Flow dynamics in a stented ureter

JENNIFER H. SIGGERS†
Department of Bioengineering, Imperial College London, London SW7 2AZ, UK

SARAH WATERS
Oxford Centre for Industrial and Applied Mathematics, Mathematical Institute,
24–29 St Giles’, Oxford OX1 3LB, UK

AND

JONATHAN WATTIS AND LINDA CUMMINGS
Department of Mathematical Science, New Jersey Institute of Technology,
Newark, NJ 07102, USA

[Received on 19 July 2007; revised on 23 August 2008; accepted on 28 August 2008]

Vesicorenal reflux is a major side effect associated with ureteric stent placement. In a stented upper urinary tract when the bladder pressure rises, such as during bladder spasms (due to irritation caused by the stent) or voiding of the bladder, it drives urine reflux up the ureter, which, in turn, may be a contributory factor for infections in the renal pelvis. We develop a mathematical model to examine urine flow in a stented ureter, assuming that it remains axisymmetric and treating the wall as a non-linear elastic membrane. The stent is modelled as a rigid, permeable, hollow, circular cylinder lying coaxially inside the ureter. The renal pelvis is treated as an elastic bag, whose volume increases in response to an increased internal pressure. Fluid enters the renal pelvis from the kidney with a prescribed flux. The stent, ureter and renal pelvis are filled with urine, and the bladder pressure is prescribed. We use the model to calculate the total volume of reflux generated during rises in bladder pressure and investigate how it is affected by the stent and ureter properties.

Keywords: vesicorenal reflux; ureteric stent; urodynamics; mathematical modelling; fluid dynamics.

1. Introduction

In healthy individuals, urine collects in the renal pelvis, is pushed through the ureter by peristalsis and stored at low pressures in the bladder until voiding occurs (see Fig. 1). During voiding, the bladder pressure rises to expel the urine through the urethra and out of the body. To prevent vesicorenal reflux (back flow) of urine up the ureter, the vesicoureteric junction (the junction between the ureter and the bladder) closes during voiding.

Obstructions to ureteric flow can occur due to external sources of pressure (e.g. tumours) and internal blockages (e.g. kidney stones). The high urine pressures generated can be painful and may lead to renal failure due to an increased intra-renal pressure or an infection. If the obstruction cannot be removed, then the pressure must be relieved, either by a nephrostomy (drainage directly from the renal pelvis out of the body) or by inserting a ureteric stent. The most commonly used stents are long, slender, hollow, polymer

†Email: j.siggers@imperial.ac.uk
tubes extending the entire length of the ureter, e.g. see Fig. 2. They are relatively pliable longitudinally but difficult to compress radially. The most used type is the double-J or double pigtail stent, which has curls at either end to hold it in place in the renal pelvis and bladder. Ureteric stents for human use vary in circumference from 4.7 to 18 F (Walsh et al., 2002) (1 French (F) corresponds to an outer circumference of 1 mm). The walls are perforated with small holes; if there is a blockage in the stent lumen or externally to the stent, then the holes allow fluid to flow around the blockage to maintain the urine flow along the ureter.

A number of side effects are associated with the use of stents:

1. **Bladder spasms.** The stent may cause irritation through contact with the bladder wall (http://www.bui.ac.uk), which can lead to frequent bladder spasms (short duration contractions of the bladder wall), leading to a rise in fluid pressure within the bladder (Cummings et al., 2004).
2. **Vesicorenal reflux.** The presence of the stent causes the ureter to lose muscle tone and become flaccid (Ramsay *et al.*, 1985), so that the vesicoureteric junction largely ceases to function as a sphincter (Cummings *et al.*, 2004). Thus, urine reflux will occur whenever the bladder pressure rises, e.g. during voiding and spasms. Peristalsis is also weakened significantly after stent insertion (Ryan *et al.*, 1994; Payne & Ramsay, 1988).

3. **Infection and encrustation.** Salts dissolved in the urine may precipitate, encrusting the stent surface (Band *et al.*, 2008). If the encrustation is severe, stent removal becomes difficult and painful, especially if encrustation occurs in the renal pelvis. It is thought that reflux may exacerbate this problem because bladder urine has a higher salt concentration than that in the kidney and also possibly because reflux can transport bacteria up the ureter resulting in bacterial biofilm growth on the stent surface, which as well as leading to infections may produce chemicals that change the pH, leading to precipitation (Reid *et al.*, 1995).

Previous theoretical models of urine flow in a healthy ureter include work to characterize the effect of peristalsis. The equations for the flow in the ureter are simplified using lubrication theory and the flow is coupled to the wall motion using kinematic boundary conditions. Lykoudis & Roos (1970) and Roos & Lykoudis (1971) assumed the ureter to be axisymmetric and prescribed the wall position, while Griffiths (1987) and Griffiths *et al.* (1987) considered a linearly elastic ureter wall, in which the peristalsis is modelled as a prescribed pressure pulse. Carew & Pedley (1997) modelled the ureter as an axisymmetric tube with non-linear elastic wall, in which stress arises as a result of prescribed active peristaltic contractions and also passive contributions, due to the local stretching in the wall.

There has also been some work on stented and catheterized systems. Most relevant to the present paper is the work of Cummings *et al.* (2004) and Waters *et al.* (2008), who studied models of an axisymmetric stented ureter. Cummings *et al.* used asymptotic methods to investigate the effect of a rise in bladder pressure on the urine flow in the ureter when the ureter wall is linearly elastic. They found that a highly permeable stent gives rise to less reflux than an impermeable one. Waters *et al.* included the non-linear elastic properties of the ureter wall and performed a numerical investigation of the effect of a pressure pulse in the bladder on the fluid flow in the ureter.
In this paper, we extend the models (Cummings et al., 2004; Waters et al., 2008) to incorporate the non-linear elastic properties of the ureter wall, urine production by the kidney and the effect of axial variations in the material properties of the stent and ureter (e.g. due to encrustation or blockage). In particular, we investigate whether stent design can help reduce the severity of reflux. In Section 2, we derive the governing equations and boundary conditions for the model system. We assume that the ureter and stent are axisymmetric and concentric and neglect peristalsis (which is weak in stented ureters; Payne & Ramsay, 1988; Ryan et al., 1994). We treat the stent as a rigid hollow cylinder of circular cross section with a permeable wall that allows fluid flow across it. We model the renal pelvis as a linearly elastic bag, into which fluid flows at a constant rate from the kidney and flows out (or in during reflux) through the ureter (we expect variations in the renal pelvis volume to be small). The bladder pressure is prescribed and drives the reflux. In Section 4, we solve the model numerically and determine the flow. We compare this with an analytical solution in the limit of small urine flux from the kidney and also determine how the total reflux changes as the model parameters are varied. Finally in Section 5, we discuss the implications of these results for stent design and patient health.

2. Model

2.1 Overview

In this section, we derive the governing equations for a model of a stented ureter. A list of the notation used is given in Table 1. We use stars to distinguish dimensional variables from dimensionless ones. We work in polar coordinates, \((r^*, \theta, z^*)\), with associated unit vectors \(e_r\), \(e_\theta\) and \(e_z\) and assume that gravity acts in the \(e_z\) direction, i.e. the ureter is vertical. We assume that the flow and the positions of the ureter and stent remain axisymmetric (i.e. independent of \(\theta\)) with a common vertical axis, as illustrated in Fig. 3. The ureter has length \(l^*\) and radius \(b^*(z^*, t^*)\), where \(t^*\) is the time, and the ureteropelvic junction (the junction between the renal pelvis and the ureter) is at \(z^* = 0\) and the vesicoureteric junction is at \(z^* = l^*\).

The stent and ureter are filled with urine, which we treat as a homogeneous, incompressible Newtonian fluid, with viscosity \(\mu^*\) and density \(\rho^*\) equal to that of water (see Table 2). The pressures within the ureter inside and outside the stent are denoted \(p^*_s\) and \(p^* u\), respectively (the subscript \(s\) is used to denote quantities within the stent, while \(u\) is used for the corresponding quantities in the ureter but outside the stent), and the axial and radial velocities are denoted \(w^*\) and \(u^*\), respectively. We assume that the pressure external to the ureter, \(p^*_e(z^*)\), is hydrostatic and equal to \(p^*_c + \rho^* g^* z^*\), where \(g^*\) is the acceleration due to gravity and \(p^*_c\) is a constant; however, if there is an additional source of pressure external to the ureter, e.g. due to a tumour, this may be modelled by increasing \(p^*_c\) locally. We work in terms of reduced pressures, \(\hat{p}^*\), related to \(p^*\) by subtracting the hydrostatic pressure: \(\hat{p}^* = p^* - p^*_c\).

The stent has inner radius \(a^*_i(z^*)\) and outer radius \(a^*_o(z^*)\). It is assumed that the flux per unit area across the stent wall is proportional to the pressure difference across the wall, with constant of proportionality given by the wall permeability. Encrustation on the stent surface can decrease the permeability due to hole blockage. Furthermore, encrustation on the outside of the stent increases the effective outer radius, \(a^*_o\), in that region, while on the inside it decreases \(a^*_i\). We apply no-slip boundary conditions at the stent surface.

The ureter wall is modelled as an elastic membrane with natural radius \(b^*_n(z^*\), \(t^*)\) (the radius when the transmural pressure is zero). It is assumed to move only in the radial direction, and the membrane tension is taken to be a non-linear function of the radial stretch, given by (see Fung, 1967, for details)
**Table 1** List of notation used in the paper

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Dimensionless equivalent</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$t^*$</td>
<td>$t$</td>
<td>Time</td>
</tr>
<tr>
<td>$r^*$</td>
<td>$r$</td>
<td>Radial coordinate</td>
</tr>
<tr>
<td>$\theta$</td>
<td>$\theta$</td>
<td>Polar angle</td>
</tr>
<tr>
<td>$z^*$</td>
<td>$z$</td>
<td>Axial coordinate</td>
</tr>
<tr>
<td>$u^<em>(r^</em>, z^<em>, t^</em>)$</td>
<td>$u(r, z, t)$</td>
<td>Radial velocity</td>
</tr>
<tr>
<td>$w^<em>(r^</em>, z^<em>, t^</em>)$</td>
<td>$w(r, z, t)$</td>
<td>Axial velocity</td>
</tr>
<tr>
<td>$b^<em>(z^</em>, t^*)$</td>
<td>$b(z, t)$</td>
<td>Ureteric radius</td>
</tr>
<tr>
<td>$q_s(z, t)$, $q_u(z, t)$</td>
<td>Fluxes down stent and ureter</td>
<td></td>
</tr>
<tr>
<td>$a_i^<em>(z^</em>)$, $a_o^<em>(z^</em>)$</td>
<td>Inner, outer stent radius</td>
<td></td>
</tr>
<tr>
<td>$b_n^<em>(z^</em>)$</td>
<td>$d(z)$</td>
<td>Natural ureteric radius</td>
</tr>
<tr>
<td>$g^*$</td>
<td>—</td>
<td>Acceleration due to gravity</td>
</tr>
<tr>
<td>$\mu^*$</td>
<td>—</td>
<td>Urine viscosity</td>
</tr>
<tr>
<td>$\rho^*$</td>
<td>—</td>
<td>Urine density</td>
</tr>
<tr>
<td>$l^*$</td>
<td>—</td>
<td>Length of ureter</td>
</tr>
<tr>
<td>$k_w^*$</td>
<td>$k_w, \gamma$</td>
<td>Stiffness of renal pelvis</td>
</tr>
<tr>
<td>$q_k^*$</td>
<td>$q_k$</td>
<td>Urine flux from kidney</td>
</tr>
<tr>
<td>$\epsilon = b_n^* / l^*$</td>
<td>Aspect ratio of ureter</td>
<td></td>
</tr>
<tr>
<td>$v_k, v_{kn}$</td>
<td>Volume of renal pelvis and equilibrium, volume of renal pelvis</td>
<td></td>
</tr>
<tr>
<td>$p^*$, $\tilde{p}$</td>
<td>$p, \tilde{p}$</td>
<td>Pressure and reduced pressure</td>
</tr>
<tr>
<td>$p_{hyd}(z^<em>)$, $p_c^</em>$</td>
<td>Hydrostatic pressure and pressure outside renal pelvis</td>
<td></td>
</tr>
<tr>
<td>$p_s(z, t)$, $\tilde{p}_s(z, t)$</td>
<td>Absolute and reduced pressure in stent</td>
<td></td>
</tr>
<tr>
<td>$p_u(z, t)$, $\tilde{p}_u(z, t)$</td>
<td>Absolute and reduced pressure in ureter outside stent</td>
<td></td>
</tr>
<tr>
<td>$p_e(z, t)$, $\tilde{p}_e(z, t)$</td>
<td>Absolute and reduced pressure external to ureter</td>
<td></td>
</tr>
<tr>
<td>$p_k(t)$, $\tilde{p}_k(t)$</td>
<td>Absolute and reduced pressure in the renal pelvis</td>
<td></td>
</tr>
<tr>
<td>$p_b(t)$, $\tilde{p}_b(t)$</td>
<td>Absolute and reduced pressure in the bladder</td>
<td></td>
</tr>
<tr>
<td>$\tilde{p}_{kn}$</td>
<td>$\tilde{p}_{kn}$</td>
<td>Normal equilibrium reduced pressure in renal pelvis</td>
</tr>
<tr>
<td>$\tilde{p}<em>r$, $\tilde{p}</em>{max}$</td>
<td>$\tilde{p}<em>r$, $\tilde{p}</em>{max}$</td>
<td>Resting and maximum reduced bladder pressures</td>
</tr>
<tr>
<td>$f$</td>
<td>Function characterizing elastic properties of ureteric wall</td>
<td></td>
</tr>
<tr>
<td>$e_r$, $e_\theta$, $e_z$</td>
<td>Unit coordinate vectors</td>
<td></td>
</tr>
</tbody>
</table>
Fig. 3. Schematic diagram of the mathematical model showing the ureter, stent and renal pelvis. Dashed lines indicate the ends of the ureter, while the dotted lines indicate the cross sections shown above.

**Table 2** Typical values of dimensional parameters in humans

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Typical value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine viscosity</td>
<td>( \mu^* )</td>
<td>0.00654 g cm(^{-1}) s(^{-1})</td>
<td>Batchelor (1967)‡</td>
</tr>
<tr>
<td>Urine density</td>
<td>( \rho^* )</td>
<td>1 g cm(^{-3})</td>
<td>Batchelor (1967)</td>
</tr>
<tr>
<td>Acceleration due to gravity</td>
<td>( g^* )</td>
<td>981 cm s(^{-2})</td>
<td></td>
</tr>
<tr>
<td>Ureretic length</td>
<td>( l^* )</td>
<td>25 cm</td>
<td>Cummings et al. (2004)</td>
</tr>
<tr>
<td>Inner stent radius</td>
<td>( a_i^* )</td>
<td>0.065 cm</td>
<td>†</td>
</tr>
<tr>
<td>Outer stent radius</td>
<td>( a_o^* )</td>
<td>0.095 cm</td>
<td>†</td>
</tr>
<tr>
<td>Natural internal radius of ureteric wall</td>
<td>( b^* )</td>
<td>0.2 cm</td>
<td>Cummings et al. (2004)</td>
</tr>
<tr>
<td>Body pressure outside renal pelvis</td>
<td>( p_c^* )</td>
<td>4-cm H(_2)O</td>
<td>Cummings et al. (2004)</td>
</tr>
<tr>
<td>Rate of urine production</td>
<td>( q_k^* )</td>
<td>( 8.3 \times 10^{-3} ) cm(^3) s(^{-1})</td>
<td>Cummings et al. (2004)</td>
</tr>
</tbody>
</table>

† Measured from stents supplied by Boston Scientific.
‡ Note that an incorrect value for the urine viscosity was used in Cummings et al. (2004).

\[ f^*(\frac{b^*}{b_o^*}) = k_w^*(e^{b^*(b^*/b_o^*-1)} - 1). \] (2.1)

Here, the stiffness of the ureter wall is characterized by \( k_w^* \) and the parameter \( \gamma \) is a positive constant characterizing how rapidly the membrane tension varies with ureter radius. The fluid and wall motion are coupled through boundary conditions at the wall of the ureter: no-slip velocity boundary conditions are applied to the fluid, which in turn imposes a traction on the elastic wall.

We model the renal pelvis as an elastic bag of constant stiffness \( k_p^* \) and assume that the pressure there depends only on the volume of fluid contained within it (Section 2.3). Fluid flows into the renal pelvis from the kidney at a constant rate, \( q_k^* \), and flows out (or in during reflux) at a variable rate through the ureteropelvic junction. The bladder is treated as a reservoir of fluid at a prescribed pressure.

Where available, typical physiological values of the parameters in human adults are given in Table 2. However, physiological estimates of the wall stiffness of a stented ureter and renal pelvis, and estimates of the time and pressure scales involved for the bladder pressure, are limited.
2.2 Stent–ureter system

2.2.1 Scaling and non-dimensionalization of the fluid flow. We non-dimensionalize as follows:

\[ z^* = l^*z, \quad (r^*, b^*, a^*_1, a^*_o) = b^*_a(r, b, a_1, a_o), \quad f^* = k_w^*f, \quad \hat{p}^* = \frac{k_w^*}{b^*_a} \hat{p}, \]

\[ u^* = \frac{\epsilon^2 k_w^*}{\mu^*} u, \quad w^* = \frac{\epsilon k_w^*}{\mu^*} w, \quad t^* = \frac{\mu^* b^*_w}{\epsilon^2 k_w^*} t, \]

where \( \epsilon = b^*_w/l^* \) is the aspect ratio, and \( z = 0 \) at the ureteropelvic junction and \( z = 1 \) at the vesicoureteric junction. Note that \( \mu^* b^*_w/(\epsilon^2 k_w^*) \) is the timescale for the fluid to move in response to the pressure gradient due to wall deformation.

2.2.2 Governing equations and boundary conditions. In the following sections, we assume that \( \epsilon^2 \ll 1 \) and \( \epsilon^2 Re \ll 1 \), where \( Re = \rho^* \epsilon^2 l^* k_w^*/\mu^* \) is the Reynolds number. Using the values in Table 2, we find that \( \epsilon^2 \approx 1 \times 10^{-5} \). In the expression for the Reynolds number, the parameter \( k_w^* \) is unknown. Requiring that \( \epsilon^2 Re \ll 1 \) therefore puts a limit on the size of \( k_w^* \). Further experimental work is required to ascertain the size of this parameter, particularly in a stented system where we expect the value to be lower than that for healthy ureters due to the loss of muscle tone associated with stenting (Ramsay et al., 1985). Once the size of \( k_w^* \) is known, the applicability of this model can be determined by considering the size of \( \epsilon^2 Re \ll 1 \).

The dominant balance in the Navier–Stokes equations is then between the axial pressure gradient and viscous terms (see, e.g., Ockendon & Ockendon, 1995), and the dimensionless axisymmetric equations are

\[ \frac{\partial \hat{p}}{\partial r} = 0, \quad \frac{\partial \hat{p}}{\partial z} = \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial w}{\partial r} \right), \quad \frac{1}{r} \frac{\partial}{\partial r}(ru) + \frac{\partial w}{\partial z} = 0, \]

(2.2a–c)

where the symbol \( \hat{p} \) should be interpreted as \( \hat{p}(z, t) \) within the stent (\( 0 \leq r \leq a_i \)) and \( \hat{p}_a(z, t) \) in \( a_o \leq r \leq b \). We assume that the flux through the holes in the stent is proportional to the pressure drop across the wall:

\[ a_i u|_{r=a_i} = a_o u|_{r=a_o} = \frac{a_i^4 d}{16} (\hat{p}_s - \hat{p}_a), \]

(2.3)

where \( d(z) \) is the stent permeability or open porosity, and the factor \( a_i^4/16 \) has been inserted for convenience later. The value of \( d \) depends on the size and number of holes in the stent; the estimate in Cummings et al. (2004) yields the value \( d \approx 900 \). We also impose no-slip axial velocity at the stent wall. At the ureter wall, \( r = b \), we impose the no-slip and no-penetration conditions

\[ w = 0 \quad \text{and} \quad u = \frac{\partial b}{\partial t}. \]

(2.4a,b)

Equation (2.2) gives

\[ w = \begin{cases} \frac{1}{4} \frac{\partial \hat{p}_s}{\partial z} (r^2 - a_i^2), \\ \frac{1}{4} \frac{\partial \hat{p}_a}{\partial z} \left( (r^2 - a_o^2) - (b^2 - a_o^2) \log(r^2/a_o^2)/(b^2/a_o^2) \right), \end{cases} \]

(2.5)
\[
\begin{aligned}
&u = \left\{ \begin{array}{l}
\frac{r}{16} \frac{\partial}{\partial z} \left( (2a_i^2 - r^2) \frac{\partial \hat{p}_s}{\partial z} \right), \\
\frac{1}{16r} \frac{\partial}{\partial z} \left( \frac{\partial \hat{p}_u}{\partial z} \left( -(r^2 - a_o^2)^2 + 2 \frac{(b^2 - a_o^2)}{\log(b^2/a_o^2)} \left( r^2 \log \frac{r^2}{a_o^2} - r^2 + a_o^2 \right) \right) + a_i^4 \frac{\partial \hat{p}_s}{\partial z} \right),
\end{array} \right.
\end{aligned}
\]  
(2.6)
in the two regions \(0 \leq r \leq a_i\) and \(a_o \leq r \leq b\), respectively. Substituting (2.6) into (2.3) provides an expression relating the pressures \(\hat{p}_s\) and \(\hat{p}_u\):

\[
\frac{\partial}{\partial z} \left( a_i^4 \frac{\partial \hat{p}_s}{\partial z} \right) = a_i^4 d (\hat{p}_s - \hat{p}_u),
\]
(2.7)
while (2.4b) yields the mass conservation equation

\[
\frac{\partial (\pi b^2)}{\partial t} = -\frac{\partial}{\partial z} (q_s + q_u),
\]
(2.8)
where \(q_s\) and \(q_u\) are the non-dimensional axial fluxes down the stent and the ureter, given by

\[
\begin{aligned}
q_s(z, t) &= -\frac{\pi}{8} \frac{\partial \hat{p}_s}{\partial z} a_i^4, \\
q_u(z, t) &= \frac{\pi}{8} \frac{\partial \hat{p}_u}{\partial z} \left( (b^2 - a_o^2)^2 + 2 \frac{(b^2 - a_o^2)}{\log(b^2/a_o^2)} \left( b^2 - a_o^2 - b \log \frac{b}{a_o} \right) \right),
\end{aligned}
\]
(2.9a,b)
respectively. Note that negative values of \(q_s\) and \(q_u\) indicate reflux.

We relate the wall position, \(b\), to the transmural pressure, \(\hat{p}_u - \hat{p}_c\), via the following wall law (which may be derived from a force balance on the wall neglecting axial stretch and wall inertia):

\[
\hat{p}_u = \frac{1}{b} f(b) + \hat{p}_c,
\]
(2.10)
where, using (2.1),

\[
f(\lambda) = e^{\gamma(\lambda-1)} - 1.
\]
(2.11)
In the absence of data determining \(\gamma\), we consider \(\gamma = 1\) throughout. Note that \(f(1) = 0\), corresponding to zero stress in the unstretched state.

2.3 Renal pelvis

We assume that the renal pelvis pressure, \(\hat{p}_k\), depends only on \(v_k(t)\), the renal pelvis volume. As reflux occurs \(v_k\) will increase, which causes \(\hat{p}_k\) to increase, acting to suppress further reflux.

We expect variations in \(v_k\) to be small, allowing a linear approximation, and assume the non-dimensional and corresponding dimensional relationships

\[
\hat{p}_k = \hat{p}_{kn} + k_k (v_k - v_{kn}), \quad \hat{p}_k^* = \hat{p}_{kn}^* + k_k^* (v_k^* - v_{kn}^*),
\]
(2.12)
where \(v_{kn}^*\) and \(\hat{p}_{kn}^*\) are the equilibrium values of \(v_k^*\) and \(\hat{p}_k^*\), respectively, under normal renal operation, \(k_k^*\) is the stiffness of the renal pelvis wall and

\[
k_k = \varepsilon^{-4} l_s^4 k_w^*/k_w^*.
\]
We denote the non-dimensional net rate of urine excretion from the kidney into the renal pelvis by

\[ q_k = \frac{\mu^* q_k^*}{\varepsilon^3 \gamma^2 k_w}. \]  

(2.13)

Its value depends on patient physiology and fluid intake, and we assume that it is constant on the timescale of variations in the bladder pressure. Thus, \( \nu_k \) satisfies

\[ \frac{d \nu_k}{dt} = q_k - q_s(0, t) - q_u(0, t), \]

where \( q_s(0, t) \) and \( q_u(0, t) \) are fluxes given by (2.9a,b), and hence

\[ \frac{d \hat{p}_b}{dt} = k_k(q_k - q_s(0, t) - q_u(0, t)). \]  

(2.14)

2.4 Model equations

We have obtained a system of three partial differential equations (2.7), (2.8) and (2.10) governing the three non-dimensional variables \( \hat{p}_s(z, t) \) and \( \hat{p}_u(z, t) \), the reduced pressures inside the stent and outside the stent, respectively, and \( b(z, t) \), the radial position of the wall. At the ureteropelvic junction \( (z = 0) \) and vesicoureteric junction \( (z = 1) \), the ureter is free to move radially only (no movement in the axial direction), and we assume continuity of pressure; thus,

\[ \hat{p}_s(0, t) = \hat{p}_u(0, t) = \hat{p}_k(t) \quad \text{and} \quad \hat{p}_s(1, t) = \hat{p}_u(1, t) = \hat{p}_b(t), \]  

(2.15a,b)

and the renal pelvis pressure is governed by (2.14). To complete the model, the reduced bladder pressure, \( \hat{p}_b(t) \), must be specified. This increases during voiding and bladder spasms, which drive the reflux.

The bladder pressure is prescribed as shown in Fig. 4. Two types of bladder pressure pulse are considered to model bladder spasms and bladder voiding. Type I (spasm) corresponds to Fig. 4(a). During Phase 0, the bladder pressure, \( \hat{p}_b \), is fixed at its resting value, \( \hat{p}_t \), and a steady state is assumed. In Phase I, \( \hat{p}_b \) increases linearly from \( \hat{p}_t \) to the maximum pressure, \( \hat{p}_{\text{max}} \), over a time \( t_1 \), and in Phase II it decreases for a time \( t_2 - t_1 \), until it returns to the constant value \( \hat{p}_t \) and Phase III begins, during which the system relaxes back to the initial state. Type II (voiding) corresponds to Fig. 4(b), where after the

![Fig. 4. Sketches of Type I and Type II bladder pressure pulses.](image-url)
initial pressure rise, the pressure then remains fixed and elevated for a period of time. Note that the (dimensional) pulse duration has been non-dimensionalized with respect to the timescale, \( \mu b^*/(\epsilon^2 k^*_w) \), which corresponds to the timescale over which the fluid moves in response to pressure gradients arising from wall deformations.

We define the total reflux at the point \( z \) to be the total volume of fluid passing through \( z \) in the negative \( z \) direction, i.e.

\[
- \int \min(q_s(z, t), 0) + \min(q_u(z, t), 0) dt,
\]

where the integral is taken over the duration of the bladder pulse. Usually, we consider the total reflux at the ureteropelvic junction (i.e. at \( z = 0 \)).

Note that the systems of equations found by Cummings et al. (2004) may be obtained by taking (2.7), (2.8) and (2.10), setting \( \hat{p}_e = 0 \) and assuming that \( b/a \), \( a_i \) and \( a_o \) are constant. Cummings et al. also assumed \( a_i = a_o \) and linearized (2.10) about the unstretched state with \( b = b_a \).

3. Analytical steady-state solution

In this section, we seek a steady solution to the system described in Section 2.4 in the limit of small \( q_k \) (see (2.13)). We assume that the stent and ureter properties are constant along their length (i.e. \( a_i, a_o, d \) and \( \hat{p}_e \) are constants).

When \( q_k = 0 \), there is an exact solution, in which the pressures in the ureter everywhere equal the bladder pressure, while the ureter has a uniform radius, i.e.

\[
\hat{p}_s = \hat{p}_u = \hat{p}_b,
\]

and \( b = b_0 \), independent of \( z \) and \( t \), where \( b_0 \) satisfies

\[
\frac{1}{b_0} f(b_0) - \hat{p}_b + \hat{p}_c = 0.
\]

For small \( q_k \), the above solution provides the first term in a series solution to the steady problem in powers of \( q_k \). We write

\[
\hat{p}_s = \hat{p}_b + q_k \hat{p}_{s1} + \cdots, \quad \hat{p}_u = \hat{p}_b + q_k \hat{p}_{u1} + \cdots, \quad b = b_0 + q_k b_1 + \cdots,
\]

where the correction terms are independent of \( t \) but not necessarily uniform in \( z \). To find the next terms in the series above, we substitute these expansions into (2.7), (2.8) and (2.10). The \( \mathcal{O}(q_k) \) contributions to (2.7) and (2.8) are

\[
\frac{\partial^2 \hat{p}_{s1}}{\partial z^2} = d (\hat{p}_{s1} - \hat{p}_{u1}), \quad -\gamma_1 \frac{\partial \hat{p}_{s1}}{\partial z} - \gamma_2 \frac{\partial \hat{p}_{u1}}{\partial z} = 1,
\]

respectively, where

\[
\gamma_1 = \frac{\pi}{8} a_i^4, \quad \gamma_2 = \frac{\pi}{8} (b_0^2 - a_o^2) \left( \frac{b_0^2 + a_o^2}{\log(b_0^2/a_o^2)} - \frac{2(b_0^2 - a_o^2)}{\log(b_0^2/a_o^2)} \right)
\]

(3.3)
are positive and constant along the length of the ureter, and the boundary conditions at this order are ${\hat{p}}_{s1} = {\hat{p}}_{u1}$ at $z = 0$ and ${\hat{p}}_{s1} = 0$ at $z = 1$. Integrating (3.2b), we obtain $\gamma_1 {\hat{p}}_{s1} + \gamma_2 {\hat{p}}_{u1} = 1 - z$, and (3.2a) together with the boundary conditions gives the solutions

$$\hat{p}_{s1} = \hat{p}_{u1} = \frac{1 - z}{\gamma_1 + \gamma_2},$$

for the corrections to the pressures, indicating that the pressure is slightly higher at the renal pelvis than the bladder. Equation (2.8) implies that

$$b_1 = \frac{1 - z}{(\gamma_1 + \gamma_2)(\hat{c}(b^{-1} f(b/b_a))/\hat{c}b)|_{b=b_0}},$$

so, as expected, the ureter radius is slightly larger at the renal pelvis (corresponding to the higher pressure there). We note that using a similar model but with axial stretch incorporated, we found that there is slight stretching of the ureter near to the renal pelvis and compression near to the bladder, due to the drag of fluid flowing down the ureter.

4. Numerical results

We solve the full time-dependent problem described in Section 2.4 numerically. We discretize the equations using a uniform mesh in $z$, use second-order central differences and perform the time stepping using the Matlab built-in function ode15s. The code is validated by refining the mesh, using shorter time steps and checking against analytical results from Section 3.

Throughout this section, we use the parameter values listed in Table 3, unless stated otherwise. These parameter values are based on the physiological estimates where possible. However, physiological

| Table 3 Table of default non-dimensional parameter values used in the model |
| --- | --- | --- |
| Quantity | Symbol | Typical value used in simulations |
| Inner stent radius | $a_i$ | 0.3 |
| Outer stent radius | $a_o$ | 0.5 |
| Natural radius of ureter | $b_a$ | 1.0 |
| Stent permeability | $d$ | 900 |
| Ureter wall parameter (see (2.11)) | $\gamma$ | 1 |
| Urine excretion rate from kidney | $q_k$ | 0.06 |
| Renal pelvis stiffness | $k_k$ | 1 |
| Bladder pressures | --- | --- |
| During resting | $\hat{p}_r$ | 0 |
| Maximum | $\hat{p}_{\text{max}}$ | 1 |
| Times: Type I | --- | --- |
| Pressure rise | $t_1$ | 10 |
| Pressure fall | $t_2 - t_1$ | 10 |
| Times: Type II | --- | --- |
| Pressure rise | $t_1$ | 10 |
| Constant pressure | $t_2 - t_1$ | 30 |
| Pressure fall | $t_3 - t_2$ | 10 |
estimates of the wall stiffness of a stented ureter and renal pelvis, and estimates of the time and pressure scales involved for the bladder pressure, are non-existent. We choose parameter values that are consistent with our assumption of lubrication theory and enable all the incorporated effects in the model to be retained.

4.1 Examples of typical behaviour

We start by considering a Type I pressure profile (see Section 2.4 and Fig. 4a), with the parameter values given in Table 3. The ureteric radius, \( b(z, t) \), is shown in Fig. 5(a–d) at various times during the simulation, and the corresponding pressures in the bladder and renal pelvis and the fluxes at the ureteropelvic junction and vesicoureteric junction are shown in Fig. 5(e) and (f). During resting, the bladder pressure, \( \hat{p}_b \), the elevated renal pelvic pressure, \( \hat{p}_k \), and the axial variation in the ureteric radius arise due to the non-zero urine flux, \( q_k \), from the kidney into the renal pelvis (Fig. 5a). As \( \hat{p}_b \) increases (Fig. 5b), a pulse of fluid flows into the ureter and the ureteric radius quickly increases at \( z = 1 \). The radius at \( z = 0 \) increases as the pulse wave reaches the renal pelvis and continues to increase until approximately halfway through Phase II, before dropping gradually back to the resting value. It takes

![Graphs](https://example.com/graphs.png)

**Fig. 5.** Type I simulation using parameter values in Table 3. Phase I starts at \( t = 0 \), Phase II at \( t = 10 \) and Phase III at \( t = 20 \). (a)–(d) Snapshots of ureter radius \( b(z, t) \) (solid curve) at various times. Also shown are the natural ureter radius (dotted), and \( a_i \), \( a_o \) (dashed). The renal pelvis is at \( z = 0 \) and the bladder is at \( z = 1 \). (e) Bladder pressure, \( \hat{p}_b \), (dashed) and renal pelvic pressure, \( \hat{p}_k \), (solid) against time. (f) Non-dimensional flux at the ureteropelvic junction, \( z = 0 \) (solid), and at the vesicoureteric junction, \( z = 1 \) (dashed). The vertical dotted lines in (e) and (f) indicate the ends of the phases.
a comparatively long time for the steady resting state to be restored after the transient rise in bladder pressure, but for sufficiently long times the renal pelvic pressure indeed returns to the steady-state value.

Figure 5(e) shows the pressures in the bladder (dashed) and the renal pelvis (solid), during the same scenario. The pressure in the renal pelvis starts rising towards the end of Phase I and peaks approximately halfway through Phase II. Figure 5(f) shows the corresponding fluxes at the ureteropelvic junction (solid) and vesicoureteric junction (dashed). We see that the reflux into the ureter from the bladder exceeds that from the ureter into the renal pelvis. Most of the reflux occurs during Phase I, with the remainder occurring at the start of Phase II. However, for sufficiently small values of $t_1$ and $t_2$, reflux at the ureteropelvic junction continues into the start of Phase III. Note that there is a significant delay before reflux is observed at the ureteropelvic junction; this is a consequence of the fact that for a bladder pressure pulse duration of this type, a pulse of fluid travels up the ureter towards the renal pelvis on the timescale of the prescribed bladder pressure rise.

Figure 6 illustrates a Type I pressure profile, for which $t_1 = 100$ and $t_2 = 200$ (the remaining parameters are given in Table 3). The duration of the bladder pressure rise is now long compared to the typical timescale for the fluid to move in response to pressure gradients due to wall deformations, and as the bladder pressure increases from zero, the ureter inflates and the renal pelvis pressure increases due to reflux through the ureteropelvic junction (Fig. 6b,e). A nearly axially uniform inflated state is achieved (Fig. 6b). Note that for this longer bladder pressure pulse duration, any time-dependent variations in

Fig. 6. Type I simulation using parameter values in Table 3 (except for timescales). Phase I starts at $t = 0$, Phase II at $t = 100$ and Phase III at $t = 200$. The graphs and line styles correspond to those in Fig. 5.
bladder pressure are rapidly transmitted to the renal pelvis; this is revealed in the fact that the maximum pressure in the renal pelvis in Fig. 6(e) is achieved at \( t \approx 101.9 \), i.e. after a delay of 1.9 from the maximum in the bladder. For the shorter duration pulse shown in Fig. 5(e), the maximum renal pelvis pressure is achieved at \( t \approx 13.2 \), i.e. after a longer delay of 3.2 after the bladder maximum. As a consequence, for the longer bladder pressure pulse duration the maximum renal pelvis volume is achieved more quickly after the maximum bladder pressure is reached (see also (2.12)).

Next, we consider a Type II scenario (see Fig. 4b), with parameters given in Table 3 (see Fig. 7). As \( \hat{p}_b \) starts to increase, \( b(1, t) \) increases, and later \( b(0, t) \) increases, corresponding to an increase in \( \hat{p}_k \) and soon afterwards the start of reflux through the ureteropelvic junction (Fig. 7b,e,f). During much of Phase II, the radius \( b(z, t) \) is approximately independent of \( z \) (Fig. 7c), and a new steady state is established, which persists until the start of Phase III. In this state, \( \hat{p}_k \) and \( \hat{p}_b \) are almost equal, and the flux down the ureter equals \( q_k \) (Fig. 7e,f). In Phase III, \( \hat{p}_b \) begins to fall, meaning that a positive flux occurs at both the vesicoureteric junction and the ureteropelvic junction (Fig. 7f). In Phase IV, when the bladder pressure is close to its resting value it takes a relatively long time for an equilibrium to be re-established, compared with the start of Phase II, see the solid curve in Fig. 7(e). A possible reason is that the ureteric radius is smaller at the start of Phase IV than at the start of Phase II, and since the resistance to flow in a cylinder is proportional to the fourth power of the cylindrical radius, both the flux and the rate of equilibration in Phase II will be larger than at the start of Phase IV.

![Graphs illustrating bladder pressure transmission to renal pelvis and reflux through ureteropelvic junction](image)

**Fig. 7.** Type II simulation using parameter values in Table 3. The graphs and line styles correspond to those in Fig. 5.
4.2 Comparison with analytical solution

In the Type II scenario outlined above, almost all the reflux occurs during Phase I, with a small amount occurring at the start of Phase II. For the rest of Phase II, the system is in a near steady state, and so we may use the analytical solution presented in Section 3 to estimate the reflux. We use (2.12) to calculate series solutions for the volume of the renal pelvis, \( v_k \), in the two steady states corresponding to Phase 0 (resting phase with \( \hat{p}_b = 0 \)) and Phase II (in which \( \hat{p}_b = \hat{p}_{\text{max}} \)). The total reflux at the ureteropelvic junction equals the difference between the volumes of the renal pelvis in the two steady states minus the amount of urine entering the renal pelvis from the kidney in that time. Hence,

\[
\text{Reflux} = v_k^{\text{II}} - v_k^0 - q_k \Delta t = \frac{\hat{p}_k^{\text{II}} - \hat{p}_k^0}{k_k} - q_k \Delta t = \frac{\hat{p}_{\text{max}} - \hat{p}_b}{k_k} - q_k \Delta t + \frac{q_k}{k_k} h + \mathcal{O}(q_k^2),
\]

where \( \hat{p}_k^0, \hat{p}_k^{\text{II}}, v_k^0 \) and \( v_k^{\text{II}} \) are the renal pelvis pressures and volumes in Phases 0 and II, respectively, \( \Delta t \) is the time between the end of Phase 0 and the establishment of the steady Phase II state, and

\[
h = \frac{1}{(\gamma_1 + \gamma_2)^{\text{II}}} - \frac{1}{(\gamma_1 + \gamma_2)^0},
\]

where \( \gamma_1^0, \gamma_2^0, \gamma_1^{\text{II}} \) and \( \gamma_2^{\text{II}} \) are the values of \( \gamma_1 \) and \( \gamma_2 \) in the respective phases (see (3.3)). Note that \( h \) is negative and \( \mathcal{O}(1) \). The term \( q_k h / k_k \) appearing in the expression for the reflux (4.1) arises due to the fact that the pressure difference along the ureter (needed to drive the flux \( q_k \)) is smaller in Phase II than in Phase 0. Thus, the difference in renal pelvic pressure between Phase 0 and Phase II is slightly less than the difference in bladder pressure, giving the negative correction \( q_k h / k_k \) to the reflux. At leading order, the only parameters affecting the total reflux are the bladder pressure difference between Phase 0 and Phase II states and the stiffness of the renal pelvis. The stent design in particular only affects the total reflux at \( \mathcal{O}(q_k) \).

Figure 8(a) shows the dependence of the \( \mathcal{O}(q_k) \) correction to the reflux \( h \) (given by (4.2)) on the stent radius, \( a_0 \) (keeping the stent wall thickness \( a_0 - a_i \) fixed), which indicates that there is an optimal stent size to minimize reflux \( h \) is the first correction term to the reflux volume for small kidney production rates \( q_k \). For a given ureter radius, the existence of an optimal stent radius can be explained as follows: for a small stent, most of the urine travels down the outside, so increasing the stent radius leads to an increase in the resistance of the stent–ureter system and a reduction in reflux. Conversely, for a large stent most of the urine travels down the inside of the stent lumen and so a small decrease in the stent radius leads to an increase in resistance and a reduction in reflux. Thus, for any given ureter radius, there is an optimum stent radius which offers the maximum resistance to flow; for this optimal stent size, the flow down the outside of the stent is of a similar magnitude to that down the stent lumen. The difference between normal operation and reflux is that in normal operation (the resting state), the bladder pressure is low and the ureter is unstretched and offers a high resistance to flow; in the reflux steady state, the bladder is at high pressure, causing the whole of the ureter to become inflated and the system then offers a low resistance to flow. A small stent presents a low resistance in both steady states, whereas a larger stent can have a low resistance in normal operation (an unstretched ureter and most flow occurring down the stent lumen), but offer a larger resistance to reflux when the ureter is inflated and there are similar amounts of flow along the lumen and the outside of the stent. Keeping \( a_0 \) fixed, decreasing \( a_i \) thickens the stent, increases the resistance of the stent–ureter system and so leads to a reduction in reflux: this effect is illustrated in Fig. 8(b).
4.3 Influence of stent design and model parameters on reflux

4.3.1 Uniform properties. In Section 4.2, we showed that stent design has only a weak effect on the reflux during those Type II pressure variations in which a steady state is established during Phase II; we now investigate numerically how reflux depends on the system parameters in general. We consider the total reflux at the ureteropelvic junction, i.e. at \( z = 0 \), defined by (2.16). We consider both Types I and II pressure pulses.

In Fig. 9(a), we see that for both types of bladder pressure pulse, increasing the maximum reduced bladder pressure increases the total reflux, as expected. The reflux corresponding to Type II (voiding, dashed line) is slightly higher compared to Type I (spasm, solid line), reflecting the fact that the bladder pressure is held at its maximum value for longer. Note also that it is necessary for \( \hat{p}_{\text{max}} \) to exceed a certain value for significant reflux to occur. The effect of increasing \( q_k \) is to decrease the total reflux, as shown in Fig. 9(b). For sufficiently large values of \( q_k \), the renal pelvis (reduced) pressure exceeds that in the bladder, and there is no reflux. Increasing the pressure outside the ureter decreases the reflux for both Type I and Type II pulses (Fig. 9c).

![Fig. 8](image)

**Fig. 8.** (a) Analytical results show how \( h \), the \( O(q_k) \) contribution to the total reflux at the ureteropelvic junction in (4.1), changes with \( a_o \) (keeping \( a_o - a_l \) fixed); (b) graph of \( g \) against \( a_l \) (keeping \( a_o \) fixed).

![Fig. 9](image)

**Fig. 9.** Graphs showing how the total reflux depends on physiological parameters for Type I (solid) and Type II (dashed) bladder pulses. The parameters considered are (a) the maximum reduced bladder pressure, \( \hat{p}_{\text{max}} \), (b) the imposed flux \( q_k \) produced by the kidney, \( q_k \), and (c) reduced external pressure (constant along the ureter length), \( \hat{p}_e \). All parameter values except for the one under investigation are given by the values in Table 3.
Figure 10 shows the effect of the timescale of the bladder pressure pulse on the degree of reflux for a variety of urine production rates $q_k$; in each case the horizontal axis is $t_1$. For Type I pulses (solid lines), $t_2 = 2t_1$, while for Type II pulses (dashed lines) $t_2 = t_1 + 30$ and $t_3 = t_1 + t_2$.

We start by considering Fig. 10(a), corresponding to $q_k = 0$. For Type I pulses (solid line), the total reflux increases as the duration of the pressure rise (and fall) increases. For short duration pulses, the fluid pulse entering the ureter from the bladder does not reach the renal pelvis before the bladder pressure starts to decrease and the reflux is small. Thus, increasing the duration of the pressure rise enables the fluid pulse to reach the renal pelvis, and correspondingly the volume of reflux increases. For larger pressure rise duration, the total reflux asymptotes to a constant value. In this case, the pressures within the ureter and stent are approximately axially uniform. The flux at the renal pelvis is then given by $(d\hat{p}_b/dt)/k_k$, and hence the total reflux (obtained by integrating over the duration for which $p_b$ is increasing in time) is $(\hat{p}_{\text{max}} - \hat{p}_r)/k_k$. The additional fluid entering the system from the bladder is accommodated within the ureter. For Type II pulses (dashed line in Fig. 10a), a steady state is achieved when $\hat{p}_b = \hat{p}_{\text{max}}$, and so the reflux volume is just the difference between the renal pelvis volume in the resting and the maximum bladder pressure phases (and is independent of the timescale of the pressure rise); see (4.1) and the dotted line in Fig. 10(a) which shows this asymptotic value (the dashed and dotted lines are indistinguishable).

In Fig. 10(b–d), $q_k$ takes values of 0.001, 0.01 and 0.06, respectively. The effect of increasing $q_k$ (in the absence of a prescribed bladder pulse) is to raise the renal pelvic pressure and set up an axially non-uniform ureter radius steady state (see (2.14)). Considering Type I pulses (solid lines), for short durations, the fluid pulse does not have time to reach the renal pelvis and there is very little reflux—indeed for the case $q_k = 0.06$ (Fig. 10d), the value of $q_k$ is sufficiently large that it overcomes the flow from the ureter into the renal pelvis and there is a range of short pulse durations for which the
reflux is identically zero. As the duration increases, the fluid pulse has time to reach the renal pelvis and the reflux increases. However, for pulses of sufficiently long duration (the rate-of-pressure-increase decreasing), bladder pressure rises very slowly, and the rate of fluid secretion by the renal pelvis can generate pressures sufficient to suppress, partially, the reflux from the bladder. Hence, the degree of reflux into the renal pelvis starts to fall, the extra urine being accommodated within the ureter. If we raise the bladder pressure slowly enough, then all the fluid expelled from the bladder can be accommodated by the ureter, with none entering the renal pelvis. Again, this is due to the rate of urine secretion by the renal pelvis (which is now large relative to the rate of bladder pressure rise), which generates higher pressures within the renal pelvis sufficient to overcome the bladder pressure driving the reflux. This phenomenon of reflux suppression as the duration of Phase I is increased is not observed at all for \( q_k = 0 \). For Type II pulses (dashed lines), the reflux is maximized when the pressure rises have short duration (i.e. duration of Phase I), as there is no significant pressure rise due to urine production by the renal pelvis during this time, so most of the fluid refluxed from the bladder is able to reach the renal pelvis. Again, as the duration of the pressure rise increases, a larger quantity of fluid is secreted by the renal pelvis, leading to higher pressures there; thus more of the refluxed fluid has to be accommodated within the ureter and the total reflux falls. Note that in Fig. 10(b), where \( q_k = 0.001 \), the small \( q_k \) prediction (4.1) for the reflux for a Type II pulse agrees well with the numerical value (compare the dotted and dashed lines). The agreement is less good in Fig. 10(c), presumably due to a steady state not being achieved during Phase II in this case.

In Fig. 11, we examine the effect of stent properties on the degree of reflux. In Fig. 11(a–c), the outer stent radius, \( a_0 \), is varied while the stent wall thickness is kept fixed at 0.2. The total reflux is shown

![Fig. 11](image-url)
in Fig. 11(a) for \(q_k = 0.06\) and Fig. 11(b) for \(q_k = 0\), respectively. Figure 11(c) shows the percentage reflux through the lumen for \(q_k = 0.06\). When \(q_k = 0\) (Fig. 11b), as the outer stent radius, \(a_o\), increases, the total reflux at the ureteropelvic junction for a Type I pulse at first decreases and then increases. One possible explanation for this is that as \(a_o\) increases, the resistance of the stent–ureter system first increases and then decreases (as explained in Section 3). With a higher resistance system, for a given pressure difference between the bladder and the renal pelvis, less reflux will occur, giving rise to the observed results. For a Type II pulse, the total reflux is independent of \(a_o\), as the reflux just depends on the difference between the maximum and resting values of the reduced bladder pressure. For \(q_k = 0.06\), the total reflux is now sensitive to \(a_0\) for both types of bladder pressure pulse (Fig. 11a,c). As the outer stent radius increases, a higher percentage of fluid flows through the stent lumen (Fig. 11 c) as expected.

In Fig. 11(d), the outer stent radius is fixed at 0.5, while the inner stent radius increases (corresponding to the thickness of the stent wall decreasing). For \(q_k = 0.06\), we see that the total reflux increases as \(a_i\) increases; the stent occupies less of the total volume of the system and correspondingly the reflux increases. Varying the stent permeability has a minimal effect on the total reflux for both types of bladder pressure pulse (Fig. 11e). This agrees with the analytical solution, which also predicts that reflux is not dependent on stent permeability, \(d\), even at \(\mathcal{O}(q_k)\), see (4.2).

4.3.2 Non-uniform properties. So far we have only considered parameter values that are independent of \(z\). Now, we consider a ureter with an obstructed region \([z_1, z_2]\) in which the stent radii, \(a_i\) and \(a_o\), the stent permeability, \(d\), and the external reduced pressure, \(\tilde{p}_e\), may vary in the manner indicated in Fig. 12. Outside the interval \([z_1, z_2]\) each parameter takes its usual value, while within \([z_1 + h_1, z_2 - h_2]\) one or more parameters take a different constant value. The obstruction has end regions of lengths \(h_1\) and \(h_2\)

![Fig. 12. Sketch graph showing the parameter variation used to simulate obstructions in the ureter.](image-url)
(we use $h_1 = h_2 = \frac{z_2 - z_1}{5}$) in which the parameter varies sinusoidally over half a period as shown. Thus, the parameter and its first derivative with respect to $z$ are everywhere continuous.

Figure 13(a) shows the flow in a ureter with a region under external compression (i.e. an increased value of $\hat{p}_e$) in the resting state, indicating that there is a substantial flow through the stent walls just before and after the obstruction. Figure 13(b) shows the corresponding pressure field along the ureter, which indicates that there is a larger pressure gradient at the constriction. Note that there is a significant inflation of the ureter upstream of the compression, but little expansion downstream.

Figure 14 shows the corresponding graphs for a stent with an encrusted region, modelled by a thicker stent wall with lower permeability. Fluid flows from inside the stent to outside, around the encrustation and back inside the stent (Fig. 14a). Figure 14(b) shows that there is a larger pressure gradient across the obstruction than elsewhere in the ureter.

Figures 15 and 16 show the total reflux at the ureteropelvic junction for a Type I bladder pressure rise when the ureter is obstructed. In Fig. 15, the effect of the severity of external compression in the region $0.4 < z < 0.6$ is shown. An increase in external pressure decreases the total reflux at the ureteropelvic junction (Fig. 15a), as does an increase in the maximum encrustation thickness (Fig. 15b). In Fig. 16, the effect of the location of the obstructed region on reflux is shown. Both a region of elevated external pressure (solid curve) and also a region of encrustation (dotted–dashed curve) are considered. Figure 16(a) shows the effect of blockage width on the degree of reflux; as expected the reflux decreases as the blockage width increases (less available volume for the fluid flow). Figure 16(b) shows the effect of different obstruction locations on the total reflux for a fixed-size obstruction. The total reflux compared with that obtained in the unobstructed state decreases, and the reflux is most suppressed when the obstruction is located near to the bladder end of the ureter.

![Figure 13](image-url)

**Fig. 13.** The steady resting state in the ureter when the region $0.3 < z < 0.7$ has an increased value of $\hat{p}_e$. In $0.38 < z < 0.62$, $\hat{p}_e = 700$, and for $z < 0.3$ or $z > 0.7$, $\hat{p}_e = 0$. (a) Streamlines: solid curves; $b$: thick solid curve; $b_a$: dotted–dashed line; stent walls: dashed lines. (b) $\hat{p}_e$: solid curve; $\hat{p}_{bc}$: dashed curve. The points $z_1, z_1 + h_1, z_2 - h_2, z_2$ are indicated by dotted lines, and all parameters except $\hat{p}_e$ take values in Table 3.
FIG. 14. The steady resting state in the ureter when the region $0.3 < z < 0.7$ has a thicker stent wall and lower permeability, modelling a region of encrustation. In $0.38 < z < 0.62$, we set $a_i = 0.35$, $a_0 = 1.25$ and $d = 1$ (for $z < 0.3$ and $z > 0.7$ these parameters equal 0.65, 0.95 and 900, respectively). The parts of this figure show the same quantities as in Fig. 13, and all parameters except $a_i$, $a_0$ and $d$ have the values in Table 3.

FIG. 15. Graphs showing how an obstruction in the ureter affects the total reflux for a Type I pulse. (a) We consider a region under external pressure. In all cases, $\hat{p}_e$ is elevated in $[0.4, 0.6]$ and zero elsewhere; the $x$-axis plots the maximum value. (b) We consider a stent that is encrusted in $[0.4, 0.6]$. In the central part of this region $d = 1$, while two-thirds of the encrustation is outside and one-third inside (thus $a_i = 0.3 - x/3$, $a_0 = 0.5 + 2x/3$, where $x$ is the thickness of encrustation). All other parameters take the values in Table 3.

5. Discussion

We have developed and solved a mathematical model of a stented ureter to determine the urine flow and ureteric distension. In particular, we have investigated the degree of reflux generated when the bladder pressure rises in a prescribed manner. The numerical code enables investigation of the complex flows in the system. In the analysis presented in Section 3, we found an asymptotic solution for the steady state when the excretion rate for the kidney, $q_k$, is small, the bladder pressure is constant and the stent and ureteric wall properties are spatially uniform. Since a steady state is obtained in our simulations during
Phase II of Type II bladder pressure pulses, we were able to approximate the total reflux analytically. To leading order, the total reflux is proportional to the pressure difference between the resting (Phase 0) and elevated pressure (Phase II) states, meaning that stent design and ureteric wall properties have an effect that is smaller by a factor proportional to the net rate of urine excretion from the kidney into the renal pelvis, $q_k$.

For the cases of external compression and stent encrustation (Figs 13–16), the total reflux during Type I bladder pulses was reduced significantly. The reflux is smallest when the obstruction is situated near the bladder, which in turn suggests that the total reflux depends heavily on the functionality of the vesicoureteric junction. As described in Section 1, the ureter loses muscle tone after stent insertion and the vesicoureteric junction ceases to contract.

In our model, we neglected peristaltic contractions of the ureteric wall. There has been little research on the effects of stents on these contractions, although Payne & Ramsay (1988) found that peristaltic waves in pig ureters become much weaker after stent insertion. In addition, we have not found any research on the effect of bladder pressure rises on peristalsis in the literature, in either unstented or stented systems (in particular, we would expect there to be some effect in the stented system, where the ureter is not shielded from the bladder pressure elevation). However, as long as the peristaltic waves have a sufficiently small amplitude and wave speed, we would expect only a small effect on the total reflux. Investigating the effects of weak peristalsis on the reflux is a subject for future research.

In summary, our results suggest that to reduce reflux the priorities should be to encourage patients not to squeeze hard while voiding (so that $\hat{p}_{\text{max}}$ is not large), to increase bladder pressure slowly (so that a greater proportion of the urine expelled from the bladder is accommodated with the ureter—see Fig. 10) and to ensure that the stent dimensions are such that reflux is minimized (see Fig. 8a). To obtain a further reduction in the total reflux, a stent that either allows the vesicoureteric junction to retain better functionality or causes less bladder irritation could be used, thereby reducing the frequency and strength of bladder spasms. A possible way to achieve this is by using tail stents (Dunn et al., 2000), whose outer radius decreases to 3 F at the bladder end, thereby causing less irritation and also probably allowing better functionality of the vesicooureteric junction. Another development is the mesh stent (Olweny et al., 2000), which was developed to try to improve luminal flow and reduce bladder irritation. However, the use of these two stent types is not widespread. The research presented here lays the foundation for future research into the effects of stent design on reflux.
Acknowledgements

The authors would like to thank Prof. M. Heil, Prof. T. J. Pedley, Dr J. M. Oliver, Dr K. Heaton, Dr R. Bayston, Mr M. C. Bishop and Prof. D. M. Grant for helpful comments and discussion. We also thank Boston Scientific for providing stent samples for the research. Most of the work was carried out while all authors were employed at the University of Nottingham.

Funding

BBSRC (E20379) to J.H.S.; Royal Society Dorothy Hodgkin research fellowship (sponsored by National Grid plc) to L.J.C.; EPSRC in the form of an Advanced Research Fellowship (EP/D070635/2) to S.L.W.; EPSRC-funded Springboard Fellowship (EP/E032362/1) to J.A.D.W.

REFERENCES


