Dynamics and modulation of ureteric peristalsis
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CHAPTER 4

AN ELECTRO-MYOGRAPHIC STUDY OF THE DISTAL PORCINE URETER

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ABSTRACT

Purpose: The accumulation of urine in the renal pelvis causes depolarisation of non-specific muscular pacemaker cells. The wave of depolarisation spreads distally in the ureteric smooth muscle cells via gap junctions. This wave of excitation causes a co-ordinated peristaltic contraction, which transports the urine bolus distally to the bladder. The EMG activity in the distal porcine ureter was studied and analysed to establish the characteristics of ureteric excitation.

Material and methods: Ten female New Yorkshire pigs (50-60 kg) were studied in two groups under light halothane anaesthesia (5% at induction and 1% for maintenance anaesthesia). In both groups, each pig is studied in two separate sessions at a week’s interval. In group I (n=5), bipolar needle electrodes (ø: 0.09mm) were implanted through a lower mid-line abdominal incision in the posterior bladder wall, the trigone and in the pelvic ureter at intervals of 3 and 8 cm, respectively, from the ureteric orifice. In group II (n=5), EMG spike burst activity was studied using a twin bipolar ring-electrode attached to an endoluminal ureteric catheter. EMG complexes were recorded using 0-30 Hz filters. The duration of spike burst complexes and their intervals were analysed using a Nicollet, Pathfinder II® machine and a Poly® 4.9 digital signal processing program.

Results: Two types of spike burst activity could be distinguished between the electrodes: A: the migrating type and B: the non-migrating type. Frequency distribution analysis of spike burst duration revealed two main classes in experimental group II. A short spike burst (96%) which lasted 4.5 ± 1.8 sec and a longer one lasting 13.4 ± 1.5 sec. The conduction velocity of the migrating spike bursts (n=177, 42% of total) between the proximal and the distal electrode had an average of 2.3 ± 1.3 cm/sec. No relationship was found between the duration of the proximal spike burst and the conduction velocity. Data from experimental group I correlate well with that from group II.

Conclusions: The results of our EMG study in the distal ureter reveal an approximately 9 cm long electrically active zone in ≥ 90% of EMG activity recordings. The duration of activity lasts for approximately 5 sec. Such an excited segment of ureter leads to a contraction, which occludes the ureter and can prevent retrograde leakage of intraluminal contents.
INTRODUCTION
Urine produced in the kidney accumulates temporarily in the renal pelvis before being transported through the ureter into the bladder by periodic peristaltic activity. The ureter is a muscular tube between the low-pressure renal pelvis and the bladder cavity where pressure varies widely depending on the micturition cycle. Transport of the urinary bolus is essentially unidirectional. Reflux of urine from the bladder to the upper tract (ureter, pyelum or kidney) is by definition pathological and can damage renal function especially in a developing kidney in the presence of bacteriuria or urinary tract infection. Vesicoureteral reflux is an important cause of terminal renal failure and associated renal hypertension in adults.

The ureterovesical junction (UVJ) is the most distal portion of the ureter, which traverses the posterior bladder wall to reach the ureteric orifice in the bladder lumen. The most important function of the UVJ is considered the prevention of vesico-ureteric reflux. The diagonal passage of ureter through the bladder wall and the length of its submucosal portion are the two main factors responsible for the “passive” valvular function of the UVJ.

An “active” valvular function at the UVJ level has also been reported by several authors including Gil-Vernet and Tanagho. The sophisticated urodynamic study reported by Blok and co-workers also lends credence to the presence of an active occluding mechanism. On the basis of their ureterovesical pressure profile (UVPP) study, they postulated that the base line of UVPPs represents the resistance to flow through the UVJ.

We have first studied the functional anatomy of the porcine and later the human UVJ using enzyme-histochemical techniques to visualise Acetylcholinesterase (AChE) and Butyryl cholinesterase (BChE, non-specific cholinesterase) activity. Based on the above evidence we postulated that active contraction of the distal ureteric circular muscle at the juxtavesical level during the passage of a peristaltic wave transporting an urine bolus functions as the “active” valvular mechanism to prevent reflux while the ureteric orifice is opened to deposit a urinary bolus into the bladder lumen by an active shortening of the longitudinal muscle fibers over the urinary bolus lying within the transmural and submucosal segments of the terminal ureter. Distal spread of ureteric “peristalsis” into the superficial trigone again restores the submucosal ureteric length and re-establishes the passive anti-reflux mechanism (Fig. 1). To test the validity of this dynamic hypothesis based on our morphological studies, we undertook ureteric peristalsis studies in the pig model.
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Figure 1: The sequence of excitation, contraction and pressure rise within the ureter. Asterisk represents a pace maker cell in the muscular wall of the renal pelvis.

This article discusses the characteristics of ureteric excitation in the distal porcine ureter and the effect of intra-ureteral instrumentation on these characteristics. A study of ureteric contractions and the resulting pressure change in the ureter is the subject of a separate publication.

MATERIALS AND METHODS

Experimental animals and the anaesthetic procedure. Ten New Yorkshire female pigs were studied in two groups. EMG of the ureteric muscle in group I (n=5, 55±3 kg) was obtained using a lower abdominal laparotomy to place needle electrodes in the ureteric wall under vision (“open” procedure). In group II (n=5, 57±7 kg) a minimally invasive endoluminal approach using a self-made EMG catheter was used. During anaesthesia, optimal muscular activity for EMG measurements was maintained. As pre-medication 20 mg Azaperon plus 150 mg Ketamine per 10 kg body weight and 1 mg Atropine were administered intravenously. General anaesthesia was induced using 5% halothane and maintained on 1% halothane and a mixture of 50% N₂O and 50% O₂. One mg sulfontanil was administered intravenously prior to the abdominal incision in the group I series at least an hour prior to commencement of registration of EMG recordings. Peri-operative antibiotic cover was provided by 1200 mg Augmentine intravenously while hydration was maintained in group I by infusion of physiologic saline solution at the rate of 0.48 ± 0.10 L/hour and in group II 0.35 ± 0.08 L/hour. This rate of infusion was chosen because the animal was fasting for more than 12 hours preoperatively and general anaesthesia produces a drop in blood pressure ⁶. To deal with these problems and in order to maintain an optimal stable circulation these infusion rates were needed. Urine produced was macroscopically undiluted. Continuous monitoring of blood pressure,
electrocardiographic and pulmonographic parameters was maintained during the whole procedure to ensure optimal metabolic functioning. Each animal was studied in two sessions at an interval of one week to study the effect of instrumentation on the ureter. Analgesics and antibiotic drugs were administered to the animal postoperatively as required to reduce the discomfort from instrumentation and as chemoprophylaxis.

**Electromyographic recordings device and EMG electrodes used.** *Group I:* Bipolar copper needle electrodes ($\Omega = 0.09$ mm, L: ca 5 mm, Teflon insulation by Drijfhoud company in the Netherlands) were inserted in the ureteric muscle under direct vision. Electrodes were placed at distances of approximately 3 and 8 cm from the ureteric orifice and into the posterior aspect of the bladder wall as well as the ipsilateral trigone after opening the bladder through a small anterior vesicotomy. The indifferent electrode was connected to an abdominal forceps. The impedance of the electrodes and tissue was ~12 KΩ and was checked before the recordings were undertaken. The commercially available EEG device Pathfinder II Nicollet® was used to measure the potentials. The band pass was maintained between 1 to 30 Hz to minimise the environmental artefacts. The recordings were stored on the data disk and plotted on paper. The software of the Nicollet® was able to measure the duration of each EMG complex in each channel and also the interval between the sequence complexes in different channels. The amplitude of the complexes was set to fit the graphic facilities for recording. The data obtained from the recordings (duration of each EMG complex in each channel and the time interval between two progressing complexes from the proximal and distal channels) was stored and analysed using the Microsoft Exel 7.0 spreadsheet program running on an 586 IBM compatible machine. Any deviation from the base line visible simultaneously in different channels was considered an artefact and excluded from further analysis. No correlation was found between respiration, heart rate and the occurrence of EMG spikes.

*Group II:* EMG complexes were obtained endoluminally using two bipolar gold (99.99% pure) ring electrodes ($\Omega = 0.15$ mm, L: ca 6 mm, polycarbonate insulation: Tremel-coated by Drijfhoud company in the Netherlands) attached to a 6F ureteric catheter at an interval of 10 cm from each other. The EMG catheter was introduced through the working channel of a 22F rigid cystoscope. Ureteric electrical activity was recorded at 3 and 13 cm levels from the ureteric orifice. The indifferent electrode was connected to the cystoscope. The second working channel of the cystoscope
was used to ensure that the bladder remained empty throughout the registrations. The Pathfinder II Nicollet® was used as described in group I. The impedance measured was ~4 KΩ which is much less than in the group I. DAS-8-PGA, an analogue / digital interface with 12-bit resolution was used to digitise the EMG signal. A sampling frequency of 100Hz was established according to the Nyquist principle. Digital signal processing was undertaken using the Poly 4.9 digital signal-processing program running on a 586-100 MHz IBM compatible machine. Fast Fourier transformation analysis of the frequency power spectrum was carried out. To exclude the possibility of signals being recorded and caused by movement artefacts uncoupling of excitation- contraction cycle using a Ca\(^{2+}\) antagonist agent (Nifidepine) was also undertaken. We resorted to a rapid intra-aortic bolus infusion of 60 mg of Nifidepine and recorded continuously endoluminal ultrasonographic and mechanical ureteric activity in the contra-lateral ureter, which revealed a complete absence of any mechanical activity. The logistics of our current experimental set-up did not permit us to undertake any intracellular measurements to establish beyond any shadow of doubt an uncoupling effect.
RESULTS

Description of spike bursts in the ureter, trigone and detrusor using the open and endoluminal approaches. The form of ureteric EMG spike burst activity did not differ in either the open or the endoluminal measurements or between the recordings in the virgin or post-instrumented ureter. Two types of EMG complexes could be observed. The first type migrated from one electrode to the other (group I: 110/202; 54%, group II: 177/421; 42%), while the other type was stationary and recording was observed in only one electrode. The direction of the migrating spikes was mainly from the kidney towards the bladder, but retrograde migration of spikes was also observed occasionally. The duration of the ureteric EMG spike bursts also differed. Some were of a short duration (short spike bursts) while others.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Progressing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short spike</td>
<td></td>
</tr>
<tr>
<td>bursts</td>
<td>Migrating short spike bursts</td>
</tr>
<tr>
<td>Long spike</td>
<td>Migrating long spike bursts</td>
</tr>
<tr>
<td>bursts</td>
<td>Non-migrating short spike</td>
</tr>
<tr>
<td></td>
<td>bursts</td>
</tr>
<tr>
<td></td>
<td>Non-migrating long spike</td>
</tr>
<tr>
<td></td>
<td>bursts</td>
</tr>
</tbody>
</table>

Table 1: Description of different kinds of ureteric EMG spike bursts

The uncoupling of excitation and contraction realised by the intra-aortic administration of the 60 mg Nifidipine revealed the preservation of EMG complexes in the absence of ureteric peristalsis as revealed by endoureteric ultra-sonographic monitoring in the contra-lateral ureter simultaneously. The positive correlation between ureteric EMG activity and peristalsis could also be studied and established by visual control in the first five pigs (group I) by two independent observers.

The trigonal EMG was technically difficult to record, but 70 spike bursts in the trigone were recorded. The study of these revealed similarities of form and of migration pattern to that of the ureteric EMG. Recordings of the detrusor EMG revealed periodic activity that was totally independent of ureteric activity.
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Panel 2a: Simultaneous identical recording in two different channels represents an artifact. The second signal in A1 is a real non-migrating ureteric signal in the proximal recording electrode.

Panel 2b: No relationship is found between the ureteric EMG (A1 and A2) and the bladder and anus pressure. P bladder (P Bl.) and P anus (P An.) are synchronous with respiration. Notice the different patterns of duration and progression of the EMG spike burst activity.

Panel 2c: A1 (proximal ureteric), A2 (distal ureteric) and A3 (trigonal) recordings are quiet while A4 (detrusor) recording shows clearly periodic activity. Notice that these recordings are of a relatively short duration.

Panel 2d: recorded some minutes after 2c: The migrating ureteric EMG signal (A1-A2) has a 2.2 sec latency, appears later in the trigone (A3), and has a longer conduction latency (7.3 sec). The detrusor EMG activity (A4) has in the meanwhile increased. It remains, however, independent of the ureteric recordings.

Panel 2e: Magnification of recording of panel 2c; Notice the stable base line in A4. One can observe that the ureteric EMG signals in A1 and A2 are totally independent to the detrusor activity in A4. See also panels 2c, d, f.

Panel 2f: The migration of the EMG signal from A1 to A2 and subsequently to A3 is clearly recorded here. Note that the conduction velocity in the intramural ureter is decreased and the detrusor signal is quiet.

Panel 2g: Endoluminal EMG recording of the porcine ureter; Note the different duration patterns.

Figure 2: Examples of different kind of spike bursts as recorded in current study.
Quantitative analysis of the EMG complexes in the open approach (group I). During experiments with the first five pigs, we developed skills to continue the study (in group II) by minimally invasive endoluminal approach. Comparison of results between group I and II revealed the effect of open surgery on EMG characteristics.

The ureter: A total of 202 spike bursts from both proximal and distal electrodes were recorded. The frequency-distribution analysis of spike burst duration revealed good correlation between data obtained from the proximal and the distal electrodes (Table 2 and Fig.3). Migration of spike burst activity between the two electrodes was observed in 110 out of 202 (54%) studied cases. The conduction velocity of the migrating spike bursts between the proximal and the distal electrode (5 cm distance) was calculated to be 2.1 ± 0.9 cm/sec. A length of ~7 cm ureter was thus electrically active during 90% of ureteric excitations.

<table>
<thead>
<tr>
<th>Proximal spike bursts (n= 154)</th>
<th>Distal spike bursts (n= 123)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spike range (sec)</td>
<td>Number (% of total)</td>
</tr>
<tr>
<td>1-7</td>
<td>136 (88%)</td>
</tr>
<tr>
<td>7-11</td>
<td>13 (9%)</td>
</tr>
<tr>
<td>11-14</td>
<td>5 (3%)</td>
</tr>
</tbody>
</table>

Table 2: Three classes of the spike burst duration were observed by the open approach at laparotomy. The majority (90% of both recordings) of ureteric activity ranges between 1 and 7 sec with an average of 3.3 ± 1.1 sec.

The trigone: Needle electrodes were inserted in the submucosa of the trigone to obtain the trigonal EMG. Haematoma formation due to the introduction of the needle electrodes was the major problem. We were, however, able to record 70 spike bursts at a point one cm distal to the ureteric orifice. Analysis of data revealed close similarity to the ureteric results (Tables 2, 3 and Fig. 3). Migration of distal spike burst to the superficial trigone was recorded in 34/70 (49%) of the recordings. The conduction velocity of these signals is 1.2 ± 0.4 cm/sec, or 57% of the conduction velocity in pelvic ureter (Fig. 4).
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Frequency distribution of the proximal, distal and superficial trigonal spike burst duration (open approach)

Figure 3: Distal EMG signal is shifted slightly to the left (open approach). A similarity of 80% (Student’s t test: 0.8) is found between the range 1-7 second in proximal and distal group. The trigonal EMG is similar to the distal EMG signal.

The detrusor: Simultaneous recording of detrusor EMG activity was performed to study ureteric functional autonomy, which we assumed must be present on the basis of the hypothesis postulated by us based from our morphological findings. Most detrusor EMG signals lasted more than 100 sec which was above the maximal measurement capacity of our Nicollet® instrument. The duration of other (short) signals from the detrusor that we could study was much longer (8.2 ± 5.3 sec) than the ureteric one (3.3 ± 1.1 sec) and no migration pattern could be found.

<table>
<thead>
<tr>
<th>Distal spike bursts (n=123)</th>
<th>Trigonal spike bursts (n=70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spike range (sec)</td>
<td>Number (% of total)</td>
</tr>
<tr>
<td>1-7</td>
<td>113 (92%)</td>
</tr>
<tr>
<td>7-9</td>
<td>6 (5%)</td>
</tr>
<tr>
<td>9-12</td>
<td>4 (3%)</td>
</tr>
</tbody>
</table>

Table 3: Frequency distribution of spike burst data shows a tri-phase in the distal and in superficial trigone (open approach)
Conduction velocity (open approach) of the EMG signal in pelvic ureter and UVJ

**Figure 4:** Conduction velocity of the EMG signal (open approach) decreases by 43% when passing through the intramural ureteric segment. Error bars are standard deviations. P value by student t-test: 0.000001

*Quantitative analysis of the EMG complexes using endoluminal (minimally invasive) approach (group II).* The advantage of this technique is that it is easier to perform, that it is minimally invasive and that the data are less affected by artefact from open operative manipulation.

A total number of 421 spike bursts in the proximal and distal recordings were studied. The frequency distribution of the spike burst duration was similar to that in group I and revealed that spike burst activity was distributed in two main classes (Table 4, Fig. 5). Migration of the spike bursts between the two electrodes was observed in 177 (42%) of the 421 recordings. The conduction velocity of the migrating spike bursts between the proximal and the distal electrode (10 cm distance) was averaged to be 2.3 ± 1.3 cm/sec. No relationship was found between the duration of the proximal spike bursts and the conduction velocity (Fig. 6). The average of the spike burst duration was 4.8 ± 1.8 sec in the measurements before manipulation and 4.0 ± 1.7 sec at one week post intraureteric instrumentation, which is not statistically different (P_{student t-test} = 0.45) (Fig.7). The measurement of the migration time of the ureteric spike burst in the ureter before and after manipulation revealed that the conduction velocity increased
from 2.0 ±1.6 cm/sec (n= 88) to 2.5 ±1.9 cm/sec (n=26) before and after the manipulation respectively (P_{\text{student t-test}}: 0.01, Fig. 8).

<table>
<thead>
<tr>
<th>Proximal spike bursts</th>
<th>Distal spike bursts</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n= 334)</td>
<td>(n= 263)</td>
</tr>
<tr>
<td>Spike range (sec)</td>
<td>Spike range (sec)</td>
</tr>
<tr>
<td>Number (% of total)</td>
<td>Number (% of total)</td>
</tr>
<tr>
<td>Duration ± SD (sec)</td>
<td>Duration ± SD (sec)</td>
</tr>
<tr>
<td>1-11</td>
<td>1-12</td>
</tr>
<tr>
<td>322 (96%)</td>
<td>260 (99%)</td>
</tr>
<tr>
<td>4.5± 1.8</td>
<td>4.7± 2</td>
</tr>
<tr>
<td>11-16</td>
<td>12-16</td>
</tr>
<tr>
<td>12 (4%)</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>13.4± 1.5</td>
<td>13.2± 1</td>
</tr>
</tbody>
</table>

**Table 4:** Two classes of the spike burst duration as observed by endoluminal approach. The majority (96%) of ureteric activity ranges between 1 and 11 sec with an average of 4.6± 1.9 sec.

![Frequency distribution of proximal and distal spike bursts duration](image)

**Figure 5:** Spike burst are distributed in two main classes (endoluminal approach). Proximal and distal recording show same distribution pattern.

**Fast Fourier transformation (FFT) analysis to study the power spectrum.** The validity of the band pass (1-30 Hz) used by us was further established by power spectrum analysis using fast Fourier transformation of initial wide input band pass (0.1-1000 Hz). It revealed that the peak EMG activity of the ureter is at 20 Hz. No other higher or lower frequencies were observed.
Signal conduction velocity as the function of proximal signal duration

![Graph showing signal conduction velocity as a function of proximal signal duration. The graph displays two lines: one for real conduction velocity and another for average conduction velocity.]}

**Figure 6:** Conduction velocity of ureteric EMG signal decreases slightly (non-significant) when the (proximal) signal becomes longer.

**DISCUSSION AND CONCLUSION**

The transport of a urine bolus through the ureter results from a sequence of electrical excitation of the muscular components, followed by their contraction (a constriction where circular muscle bundles are present) and the segmental rise of ureteric wall tension in order to generate a peristaltic wave movement (Fig 1). Djurhuus and co-workers found evidence that the renal pelvis control ureteric activity. The frequency of the pelvic EMG activity increases with intra-pelvic pressure. They assume that pelvic function follows a stretch-response curve. According to “Multiple coupled oscillators” model as explained by Golenhofen (1973) and Constantinou (1981), multiple renal pelvic pacemaker cells are able to oscillate independently. The propagation of these signals to the ureter depend to 1) the number and the power of these oscillations and 2) whether these oscillations are synchronised. Each action potential is characterised by a rapid depolarisation followed by a plateau and a slow repolarisation phase. Spikes were observed at the plateau phase of some spieces. The propagation of the signal through the ureter is purely myogenic depended to the gap junctions between the smooth muscle cells. The rise in wall tension and, thus, the bolus transport, depends on two elements: a, the contractile components which ensure the power that is needed for the rise in tension (the motor
role) and b, the compliance of the tube (the internal freedom or resistance
to change of form) which is mainly represented by the quality and quantity
of the connective tissue\textsuperscript{12-13}. The electrically active zone displaces itself
distally via gap junctions to eventually reach the pelvic ureter and the UVJ.

The length (velocity of signal conduction differentiated in time) and the
duration of this active zone in the distal ureter are the parameters which
determine the length of a \textit{"contracted"} ureteric segment during ureteric
peristalsis and thus guarantee unidirectional fluid transport. A rapid
conduction velocity is associated with a longer contracted segment, but a
slower conduction velocity is associated with an increased number of
activated smooth muscle cells, so that the contraction force is stronger. This
inverse relationship between the EMG signal propagation velocity and force
has been elegantly demonstrated in an in vitro study\textsuperscript{14}.

\textit{Study of ureteric excitation supports its underlying morphological basis.}
From the endoluminal measurements undertaken in an earlier non-
catheterised “virgin” ureter a length of $\sim 10$ cm is calculated to be excited.
The average excitation duration is calculated to be 4.5 sec. These twin
characteristics play an important role in ureteric contraction, its
unidirectionality as well as the discharge of urine bolus from the most distal
portion of the ureter into the bladder cavity. The ureter contraction occurs
0.2 sec after its excitation\textsuperscript{14}.

\textit{The effect of intraureteric instrumentation.} To visualize the effect of
intraureteric instrumentation we compared the distribution of spike burst
duration before and after one week of intraureteric manipulation
(catheterisation). As illustrated in fig. 6, some spike bursts in the range of 2
to 9 sec were suppressed at one week. The conduction velocity increased
from $2.0 \pm 1.6$ cm/sec (n=88) to $2.5 \pm 1.9$ cm/sec (n=26) before and after the
manipulation respectively ($P_{\text{Student \ t-test}}: 0.01$, Fig. 7). This significant increase
of the propagation velocity of the EMG signal may be due to a
hypopolarised state of the cellular membrane as a result of local
inflammation after manipulation. Intracellular potential measurements could
not be undertaken given the limits of our in vivo experimental set-up. The
rise in the conduction velocity apparently compensates for the decrease in
the average of spike burst duration after manipulation to establish a certain
length ($\sim 10$ cm) of excited ureter.
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Figure 7: The effect of intraureteral instrumentation on the frequency distribution of ureteric spike bursts. Some spike bursts in the range from 2 to 9 sec in duration reveals a shift to the left.

From our morphological studies, it appears that the intramural segment of the ureter can contract and open the ureteric orifice during the passage of a urine bolus. A length of ±3 cm (maximum length of the UVJ) is excited and retracts in 2.5 sec (3 cm/ 1.2 cm/sec), which is within the latent period of excitation of the most distal portion of the extravesical ureter.

It is known that the contraction duration of smooth muscle is relatively long. The periods of excitation recorded by us would, under normal physiological conditions, probably be slightly longer, since general anaesthesia is known to decrease the ureteric peristalsis activity.
Conduction velocity of EMG (cm/sec)

![Graph showing conduction velocity before and after intraureteric manipulation]

**Figure 8:** Conduction velocity of the EMG signal increases after intraureteric manipulation (catheterisation) by a factor of 1.25. Error bars are standard deviations. P value by student t-test: 0.003

**Smooth muscle EMG and its urological application.** Data concerning the electrical properties of smooth muscle EMG in urology is limited to in vitro study of muscle strips and a limited number of in vivo publications on the corpus cavernosum EMG in order to study and find therapeutic clues to erectile dysfunction. Data acquisition is a major problem in studying smooth muscle EMG generally and in studying the upper urinary tract especially. The positioning of reliable minimally invasive electrodes within the upper urinary tract in standard locations has until now not been possible. This technical problem, the fact that the available hardware is not designed for smooth muscle EMG studies, and the absence of accepted standard values are responsible for controversies surrounding the results obtained by different groups of investigators. The introduction of fast Fourier transformation analysis of smooth muscle electrical activity by Merckx and co-workers was a major step forward in solving these problems.

Shafik studied EMG activity and intra-luminal pressure rise in the human ureter in patients who underwent rectopexy due to rectal prolapse. He was able to distinguish separate pacesetter- and action potentials in different segments of the ureter. Mechanical activity is accompanied by ureteric action potentials. The duration and conduction velocity of the action
potentials was not reported by him but he did report that pacesetter potentials were conducted at 5.9 ± 1.2 cm/sec in his study. This is slightly faster than the results in the pig model. There may be an underlying species or operation-related factor responsible for this difference.

Simultaneous recording of EMG, ureteric wall movement and or intraureteric pressure rise delivers useful information about the coupling of the excitation and contraction cycle. It is however not easy to acquire reliable data on ureteric movement. X-ray imaging techniques using contrast media are slow and reveal relatively static momentary images. Implantation of movement detectors on the ureter is invasive and would undoubtedly affect ureteric physiology. The use of colour Doppler ultrasound as described by Summers and co-workers (1992) or radioisotope imaging as reported by Lepej and co-workers (1991) and Wemyss-Hold (1993) may be useful non-invasive techniques. We have studied ureteric motility patterns using endoluminal ultrasonography and our data reveals that correlation exists between ureteric excitation as revealed by EMG and peristaltic activity as recorded by endoluminal ultrasonography.

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