HE (1995) Oral diclofenac in the prophylactic treatment of recurrent renal colic: A double blind comparison with placebo. Eur Urol 28:108–111
4. Hanley HG (1953) The electro-ureterogram. Br J Urol 25(4):358–365
5. Corey EL, Fite EH Jr, Vest SA (1956) Electropotential changes in the normal human ureter. J Urol 75(2):244–249
6. Weinberg SR, Maletta TJ, Brooklyn NY (1961) Measurement of peristalsis of the ureter and its relation to drugs. JAMA 175(1):15–18
7. Ross JA, Edmond P, Coull J, Simpson D (1965) The 'dynamic function' of the intact human renal pelvis and ureter. Br J Surg 52(8):617–621
8. Ross JA, Edmond P, Coull J, Griffiths J (1967) Observations on the physiology of the human renal pelvis and ureter. J Urol 97:449–454
9. Edmond P, Ross JA, Kirkland IS (1970) Human ureteral peristalsis. J Urol 104:670–674
10. Roshani H, Dabhoiwala NF, Dijkhuis T, Kurth KH, Lamers WH (2000) An in vivo endoluminal ultrasonographic study of peristaltic activity in the distal porcine ureter. J Urol 163:602–606
11. Helin HL, Kirkinen P, Purhonen S, Penttinen J (1998) Ultrasonography of the ureter during laparoscopic, abdominal and radical hysterectomy. Ultrasound Obstet Gynaecol 12(3):207–210
12. Young AJ, Archer PL, Lynn B, McCahy PJ, Miller RA (2007) Evaluation of novel technique for studying ureteral function in vivo. J Endourol 21(1):94–99
13. Davenport K, Timoney AG, Keeley FX (2006) A comparative in vitro study to determine the beneficial effect of calcium-channel and $a_1$-adrenoceptor antagonism on human ureteric activity. BJU Int 98(3):651–655
14. Morita T, Wada I, Saeki H, Tsuchida S, Weiss RM (1987) Ureteral urine transport: changes in bolus volume, peristaltic frequency, intraluminal pressure and volume of flow resulting from autonomic drugs. J Urol 137(1):132–135
15. Danuser H, Weiss R, Abel D, Walter B, Scholtysik G, Mettler D, Studer UE (2001) Systemic and topical drug administration in the pig ureter: effect of phosphodiesterase inhibitors, $\alpha_1$, $\beta$ and $\beta_2$ adrenergic receptor agonists and antagonists on the frequency and amplitude of ureteral contractions. J Urol 166(2):714–720
16. Hertle L, Nawrath H (1984) Calcium channel blockade in smooth muscle of the human upper urinary tract. 1. Effects on depolarisation-induced activation. J Urol 132:1265–1269
17. Kekec Z, Yilmaz U, Sozuer E (2000) The effectiveness of tenoxicam versus isosorbide dinitrate plus tenoxicam in the treatment of acute renal colic. BJU Int 85:783–785
18. Mastrangelo D, Baertschi AJ, Roatti A, Amherdt M, Iselin CE (2003) Nitric oxide production within rat urothelial cells. J Urol 170(4):1409–1414
19. Santis WF, Peters CA, Yalla SV, Sullivan MP (2003) Ureteral function is modulated by a local renin angiotensin system. J Urol 170(1):259–263
20. Cole RS, Fry CH, Shuttleworth KED (1988) The action of the prostaglandins on isolated human ureteric smooth muscle. Br J Urol 61:19–26
21. Lennon GM, Bourke J, Ryan PC, Fitzpatrick JM (1993) Pharmacological options for the treatment of acute ureteric colic. Br J Urol 71:401–407
22. Sivrikaya A, Celik OF, Sivrikaya N, Ozgur GK (2003) The effect of diclofenac sodium and papaverine on isolated human ureteric smooth muscle. Int Urol Nephrol 35:479–483
23. Nakada SY, Jerde TJ, Bjorling DE, Saban R (2000) Selective cyclooxygenase-2 inhibitors reduce ureteral contraction in vitro: a better alternative for renal colic?. J Urol 163:607–612
24. Jerde TJ, Calaman-Dixon JL, Bjorling DE, Nakada SY (2005) Celecoxib inhibits ureteral contractility and prostanoid release. Urol 65:185–190
25. Venkatesh R, Landman J, Minor SD, Lee DI, Rehman J, Vanlangendonck R, Ragab M, Morrissey K, Sundaram CP, Clayman RV (2005) Impact of a double-pigtail stent on ureteral peristalsis in the porcine model: Initial studies using a novel implantable magnetic sensor. J Endourol 19(2):170–176
26. Davis DM, Zimskind PD, Paquet J-P (1963) Studies on urodynamics: new light on ureteral function. J Urol 90:150–159
27. Butcher H, Sleator W, Schmandt W (1957) A study of the peristaltic conduction mechanism in the canine ureter. J Urol 78:221–231
28. Young CJ, Attele A, Toledano A, Nunez R, Moss J (1994) Volatile anaesthetics decrease peristalsis in the guinea pig ureter. Anaesthesiology 81:452–458
29. Stower MJ, Clarke AG, Wright JW, Hardcastle JD (1984) Effect of glucagons on ureteric peristalsis in man, pig, rabbit and rat. J Urol 131:822–824