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INVITED EDITORIAL

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Progress in urodynamic research on the upper urinary tract: implications for practical urology

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Abstract The development of new surgical techniques for bladder substitution and continent urinary diversion has extended interest in urodynamics of the upper urinary tract. From a subdiscipline attracting mainly scientists and bioengineers, renal pelvic kinetics and ureteral peristalsis have evolved as important factors in routine clinical urology. The observed changes in peristaltic pattern during high diuresis, obstruction and urinary reflux have influenced management of stone disease and neurogenic bladder. The demonstration that high intravesical pressure is reflected to the kidney not only when the ureteric orifice is incompetent, but also during high diuresis, established the necessity for low pressures in neobladders. Much further clarification of urinary transport from the renal tubules to the bladder should be achievable by refined techniques of fluoroscopy, isotopic renography and manometry.

Key words Urodynamics · Upper urinary tract · Ureteric peristalsis · Ureteric reflux · Renal pelvic pressure

Introduction

In 1957 Fredrik Kiil [16] published a remarkably elaborate study of urinary transit from the renal pelvis to the bladder, the pressure required and the pattern of ureteric peristalsis. Using thin fluid-filled catheters, he demonstrated contractions in the ureters with a frequency ranging from one every 4th min to 10 times/min and propulsion of the urine to the bladder with varying pressure amplitudes, up to 100 cm H_2O. The peristalsis was irregular and without correlation between left and right sides. Lutzeyer and Melchior [21] later tried to measure ureteric pressure and flow simultaneously by means of specially constructed catheters, using a thermoconducting technique and measuring heat losses. Whitaker's [36] work on pelvic manometry in paediatric hydronephrosis in the 1970s evoked increased interest in the urodynamics of the upper urinary tract, and since then understanding of the physiology and pathophysiology of the ureter and renal pelvis has expanded. Griffiths [11] demonstrated the basic patterns of flow and pressure during passage through distensible tubes undergoing active peristalsis. Constantinou [5] studied pelvic pacemaker control of ureteric peristalsis, and in experiments on pigs Djurhuus et al. [8] and Griffiths et al. [12], measuring the pressure elicited in different calyces during contractions, found interesting disruption of the peristaltic rate at the pyeloureteric junction in hydronephrosis, which hindered the urinary transport.

Progress in urodynamics took place in parallel with advances in clinical urology, and there was a lively interchange of ideas between scientists and clinicians. Around 1980 the accepted treatment of urinary calculi changed from open surgery with huge incisions and a long hospital stay, first to percutaneous lithotripsy and later to extracorporeal shock wave lithotripsy (ESWL) performed as an out-patient procedure. Percutaneous puncture became accepted as affording easy and generally uncomplicated access to the renal pelvis. Ureteric stents were evolved to ensure transit of urine from kidney to bladder, even for long-term use in the presence of ureteric stone. Techniques for continent urinary diversion greatly improved the quality of life after cystectomy for advanced bladder cancer. Augmentation cystoplasty and construction of substitute bladders were increasingly performed in patients with urinary incontinence due to neurological disease. New principles were introduced for relief of ureteric colicky pain. Foetal hydronephrosis could often be disclosed by routine sonography in pregnant women.

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To what extent has the increased understanding of upper urinary tract urodynamics influenced clinical urology as regards indications and choice of treatments? A summary of the main results of the relevant research may provide an appropriate background to some personal reflections on these matters.

Research results

Urine transport and ureteric peristalsis

Early videographic studies showed that urine is propelled by contractions which arise in one of the fornices, spread out over the pelvis, pass the pyeloureteral junction and continue along the ureter [19]. The process generally starts in one of the upper calices, but after two or three contractions another calyx often takes over and initiates the next contraction. Sometimes, especially in dehydration, the interval between contractions may be some minutes, but usually the frequency is two to five contractions per minute. The basal pressure in the renal pelvis is normally below 10 cmH_2O. A pressure rise occurs during high diuresis, but the pressure does not normally exceed 15–20 cmH_2O. When a contraction runs along the pelvis, its amplitudes are higher in a small than in a wide pelvis and generally attain 2–5 cmH_2O [1, 16].

A much-discussed question is whether the start of contractions is simply an effect of pelvic wall distension or is due to depolarization of an action potential at a pacemaker site in the pelvis [33]. In the latter case, the electrical activity could propagate distally and evoke a contraction wave along the ureter. One theory maintains that the contraction is wholly myogenic, without influence from the autonomic nervous systems [2]. Observations that peristalsis occurs in normal fashion also in the ureter of a transplanted kidney, over a ureteric anastomosis and even in a reversed ureteric segment, support the theory that overall nervous regulation is not necessary [25].

On electron microscopy of the ureteral wall, Notely [24] noted sites of intimate contact between the membranes of adjacent muscle cells at which conduction of electrical impulses and myogenic activity could be facilitated. With better staining techniques, Gosling and Dixon [10] found large, cytoplasm-rich cells lining the forniceal wall close to the papillae, and proposed that these cells trigger the onset of peristalsis.

Any part of the ureter, and also the ureterovesical orifice, however, seems capable of pacemaker action. If a ureteric site is irritated by a calculus or pinched by a forceps at surgery, a contraction often arises. In the presence of nephrostomy, retrograde peristalsis from the bladder to the renal pelvis is common.

Both cholinergic and adrenergic nerves are present in the upper urinary tract. α-Adrenergic nerves dominate in the renal pelvis, but become fewer at more distal sites, so that in the ureter cholinergic nerves are more common [32]. The frequency-stimulating action of α-adrenergic drugs on ureteric contractions and the relaxing action of β-adrenergic agonists are well known [13].

Even if peristalsis can be evoked without neurostimulation, the neurotransmitters may affect the amplitude of the contractions. Another proposed alternative is that α-adrenergic nerve terminals act as superregulators only in situations with disorganized or very slow peristalsis.

The urine is propelled downwards, driven in front of the contracion wave along the pelvis and out in its calyces (Fig. 1). The contraction wholly empties the ureter and causes collapse of its walls during the transit to the bladder. As the contraction speed is around 2 cm/s, 13–15 s is required for passage of a wave from the kidney to the ureteric orifice [26, 27]. The front of the urine column, however, travels at much greater speed and starts as soon as the contraction begins its passage over the pelvis. If the ureter is unobstructed, the urine quickly passes down to the orifice, often with some retardation at the site of crossing of the iliac vessels [27]. Formerly, each urine fraction was described as a bolus but, because of its very rapidly progressing front, urine propagation differs from the oesophageal bolus pattern. At low urinary output, the urine fractions are small and each can be observed on fluoroscopy as a thin, needle-shaped contrast column. At high diuresis each urine bolus is longer and wider.

This peristaltic pattern is typical when the rate of urine production is normal. The ureter responds to increased output mainly with augmented volume in each urinary fraction [12]. Only at the start of moderately increased excretion is there a temporary rise in peristaltic frequency, maximally up to 12 waves/min. At that frequency two separate contraction waves can travel simultaneously along the pelvis and ureter. With still greater diuresis the peristaltic frequency falls again and the urine fractions are both longer and wider. At output exceeding ca. 2 ml/min a complete contraction coapting the ureteric walls would cause partial obstruction, but such coapting is prevented by the urine pressure. As a result, "leaky boluses" form. When human diuresis exceeds ca. 9 ml/min, fluoroscopic detection of the contractions becomes difficult. That contractions still occur was proved by Pohl et al. [30], using specially sensitive catheters to measure pressure and force. These contractions, however, are shallow and the ureter functions as an open tube. Cystoscopic viewing shows a continuous stream of urine from the orifice. With the concept of the ureter as an open tube at high diuresis, there is obviously a close connection between vesical and ureteric mechanical activity under these conditions. This revelation may be the most important result of urodynamic studies of the upper urinary tract, with implications for vesicoureteral reflux, neurogenic bladder and reconstructive bladder surgery.

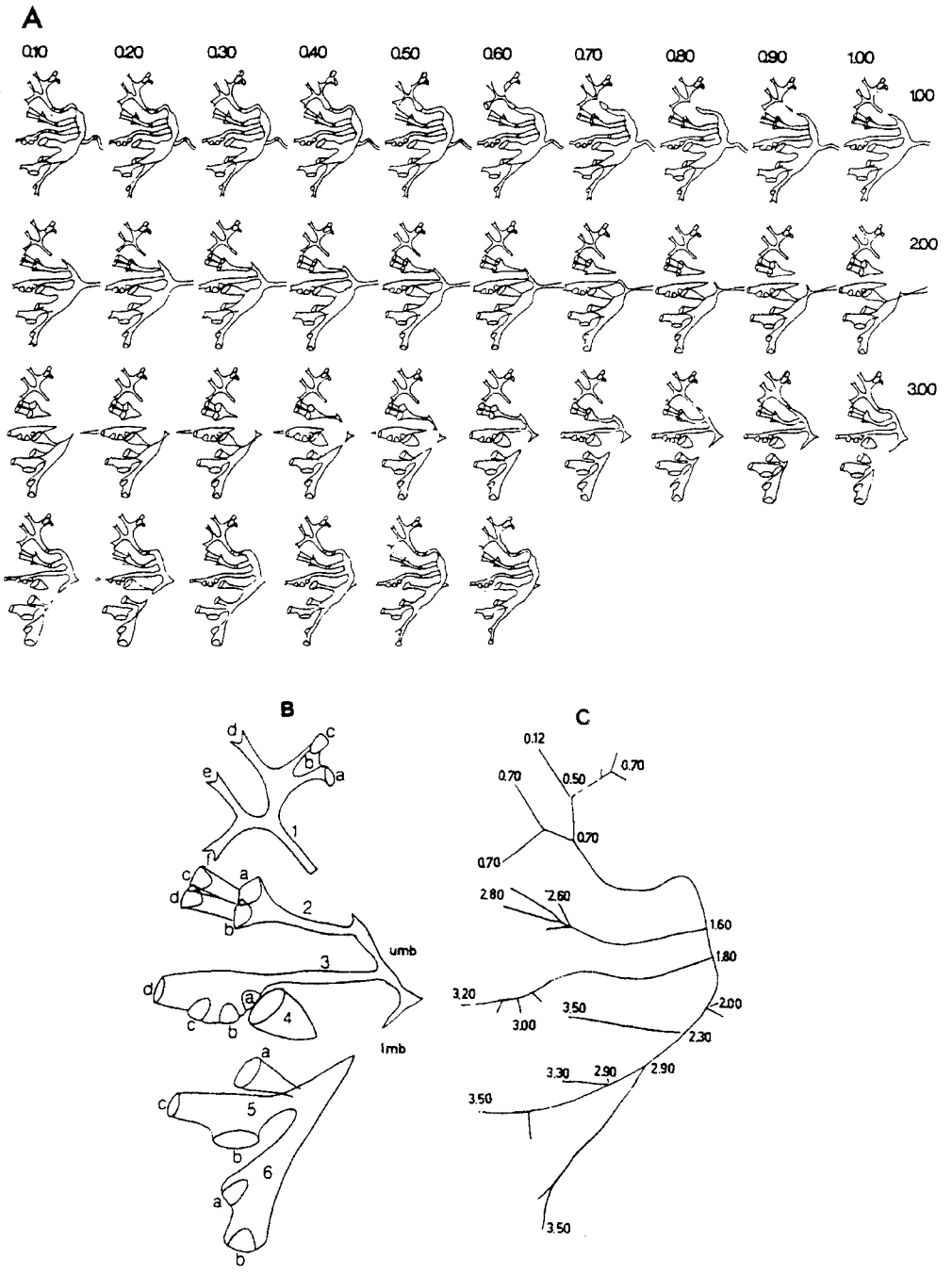

Fig. 1 **A** Kinetic urography of a normal right pyelocalyceal system in the upright position (frontal projection). Contraction interval 8.5 s, urinary output 0.3 ml/min per kidney. Scale 1:4. Interval between reproduced pictures 0.10 s. **B** The same as in **A** at 2.60 s. Calyceal branches identified by *arabic numerals* and papillae by *lowercase letters*. **C** Same as in **A**, showing time (s) for progress of the contraction wave. Contraction starts in fornix *1d* at 0.12 s. When the contraction completely closes the branch, the urine in this calyx is pushed into adjoining calyces. When the contraction at 0.70 s enters the confluence of the calyces, calyx *1d* opens up and fills again. After 1.20 s the contraction extends into branch 2 and after 1.30 s also into branch 3. After 3.60 s the contraction is completed and urine is again distributed in the calyces as at 0.10 s [from Ohlson L (1985) Kinetic urography. See Ref. 26]

Abnormalities of transport and peristalsis

A hierarchic system of renal pelvic pacemakers was proposed by Constantinou [5] and Djurhuus et al. [8], who found that the contraction frequency was highest in the peripheral calyces. In porcine experiments, the ureteric peristaltic rate was normally lower, but there was always integration between calyceal and pelvic contractions. In idiopathic hydronephrosis, on the other hand, the normally regular propagation of contractions became distorted or was interrupted and sometimes even extinguished at the junction, causing functional obstruction. Other investigators, however, found evidence of retrograde peristalsis on fluoroscopy in patients with hydronephrosis [17]. Some of the contractions were inhibited at the obstructed junction and instead reflected backwards to the calyces. The efficacy of urinary transport was impaired by both the narrowness of the upper ureter and the disordered contractions when an antegrade wave interfered with a retrograde wave.

The horseshoe-shaped arrangement of muscle fibres around the ureteric orifice was previously regarded as an effective closure mechanism in normal ureters,

whereas in reflux the open orifices transmitted the raised intravesical pressure to the pelvis during micturition. In infravesical obstruction, e.g. benign prostatic hyperplasia, and in neurogenic bladder with hyperactive detrusor, it was recognized that high intravesical pressure could inhibit urinary flow, with fatal uraemia as a possible outcome.

The mechanism of closure, however, has been incompletely understood. Formerly it was thought that rigidity of the bladder wall could cause a functional obstruction and that intravesical pressure exceeding 40 cmH_2O would counteract the net filtration pressure of the kidneys [20]. Elucidation of ureteric peristalsis has shown that, when diuresis is sufficient to transform the ureter to an open tube, the bladder pressure is always transmitted to the renal pelvis whether or not there is reflux [14]. This evidence has intensified efforts to relieve high bladder pressure by active treatment, reducing the risk of prolonged high-pressure transmission to the kidneys. In the absence of reflux, the ureter is empty for most of the time at low urinary excretion and the renal pelvis is not exposed to pressure changes in the bladder. If reflux is present, the backward filling of the ureter can transmit high bladder pressure to the renal pelvis. The implications are that children with vesicoureteric reflux during the storage phase are at greater risk than those with reflux only during voiding and that slow-acting diuretics should be preferred to fast-acting diuretics in cases of neurogenic bladder with overactive detrusor.

The above-mentioned studies [14] further demonstrated that retrograde peristalsis in the ureter is pathological. If retrograde peristalsis starts in the ureter itself, it usually indicates presence of stricture or stone; if it arises at the orifice, detrusor hyperactivity and possibly intravesical hypertension can be suspected. Fluoroscopic observation of ureteric peristalsis can show whether or not a calculus causes obstruction, and permits early detection of partial intraureteric stenosis or compression by neighbouring retroperitoneal tissues. In the case of a stone, the characteristics of the peristaltic wave – weak, absent or retrograde – and impairment of urinary transport can indicate whether a ureter that appears wide on a urogram is in fact obstructed.

Clinical implications

The main clinical implications of urodynamic observations relate to hydronephrosis, vesicoureteric reflux, calculus disease and neobladders.

Hydronephrosis

The difficulty of judging from urography whether or not widening of the renal pelvis is caused by obstruction has long prompted a search for better diagnostic methods. In children with megaureters, the ureteric pressure was measured from the opened bladder in order to identify those with obstruction who required reimplantation in the bladder [1, 36]. The hydrostatic pressure within the renal pelvis in cases with long-standing hydronephrosis is low and indistinguishable from those found in normal pelvis [16]. Renal pelvic manometry in hydronephrotic children, performed with the percutaneous technique during infusion of saline solution, was reported by Whitaker in 1975 [37]. In many children with dilated pelvis, the intrapelvic pressure rose in response to increasingly rapid pelvic infusion. The hypothesis was that obstruction at the pyeloureteric junction could be demonstrated if the pressure difference between renal pelvis and bladder exceeded 22 cmH_2O when the infusion rate was 10 ml/min, but if this difference remained less than 15 cmH_2O there was no obstruction.

Although criticized, Whitaker's method attracted much clinical interest. The report was published at a time when radiologists had begun to undertake routine nephrostomies, a circumstance that favoured adoption of the method. It still has some practical use, but has been largely supplanted by modern techniques of isotope renography.

One criticism of Whitaker's technique was founded on observations of considerable hysteresis in the pelvic and ureteric wall [6]. Hysteresis is a well-known phenomenon in the bladder, where the pressures are not equivalent in filling and in emptying at the same intravesical volumes. Distension of a large renal pelvis requires a large infused volume, but, once overdistension has occurred, a still greater volume is required to achieve the same pressure as before. In a large pelvis equilibrium is relatively easily reached, but a child's small kidney is more vulnerable to obstruction [18]. Hysteresis seems to protect the kidney and reduce the high pressure in obstruction. The phenomenon also makes comparison of pressures difficult, as the pressure can vary at different times in the same patient, even if urinary output is constant.

The number of patients with renal pelvic pressure in Whitaker's intermediate zone (15–22 cmH_2O above bladder pressure) is substantial. Furthermore, some authors did not find statistically significant differences, even during infusion of fluid, between pressures in normal pelvis and in those with urographically suspect hydronephrosis. Bratt et al. [4] questioned whether an obstructed kidney could filter an amount of urine equivalent to the volume recommended by Whitaker for infusion, and suggested instead a high dose of fast-acting diuretic intravenously in order to stress the kidney with its own maximal urine production.

With the brilliant work of O'Reilly et al. [28] combining injection of radioactive iodohippuran and furosemide in patients with dilated renal pelvis and slow drainage of urine, it became possible to determine the

renal function from the ascending limb of the curve simultaneously with the transport capacity from the slope of the descending limb. When results from diuretic renography were compared with pelvic pressure recordings, an obvious difference was found in about one-third of the patients, with one method indicating obstruction and the other not. The pioneers of both methods tried to explain the reasons for the differences [15, 28, 31, 38, 39]. In patients with greatly impaired renal function, diuretic renography could be expected to give a false-negative response. Location of the pelvis and correct positioning of the collimators on the patient's back at renography could be difficult and give misleading results. In a very large pelvis the result could be falsely negative if the volume of infused fluid was less than the renal pelvic capcity. Mixture of the infused fluid with contrast medium and fluoroscopic control of the procedure were therefore recommended. Further, if the infused volume was much larger than possible output from the kidney, the renal pressure could be artificially elevated.

The intense interest in hydronephrosis since the 1970s has prompted studies of the condition's natural history. With repeated renography and chrome-ethylenediaminetetra-acetic acid (EDTA) clearance tests, separate renal filtration function could be reliably monitored for years. Several follow-up studies showed that idiopathic hydronephrosis as a rule does not progress to renal failure. These findings have particularly encouraged paediatric urologists to switch to a more conservative attitude toward surgical treatment during infancy. The previously heated debate on the use of intrauterine pyeloplasty has ceased.

It is known, however, that a "silent" kidney with radiological signs of obstructed pyeloureteric junction is occasionally found in a patient without much history of pain. Pyeloplasty therefore should not be reserved only for patients with recurrent pain, bouts of acute pyelonephritis or complicating stone. Diuretic renography, being non-invasive, is an excellent primary diagnostic test of transit capacity over the pyeloureteric junction. Intravenous pyelography performed during acute pain, which most patients experience, may still be sufficiently informative, with additional manometry reserved for patients with doubtful symptoms. Colour Doppler sonographic evaluation of urine bolus volume, dynamics and shape on emergence at the ureteric orifice may prove useful in future [7, 35]. The asymptomatic dilatation of the upper urinary tract often unexpectedly found on abdominal sonographic screening should probably be managed with regular surveillance for a few years, provided that the ipsilateral function is good.

Vesicouretric reflux

Intensified interest in the ureter's anatomy and its distal tunnelling through the bladder wall is exemplified by pressure profiles for studying the tightness of the orifice [3]. A method for detecting retrograde peristalsis in reflux while restricting radiation exposure in children, evolved by Müller-Schauenberg [22], uses fast-frame views of radioisotope activity in different parts of the ureter to map the direction and magnitude of urine fractions. With another technique for study of vesicoureteric reflux [9], the refluxed volume is determined from radioactivity count over ureters and kidneys after infusion of technetium isotope into the bladder during cystometry. The correlation between bladder volume and pressure at reflux is continuously recorded. These urodynamic techniques, though not yet in common use, can be valuable in surveillance of children with low-grade, uncomplicated reflux. They can also be useful in patients with urinary diversion via intestinal loops, to check the reflux-preventing efficacy of ureteric anastomoses.

Indwelling stents are often inserted to relieve ureteric obstruction, mostly caused by malignant tumour or irradiation. A stent may also be used after reimplantation to the bladder, while awaiting spontaneous migration of a stone or between ESWL sessions. Patients with ureteric stent not uncommonly complain of accentuated micturition urge and flank pain. As the numerous holes in the stent allow continuous urine transit and the sphincter function of the orifice is disrupted, voiding and straining cause reflux to the renal pelvis. To prevent reflux and concomitant pain, it may be advantageous to use a stent fitted with silicone flaps distally.

Calculus disease

In acute ureteric colic the obstructed ureter dilates and fluoroscopic visualization of peristalsis is impeded by reduced amplitude of the contractions. The pain was formerly thought to be caused by intensive contractions, which could help to propel the stone towards the bladder. Work in the past 20 years, however, has shown that the cause of pain is increased tension in the pelvic and ureteric walls. The essential aim of treatment in acute pain thus should be to relieve this hypertension by means of drugs or insertion of catheters. A flexible "pigtail" catheter provided with multiple drainage holes in its curved tail is generally well tolerated, if it is not too wide. Studies in animals showed that catheters wider than 4 Ch considerably disturb peristalsis [29], and it may be advisable to use finer stents in patients with stones small enough for spontaneous transit, since elimination of the contractions may inhibit stone passage. For larger stones which will later require ESWL or sonographic or laser fragmentation, a wider stent is advisable to guarantee passage of urine.

The pharmacological management of ureteric colic has also been influenced by the recognition of high mural tension as the cause. The increased synthesis of prostaglandins that occurs in acute renal obstruction

can be counteracted by prostaglandin inhibitors, whereby high intrapelvic pressure is lowered by reduction of urinary output [33].

Bladder substitutes and continent diversion

Close collaboration between surgical pioneers in reconstructive surgery and urologists with sound basic knowledge of urodynamics has been a prerequisite for the construction of new and well-functioning bladder substitutes or devising methods of continent diversion. The caecal or sigmoid loops previously used were found to generate high intraluminal pressure because of circular contractions in the muscular wall, and cause renal deterioration. With improved understanding of the urinary tract as a communicating system involving risk of pressure propagation from intestinal conduits to kidneys, techniques for diversion were refined. The bowel was split open in flat patches and folded perpendicular to the direction of the muscle fibres. This procedure damps the peristaltic waves, impedes reflux and reduces the pressure in the conduit, thereby protecting renal function.

In many neurogenic bladders it was evident that urine leakage acted as a protective mechanism limiting intravesical pressure and preventing backward propagation of pressure to the kidneys. Before an artificial urethral sphincter is implanted or a continent outlet for self-catheterization is constructed, it is essential to ensure that there will not be an elevated pressure in the neobladder or enlarged bladder. Mundy and Stephenson [23] showed that in many cases it is sufficient to limit bladder augmentation to suturing a wide patch of opened bowel in the sagittally opened bladder. The interruption of the detrusor's contraction by the inserted bowel will reduce detrusor hyperactivity. Lifelong monitoring of urodynamics and renal function is important in these patients, however.

In conclusion, progress in ureteric and renal urodynamic research has matched rapid and major developments in urological surgery. The main gain in urodynamics has been deepened understanding of the close relationship between intravesical and intrapelvic pressure. Knowledge of the ease with which a hyperactive bladder can impede transit of urine should help to conserve good renal function in many patients. Still finer catheters with stable pressure transducers may in the future permit study of urine transit during longer periods in ambulatory patients. With new advances in sonography and laser techniques, non-invasive methods for study of peristalsis can be anticipated. Correlation of urodynamic data with renal function can probably be facilitated by development of more sensitive Doppler techniques for calculations of renal blood flow and resistance. Urodynamic research is still in its early stages and will continue to influence treatment of urological disorders.

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