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# Urological complications following renal transplantation

## A study of 1016 consecutive transplants from a single centre

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**Abstract** A total of 1016 consecutive renal transplants performed between 1976 and 1990 were analysed retrospectively to determine the incidence of urological complications and possible predisposing factors. Some 189 episodes of ureteric obstruction and/or urinary leak occurred in 143 patients (overall incidence 14.1 %). The median annual rate of urinary leak was 5.1 %; that of ureteric obstruction was 4.5 % pre-1986 and 16.1 % post-1986. Sixty-three episodes of urinary leak occurred in 54 patients from 1 day to 3 months post-transplant and 60 % involved the distal ureter. Thirty were treated primarily by reconstructive surgery, ten required nephrectomy and three died of associated sepsis. A total of 126 episodes of ureteric obstruction occurred in 104 patients from 1 day to 12 years post-transplant and 86 % involved the distal ureter. Prior to 1986, 10/11 patients with ureteric obstruction were treated by reconstructive surgery, but since then 88 (95 %) have been treated by percutaneous nephrostomy, with or without stenting, with only one graft lost and no deaths. Children had a significantly increased incidence of ureteric obstruction ($P < 0.001$) whilst male recipients had an increased incidence of urinary leak ($P = 0.04$). More patients with ureteric obstruction than those without had two or more episodes of rejection ($P = 0.03$). No single cause for the increased incidence of ureteric obstruction since 1986 has been identified. Continued attention to technical detail and further study of this trend is warranted.

**Key words** Kidney transplantation, urolocial complications
Urological complications, kidney transplantation

## Introduction

The ultimate aim of renal transplantation is to restore normal renal function to patients with end-stage renal failure. However, the specific urological complications of urinary leak and ureteric obstruction may hinder this restoration and in certain cases lead to graft loss or patient mortality. The larger published series report an incidence ranging from 2.5 % to 13.2 % [11, 17, 21, 25, 27, 29] and this represents a significant post-transplant morbidity. The aims of this study were twofold. The first was to audit retrospectively 1016 consecutive renal transplants performed in the Newcastle upon Tyne Transplant Unit from 1976 to 1990 to determine the incidence, aetiology and management of both urinary leak and ureteric obstruction. Over the last 4 years in this unit we have been concerned about an increased incidence of ureteric obstruction following renal transplantation. The second aim of this study was to identify possible reasons for this increase.

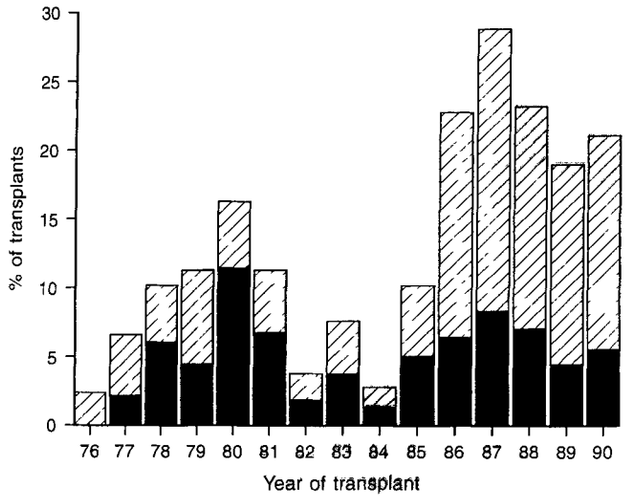


**Fig. 1** Annual incidence of urinary leak ( ■ ) and ureteric obstruction ( ▨ ) after renal transplants performed between 1976 and 1990

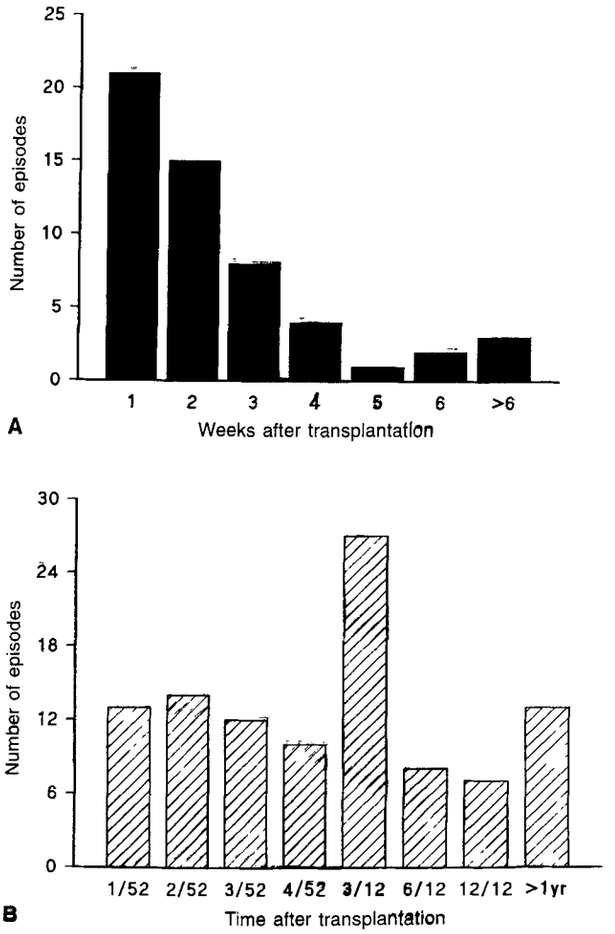


**Fig. 2 A, B** Time after transplantation for occurrence of: **A** primary urinary leaks and **B** primary ureteric obstruction

## Patients and methods

Between January 1976 and October 1990, 1016 consecutive renal transplants (911 cadaveric and 105 live donors) were performed on 597 male and 419 female patients with a mean age of 39.7 years (range 2–80 years). There were 856 first, 141 second, 18 third and 1 fourth transplant. Fifty-one transplants were performed on children (age ≤ 16 years). Follow-up ranged from 6 months to 16 years.

Two basic regimens of immunosuppression were used over the study period. From 1976 to 1985, patients received azathioprine (2 mg/kg) and low-dose prednisolone (30 mg). Since 1986 patients have received cyclosporin (initial dose of 10–15 mg/kg and thereafter kept within therapeutic range of 200–400 ng/ml). Prednisolone (25 mg) is added for those having second grafts and triple therapy for those having subsequent grafts.

After revascularization of the graft, most patients transplanted from 1976 to 1988 had a tunnelled ureteric anastomosis using a modified Leadbetter-Politano technique. From 1989 to the end of the study period, a direct pull-through technique was used. This required a cystotomy and the ureter brought directly through the bladder wall at a separate site. Then, as with the tunnelled technique, the end was spatulated and sutured to the inside of the bladder using four interrupted sutures of 4/0 chromic catgut. A two-layer bladder closure was performed using 2/0 chromic catgut and latterly using 2/0 polydioxanone. Three patients required primary uretero-ureterostomy for a short donor ureter and four implantation of the ureter into an existing ileal conduit. Only the uretero-ureterostomies were routinely splinted. Two suction drains were placed in the region of the vascular and ureteric anastomoses and removed after 24–48 h, unless drainage was high, when biochemical analysis of the fluid was determined. A Foley catheter was left in the bladder for 5–7 days postoperatively. All operations were performed by a consultant, senior registrar or research registrar experienced in renal transplantation.

In the early post-transplant period renal function was monitored by daily serum creatinine estimations. For patients with graft dysfunction, a $^{99m}$Tc diethylene triamine penta-acetetic acid (DTPA) isotope scan and ultrasonography were performed. In our experience urinary leaks within the first 14 days post-transplant were not reliably diagnosed by radiological methods but were usually evident on clinical and biochemical grounds. Later leaks were more evident with imaging. Our current treatment of choice for early urinary leak is reconstructive surgery. Ureteric obstruction was diagnosed by ultrasound demonstrating pelvicalyceal dilatation associated with an elevated serum creatinine, having excluded other causes of graft dysfunction. Since 1986 the diagnosis has been confirmed by antegrade pyelography with a subsequent Whittaker test [32] and a consequential decrease in serum creatinine. If the Whittaker test was positive, a 7 Fr double-J silicone ureteric stent was inserted across the obstruction percutaneously.

There is little doubt that technical reasons and distal ureteric ischaemia are major aetiological factors for the development of urinary leak and ureteric obstruction. Other possible contributory factors, including sex, age, source of transplant [cadaveric (local/imported) or live], number of transplant, duration of cold ischaemia and number of episodes of rejection in the 1st month were analysed. The number of rejection episodes, duration of cold ischaemia and source of cadaveric kidneys could only be analysed in those patients transplanted from 1986 to 1990 [$n$ = 451 (44.4%)]. The first two episodes of rejection were usually diagnosed clinically and subsequent episodes by histology. Statistical analysis was performed using the chi-squared test with continuity correction.

## Results

There were 189 episodes of urinary leak and/or ureteric obstruction in 143 patients, giving an incidence of 14.1%. Fifteen patients had both complications (11 synchronous and 4 separate). The median annual rate of urinary leak was 5.1% (range 0%–11.5%); that of ureteric obstruction was 4.5% pre-1986 (range 1.4%–6.8%) and 16.1% post-1986 (range 14.5%–20.5%; Fig. 1).

### Urinary leak

There were 63 episodes of urinary leak in 54 patients, giving an incidence of 5.3%. A total of 48/54 (86%) primary urinary leaks occurred in the 1st month following transplantation, with the latest occurring at 3 months (Fig. 2a). Some 32/54 (60%) urinary leaks occurred at the vesico-ureteric junction (VUJ) and distal third of the ureter, with the remainder distributed equally through the rest of the urinary tract (Table 1).

A total of 30/54 primary urinary leaks were treated by reconstructive surgery: ureteric reimplantation ($n = 20$), pelvivesicotomy ($n = 2$), Boari flap ($n = 1$), uretero-ureterostomy ($n = 2$), ileal conduit ($n = 1$), repair of bladder ($n = 3$) and resection of necrotic upper pole of kidney ($n = 1$). Some 10/54 patients were treated by percutaneous nephrostomy (PCN); (alone $n = 6$ and with subsequent stenting $n = 4$), but 8 of these had combined ureteric obstruction and leak. The two patients with leak alone who were treated by PCN both presented more than 6 weeks post-transplant and were not considered fit enough for reconstructive surgery. Both subsequently died of sepsis. Six patients from the early part of the study were treated conservatively with success, although our current policy is to manage leaks surgically. Eight patients required nephrectomy for unsalvagable pelvi-ureteral necrosis ($n = 6$) or sepsis ($n = 2$).

Seven patients had multiple episodes of urinary leak: five patients two episodes and two patients three episodes. These occurred in a similar distribution to primary leaks and were primarily corrected surgically. There was a successful outcome in five patients; two patients required nephrectomy, one of whom died of sepsis.

Graft loss as a direct result of urinary leak occurred in ten patients, all of whom required transplant nephrectomy of a functioning graft. In six of these there was total necrosis of the pelvis and ureter precluding any surgical reconstruction, three had sepsis and one patient refused an ileal conduit where other means of surgical reconstruction were not technically possible. Three patients died of sepsis resulting from urinary leak.

**Table 1** Site of primary complication. VUJ, Vesico-ureteric junction; PUJ, Pelvi-ureteric junction

| | |
|---|---|
| Urinary leak | |
| Ureter: Distal third and VUJ | 32[a] |
| Middle third | 1 |
| Total pelvis and ureter | 8 |
| Uretero-ureterostomy | 3 |
| Kidney | 3 |
| Bladder | 6[a] |
| Not known | 3 |
| Ureteric obstruction | |
| Ureter: Distal third and VUJ | 89 |
| Middle third | 10 |
| PUJ | 4 |
| Uretero-ureterostomy | 1 |

[a] Including two bladder and VUJ combined

### Ureteric obstruction

There were 126 episodes of ureteric obstruction in 104 patients, giving an incidence of 10.2%. A total of 49/104 (47%) primary episodes arose in the 1st month post-transplant with 76/104 (73%) occurring by 3 months (Fig. 2b). Thirteen episodes of obstruction were diagnosed more than 1 year following transplantation with one occurring at 12 years. Some 89/104 primary episodes occurred at the VUJ and distal third of the ureter with the rest distributed equally throughout the urinary tract (Table 1).

Prior to 1986 only 11 patients were diagnosed to be suffering from ureteric obstruction; 10 were treated surgically (reimplantation of ureter $n = 6$, transvesical procedures $n = 3$, uretero-ureterostomy $n = 1$) and one conservatively. The four patients treated by a transvesical approach or conservatively developed further ureteric obstruction (see below). In 1986 new radiological facilities became available to us for the diagnosis and treatment of ureteric obstruction. Since then 88/93 patients (95%) have been treated with PCN alone ($n = 14$) or with subsequent stenting ($n = 74$). The remaining five were treated by ureteric reimplantation ($n = 3$), operative placement of stent ($n = 1$) and drainage of lymphocoele ($n = 1$). Stenting failed in seven patients because of technical difficulties in placing or replacing the stent. These patients were successfully managed surgically (pelvi-vesicotomy $n = 4$, reimplantation of ureter $n = 1$, uretero-ureterostomy $n = 1$), although one required nephrectomy as he refused an ileal conduit.

Nineteen patients had multiple episodes of ureteric obstruction: 16 patients two episodes and 3 patients three episodes. Of these patients, 4/19 presented before 1986 and 3 had an initial transvesical procedure (VUJ "sphincteroplasty" and stent $n = 2$, resection of ureteric orifice $n = 1$) and 1 was managed conservatively. Of these 4, 3 were successfully managed surgically and 1 by PCN and stenting. The remaining 15 patients have presented since 1986; 7 were initially treated by PCN alone, 7 by PCN and stenting, and 1 patient whose ureter was divided during drainage of a lymphocoele by end-to-end ureteric anasto-

**Table 2** Summary of donor and recipient variables with relationship to urinary leak and ureteric obstruction

| | Age (years) | | Sex | | Source | | | | Number | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | ≤ 16 | > 16 | Male | Female | Cadaver | Live | Local | Imported | 1 | 2 | 3/4 |
| No leak | 48 | 914 | 557 | 404 | 867 | 95 | 287 | 154 | 813 | 131 | 18 |
| Leak | 3 | 51 | 40 | 15 | 44 | 10 | 21 | 9 | 43 | 10 | 1 |
| | $\chi^2 = 0.02$ | $P = 0.9$ | $\chi^2 = 4.68$ | $P = 0.04$ | $\chi^2 = 3.24$ | $P = 0.07$ | $\chi^2 = 0.12$ | $P = 0.73$ | $\chi^2 = 0.42$ | $P = 0.66$ | |
| No obstruction | 36 | 876 | 528 | 384 | 820 | 92 | 258 | 140 | 764 | 130 | 18 |
| Obstruction | 15 | 89 | 69 | 35 | 91 | 13 | 50 | 23 | 92 | 11 | 1 |
| | $\chi^2 = 19.35$ | $P < 0.001$ | $\chi^2 = 2.41$ | $P = 0.12$ | $\chi^2 = 0.35$ | $P = 0.55$ | $\chi^2 = 0.22$ | $P = 0.64$ | $\chi^2 = 0.84$ | $P = 0.36$ | $P = 0.36$ |

mosis. All seven patients treated by PCN alone had a subsequently successful PCN with stenting. Of the seven with initial PCN and stenting, four required a further PCN and stent insertion whilst three required subsequent pelvivesicotomy because the stent could not be replaced. The patient with a lymphocoele developed a stricture at the ureteric anastomosis and required a uretero-ureterostomy. Only one graft has been lost as a direct result of ureteric obstruction and that in the patient who refused an ileal conduit; there have been no deaths.

### Synchronous urinary leak and ureteric obstruction

Eleven patients had synchronous complications. Six occurred at the VUJ within 2 weeks of transplantation. These were managed surgically by reimplantation of the ureter ($n = 2$), PCN alone ($n = 2$) and PCN and stenting ($n = 2$). Two of those treated interventionally required subsequent surgery (ureteric reimplantation $n = 1$, pelvivesicotomy $n = 1$) for a recurrent complication. Two patients had VUJ obstruction and developed an iatrogenic urinary leak around the PCN that settled spontaneously within 1 week. Two further patients developed upper pole calyceal leaks secondary to VUJ and middle third ureteric obstruction, respectively, and one patient had a synchronous leak and obstruction at the primary uretero-ureterostomy.

### Possible aetiological factors

The influence of recipient age, sex and source of kidney is summarised in Table 2. Children ( ≤ 16 years old) had a significantly increased incidence of ureteric obstruction whilst male recipients had a significantly increased incidence of urinary leak. The number of transplant and source of the kidney had no effect. Patients with ureteric obstruction who were transplanted from 1986 to 1990 were more likely to have had two or more episodes of rejection when compared to those without obstruction (38/177 vs 36/274 with less than two episodes $\chi^2 = 4.85$, $P = 0.03$). Patients with urinary leak showed no such relationship. Eighty-seven percent of complications occurred in only one kidney of the retrieved pair. The mean cold ischaemia time for patients with urological complications was 1262 min (SD 492 min) compared to 1272 min (SD 438 min) for those without.

## Discussion

Urological complications have resulted in significant patient morbidity and mortality since the inception of renal transplantation. Many transplant centres have reported their experience, with the incidence of complications ranging from 0.9% to 29.6% [17], although patient numbers are often small and few studies are contemporary [2, 15, 18, 31]. This present series is one of the largest reported and our incidence of 14.1% compares favourably with the 13.2% reported by the Boston group ($n = 718$) [17] and the 12.5% by the SE and SW Thames group ($n = 1000$) [21]. It is, however, higher than the 7% reported by the Oxford group ($n = 600$) [11] and the 2.5% by the Brooklyn group ($n = 1097$) [29] in the more contemporary series. Our complication rate of 14.1% is high because of the marked increase in episodes of ureteric obstruction seen since 1986. Most of these authors have a similar message. Firstly, great care should be taken to preserve the ureteric vasculature during kidney retrieval and implantation. Secondly, high-dose steroids as used in the pre-cyclosporin era are associated with an increased incidence of urinary leak and ureteric obstruction. Lastly, early diagnosis and management are essential to minimize the morbidity and mortality.

The two major aetiological factors responsible for urinary leak and early ureteric obstruction are technical factors and ureteric ischaemia. Technical factors include poor techniques of organ retrieval and ureteric implantation; damage to accessory renal arteries; leaving the ureter too long, too short or twisted and an inadequate bladder closure. Ureteric ischaemia may also be due to technical factors and it is the distal ureter that is most vulnerable due to its precarious blood supply. In situ the ureter receives its blood supply from the renal, aortic, gonadal, common iliac and vesical arteries [33]. Following harvesting of the kidney the ureter receives its entire blood supply from the ureteric branch of the renal artery.

Any damage to this by traction, stripping or diathermy during harvesting, bench preparation or implantation is liable to render the ureter, in particular the distal portion, ischaemic. Other aetiological factors are also responsible for specific urological complications. Acute allograft rejection involves the kidney and ureter equally [8, 23, 24] and the resultant oedema with or without ischaemia may lead to ureteric obstruction. Our data has shown that more patients with ureteric obstruction had two or more episodes of acute rejection. Several patients developed marked ureteric dilatation associated with acute rejection and the dilatation settled with anti-rejection therapy, suggesting ureteric oedema. Furthermore, the sequelae of vascular damage due to rejection is fibrosis and this may result in later ureteric obstruction [27]. Rejection should therefore be promptly recognised and treated. Our data and that of others [10] suggest that rejection is not implicated in urinary leak, although severe ureteral rejection may lead to ureteric necrosis [23]. Late obstruction can occur more than 1 year post-transplant and this can be due to fibrous encasement of the ureter [14]. Other aetiological factors for obstruction include external compression from lymphocoele, spermatic cord, abscess or haematoma; intraluminal blockage from clots, stones or tumours; and classical pelvi-ureteric junction (PUJ) obstruction [11, 21, 27].

Donor and recipient variables have also been examined in this and other studies. Children under 16 years of age had a high incidence (30%) of ureteric obstruction and all occurred in the distal third of the ureter and VUJ. Most children in our centre receive a donor kidney from another child and the ureter will be of small calibre, making technical problems more likely. Male recipients in this study had an increased incidence of urinary leak. The bladders of anuric males, in particular, are often shrunken and the technical difficulty of implanting the ureter in these patients could account for the increased incidence. Other studies have failed to show that age or sex affect the incidence, although children have not specifically been studied [10, 21]. Second, third or fourth transplants were not associated with an increased incidence of urological complications in this or other studies [11, 29], suggesting that immunological or technical factors alone are not responsible. This study has shown that urinary leaks alone tend to be increased, although not statistically so, in live donor compared to cadaveric transplants and this has been found in other series [10]. One series [17] has found both leaks and obstruction to be increased in live donor transplants, whilst others have shown no relationship [21, 29]. Careful hilar dissection and removal of an adequate length of ureter with an adventitial cuff should decrease the incidence of these complications in live donor transplants.

Ultrasound has now become our first line investigation for patients with suspected ureteric obstruction. Its use is widely documented [1, 9, 13, 19]. The presence of mild pelvi-calyceal dilatation is a normal finding in 11%–31% of transplant kidneys [12, 28]. Only if this progresses or if moderate/severe dilatation is present associated with a rise in serum creatinine can obstruction be diagnosed. Prior to 1986 all episodes of obstruction required surgical treatment and this is in keeping with the other large series. This surgery is difficult but consequent graft loss is minimal [11, 17, 21]. The use of interventional radiology has revolutionised the treatment of ureteric obstruction. A total of 88/93 (95%) of our patients with conclusive ureteric obstruction on ultrasound have been treated by PCN, with 81 (74 primary and 7 recurrent obstructions) having subsequent percutaneous stent insertion with or without balloon dilatation of the stricture. Our current policy is to change the stent every 3 months because of the risk of encrustation and blockage or before if indicated, and then to remove it if and when the obstruction resolves. Whether this technique is curative or merely a holding measure remains to be answered. A Whittaker test is now always performed prior to stent insertion to show the functional significance of the antegrade pyelography findings. A number of patients with obstruction diagnosed by ultrasound and antegrade pyelography have gone on to have a negative Whittaker test and have required to further treatment. Only 7/81 (9%) required subsequent revisional surgery because of failure to insert or replace the stent. Detailed follow-up data for stented patients is currently being evaluated and is beyond the remit of this paper. These procedures are now in widespread use [3, 4, 16, 29] and have generally obviated the need for primary revisional surgery.

In our experience, the diagnosis of urinary leak is often more difficult. In the first 2 weeks post-transplant the diagnosis has usually been made on clinical and biochemical grounds. Imaging techniques have often not localized the leak and simply delayed surgical treatment, although this is contrary to other reported experience [3, 26]. Currently, patients developing unexplained graft pain without evidence of infection or rejection are usually explored on suspicion of a leak. Corroborative biochemical evidence may be available. To date this policy of early intervention has not yielded any false-positive diagnoses. Those leaks presenting later were more reliably diagnosed by ultrasound, intravenous urogram (IVU), antegrade pyelography and cystography. We feel early surgical intervention is required for urinary leaks to minimise the risk of sepsis and graft and patient mortality. Reimplantation of the ureter with stenting or another reconstructive procedure is our treatment of choice. Conservative treatment has been advocated by some [5] but is rarely practised by us, except for some bladder leaks treated by bladder catheterisation [2, 6]. Other centres report some successful results using PCN and stenting for urinary leaks [3, 16], although the duration of treatment is prolonged. Our experience and that of others [16, 22] suggests tha interventional techniques should be used in those patients unfit or being prepared for surgery.

Why has there been a marked increase in ureteric obstruction since 1986? There may be three reasons. Firstly, technical factors should be considered. In our centre machine preservation ceased in 1979 and in situ cold perfusion with Marshall's solution has been used since. Our technique of kidney retrieval has not altered; in particular great care is taken to dissect the ureter with a large cuff of adventitia. Organ retrieval has been performed by a succession of experienced registrars and senior registrars who rotate through the unit every 2 years. A learning curve for each does not appear to account for the increased incidence of complications. The incidence of complications is no different in those kidneys that we ourselves retrieve locally and in those imported from other centres. The incidence of ureteric obstruction has risen since 1986 for all grades of recipient surgeon, including consultants who had used the same techniques over many years. If technical factors alone were responsible, one might expect the incidence of urinary leak to have also increased. This was not so. A modified Leadbetter-Politano technique was used for the ureteric anastomosis until 1989, since when a direct pull-through technique has been employed. Many centres now perform an extravesical ureteroneocystotomy. The only one of the large series [11, 17, 21, 25, 27, 29] to have regularly used this technique is the Brooklyn group, who have the lowest incidence of complications (2.5%) [29]. Other smaller studies have shown that there are fewer urological complications associated with an extravesical technique than with the Leadbetter-Politano technique [25, 30]. Secondly, new facilities for the diagnosis and management of ureteric obstruction became available in 1986, and it may be that the problem was previously under-recognised. IVU and latterly ultrasound had been the investigations of choice with their attendant limitations. Since 1986 it has been possible to perform PCN and use antegrade pyelography and Whittaker testing to confirm or refute obstruction. Fourteen patients with ureteric obstruction were treated by PCN alone without the need for stenting. It could be argued that these patients had false-positive diagnoses. However, seven went on to develop further obstruction requiring stent insertion, two required the PCN for post-biopsy clot obstruction and the remaining five had an associated decrease in serum creatinine. Thirdly, cyclosporin was introduced as first line immunosuppression in our centre in 1986. Cyclosporin is known to produce an arteriolopathy in the kidney, resulting in narrowing of arterioles and small arteries [7, 20]. It is possible that this process also involves the transplant ureter, resulting in ischaemia, fibrosis and consequent obstruction. Since most ureteric obstruction is now treated percutaneously, there is a dearth of histological material to test this hypothesis. Other series have not noted an effect with cyclosporin [11, 29].

In conclusion, meticulous attention to technical detail is imperative during kidney retrieval, bench preparation and implantation to preserve ureteric vasculature. The distal ureter is most vulnerable. Only in this way can the incidence of urinary leak and ureteric obstruction be kept to a minimum. Particular care should be taken in those groups at greater risk; live donor transplant, male and child recipients and those in whom multiple renal arteries are present. Early aggressive diagnosis and management of urological complications should be undertaken to minimise the morbidity and mortality of both graft and patient. Urinary leak should be managed surgically where possible by reconstructive surgery. Exploration of the graft on clinical suspicion alone may be required in the early post-transplant period where imaging techniques are equivocal. Ureteric obstruction can be managed by PCN and subsequent stenting where the necessary facilities and expertise are available. No definite cause for our recent increase in ureteric obstruction has been identified and this demands further investigation. We are currently conducting a prospective randomised trial comparing external ureteroneocystotomy with the direct pull-through anastomosis and with or without a prophylactic ureteric stent for 3 months. This study may show us the way forward for reducing the incidence of ureteric obstruction.

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