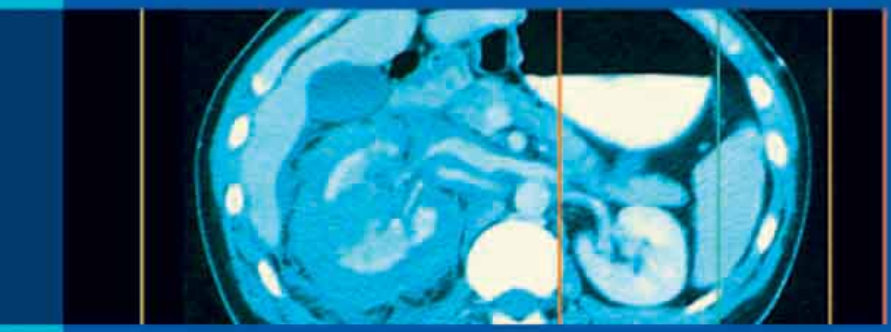
Hashim Hashim
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Hashim Hashim, John Reynard, and Nigel C. Cowan



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Foreword

The specialty of urology has evolved into a less surgical and more cognitive discipline. Indeed, most of what we do in our daily clinical practice involves nonoperative patient care. However many of our patients present with, what are perceived as, emergencies. Such 'emergencies' encompass a broad spectrum of diagnoses, ranging from the often mundane hematuria and orchalgia to the more striking renal colic, symptomatic urinary retention, Fournier's gangrene and testicular torsion, to name but a few. Frequently these emergencies require swift but prudent judgment in order to achieve a satisfactory outcome.

Despite the plethora of these daily encountered 'emergencies,' their descriptions are diluted in the voluminous urologic textbooks available. By assembling this textbook specifically on urologic emergencies, these distinguished authors have contributed a unique and valuable addition to our urologic literary armamentarium. Their objectives are to present diagnostic and treatment-oriented information that can be accessed rapidly and efficiently. These goals are accomplished without comprising thoroughness.

The book consists of 10 broad chapters divided into specific sections making the information easily retrievable. Diagrams and photographs are incorporated appropriately to highlight important points. Diagnostic and therapeutic tips of practical significance are offered throughout the book. This superb organizational format provides a clear, logical and efficient approach to urologic emergencies and should serve as a principal reference for any physician dealing which these ubiquitous problems.

I congratulate the authors and am confident that their gallant efforts will serve to better educate physicians and ultimately improve patient care.

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Chapter 1

Presenting Symptoms of Urological Emergencies

Hashim Hashim and John Reynard

FLANK PAIN

Flank pain is regarded as a classic symptom of renal or ureteric pathology. Indeed, it is often immediately assumed that a patient who presents with flank pain has a stone in the ureter or kidney. However, only 50% of patients who present with flank pain have a ureteric stone confirmed on imaging studies (Smith et al. 1996, Thompson et al. 2001). The other 50% have non–stone-related disease (and more often than not nonurological disease), the differential diagnosis of which is long and dependent on the age, the side of the pain, and the sex of the patient.

The multiple causes of flank pain, to an extent, reflect the fact that the nerve roots subserving pain sensation from the kidney also subserve pain sensation from other organs. Pain sensation from the kidney primarily is transmitted via preganglionic sympathetic nerves that reach spinal cord levels T11 to L2 through the dorsal nerve roots. These same nerve roots supply pain fibres to other intraabdominal organs. Similarly, pain derived from the T10 to T12 costal nerves can also be confused with renal colic.

Causes

This list of causes of flank pain is not exhaustive. Some of these alternative causes may seem bizarre, but we have seen examples of all of these conditions, which were initially referred to us as 'ureteric stone pain,' but where the final diagnosis was some other cause.

Pain on either side

- Urological causes: ureteric stones, renal stones, renal or ureteric tumours, renal infection (pyelonephritis, perinephric abscess, pyonephrosis), pelviureteric junction obstruction.
- Medical causes of flank pain: myocardial infarction, pneumonia, rib fracture, malaria, pulmonary embolus.

Gynaecological and obstetric disease: twisted ovarian cysts, ectopic pregnancy, salpingitis.
Other nonurological causes: pancreatitis, diverticulitis, inflammatory bowel disease, peptic ulcer disease, gastritis.

Right-side flank pain
Biliary colic, cholecystitis, hepatitis, appendicitis.

When flank pain has a urological origin, it occurs as a consequence of distention of the renal capsule by inflammatory or neoplastic disease (pain of constant intensity) or as a consequence of obstruction to the kidney (pain of fluctuating intensity). In the case of ureteric obstruction by a stone, pain also arises as a consequence of obstruction to the kidney and from localised inflammation within the ureter.

Characteristics of flank pain due to ureteric stones: this pain is typically of sudden onset, located below the costovertebral angle of the 12th rib and lateral to the sacrospinalis muscle, and it radiates anteriorly to the abdomen and inferiorly to the ipsilateral groin. The intensity may increase rapidly, reaching a peak within minutes or may increase more slowly over the course of 1 to 2 hours. The patient cannot get comfortable, and tries to move in an attempt to relieve the pain. The pain is not exacerbated by movement or posture. Associated symptoms, occurring with variable frequency include nausea, vomiting, and haematuria.

Patients with pathology that irritates the peritoneum (i.e., peritonitis) usually lie motionless. Any movement, or palpation, exacerbates the pain. Patients with renal colic try to move around to find a more comfortable position. The pain may radiate to the shoulder tip or scapula if there is irritation of the diaphragm (the sensory innervation of which is by the phrenic nerve, spinal nerve root C4). Shoulder-tip pain is not a feature of urological disease.

HAEMATURIA

While haematuria is only relatively rarely an emergency (presenting as clot retention, clot colic, or anaemia), it is such an alarming symptom that it may cause a patient to present to the emergency department.

Blood in the urine may be seen with the naked eye (variously described as macroscopic, frank, or gross haematuria), or may be detected on urine dipstick (dipstick haematuria) or by microscopic examination of urine (microscopic haematuria, defined as the presence of >3 red blood cells per high power microscopic

field). Just 5 mL of blood in 1 L of urine is visible with the naked eye. Dipstick tests for blood in the urine test for haemoglobin rather than intact red blood cells. A cause for the haematuria cannot be found in a substantial proportion of patients despite investigations in the form of flexible cystoscopy, renal ultrasonography, and intravenous urography (IVU) (no cause for the haematuria is found in approximately 50% of patients with macroscopic haematuria and 60% to 70% of patients with microscopic haematuria; Khadra et al. 2000).

Haematuria has nephrological (medical) or urological (surgical) causes. Medical causes are glomerular and nonglomerular, for example, blood dyscrasias, interstitial nephritis, and renovascular disease. Glomerular haematuria results in dysmorphic erythrocytes (distorted during their passage through the glomerulus), red blood cell casts, and proteinuria, while nonglomerular haematuria (bleeding from a site in the nephron distal to the glomerulus) results in circular erythrocytes, the absence of erythrocyte casts, and the absence of proteinuria.

Surgical/urological nonglomerular causes include renal tumours, urothelial tumours (bladder, ureteric, renal collecting system), prostate cancer, bleeding from vascular benign prostatic enlargement, trauma, renal or ureteric stones, and urinary tract infection. Haematuria in these situations is usually characterised by circular erythrocytes and absence of proteinuria and casts.

Haematuria can be painless or painful. It can occur at the beginning of the urinary stream, at the end of the urinary stream, or be present throughout the stream. Haematuria at the beginning of the stream may indicate urethral or prostatic pathology. Haematuria at the end of the stream may indicate prostatic urethra or bladder neck pathology and that present throughout the stream of urine may indicate renal or bladder pathology.

Associated symptoms help determine the cause. Associated renal angle pain suggests a renal or ureteric source for the haematuria, whereas suprapubic pain suggests a bladder source. Painless frank haematuria is not infrequently due to bladder cancer.

As stated above, while patients sometimes present acutely to their family doctors or to hospital emergency departments with haematuria, it is seldom a urological emergency, unless the bleeding is so heavy that the patient has become anaemic as a consequence (this is rare), or the bladder or a ureter has become blocked by clots (in which case the patient presents with retention of urine or with ureteric colic, which may mimic that due

to a stone). We investigate all patients with haematuria, and recommend, as a bare minimum, urine culture and cytology, renal ultrasonography, and flexible cystoscopy, with more complex investigations such as an IVU or computed tomography (CT) scan in selected groups.

OLIGURIA, ANURIA, AND INABILITY TO PASS URINE

Anuria is defined as complete absence of urine production and usually indicates obstruction to the urinary tract. The level of obstruction may be at the outlet of the bladder, or at the level of the ureters bilaterally. Unrelieved bilateral urinary tract obstruction leads rapidly to acute renal failure, which may have very serious consequences (e.g., hyperkalaemia, fluid overload).

If the level of obstruction is at the outlet of the bladder, abdominal examination will reveal a percussable and palpably distended bladder. Urine will be present in the bladder on catheterisation, and urine output will resume once a catheter has bypassed the obstruction. The commonest cause is benign prostatic enlargement and less commonly malignant enlargement of the prostate.

If the obstruction is at the level of the lower ureters or ureteric orifices, the bladder will not be palpable or percussable. Catheterisation will reveal no or a very low volume of urine in the bladder and there will be no improvement in urine output, or of renal function post-catheterisation. Causes include locally advanced prostate cancer, extensive involvement of the trigone of the bladder by bladder cancer, and locally advanced cervical or rectal cancer. Rectal or vaginal examination may reveal a cervical, prostatic, or rectal cancer and cystoscopic examination of the bladder may demonstrate a bladder cancer.

Bilateral obstruction higher up the ureters may be due to extensive lymph node metastases to the pelvic and para-aortic nodes from distant malignancy, retroperitoneal fibrosis, and rarely bilateral ureteric stones. Evidence of a malignancy elsewhere may be found on clinical examination. The diagnosis is usually made on the basis of excluding obstruction at the outlet of the bladder and in the lower ureters and by radiographic imaging (ultrasound and abdominal CT).

Oliguria is scanty urine production, and more precisely is defined as urine production of less than 400 mL/day in adults and less than 1 mL/kg of bodyweight per hour in children. The causes are prerenal (e.g., hypovolaemia, hypotension), renal (e.g., acute vasculitis, acute glomerular lesions, acute interstitial nephritis, and acute tubular necrosis from nephrotoxic drugs, toxins, or

sepsis), and postrenal causes (as for anuria, but where the degree of obstruction has not yet reached a level critical enough to stop urine production completely).

SUPRAPUBIC PAIN

Suprapubic pain can be caused by overdistention of the bladder, and inflammatory, infective, and neoplastic conditions of the bladder. All such conditions may present as an emergency. Bladder overdistention may result from bladder outflow obstruction, e.g., by enlarged prostate, urethral stricture, etc. Painful inability to empty the bladder is defined as urinary retention.

Urinary tract infection is usually associated with urethral burning or scalding on voiding; frequent, low-volume voiding; and a feeling of incomplete bladder emptying with an immediate desire to void again. The urine may be offensive to smell.

Inflammatory conditions of the bladder such as interstitial cystitis can also cause suprapubic pain as can carcinoma in situ. Gynaecological causes of suprapubic pain include endometriosis, fibroids, and ovarian pathology. Gastrointestinal causes of suprapubic pain include inflammatory and neoplastic bowel disease and irritable bowel syndrome.

SCROTAL PAIN AND SWELLING

Scrotal pain may arise as a consequence of pathology within the scrotum itself (e.g., torsion of the testicles or its appendages, epididymo-orchitis) or it may be referred from disease elsewhere (e.g., the pain of ureteric colic may be referred to the testis).

The classic presentation of testicular torsion is one of sudden onset of acute pain in the hemiscrotum, sometimes waking the patient from sleep. It may radiate to the groin and/or the loin. There may be a history of mild trauma to the testis in the hours before the acute onset of pain. Similar episodes may have occurred in the past, with spontaneous resolution of the pain, suggesting torsion with spontaneous detorsion. Patients will be in considerable pain. They may have a slight fever. They do not like the testis being touched and will find it difficult to walk and to get up on the examination couch, as movement exacerbates the pain. The testis is usually swollen, very tender to touch, and may appear abnormally tense (if the patient lets you squeeze it!). It may be high-riding (lying at a higher than normal position in the scrotum) and may lie horizontally due to twisting of the cord. The testis may feel hard and there may be scrotal wall erythema.

Epididymo-orchitis may present with similar symptoms. The localisation of tenderness in the epididymis and the absence of testicular tenderness may help to distinguish epididymo-orchitis from testicular torsion, but in many cases it is difficult to make a precise diagnosis on clinical grounds alone, and often testicular exploration is the only way of establishing the diagnosis with certainty.

Other scrotal pathology may present as acute scrotal swelling leading to emergency presentation. Rarely testicular tumours present as an emergency with rapid onset (days) of scrotal swelling. Very rarely they present with advanced metastatic disease (see Chapter 9).

PRIAPISM

Priapism is a painful persistent prolonged erection not related to sexual stimulation. Its causes are summarised in Chapter 6. Knowledge of these causes allows appropriate questions to be asked during history taking. The two broad categories of priapsim are low flow (most common) and high flow. Low-flow priapism is essentially due to haematological disease, malignant infiltration of the corpora cavernosa with malignant disease, or drugs. High-flow priapism is due to perineal trauma, which creates an arteriovenous fistula. It is painless, unlike low priapsim where ischaemia of the erectile tissue causes pain.

The diagnosis of priapism is usually obvious from the history and examination of the erect, tender penis (in low-flow priapism). Characteristically the corpora cavernosa are rigid and the glans is flaccid. Examine the abdomen for evidence of malignant disease and perform a digital rectal examination to examine the prostate and check anal tone.

BACK PAIN AND UROLOGICAL SYMPTOMS

Occasionally, patients with urological disease present with associated back pain. In some cases this may be the very first symptom of urological disease and it may be so severe that the patient may present acutely to the emergency department. In broad terms, there are two broad categories of disease that may present with back pain and urological symptoms: neurological conditions, and malignant conditions of urological or nonurological origin.

Neurological Disease

Patients with neurological disease may present with both back pain and disturbed lower urinary tract, disturbed bowel, and dis-

turbed sexual function. Such conditions include spinal cord and cauda equina tumours and prolapsed intervertebral discs. In all of these conditions back pain is the most common early presenting symptom. It is usual gradual in onset and progresses slowly, but relentlessly. Associated symptoms suggestive of a neurological cause for the pain include pins and needles in the hands or feet, weakness in the arms (cervical cord) or legs (lumbosacral spine), urinary symptoms such as hesitancy and a poor urinary flow, constipation, loss of erections and seemingly bizarre symptoms such as loss of sensation of orgasm or absent ejaculation. From time to time the patient may present in urinary retention. It is all too easy to assume that this is due to prostatic obstruction if a focused neurological history is not sought and a focused neurological examination is not performed.

Malignant Disease

Malignant tumours may metastasize to the vertebral column, where they may compress the spinal cord (spinal cord compression) or the nerve roots that comprise the cauda equina. Examples include urological malignancies such as prostate cancer, and nonurological malignancies such as lung cancer. In so doing they may cause both back pain and disturbed urinary, bowel, and sexual function. The pain of vertebral metastases may be localised to the area of the involved vertebra, but may also involve adjacent spinal nerve roots, causing radicular pain. Interscapular pain that wakes the patient at night is characteristic of a metastatic deposit in the thoracic spine.

The physical sign of spinal cord compression is a sensory level, but this tends to occur late in the day in the course of the condition. Remember, however, that a normal neurological examination does not exclude a diagnosis of cord compression. If, on the basis of the patient's symptoms, you suspect cord compression, arrange for a magnetic resonance imaging (MRI) scan without delay.

Malignant infiltration of retroperitoneal lymph nodes by, for example, testicular cancers or lymphoma can also cause back pain.

As a general rule, if a patient presents with bizarre symptoms that are difficult to explain, consider the possibility of a neurological cause.

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Chapter 2
Lower Urinary Tract Emergencies

John Reynard

ACUTE URINARY RETENTION

Definition

Painful inability to void, with relief of pain following drainage of the bladder by catheterisation.

The combination of reduced or absent urine output with lower abdominal pain is not in itself enough to make a diagnosis of acute retention. Many acute surgical conditions cause abdominal pain and fluid depletion, the latter leading to reduced urine output, and this reduced urine output can give the erroneous impression that the patient is in retention, when in fact they are not. Thus, central to the diagnosis is the presence of a *large* volume of urine, which when drained by catheterisation, leads to resolution of the pain. What represents 'large' has not been strictly defined, but volumes of 500 to 800 mL are typical. Volumes <500 mL should lead one to question the diagnosis. Volumes >800 mL are defined as acute-on-chronic retention (see Is It Acute or Chronic Retention? below).

Pathophysiology

There are three broad mechanisms:

- increased urethral *resistance*, i.e., bladder outlet obstruction (BOO)
- low bladder *pressure*, i.e., impaired bladder contractility
- interruption of sensory or motor innervation of the bladder

Causes in Men

The commonest cause is benign prostatic enlargement (BPE) due to benign prostatic hyperplasia (BPH) leading to BOO; less common causes include malignant enlargement of the prostate, urethral stricture, and, rarely, prostatic abscess.

Urinary retention in men is either *spontaneous* or *precipitated by an event*. Precipitated retention is less likely to recur once the event that caused it has been removed. Spontaneous retention is more likely to recur after a trial of catheter removal, and therefore is more likely to require definitive treatment, e.g., transurethral resection of the prostate (TURP). Precipitating events include anaesthetics and other drugs (anticholinergics, sympathomimetic agents such as ephedrine in nasal decongestants); nonprostatic abdominal or perineal surgery; and immobility following surgical procedures, e.g., total hip replacement.

Causes in Women

There are more possible causes in women, but acute urinary retention is less common than it is in men. The causes include pelvic prolapse (cystocoele, rectocoele, uterine), the prolapsing organ directly compressing the urethra; urethral stricture; urethral diverticulum; postsurgery for 'stress' incontinence; Fowler's syndrome (impaired relaxation of external sphincter occurring in premenopausal women, often in association with polycystic ovaries); and pelvic masses (e.g., ovarian masses) (Fowler 2003).

Causes in Either Sex

A wide variety of pathologies can cause urinary retention in both men and women: haematuria leading to clot retention; drugs (as above); pain (adrenergic stimulation of the bladder neck); postoperative retention; sacral (S2–S4) nerve compression or damage—so-called cauda equina compression (due to prolapsed L2–L3 disc or L3–L4 intervertebral disc, trauma to the vertebrae, benign or metastatic tumours); radical pelvic surgery damaging the parasympathetic plexus (radical hysterectomy, abdominoperineal resection); pelvic fracture rupturing the urethra (more likely in men than women); neurotropic viruses involving the sensory dorsal root ganglia of S2–S4 (herpes simplex or zoster); multiple sclerosis; transverse myelitis; diabetic cystopathy; damage to dorsal columns of spinal cord causing loss of bladder sensation (tabes dorsalis, pernicious anaemia).

Neurological Causes of Retention—A Word of Warning!

It is all too easy to assume that urinary retention in a man is due to BPH. Of course this is by far the commonest cause in elderly men, but in the younger man (below the age of 60, but even in some men older than 60), spend a few moments considering whether there might be some other cause. Similarly, in women,

where retention is much less common than in men, think *why* the patient went into retention.

Be wary of the patient with a history of constipation and be particularly wary where there is associated back pain. We all get back pain from time to time, but pain of neurological origin, such as that due to a spinal tumour or due to cauda equina compression from a prolapsed intervertebral disc (pressing on S2–S4 nerve roots, thereby impairing bladder contraction) may be severe, relentless, and progressive. The patient may say that the pain has become severe in the weeks before the episode of retention. Nighttime back pain and sciatica (pain shooting down the back of the thigh and legs), which are relieved by sitting in a chair or by pacing around the bedroom at night, are typical of the pain caused by a neurofibroma or ependymoma affecting the cauda equina. Interscapular back pain is typically caused by tumours that have metastasized to the thoracic spine.

Altered sensation due to a cauda equina compression can manifest as the inability to tell whether the bladder is full, inability to feel urine passing down their urethra while voiding, and difficulty in knowing whether one is going to pass faeces or flatus.

Male patients with a neurological cause for their retention (such as spinal tumour) may report symptoms of sexual dysfunction that may appear bizarre (and may therefore be dismissed). They might have lost the ability to get an erection or have lost the sensation of orgasm. They might complain of odd burning or tingling sensations in the perineum or penis.

It doesn't take more than a minute or two to ask a few relevant questions (Are you constipated? Have you had back pain? Do your legs feel funny or weak?), to establish whether the patient has a sensory-level sign (the cardinal sign of a cord compression) and other neurological signs and to test the integrity of the sacral nerve roots that subserve bladder function—S2 to S4. In the male patient, this can be done by squeezing the glans of the penis while performing a digital rectal examination (DRE). Contraction of the anus, felt by the physician's palpating finger, indicates that the afferent and efferent sacral nerves and the sacral cord are intact. This is the bulbocavernosus reflex (BCR). In women, once catheterised, the 'same' reflex can be elicited by gently tugging the catheter onto the bladder neck, again while doing a DRE. Again, contraction of the anus indicates that the afferent and efferent sacral nerves and the sacral cord are intact.

If you don't know about these rare causes of retention, you won't think to ask the relevant questions. Missing the diagnosis

in such cases can have profound implications for the patient (and for you!). One should have a low threshold for arranging an urgent magnetic resonance imaging (MRI) scan of the thoracic, lumbar, and sacral cord, and of the cauda equina in patients who present in urinary retention with these additional symptoms or signs.

Risk Factors for Postoperative Retention

Postoperative retention may be precipitated by instrumentation of the lower urinary tract; surgery to the perineum or anorectum; gynaecological surgery; bladder overdistention; reduced sensation of bladder fullness; preexisting prostatic obstruction; and epidural anaesthesia. Postpartum urinary retention is not uncommon, particularly with epidural anaesthesia and instrumental delivery.

Urinary Retention: Initial Management

Urethral catheterisation is the mainstay of initial management of urinary retention. This relieves the pain of the overdistended bladder. If it is not possible to pass a catheter urethrally, then a suprapubic catheter will be required. Record the volume drained—this confirms the diagnosis, determines subsequent management, and provides prognostic information with regard to outcome from this treatment.

IS IT ACUTE OR CHRONIC RETENTION?

There is a group of elderly men who are in urinary retention, but who are not aware of it. This is so-called high-pressure chronic retention. Mitchell (1984) defined high-pressure chronic retention of urine as maintenance of voiding, with a bladder volume of >800 mL and an intravesical pressure above 30 cm H_2O, often accompanied by hydronephrosis (Abrams et al. 1978, George et al. 1983). Over time this leads to renal failure. The patient continues to void spontaneously and will often have no sensation of incomplete emptying. His bladder seems to be insensitive to the gross distention. Often the first presenting symptom is bedwetting. This is such an unpleasant and disruptive symptom that it will cause most people to visit their doctor. In such cases inspection of the abdomen will show gross distention of the bladder, which may be confirmed by palpation and percussion of the tense bladder.

Sometimes the patient with high-pressure chronic retention is suddenly unable to pass urine, and in this situation so-called acute-on-chronic high-pressure retention of urine has developed.

On catheterisation, a large volume of urine is drained from the bladder (often in the order of 1 to 2 L and sometimes much greater) The serum creatinine will be elevated and an ultrasound will show hydronephrosis (Fig. 2.1) with a grossly distended bladder.

Recording the volume of urine obtained following catheterisation can help define two groups of patients, those with acute retention of urine (retention volume <800 mls) and those with acute-on-chronic retention (retention volume >800 mls). Prior to catheterisation, if the patient reports recent bedwetting you may suspect that you are dealing with a case of high-pressure acute-on-chronic retention. The retention volume will confirm the diagnosis.

Where the patient has a high retention volume (more than a couple of litres), the serum creatinine is elevated, and a renal ultrasound shows hydronephrosis, anticipate that a post-obstructive diuresis is going to occur. This can be very marked and is due to a number of factors:

- Reduction in urine flow through the loop of Henle removes the 'driving force' behind development of the corticomedullary concentration gradient. In addition, continued perfusion of the kidney effectively also 'washes out' this gradient, which is

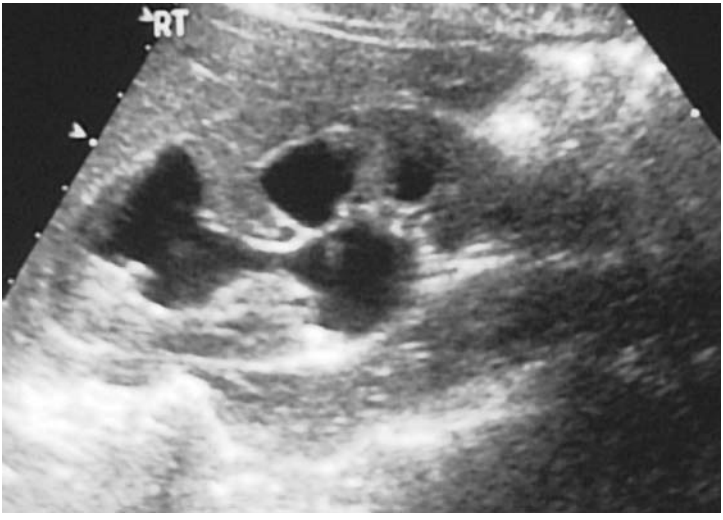


FIGURE 2.1. Hydronephrosis in a case of high-pressure chronic retention.

essential for allowing the kidney to concentrate urine. Once normal flow through the nephron has recommenced following emptying of the bladder and removal of the back pressure on the kidney, it takes a few days for this corticomedullary concentration gradient to be re-established. During this period, the kidney cannot concentrate the urine and a diuresis occurs until the corticomedullary concentration gradient is re-established.

- The elevated serum urea acts as an osmotic diuretic.
- Excessive salt and water, laid down during the period of retention, is appropriately excreted by the kidney.

Usually the patient comes to no harm from this diuresis, even when several litres of urine are excreted per 24 hours. However, occasionally the intravascular volume may fall and postural hypotension may develop. One good way of anticipating this is to record lying and standing blood pressure. If there is a large discrepancy between the two, consider intravenous fluid replacement with normal saline.

WHAT TO DO NEXT FOR THE MAN WITH ACUTE RETENTION

Precipitated retention often does not recur. Spontaneous retention often does.

Precipiated urinary retention should be managed by a trial of catheter removal. In spontaneous retention, many urologists will try to avoid proceeding straight to TURP after just one episode of retention, instead recommending a trial of catheter removal, with or without an alpha blocker, in the hope that the patient will void spontaneously and avoid the need for operation. A trial without catheter is clearly not appropriate in cases where there is back pressure on the kidneys—high-pressure retention. About a quarter of men with acute retention will void successfully after a trial without catheter (Djavan et al. 1997, Hastie et al. 1990). Of those who pass urine successfully after an initial episode of retention, about 50% will go back into retention within a week, 60% within a month, and 70% after a year. This means that after 1 year, only about one in 5 to 10 men originally presenting with urinary retention will not have gone back into retention. Recurrent retention is more likely in those with a flow rate <5 mL/s or average voided volumes of <150 mL. An alpha blocker started 24 hours before a trial of catheter removal increases the chances of voiding successfully (30% taking placebo voiding successfully, and 50% taking an alpha doing so; McNeill et al. 1999). However,

whether continued use of an alpha blocker after an episode of acute retention reduces the risk of a further episode of retention (McNeill et al. 2001) isn't yet known.

So, a trial of an alpha blocker is reasonable, but a substantial number of men with spontaenous acute retention of urine will end up going back into retention and will therefore eventually come under the care of a urologist for TURP.

RETENTION IN PATIENTS WITH A CATHETERISABLE STOMA

An increasing number of patients have undergone reconstructive surgery involving the formation of a catheterisable stoma, such as a Mitrofanoff stoma.

Patients with a Mitrofanoff catheterisable stoma are sometimes unable to pass a catheter into their stoma. This not infrequently occurs after spinal or other surgery. The spinal surgery may change the 'angle' of the stoma or their bladder may become overfull in the post-operative period which again may distort the stoma to the extent that it is difficult to pass a catheter. In this situation, attempting to pass the catheter yourself, using plenty of lubrication, is reasonable. If you fail, try to pass a floppy guidewire through the stoma (preferably under radiological control if this is available). This may pass into the bladder where the catheter will not. A catheter, with the tip cut off, can then be passed over the guidewire and into the bladder. If this fails, pass a suprapubic catheter, empty the bladder, and then usually the patient will be able to pass their catheter without any problems.

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Chapter 3
Nontraumatic Renal Emergencies

John Reynard

ACUTE FLANK PAIN—URETERIC OR RENAL COLIC

Sudden onset of severe pain in the flank is most often due to the passage of a stone formed in the kidney, down through the ureter. The pain is characteristically of very sudden onset, is colicky in nature (waves of increasing severity are followed by a reduction in severity, but it seldom goes away completely), and it radiates to the groin as the stone passes into the lower ureter. The pain may change in location, from the flank to the groin, but the location of the pain does not provide a good indication of the position of the stone, except in those cases where the patient has pain or discomfort in the penis and a strong desire to void, which suggest that the stone may have moved into the intramural part of the ureter. The patient cannot get comfortable, and may roll around in agony. Indeed, the majority of women we have seen with radiologically confirmed ureteric stones and who have also had children, describe the pain of a ureteric stone as being worse than the pain of labour.

The problem with these classic symptoms of ureteric colic is that approximately 50% of patients with the symptoms we have just described do not have a stone confirmed on subsequent imaging studies, nor do they physically ever pass a stone (Smith et al. 1996, Thomson et al. 2001). They have some other cause for their pain. The list of differential diagnoses is very long. A sample of those that we have personally seen include leaking abdominal aortic aneurysms, pneumonia, myocardial infarction, ovarian pathology (e.g., twisted ovarian cyst), acute appendicitis, testicular torsion, inflammatory bowel disease (Crohn's, ulcerative colitis), diverticulitis, ectopic pregnancy, burst peptic ulcer, bowel obstruction, and malaria (presenting as bilateral loin pain and dark haematuria—black water fever)!

The point, then, in making a diagnosis is to exclude other causes of flank pain, many of which are serious and may be life-threatening (leaking aortic aneurysm, gastrointestinal causes,

medical causes), from those cases where the pain is due to a ureteric stone, which is very rarely life-threatening.

Age of the patient can help in determining whether a diagnosis of a ureteric stone is more or less likely. Ureteric colic tends to be a disease of men (and to a lesser extent women) between the ages of roughly 20 and 60. It does affect younger and older patients, but the range of differential diagnoses at the extremes of age, and in women, is greater. Thus, a 25-year-old man who presents with sudden onset of severe, colicky flank pain probably has a ureteric stone, but an 80-year-old woman probably has something else going on.

Examination and Simple Tests

The pain from a ureteric stone is colicky in nature. It makes the patient want to move around, in an attempt to find a comfortable position. The patient may be doubled-up with pain. On the other hand, patients with conditions causing peritonitis, such as appendicitis or a ruptured ectopic pregnancy, want to lie very still. Any movement is very painful and in particular they do not like palpation of their abdomen. Thus, when you approach patients, just spend a few seconds looking at them. If they are lying very still, you may be dealing with a non-stone cause of flank pain.

Pregnancy Test

All premenopausal women with acute flank pain should undergo a pregnancy test. If this is positive, they are referred to a gynaecologist. If it is negative, they should undergo imaging to determine whether or not they have a ureteric stone. It goes without saying that any premenopausal woman who is going to undergo imaging using ionising radiation, should have a pregnancy test done first.

Dipstick or Microscopic Haematuria

While many patients with ureteric stones have dipstick or microscopic haematuria (and more rarely macroscopic haematuria), 10% to 30% of such patients have no blood in their urine (Kobayashi et al. 2003, Luchs et al. 2002). There is evidence that if a stone has been present in the ureter for 3 to 4 days, there is a greater likelihood that haematuria will not be detectable.

The sensitivity of dipstick haematuria for detecting ureteric stones presenting acutely is in the order of 95% on the first day

of pain, 85% on the second day of pain, and 65% on the third and fourth days (Kobayashi et al. 2003). Dipstick testing is slightly more sensitive than urine microscopy for detecting stones (80% versus 70%), and both ways of detecting haematuria have roughly the same specificity for diagnosing ureteric stones (about 60%). The slightly greater sensitivity of dipstick testing over microscopy reflects the fact that seeing red blood cells depends on how good the technician is at looking for them, and that they lyse, and therefore disappear, if the urine specimen is not examined under the microscope within a few hours. Thus, if you see a patient with a history suggestive of ureteric colic, and their pain started 3 to 4 days ago, they may well have no blood detectable in their urine even though they do have a stone.

The relatively poor specificity of dipstick or microscopic haematuria for detecting ureteric stones reflects the multiple other pathologies that can mimic the pain of a ureteric calculus combined with the fact that blood is detectable in a proportion of patients without demonstrable urinary tract pathology; in fact, no abnormality is found in approximately 70% of patients with microscopic haematuria, despite full investigation with cystoscopy, renal ultrasound, and intravenous urography (IVU) (Khadra 2000). Thus, blood in the urine may be a completely coincidental finding in a patient who presents with flank pain due to a non-stone cause.

Temperature

Perhaps the most important aspect of examination in patients with a ureteric stone confirmed on imaging is to measure their temperature. If patients have a stone, and they have a fever of, say, 39°C, they may well have infection proximal to the obstructing stone. A fever in the presence of an obstructing stone is an indication for urine and blood culture, intravenous fluids and antibiotics, and nephrostomy drainage if the fever does not resolve within a matter of hours of commencement of antibiotics.

Investigation of Suspected Ureteric Colic

The intravenous urogram (IVU) was for many years the mainstay of diagnostic imaging in patients with flank pain (Fig. 3.1). The last few years have seen a move toward computed tomography (CT) urography (CTU) (Fig. 3.2). CTU has the following advantages over IVU:



a

FIGURE 3.1. a: An intravenous urogram (IVU) control film. Two calcifications are seen in the left hemipelvis. Which is the ureteric stone? b: Following contrast administration, the lateral calcification is seen to lie outside the ureter; it is a phlebolith. The medial calcification is a ureteric stone.



b

FIGURE 3.1. *Continued*

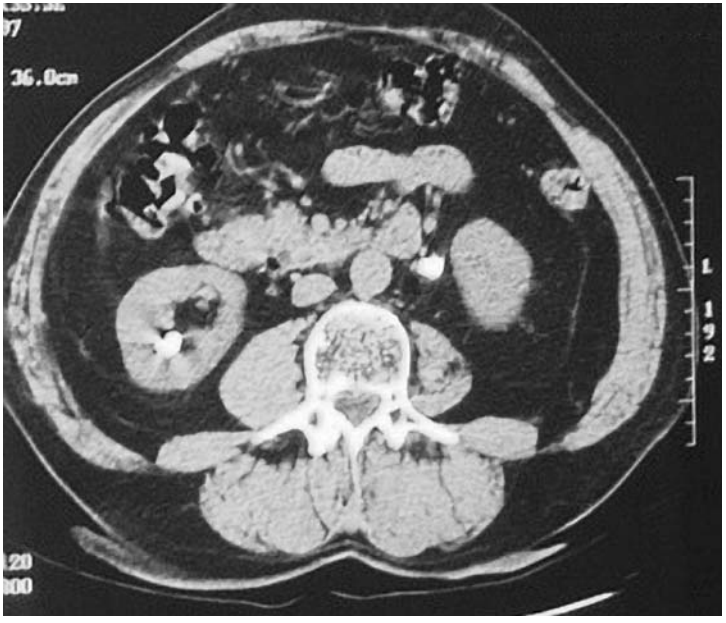


FIGURE 3.2. A computed tomography (CT) urogram (CTU). Stones 'light' up as very radiodense structures. There is one in the left ureter and one in the right kidney.

1. It has greater specificity (95%) and sensitivity (97%) for diagnosing ureteric stones than has IVU (Smith et al. 1996). CTU can identify other, non-stone causes of flank pain such as leaking aortic aneurysms (Fig. 3.3).

2. There is no need for contrast administration with CTU. This avoids the chance of a contrast reaction. The risk of fatal anaphylaxis following the administration of low-osmolality contrast media for IVU is on the order of 1 in 100,000 (Caro et al. 1991).

3. CTU is faster, taking just a few minutes to image the kidneys and ureters. An IVU, particularly where delayed films are required to identify a stone causing high-grade obstruction, may take hours to identify the precise location of the obstructing stone (Fig. 3.4).

4. In some hospitals, where high volumes of CT scans are done, the cost of CTU is equivalent to that of IVU (Thomson et al. 2001).

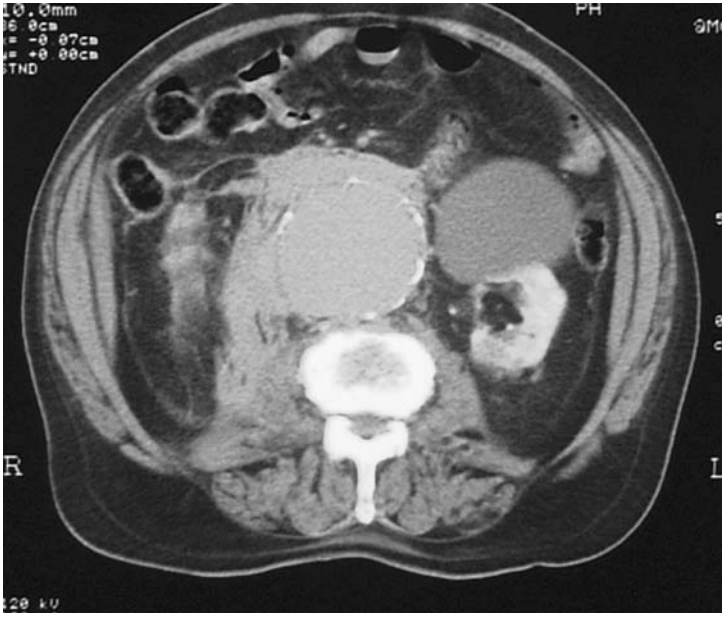


FIGURE 3.3. A leaking abdominal aortic aneurysm, referred as a ureteric stone, but correctly diagnosed by CTU.

If you only have access to IVU, remember that it is contraindicated in patients with a history of previous contrast reactions, and should be avoided in those with hay fever or a strong history of allergies or asthma who have not been pretreated with high-dose steroids 24 hour before the IVU. Patients taking metformin for diabetes should stop this for 48 hours prior to an IVU. Clearly, being able to perform an alternative test in such patients, such as CTU, is very useful.

In hospitals where 24-hour access to CTU is not possible, patients with suspected ureteric colic may be admitted for pain relief, and undergo a CTU the following morning. It is our policy, when CT urography is not immediately available (between the hours of midnight and 8 a.m.), to perform an abdominal ultrasound in all patients over the age of 50 years who present with flank pain suggestive of a possible stone. This is done to exclude serious pathology such as a leaking abdominal aortic aneurysm and to demonstrate any other gross abnormalities due to non–stone-associated flank pain.



a

FIGURE 3.4. a: On a 1-hour postcontrast film the right ureter is still not opacified. Only the outline of the kidney and renal collecting system is visible because of the distal obstruction. b: In this case it takes 2 hours for the IVU to demonstrate the stone and its position in the right lower ureter.



b

FIGURE 3.4. *Continued*

Plain abdominal x-ray and renal ultrasound are not sufficiently sensitive or specific for their routine use for diagnosing stones.

Magnetic Resonance Urography (Fig. 3.5)
This is a very accurate way of determining whether or not a stone is present in the ureter (Louca et al. 1999; O'Malley 1997).

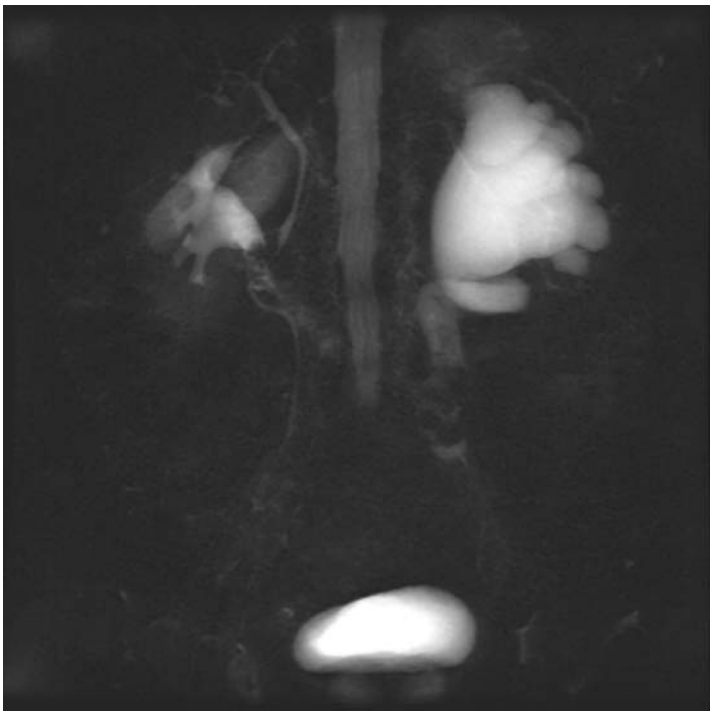


FIGURE 3.5. Magnetic resonance urogram. Stones appear as 'black holes.'

However, at the present time, cost and resticted availability limit its usefulness as a routine diagnostic method of imaging in cases of acute flank pain. This may change as MR scanners become more widely available.

Acute Management of Ureteric Stones

The management of any acutely presenting ureteric stone starts with pain relief. Nonsteroidal anti-inflammatory drugs (NSAIDs), such as diclofenac (Voltarol) given by intramuscular or intravenous injection, by mouth, or per rectum can, in many cases, provide rapid and effective pain control (Laerum et al. 1996). In other cases opiate analgesics such as pethidine or morphine are required, in addition to NSAIDs.

There is no need to encourage the patient to drink copious amounts of fluids or to give them large volumes of fluids intravenously, in the hope that this will 'flush' the stone out. Renal

blood flow and urine output from the affected kidney will tend to fall during an episode of acute partial obstruction due to a stone, and any excess fluid that is excreted will tend to cause a greater degree of hydronephrosis in the affected kidney, which will make ureteric peristalsis even less efficient than it already is. Remember, peristalsis, the forward propulsion of a bolus of urine down the ureter, can occur only if the walls of the ureter above the bolus of urine can coapt, i.e., close firmly together. If they cannot, as occurs in a ureter distended with urine, the bolus of urine cannot move distally. This is why insertion of a percutaneous nephrostomy tube can restore efficient peristalsis. By draining the hydronephrosis and hydroureter, it allows the ureteric wall to coapt and thus encourages a return to normal peristaltic function.

In many instances, small ureteric stones pass spontaneously given a period of 'watchful waiting' with analgesic supplements for exacerbations of pain. Accurate determination of stone size (on plain abdominal x-ray if the stone is so visible or by CTU) can help predict the chances that the stone will pass out of the ureter and into the bladder; 95% of stones measuring 5 mm or less pass spontaneously (Segura et al. 1997). However, it never ceases to amaze us that stones much larger than 5 mm do, from time to time, drop harmlessly out of the ureter, and that others that are only 4 mm in diameter stubbornly remain in the ureter.

Whether patients opt for watchful waiting or active intervention will, to a certain extent, depend on other factors, such as their job. Young, active patients may be very keen to opt for surgical treatment because they need to get back to work or their child-care duties, whereas some patients will be happy to sit things out. Discuss the options with patients so they are able to make a rational decision.

Indications for Intervention to Relieve Obstruction and/or Remove the Stone

1. Pain that fails to respond to analgesics, or that initially does so but then recurs and cannot be controlled with additional pain relief, is an indication for drainage of the kidney (by JJ stent insertion or percutaneous nephrostomy) or emergency definitive treatment of the stone.

2. Where there is an associated fever, one should have a low threshold for draining the kidney, and this is usually done by percutaneous nephrostomy.

3. Where renal function is impaired because of the stone (solitary kidney obstructed by a stone, bilateral ureteric stones,

or preexisting renal impairment that gets worse as a consequence of a ureteric stone), the threshold for intervention is lower.

4. Obstruction unrelieved for >4 weeks can result in long-term loss of renal function. In a study of 239 patients presenting with unilateral ureteric stones, after 2 weeks the stones were still present in 143 patients (Holm-Nielsen et al. 1981). Of these 143 patients, 50% had renal obstruction defined by isotope renography; 11 of 31 patients (35%) with obstruction for >4 weeks developed varying degrees of irreversible renal damage. The problem with current imaging for stones, which nowadays is essentially CTU, is the absence of any information on the presence of renal obstruction (most urologists do not routinely obtain isotope renograms in patients with ureteric colic). However, what we do know from the Holm-Nielsen study is that only 50% of patients with ureteric stones that are still present at 2 weeks, have renographic evidence of obstruction. It seems reasonable to limit the period of watchful waiting for spontaneous stone passage to approximately 4 weeks and to intervene to remove the stone or drain the kidney (by, for example, JJ stent placement) if it has not passed at this time.

5. Personal or occupational reasons. As stated above, some patients will not be able to wait for spontaneous stone passage and therefore may accept the risks associated with active intervention. The classic example would be the airline pilot who is unable to fly until he is stone free.

Emergency Temporising and Definitive Treatment of the Stone

Where the pain of a ureteric stone fails to respond to analgesics or where renal function is impaired because of the stone, then temporary relief of the obstruction can be obtained by insertion of a JJ stent or percutaneous nephrostomy tube. This has the advantage of not taking much time to perform. However, the disadvantage is that the stone is still present. While the stone may pass down and out of the ureter with a stent in situ, in many instances the stone simply sits where it is and subsequent definitive treatment is still required. Furthermore, though a JJ stent can relieve the pain due to the stone, it can cause bothersome irritative bladder symptoms (pain in the bladder, frequency, and urgency). Having said this, a JJ stent will usually result in passive dilatation of the ureter so that subsequent stone treatment in the form of ureteroscopy is technically easier and therefore more likely to be successful. Similarly, by allowing passive dilatation of the ureter, fragments of stone produced by extracorporeal shock-wave lithotripsy (ESWL) may be more easily able to pass out of the ureter.

General options for definitive treatment of a ureteric stone are ESWL and ureteroscopic stone removal. ESWL is suitable for stones in the upper and lower ureter. Ureteroscopy can be used to treat stones at any level in the ureter, although access and fragmentation of stones in the lower ureter is generally easier (Fig. 3.6).

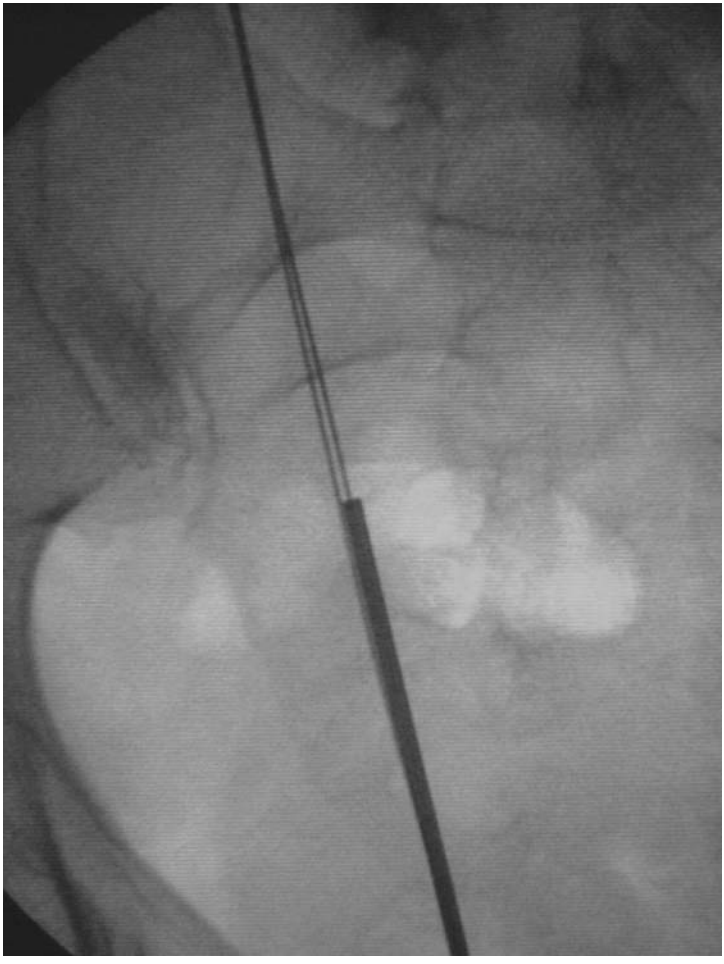


FIGURE 3.6. Ureteroscopic approach to a lower ureteric stone. Note the presence of 2 guidewires–one is a 'safety' line; the ureteroscope is passed over the other to the level of the stone.

Whether you decide to carry out definitive stone treatment, and what type of treatment you offer, will depend on local facilities and expertise. Many hospitals do not have daily access to ESWL. In others, surgeons with experience of ureteroscopic stone fragmentation are not always available.

Emergency Treatment of an Obstructed, Infected Kidney

The rationale for performing percutaneous nephrostomy (Fig. 3.7) rather than JJ stent insertion (Fig. 3.8) for an infected, obstructed kidney is to reduce the likelihood of septicaemia occurring as a consequence of showering bacteria into the circulation. It is thought that this is more likely to occur with JJ stent insertion than with percutaneous nephrostomy insertion. A discussion of subsequent management of ureteric stones that fail to pass spontaneously, or are too large to do so, is beyond the scope of this book.

Other Non-Stone Causes of Acute Flank Pain

These include pelviureteric junction obstruction (PUJO), which is called ureteropelvic junction obstruction (UPJO) in North America, and infective causes such as acute pyelonephritis, emphysematous pyelonephritis, and xanthogranulomatous pyelonephritis.

Pelviureteric Junction Obstruction

This is a functional impairment of transport of urine from the renal pelvis into the ureter. It may be acquired or congenital. The majority of cases are probably congenital in origin, but do not always present in childhood. Indeed, many present in young adults. The precise cause of the aperistaltic segment of ureter that leads to congenital cases of this condition is not known. Acquired causes of PUJO include stones (the investigation and management of which is discussed above), urothelial tumours (transitional cell carcinoma), and inflammatory and postoperative strictures.

Not infrequently PUJO may present acutely with flank pain, which may be severe enough to mimic a ureteric stone. When imaging (nowadays usually a CT scan) demonstrates hydronephrosis, with a normal-calibre ureter below the pelviureteric junction (PUJ) and no stone (or tumour) is seen, the



FIGURE 3.7. Percutaneous nephrostomy in situ.



FIGURE 3.8. JJ stent post insertion.

diagnosis of PUJO becomes likely, and a renogram (e.g., MAG3 scan) should be done to confirm the diagnosis (Fig. 3.9).

ACUTE PYELONEPHRITIS

Clinical Definition

This is a clinical diagnosis, made on the basis of fever, flank pain, and tenderness, often with an elevated white count. It may affect

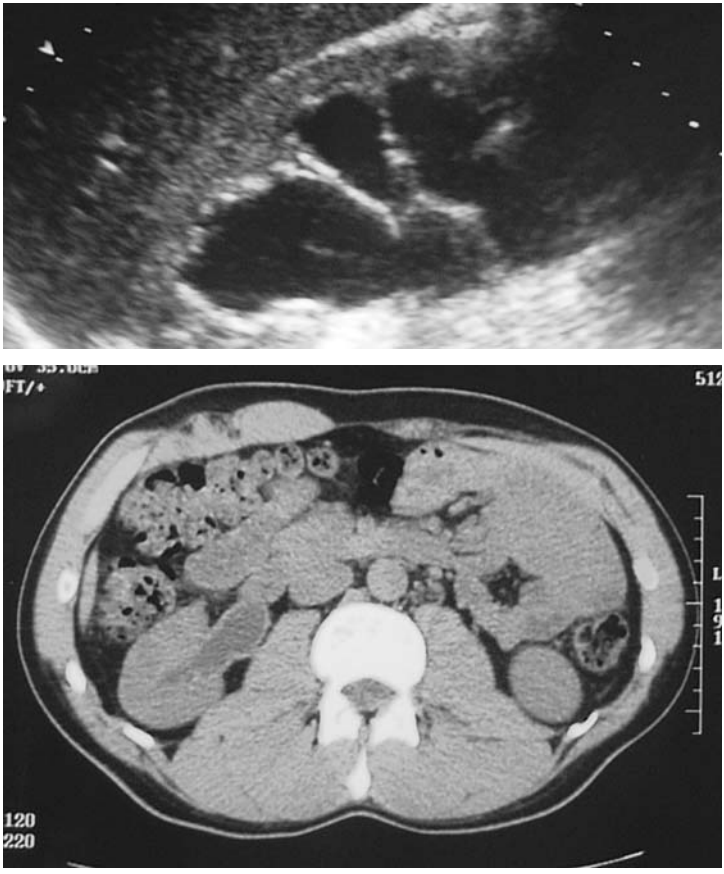
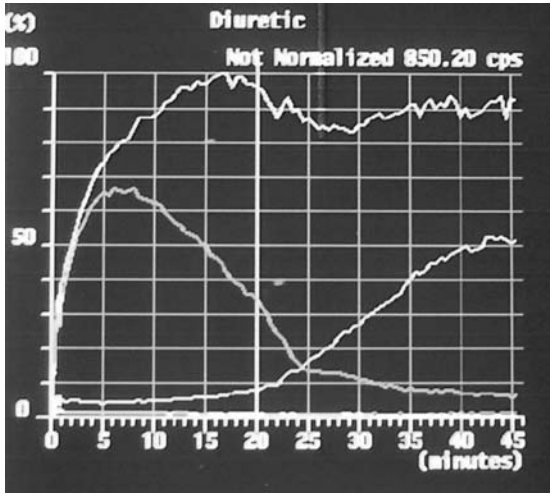


FIGURE 3.9. a: Right pelviureteric junction (PUJ) obstruction on ultrasound. b: PUJ obstruction on CT. Note the normal-calibre ureter with hydronephrosis above. c: MAG3 renogram of PUJ obstruction demonstrating obstruction to excretion of radioisotope by the kidney. (See this figure in full color in the insert.)

one or both kidneys. There are usually accompanying symptoms suggestive of a lower urinary tract infection (frequency, urgency, suprapubic pain, urethral burning or pain on voiding) that led to the ascending infection, which resulted in the subsequent acute pyelonephritis. The infecting organisms are commonly *Escherichia coli*, enterococci (*Streptococcus faecalis*), *Klebsiella*, *Proteus*, and *Pseudomonas*.



c

FIGURE 3.9. *Continued*

Urine culture is positive for bacterial growth, but the bacterial count may not always be above the 100,000 colony-forming units (cfu)/mL of urine, which is the strict definition for urinary infection. Thus, if you suspect a diagnosis of acute pyelonephritis from the symptoms of fever and flank pain, but there are only 1000 cfu/mL, manage the case as acute pyelonephritis.

Investigation and Treatment

For those patients who have a fever but are not systemically unwell, outpatient management is reasonable. Culture the urine and start oral antibiotics according to your local antibiotic policy (which will be based on the likely infecting organisms and their likely antibiotic sensitivity). We use oral ciprofloxacin, 500 mg b.i.d. for 10 days.

If the patient is systemically unwell, admit them to hospital culture urine and blood, and start intravenous fluids and intravenous antibiotics, again selecting the antibiotic according to your local antibiotic policy. We use i.v. ampicillin 1 g t.i.d. and gentamicin, 3 mg/kg as a once daily dose.

Arrange for a kidney and urinary bladder (KUB) x-ray and renal ultrasound, to see if there is an underlying upper tract abnormality (such a ureteric stone), unexplained hydronephrosis, or (rarely) gas surrounding the kidney (suggesting emphysematous pyelonephritis).



FIGURE 3.10. A CTU without contrast in a diabetic patient with left acute pyelonephritis. Note the incidental finding of a nonobstructing left renal calculus.

If the patient does not respond within 3 days to this regimen of appropriate intravenous antibiotics (confirmed on sensitivities), arrange for a CTU (Fig. 3.10). The lack of response to treatment indicates that you are dealing with a pyonephrosis (i.e., pus in the kidney, which like any abscess will respond only to drainage), a perinephric abscess (which again will respond only to drainage), or emphysematous pyelonephritis. The CTU may demonstrate an obstructing ureteric calculus that may have been missed on the KUB x-ray, and ultrasound and will show a perinephric abscess if present. A pyonephrosis should be drained by insertion of a percutaneous nephrostomy tube. A perinephric abscess should also be drained by insertion of a drain percutaneously.

If the patient responds to i.v. antibiotics, change to an oral antibiotic of appropriate sensitivity when they become apyrexial, and continue this for approximately 10 to 14 days.

PYONEPHROSIS

This is an infected hydronephrosis, the infection being severe enough to cause accumulation of pus with the renal pelvis and calyces of the kidney. The causes are essentially those of hydronephrosis, where infection has supervened. Thus, they include ureteric obstruction by stone and PUJ obstruction.

Patients with pyonephrosis are usually very unwell, with a high fever, flank pain, and tenderness. Again, a patient with this combination of symptoms and signs will usually be investigated by a renal ultrasound, where the diagnosis of a pyonephrosis is usually obvious (Fig. 3.11).

Treatment consists of i.v. antibiotics (as for pyelonephritis), i.v. fluids, and percutaneous nephrostomy insertion.



a

FIGURE 3.11. a: The appearance of a pyonephrosis on ultrasound. Note the hyperreflective material within the dilated system. b: A right pyonephrosis on CT, done without contrast. Note the presence of a stone in the kidney. c: A right pyonephrosis on CT postcontrast administration.



b



c

FIGURE 3.11. *Continued*



FIGURE 3.12. A left perinephric abscess as seen on CT.

PERINEPHRIC ABSCESS

Perinephric abscess (Fig. 3.12) develops as a consequence of extension of infection outside the parenchyma of the kidney in acute pyelonephritis, or more rarely, nowadays, from haematogenous spread of infection from a distant site. The abscess develops within Gerota's fascia—the fascial layer surrounding the kidneys and their cushion of perinephric fat. These patients are often diabetic, and associated conditions such as an obstructing ureteric calculus may be the precipitating event leading to development of the perinephric abscess. Failure of a seemingly straightforward case of acute pyelonephritis to respond to intravenous antibiotics within a few days should arouse your suspicion that there is something else going on, such as the accumulation of pus in or around the kidney, or obstruction with infection. Imaging studies, such as ultrasound and more especially CT, will establish the diagnosis and allow radiographically controlled percutaneous drainage of the abscess. However, if the pus collection is large, formal open surgical drainage under general anaesthetic will be provide more effective drainage.

EMPHYSEMATOUS PYELONEPHRITIS

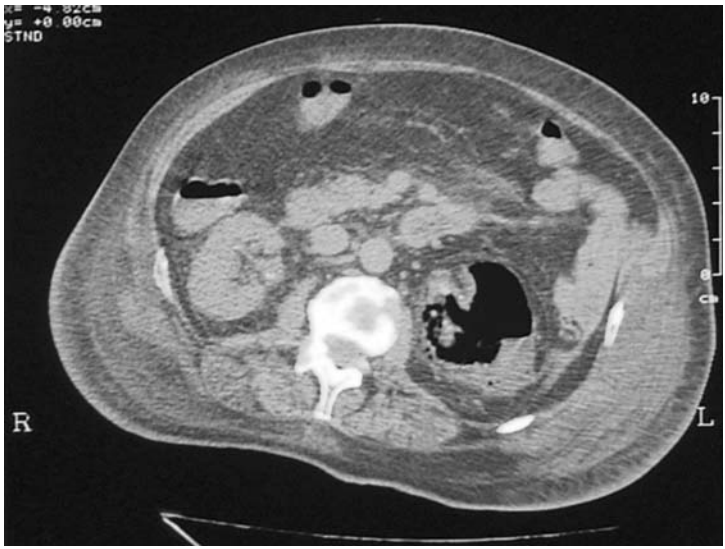
This is a rare and severe form of acute pyelonephritis caused by gas-forming organisms (Fig. 3.13). It is characterised by fever



a

FIGURE 3.13. a: A case of emphysematous pyelonephritis on plain abdominal x-ray. Note the presence of gas within the left kidney. b: A CT of the same case. The gas in the kidney (like that in the bowel) is black on CT. c: A percutaneous drain has been inserted with the patient lying prone. Note the J loop of the drain in the kidney.

b



c

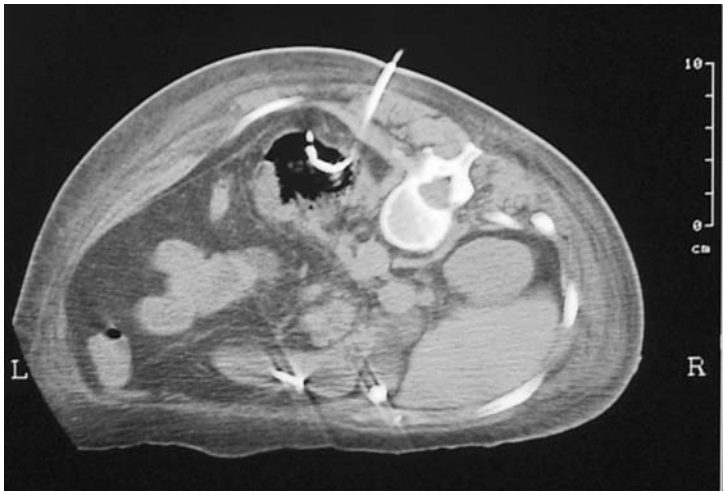


FIGURE 3.13. *Continued*

and abdominal pain, with radiographic evidence of gas within and around the kidney (on plain radiography or CT). It usually occurs in diabetics, and in many cases is precipitated by urinary obstruction by, for example, ureteric stones. The high glucose levels of the poorly controlled diabetic provides an ideal environment for fermentation by enterobacteria, carbon dioxide being produced during this process.

Presentation

Emphysematous pyelonephritis presents as a severe acute pyelonephritis (high fever and systemic upset) that fails to respond within 2 to 3 days with conventional treatment in the form of intravenous antibiotics. *E. coli* is a common causative organism, with *Klebsiella* and *Proteus* occurring from time to time. Obtaining a KUB x-ray and ultrasound in all patients with acute pyelonephritis may allow earlier diagnosis of this rare form of pyelonephritis. An unusual distribution of gas on x-ray may suggest that the gas lies around the kidney (e.g., crescent or kidney shaped). Renal ultrasonography often demonstrates strong focal echoes, indicating gas within the kidney. Intrarenal gas will be clearly seen on CT scan.

Treatment

Patients with emphysematous pyelonephritis are usually very unwell. Mortality is high. Selected patients can be managed conservatively, by intravenous antibiotics and fluids, percutaneous drainage, and careful control of diabetes. In those where sepsis is poorly controlled, emergency nephrectomy is required.

ACUTE PYELONEPHRITIS, PYONEPHROSIS, PERINEPHRIC ABSCESS, AND EMPHYSEMATOUS PYELONEPHRITIS—MAKING THE DIAGNOSIS

Maintaining a degree of suspicion in all cases of presumed acute pyelonephritis is the single most important thing in making an early diagnosis of complicated renal infection, such as a pyonephrosis, perinephric abscess, or emphysematous pyelonephritis. If patients are very unwell, or diabetic, or have a history suggestive of stones, for example, ask yourself whether they may have something more than just a simple acute pyelonephritis. They may give a history of sudden onset of severe flank pain a few days earlier, which suggests that they may have passed a stone into their ureter at this stage, and that later infection supervened.

A policy of arranging for a KUB x-ray and renal ultrasound in all patients with suspected renal infection is wise. The main clinical indicators that suggest you may be dealing with a more complex form of renal infection are length of symptoms prior to treatment and time taken to respond to treatment. Thorley and colleagues (1974) reviewed a series of 52 patients with perinephric abscess. They noted that the majority of patients with uncomplicated acute pyelonephritis had been symptomatic for less than 5 days, whereas most of those with a perinephric abscess had been symptomatic for more than 5 days prior to hospitalisation. In addition, all patients with acute pyelonephritis became afebrile after 4 days of treatment with an appropriate antibiotic, whereas patients with perinephric abscesses remained pyrexial.

XANTHOGRANULOMATOUS PYELONEPHRITIS

This is a severe renal infection usually (though not always) occurring in association with underlying renal calculi and renal obstruction. The severe infection results in destruction of renal tissue, and a nonfunctioning, enlarged kidney is the end result. *E. coli* and *Proteus* are common causative organisms. Macrophages full of fat become deposited around abscesses within the parenchyma of the kidney. The infection may be confined to the kidney or extend to the perinephric fat. The kidney becomes grossly enlarged and macroscopically contains yellowish nodules, pus, and areas of haemorrhagic necrosis. It can be very difficult to distinguish the radiological findings from a renal cancer on imaging studies such as CT (Fig. 3.14). Indeed, in most cases the diagnosis is made after nephrectomy for a presumed renal cell carcinoma.

Presentation and Imaging Studies

Patients present acutely with flank pain and fever, with a tender flank mass. Bacteria (*E. coli*, *Proteus*) may be found on culture urine. Renal ultrasonography shows an enlarged kidney containing echogenic material. On CT, renal calcification is usually seen, within the renal mass. Nonenhancing cavities are seen, containing pus and debris. On radioisotope scanning, there may be some or no function in the affected kidney.

Management

On presentation these patients are usually commenced on antibiotics as the constellation of symptoms and signs suggests infection. When imaging studies are done, such as CT, the appearances usually suggest the possibility of a renal cell carci-



FIGURE 3.14. A case of xanthogranulomatous pyelonephritis (left) as seen on CT. This can be very difficult to distinguish radiologically from a renal cancer.

noma, and therefore when signs of infection have resolved, the majority of patients will proceed to nephrectomy. Only following pathological examination of the removed kidney will it become apparent that the diagnosis was one of infection (xanthogranulomatous pyelonephritis) rather than one of a tumour.

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Chapter 4
Other Infective Urological Emergencies

Hashim Hashim and John Reynard

URINARY SEPTICAEMIA

Sepsis as a result of a urinary tract infection is a serious condition that can lead to septic shock and death. Septicaemia or sepsis is the clinical syndrome caused by bacterial infection of the blood, confirmed by positive blood cultures for a specific organism. There should be a documented source of infection with a systemic response to the infection. The systemic response is known as the systemic inflammatory response syndrome (SIRS) and is defined by as at least two of the following:

- Fever (>38°C) or hypothermia (<36°C)
- Tachycardia (>90 beats/min in patients not on beta-blockers)
- Tachypnoea (respiratory rate >20/min or $PaCO_2$ < 4.3 kPa or a requirement for mechanical ventilation)
- White cell count >12,000 cells/mm^3, <4000 cells/mm^3, or 10% immature (band) forms

Severe sepsis or sepsis syndrome is a state of altered organ perfusion or evidence of dysfunction of one or more organs, with at least one of the following: hypoxaemia, lactic acidosis, oliguria, or altered mental status. Septic shock is severe sepsis with refractory hypotension, hypoperfusion, and organ dysfunction. This is a life-threatening condition.

There are many causes of urinary sepsis, but in the hospital setting the commonest causes from a urological perspective are the presence of or manipulation of indwelling urinary catheters, urinary tract surgery, particularly endoscopic [transurethral resection of the prostate (TURP), transurethral resection of bladder tumor (TURBT), ureteroscopy, percutaneous nephrolithotomy (PCNL)] and urinary tract obstruction, particularly that due to stones obstructing the ureter. In the National Prostatectomy Audit and the European Collaborative Study of Antibiotic Prophylaxis for TURP, septicaemia occurred in

approximately 1.5% of men undergoing TURP. Diabetic patients, patients in the intensive care units (ICU), and patients on chemotherapy and steroids are more prone to urosepsis.

The commonest causative organisms of urinary sepsis are *Escherichia coli*, enterococci (*Streptococcus faecalis*), staphylococci, *Pseudomonas aeruginosa*, *Klebsiella*, and *Proteus mirabilis*.

The principles of management include early recognition, resuscitation, localisation of the source of sepsis, early and appropriate antibiotic administration, and removal of the primary source of sepsis. The clinical scenario is usually a postoperative patient who has undergone TURP or surgery for stone. Having returned to the ward, the patient becomes pyrexial, starts to shiver and shake, is tachycardic, and may be confused. On inspection the patient may initially show signs of peripheral vasodilatation (may appear flushed and warm to the touch). Look for symptoms and signs of a non-urological source of sepsis such as pneumonia. If there are no indications of infection elsewhere, assume the urinary tract is the source of sepsis.

Investigations

- Urine culture. An immediate Gram stain may aid in deciding which antibiotic to use.
- Full blood count. The white blood count is usually elevated. The platelet count may be low, a possible indication of impending disseminated intravascular coagulopathy (DIC).
- Coagulation screen. This is important if surgical or radiological drainage of the source of infection is necessary.
- Urea and electrolytes as a baseline determination of renal function.
- Arterial blood gases to identify hypoxia and the presence of metabolic acidosis.
- Blood cultures.
- Chest x-ray (CXR), looking for pneumonia, atelectasis, and effusions.

Depending on the clinical situation, a renal ultrasound may be helpful to demonstrate hydronephrosis or pyonephrosis and CT urography (CTU) may be used to establish the presence or absence of a ureteric stone.

Treatment

- Remember A (airway), B (breathing), C (circulation).
- Administer 100% oxygen via a face mask.

- Establish intravenous access with a wide-bore intravenous cannula, e.g., 16 or 18 gauge.
- Start an intravenous infusion of crystalloid e.g., normal saline or colloid e.g., Gelofusin.
- Catheterise the patient to monitor urine output.
- Start empirical antibiotic therapy (see below). This should be adjusted later when cultures are available.
- If there is septic shock, the patient needs to be transferred to the ICU. Inotropic support may be needed. Steroids may be used as adjunctive therapy in gram-negative infections. Naloxone may help revert endotoxic shock. This should all be done under the supervision of an intensivist.
- Treat the underlying cause. Drain any obstruction and remove any foreign body. If there is a stone obstructing the ureter, then either ask the radiologist to insert a nephrostomy tube to relieve the obstruction or take the patient to the operating room and insert a JJ stent. Send any urine specimens obtained for microscopy and culture.

Empirical Treatment

Empirical antibiotic treatment is the 'blind' use of antibiotics based on an educated guess of the most likely pathogen that has caused the sepsis. In urinary sepsis, the cause is often a gram-negative rod. Gram-negative aerobic rods include the enterobacteria, e.g., *E. coli*, *Klebsiella*, *Citrobacter*, *Proteus*, and *Serratia*. The enterococci (gram-positive aerobic nonhaemolytic streptococci) may sometimes cause urosepsis. In urinary tract operations involving bowel, anaerobic bacteria may be the cause of urospesis and in wound infections staphylococci, e.g., *staphylococcus aureus* and *staphylococcus epidermidis* are the usual cause.

The recommendations for treatment of urosepsis include (Naber 2001):

- A third-generation cephalosporin, e.g., cefotaxime IV, ceftriaxone IV. These are active against gram-negative bacteria, but less active against staphylococci and gram-positive bacteria. Ceftazidime also has activity against *Pseudomonas aeruginosa*. It is therefore important to get an urgent gram stain on any fluid sample sent to the laboratory. About 5% of patients who are allergic to penicillin are also allergic to cephalosporins, so enquire about penicillinallergy and consider alternative antibiotics.
- Fluoroquinolones, e.g., ciprofloxacin, can be used instead of cephalosporins. They exhibit good activity against enterobac-

taria and *P. aeruginosa*, but less activity against staphylococci and enterococci. Ciprofloxacin can be given both orally and intravenously. It is well absorbed from the gastrointestinal tract.

- Metronidazole is used if there is suspicion of an anaerobic source of sepsis.
- Other drugs that can be used if there is no clinical response to the above include a combination of piperacillin and tazobactam. This combination is active against enterobacteria, enterococci, and *Pseudomonas*.
- Gentamicin is used in conjunction with other antibiotics because it has a relatively narrow therapeutic spectrum (against gram-negative organisms). Close monitoring of therapeutic levels and renal function is important. It has good activity against enterobacteria and *Pseudomonas*, with poor activity against streptococci and anaerobes and therefore should ideally be combined with β-lactam antibiotics, e.g., cotrimoxazole but can be combined with ciprofloxacin instead.

If there is clinical improvement, intravenous treatment should continue for at least 48 hours with oral medication thereafter. Make appropriate adjustments when the sensitivity results are available from the urine cultures that were sent. It may take about 48 hours for sensitivity results to become available.

PYELONEPHRITIS AND PYONEPHROSIS

See Chapter 3.

PROSTATIC INFECTIONS AND PROSTATIC ABSCESS

Acute Bacterial Prostatitis [National Institute of Health Classification System (Krieger 1999) Category I Prostatitis]

Acute bacterial prostatitis is infection of the prostate associated with lower urinary tract infection and generalised sepsis. *E. coli* is the commonest cause. *Pseudomonas*, *Serratia*, *Klebsiella*, and enterococci are less common causes.

The presenting symptoms include acute onset of perineal and suprapubic pain with irritative (frequency, urgency, pain on voiding) and obstructive (hesitancy, poor flow, acute retention) lower urinary tract symptoms, combined with fever, chills, and malaise. The infection may be severe enough to cause septicaemia.

The patient shows signs of systemic toxicity (fever, tachycardia, hypotension), combined with suprapubic tenderness and a

palpable bladder if in urinary retention. On digital rectal examination the prostate is extremely tender.

Treatment consists of intravenous antibiotics, pain relief and relief of retention if present. Traditional teaching recommended a suprapubic catheter be inserted, rather than a urethral catheter, to avoid the potential obstruction of prostatic urethral ducts by a urethral catheter with retention of infected secretions and pus. However, in-and-out catheterisation or short periods with an indwelling catheter probably do no harm, and this is certainly an easier way of relieving retention than suprapubic catheterisation.

Prostatic Abscess

Failure to respond to the treatment regimen outlined above (persistent symptoms and persistent fever while on antibiotic therapy) suggests the development of a prostatic abscess. A transrectal ultrasound, or computed tomography (CT) scan if the former proves too painful, is the best way of diagnosing a prostatic abscess (Fig. 4.1). This may be drained by a transurethral incision or deroofing using a resectoscope.

FOURNIER'S GANGRENE

Fournier's gangrene (Fig. 4.2) is a necrotising fasciitis affecting the genitalia and perineum. It primarily affects males. Necrosis



FIGURE 4.1. A computed tomography scan of a prostatic abscess.



FIGURE 4.2. Fournier's gangrene. (See this figure in full color in the insert.)

and subsequent gangrene of infected tissues occurs. Culture of infected tissue reveals a combination of aerobic (e.g., *E. coli*, enterococcus, *Klebsiella*) and anaerobic organisms (*Bacteroides*, *Clostridium*, microaerophilic streptococci), which are believed to grow in a synergistic fashion. Conditions that predispose to the development of Fournier's gangrene include diabetes, local trauma to the genitalia and perineum (e.g., zipper injuries to the foreskin), and surgical procedures such as circumcision.

Presentation

The presentation is often dramatic. A previously well patient may become systemically unwell over a very short time course (hours) following a seemingly trivial injury to the external genitalia. A fever is usually present. The patient looks very unwell, may have marked pain in the affected tissues, and the developing sepsis may alter their mental status. The genitalia and perineum

are oedematous; on palpation of the affected area there is tenderness, and crepitus may be present, indicating the presence of subcutaneous gas produced by gas forming organisms. As the infection advances, blisters (bullae) appear in the skin and within a matter of hours areas of necrosis may develop, which spread to involve adjacent tissues, e.g., the lower abdominal wall. The condition advances rapidly, hence its alternative name of spontaneous fulminant gangrene of the genitalia.

Though blood tests may be abnormal (e.g., elevated white count), the diagnosis is a clinical one, and is based on awareness of the condition, and a low index of suspicion.

Treatment

Do not delay. While intravenous access is obtained, blood is taken for culture, intravenous fluids are started and oxygen administered, and broad-spectrum antibiotics are given to cover both gram-positive and -negative aerobes and anaerobes, e.g., ampicillin, gentamicin, and metronidazole or clindamycin. Make arrangements to transfer the patient to the operating room as quickly as possible so that debridement of necrotic tissue (skin, subcutaneous fat) can be carried out. Extensive areas of tissue may have to be removed, but it is unusual for the testes or deeper penile tissues to be involved, and these can usually be spared. A suprapubic catheter is inserted to divert urine and allow monitoring of urine output.

Where facilities allow, consider treatment with hyperbaric oxygen therapy. There is some evidence that this may be beneficial (Pizzorno et al. 1997). Repeated debridements to remove residual necrotic tissue are not infrequently required.

Mortality is on the order of 20% to 30%. There is debate about whether diabetes increases the mortality rate (Chawla et al. 2003, Nisbet and Thompson 2002).

EPIDIDYMO-ORCHITIS

This is an inflammatory condition of the epididymis, often involving the testis, and caused by bacterial infection. It presents with pain, swelling, and tenderness of the epididymis. It should be distinguished from chronic epididymitis where there is long-standing pain in the epididymis, but usually no swelling.

Infection ascends from the urethra or bladder. In men aged <35 years, the infective organism is usually *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, or coliform bacteria (causing a urethritis that then ascends to infect the epididymis). In children and older men, the infective organisms are usually coliforms.

A rare, noninfective cause of epididymitis is the antiarrhythmic drug amiodarone, which accumulates in high concentrations within the epididymis, causing inflammation (Gasparich 1984). It can be unilateral or bilateral and resolves on discontinuation of the drug.

Differential Diagnosis

Torsion of the testicle is the main differential diagnosis. A preceding history of symptoms suggestive of urethritis or urinary infection (burning when passing urine, frequency, urgency, and suprapubic pain) suggests that epididymitis is the cause of the scrotal pain, but these symptoms may not always be present in epididymitis. In epididymitis, pain, tenderness, and swelling may be confined to the epididymis, whereas in torsion the pain and swelling are localised to the testis. However, there may be overlap in these physical signs.

Where doubt exists—where you are unsure whether you are dealing with a torsion or epididymitis—exploration is the safest option. Though radionuclide scanning can differentiate between a torsion and epididymitis, this is not available in many hospitals. Colour Doppler ultrasonography, which provides a visual image of blood flow, can differentiate between a torsion and epididymitis, but its sensitivity for diagnosing torsion is only 80%, i.e., it misses the diagnosis of torsion in as many as 20% of cases (these 20% of cases have torsion, but normal findings on Doppler ultrasonography of the testis). Its sensitivity for diagnosing epididymitis is about 70%. Again, if in doubt, explore.

Treatment of Epididymitis

Culture the urine, any urethral discharge, and blood (if systemically unwell). Treatment consists of bed rest, analgesia, and antibiotics. Where *C. trachomatis* is a possible infecting organism, prescribe a 10- to 14-day course of tetracycline 500 mg four times a day or doxycycline 100 mg twice daily. If gonorrhoea is confirmed on a Gram stain of the urethral discharge (if present) and on culture, prescribe ciprofloxacin (though check the sensitivity on culture). For non–sexually transmitted disease (STD)-related epididymitis, prescribe antibiotics empirically (until culture results are available) according to your local microbiology department's advice, which will be based on local patterns of organisms isolated from urine cultures and on local patterns of antibiotic resistance. Our empirical antibiotic regimen is ciprofloxacin for 2 weeks where there is no systemic upset. When the patient is systemically unwell, we admit them for

intravenous cefuroxime 1.5 g t.i.d. and intravenous gentamicin 5 mg/kg, until they are apyrexial, at which time we switch to oral ciprofloxacin for 2 weeks.

Complications of Epididymitis

These include abscess formation, infarction of the testis, chronic pain, and infertility.

PERIURETHRAL ABSCESS

This can occur in patients with urethral stricture disease, in association with gonococcal urethritis and following urethral catheterisation. These conditions predispose to bacteria (gram-negative rods, enterococci, anaerobes, gonococcus) gaining access through Buck's fascia to the periurethral tissues. If not rapidly diagnosed and treated, infection can spread to the perineum, buttocks, and abdominal wall.

The majority (90%) of patients present with scrotal swelling and a fever. Approximately 20% will have presented with urinary retention, 10% with a urethral discharge, and 10% having spontaneoulsy discharged the abscess through the urethra.

The abscess should be incised and drained, a suprapubic catheter placed to divert the urine away from the urethra, and broad-spectrum antibiotics commenced (gentamicin and cefuroxime) until antibiotic sensitivities are known.

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Chapter 5
Traumatic Urological Emergencies

John Reynard

RENAL INJURIES (Table 5.1)

The kidneys are retroperitoneal structures surrounded by perirenal fat; posteriorly are situated the vertebral column, associated spinal muscles, and the lower ribs, and anteriorly the contents of the abdomen. As such they are relatively protected from traumatic injuries. Because of this relatively protected position, a considerable degree of force is usually required to injure a kidney. Not surprisingly, therefore, there may be associated injuries to, for example, the spleen, liver, mesentery of the bowel, or other organs. Furthermore, renal injuries may not initially be obvious, hidden as they are by other structures. Thus, to confirm (or exclude) a renal injury, one must have a high index of suspicion that such an injury could have occurred, and arrange appropriate imaging studies.

In children, there is proportionately less perirenal fat to cushion the kidneys against injury, and thus renal injuries occur with lesser degrees of trauma.

Mechanisms and Cause

The nature of the injury provides useful information about the likelihood that a renal injury has occurred. There are two broad categories of renal injury—those due to blunt trauma and those due to penetrating trauma.

Blunt injuries occur either as a result of a direct blow to the kidney or a rapid acceleration or rapid deceleration (or a combination of two or all three). The commonest cause of renal injuries in urban societies is motor vehicle accidents, either where a pedestrian has been hit by a car (direct injury combined with rapid acceleration and then deceleration) or where, for example, the occupants of a car have come to a sudden halt (rapid deceleration). Seemingly trivial injuries such as a fall from a ladder while gardening, direct falls onto the flank, or sporting injuries can lead to significant renal injuries (Fig. 5.1).

TABLE 5.1. Summary of mechanisms, causes, staging, and treatment of renal injuries

Mechanisms and cause	Blunt or penetrating Blunt—direct blow or acceleration/deceleration (road traffic accidents, falls from a height, fall onto flank) Penetrating—knives, gunshots, iatrogenic, e.g., percutaneous nephrolithotomy (PCNL)
Imaging and staging	Computed tomography—accurate, rapid, images other intra-abdominal structures Staging—American Association for the Surgery of Trauma Organ Injury Severity Scale: I, contusion; II, <1 cm laceration; III, >1 cm laceration; IV, laceration into collecting system; V, shattered kidney
Treatment	Conservative—95% of blunt injuries, 50% of stab injuries, 25% of gunshot wounds can be managed nonoperatively (cross-match, bed rest, observation) Exploration if Persistent bleeding (persistent tachycardia and/or hypotension not responding to appropriate fluid and blood replacement) Expanding perirenal haematoma Pulsatile perirenal haematoma

A penetrating injury such as a stab wound to the flank can be associated with an underlying renal injury, but remember also that lower chest and anterior abdominal stab wounds may inflict renal damage. In the case of gunshot wounds to the abdomen or chest, it is not always obvious that the kidneys might have been injured. The very fact that a patient has sustained a lower chest or abdominal gunshot wound is an indication for renal imaging, in the form of a computed tomography (CT) scan, since the bullet may pass through the kidney as it 'tumbles' around the abdomen. The bottom line is, be suspicious that the kidney has been injured until proven otherwise.

Suspect a renal injury, and arrange renal imaging, in trauma cases with:

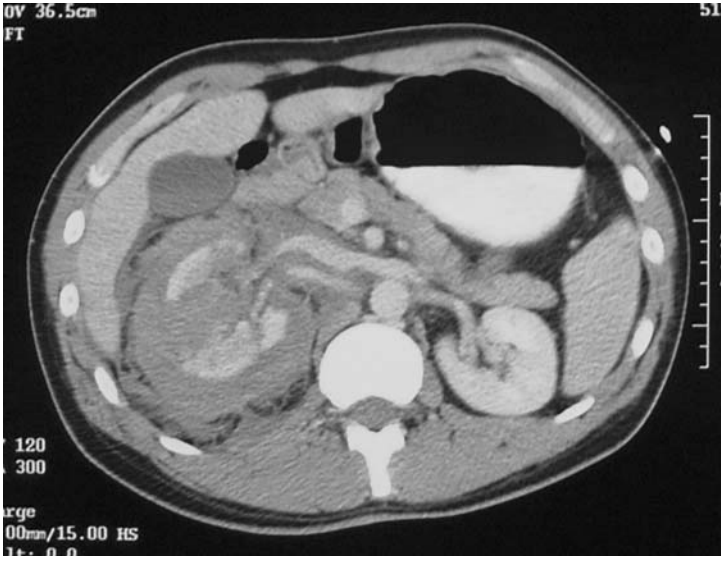


FIGURE 5.1. Computed tomography urogram (CTU) of blunt trauma to the right kidney following a fall onto the flank.

- Macroscopic haematuria
- Penetrating chest, flank, and abdominal wounds (knives, bullets)
- Microscopic [>5 red blood cells (RBCs) per high powered field] or dipstick haematuria in a hypotensive patient (hypotension is defined as a systolic blood pressure of <90 mm Hg recorded at any time since the injury) (Mee et al. 1989, Nicolaisen et al. 1985)
- A history of a rapid acceleration or deceleration
- Any child with microscopic or dipstick haematuria who has sustained trauma

Haematuria is not always present in cases of renal injury, nor does the degree of haematuria correlate with the degree of renal injury. In particular, haematuria may be absent in renal vascular injuries and those where the ureter or pelviureteric junction (PUJ) has been avulsed.

Adult patients with a history of blunt trauma and microscopic or dipstick haematuria need not have their kidneys imaged as long as there is no history of acceleration/deceleration and no

shock, since the chances of a significant injury being found are <0.2% (Miller and McAninch 1995).

What Imaging Study?

The intravenous urogram (IVU) has been replaced by the contrast-enhanced CT scan as the imaging study of choice in patients with suspected renal trauma. It provides clear definition of the injury, allowing injuries to the parenchyma and collecting system to be accurately graded (staged). The IVU is not as accurate as CT. The grade of injury provides a guide to subsequent management. Spiral CT (performed either without contrast or within a few minutes of contrast administration) does not allow accurate staging, because contrast will not yet have had time to reach the parenchyma or collecting system. A repeat CT scan 10 or 15 minutes after contrast administration will demonstrate parenchymal or collecting system injuries accurately.

Renal ultrasonography can be used in the evaluation of renal injuries. However, all of the studies upon which our current management of renal injuries are based, have used CT. It remains to be established whether, at least in some cases, ultrasonography can stage such injuries accurately enough to allow CT to be dispensed with. Ultrasound can certainly establish the presence of two kidneys and the presence of a retroperitoneal haematoma and with power Doppler can identify the presence of blood flow in the renal vessels. However, it cannot accurately identify parenchymal tears, collecting system injuries, or extravasation of urine until a later stage when a urine collection has had time to accumulate.

Contrast-enhanced CT allows the following questions to be answered:

- How deep is the parenchymal laceration?
- Does the parenchyma enhance, i.e., is it perfused?
- Is there extravasation of urine?
- How big and where is the retroperitoneal haematoma?
- Are other organs injured (bowel, spleen, liver, pancreas, etc.)?

Major injuries to either the collecting system or to the renal vessels is suggested by finding the following on CT:

- Absence of enhancement of the parenchyma suggests a renal artery injury.
- A haematoma medial to the kidney suggests a vascular injury.
- Medial extravasation of contrast suggests disruption of the PUJ or renal pelvis.

Staging (Grading)

Using CT, renal injuries can be staged (graded) according to the American Association for the Surgery of Trauma Organ Injury Severity Scale (Fig. 5.2):

Grade	Description
I	Contusion (normal CT) or subcapsular haematoma with no parenchymal laceration
II	<1 cm deep parenchymal laceration of cortex, no extravasation of urine (i.e., collecting system intact)
III	>1 cm deep parenchymal laceration of cortex, no extravasation of urine (i.e., collecting system intact)
IV	Parenchymal laceration involving cortex, medulla, and collecting system, *or* renal artery or renal vein injury with contained haemorrhage
V	Completely shattered kidney *or* avulsion of renal hilum

Intravenous Urography for Renal Imaging

Where a patient is transferred immediately to the operating theatre without having had a CT scan and a retroperitoneal haematoma is found, a single-shot abdominal x-ray taken 10 minutes after contrast administration (2 mL/kg of contrast) can be used to establish whether or not there is a renal injury (Morey et al. 1999). If the patient is hypotensive, take the image at between 20 and 30 minutes, so that there has been time for excretion of a sufficient quantity of contrast to allow opacification of the kidney. On-table IVU can also be very useful in determining the presence of a normally functioning contralateral kidney where the injury to the ipsilateral kidney is likely to necessitate a nephrectomy. In the San Francisco General Hospital experience a single-shot IVU, in many cases, has provided an image

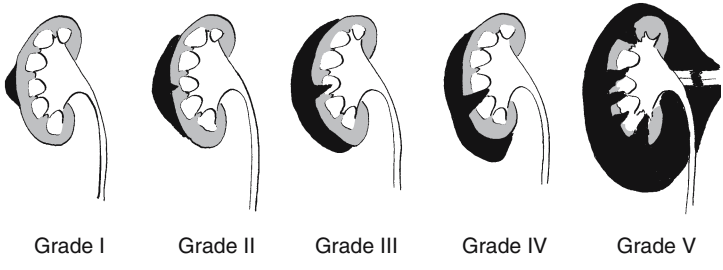


FIGURE 5.2. American Association for the Surgery of Trauma Organ Injury Severity Scale for renal injuries.

of sufficient quality to allow accurate intraoperative decision making to be made, and in approximately 30% of cases the intraoperative IVU findings obviated the need for renal exploration.

Subsequent Treatment

In general terms, renal exploration is indicated for:

Persistent bleeding (persistent tachycardia and/or hypotension failing to respond to appropriate fluid and blood replacement
Expanding perirenal haematoma (again the patient will show signs of continued bleeding)
Pulsatile perirenal haematoma

The categorisation of renal injuries into blunt and penetrating types determines the likely need to explore the kidney to stop bleeding and/or repair the renal injury. Over 95% of blunt injuries can be managed conservatively and, at least in centres where a high frequency of renal injuries is seen, a substantial proportion of penetrating injuries can be managed without renal exploration. In the San Francisco General Hospital, a centre with an international reputation for the management of renal injuries, approximately 50% of renal stab injuries and 25% of renal gunshot wounds can be managed nonoperatively.

As stated above, adult patients with a history of blunt trauma, microscopic or dipstick haematuria, no shock, and no history of acceleration/deceleration do not require renal imaging and can be discharged from the emergency department. Those with macroscopic haematuria should undergo a staging CT and be admitted for bed rest and observation, until the macroscopic haematuria resolves. Most such patients will have injuries of stage (grade) I to III.

High-grade (IV and V) injuries can be managed nonoperatively, as long as the patient is cardiovascularly stable. Urinary extravasation is not in itself necessarily an indication for exploration. Almost 90% of these injuries can heal spontaneously (Matthews et al. 1997).

Traditionally, a large volume of nonviable renal tissue is a relative indication for renal exploration and repair, as is urinary extravasation, and the finding of an expanding retroperitoneal haematoma at operation (Husmann and Morris 1990). However, a recent report from Los Angeles suggests that outcome is favourable even in patients with a devitalised segments of kidney and with urinary extravasation (Toutouzas et al. 2002). Small degrees of urinary extravasation from a minor laceration into the

collecting system of the kidney will usually resolve spontaneously. If the degree of extravasation is greater, consider placing a JJ stent. Repeat the renal imaging if the patient develops a prolonged ileus or a fever, since these signs may indicate the development of a urinoma, which can be drained percutaneously.

The Approach to Renal Exploration

The principal reason for renal exploration will be persistent bleeding causing shock. For this reason, most surgeons will elect to approach the renal pedicle first, to allow control of the renal artery and vein. This is most easily achieved by a midline incision. Such an incision has the advantage that it can be done quickly and it can also be extended up and down to allow access to the entire abdominal and pelvic cavities, for repair of injuries to other organs.

Lift the small bowel upward to allow access to the retroperitoneum. Incise the peritoneum over the aorta, above the inferior mesenteric artery (Fig. 5.3a). A large perirenal haematoma may obscure the correct site for this incision. If this is the case, look for the inferior mesenteric vein and make your incision medial to it. Once on the aorta, the inferior vena cava may be exposed, then the renal veins and the renal arteries. Pass slings around all of these vessels so you can control bleeding by compressing the renal artery and vein (Fig. 5.3b).

The kidney can now be exposed by mobilising the colon. Divide the white line of Toldt lateral to the ascending (right side) or descending (left side) colon and pull the colon upward to expose the kidney, which will be surrounded by a large haematoma.

Bleeding may be reduced by applying pressure to the vessels via the slings. Control bleeding vessels within the kidney with 4/0 Vicryl or monocryl sutures. Close any defects in the collecting system with 4/0 Vicryl. If the sutures cut out, place a strip of Surgicel over the site of bleeding, place the sutures through the capsule on either side of this, and tie them over the Surgicel. This will stop them from cutting through the friable renal parenchyma.

Iatrogenic Renal Injury: Renal Haemorrhage After Percutaneous Nephrolithotomy

Significant renal injuries can occur during percutaneous nephrolithotomy (PCNL) for kidney stones. This is the surgical equivalent of a stab wound and serious haemorrhage (necessitating some form of intervention) occurs in approximately 1% of cases (Martin et al. 2000).

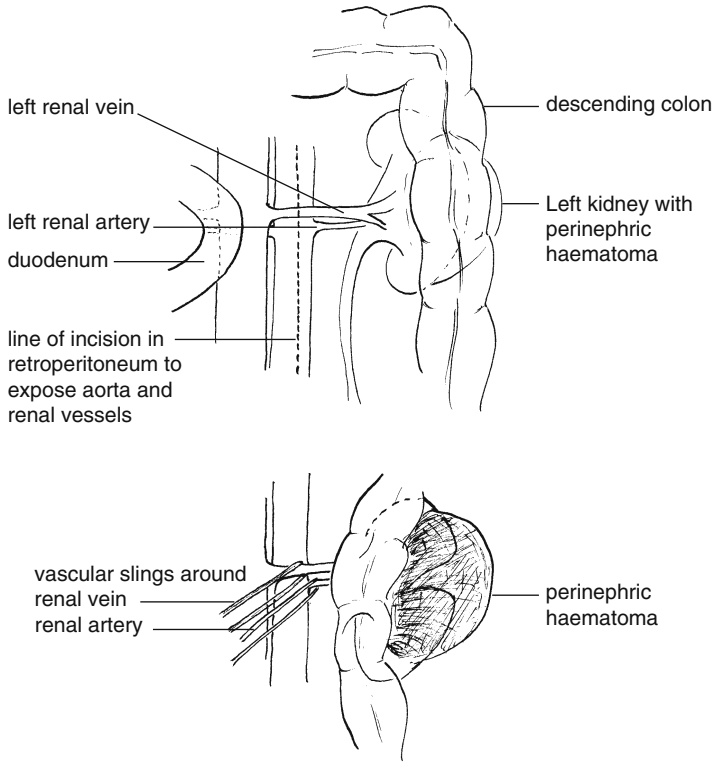


FIGURE 5.3. a: The surgical approach for control of the renal vascular pedicle. b: Gaining vascular control with slings around the renal veins and artery.

Bleeding during or after a PCNL can occur from vessels in the nephrostomy track itself, from an arteriovenous fistula or from a pseudoaneurysm that has ruptured. Track bleeding will usually tamponade around a large-bore nephrostomy tube. Traditionally persistent bleeding through the nephrostomy tube is managed by clamping the nephrostomy tube and waiting for the clot to tamponade the bleeding. While this may control bleeding in some cases, in others a rising or persistently elevated pulse rate (with later hypotension) indicates the possibility of persistent bleeding and is an indication for renal arteriography and embolisation of the arteriovenous fistula or pseudoaneurysm (Fig. 5.4). Failure to stop the bleeding by this



a

FIGURE 5.4. a: Renal arteriography after percutaneous nephrolithotomy (PCNL) where severe bleeding was encountered. An arteriovenous (AV) fistula was found. Note the 'blush' of contrast in the lower pole which represents the AV fistula. b: The AV fistula has been embolised. Note the metal embolisation coils.

technique is an indication for renal exploration and control of the bleeding by the techniques described above for penetrating renal injuries.

Arteriovenous fistulae can sometimes occur following open renal surgery for stones or tumours, and arteriography with embolisation again can be used to stop the bleeding in these cases. The bleeding in such cases usually occurs over a longer time course (days or even weeks), rather than as acute haemorrhage causing shock.



b

FIGURE 5.4. *Continued*

URETERIC INJURIES

Causes and Mechanisms of Ureteric Injury

External Trauma

The ureters are retroperitoneal in location, and as such are protected from external trauma by surrounding bony structures, muscles and other organs (Elliott and McAninch 2003). For external trauma to injure the ureters, severe force is required. External trauma to the ureter is rare, and may be blunt or penetrating. Blunt external trauma severe enough to injure the ureters will usually be associated with multiple other injuries (for the ureter to be the only organ injured in a high-velocity motor vehicle accident is very rare). Clearly, a knife or bullet wound to the abdomen or chest may damage the ureter, as well as other organs.

Very occasionally the ureter is the only organ that is injured following blunt external or penetrating trauma. For example, a fall from a height in a patient with a pre-existing PUJ obstruction can result in the hydronephrotic kidney being avulsed from the ureter. Blood may well be absent from the urine in these rare cases, and the diagnosis is made only by having a high index of suspicion and by carrying out renal imaging (CT or IVU) in all cases where there has been a rapid acceleration-deceleration injury. These injuries are said to be more frequent in children (though they are still rare), where the kidneys are more mobile because of a less well developed surrounding cushion of perinephric fat. This allows the kidneys to move more freely relative to the less mobile ureter.

Internal Trauma

Internal trauma to the ureter is uncommon, but is more common than external trauma. Surgeons are the culprits! The ureters are most vulnerable to 'surgical' injury in the pelvis. Consider the anatomical relationships of the ureters. The left ureter is crossed by the left colic vessels and by those to the sigmoid colon at the pelvic brim. In the pelvis, the ureters pass anterior to the iliac vessels, and then they turn medially at the level of the ischial spines to approach the bladder. In females, the ureter lies at the base of the broad ligament and is crossed anteriorly by the uterine artery just before entering the bladder. Not surprisingly, therefore, the ureters are vulnerable to injury during hysterectomy, oophorectomy, and sigmoid colectomy. The other obvious source of surgical injury, (probably the commonest cause) is ureteroscopy. Less commonly, the ureter may be damaged during caesarean section, aortoiliac vacular graft placement, laparoscopic procedures, and orthopaedic operations including spinal surgery and total hip replacement (Fig. 5.5). The ureter may be cut in one place, a segment may be excised along with the organ being removed, it may be ligated or angulated by a suture, or it may sustain a diathermy injury or undergo ischaemic necrosis if the blood supply to one segment is damaged.

Making the Diagnosis

This requires a high index of suspicion, particularly in cases of external trauma where the focus of attention may be on other, obvious injuries. In the case of hysterectomy or colectomy, injury to the ureter is usually, but not always, apparent at the time of surgery. The cut ureter has a characteristic appearance—familiar to those who have inadvertently divided it! It looks like no



FIGURE 5.5. Extruded cement from a total hip replacement with a JJ stent in the ureter. There was no ureteric injury in this case, but it demonstrates that the anatomical path of the ureter may be distorted, and therefore the ureter potentially injured, during total hip replacement.

other divided tubular structure, and it does not bleed in the same way as a cut vessel.

In surgical injuries to the ureter where the diagnosis is not made intraoperatively, the symptoms and signs that the ureter may have been injured include:

1. An ileus, due to the presence of urine within the peritoneal cavity
2. Prolonged postoperative fever or overt urinary sepsis
3. Persistent drainage of fluid from abdominal or pelvic drains, from the abdominal wound, or from the vagina. This fluid should be sent to the lab for creatinine estimation. If the creatinine level is higher than that of serum, the fluid is urine (the creatinine level will be at least 300 μmol/L).
4. Flank pain if the ureter has been ligated
5. An abdominal mass, representing a urinoma
6. Vague abdominal pain
7. The pathology report on the organ that has been removed may note the presence of a segment of ureter!

The diagnosis may be made within the first few days following surgery, but it may be delayed by weeks, months, or even years. In such cases, the presentation may be one of flank pain. Post-hysterectomy incontinence, which will usually be continuous in nature, may be due to a persistent leakage of urine (from a ureterovaginal fistula).

Making the Diagnosis Intraoperatively

Ureteric contusions and small ureteric perforations probably occur frequently during ureteroscopic stone fragmentation. Perforation by a laser fibre or guidewire is unlikely to result in significant extravasation, but in the latter case you might feel more comfortable in leaving a JJ stent in situ for a week or so after the procedure. If you do inadvertently push the ureteroscope through the wall of the ureter, then clearly the size of the hole in the ureter is bigger and the likelihood of extravasation of a significant volume of urine is increased. A JJ stent should be inserted in such cases, and if it is not possible to do so, because for example the safety guidewire has fallen out, then serious consideration should be given to placing a percutaneous nephrostomy tube. If a radiologist who is expert in antegrade stent placement is available, then this can be inserted at the same time that the antegrade stent is positioned. This gives the added advantage of 'dual drainage' (Fig. 5.6). In many cases, temporary urine drainage, either by a JJ stent alone or combined with a percutaneous nephrostomy, is all that is required for definitive management of such injuries.

You may be asked to give an intraoperative opinion by your gynaecological, colorectal, or vascular colleagues who suspect that they have damaged the ureter, or they may simply want reas-



FIGURE 5.6. Combined JJ stent and nephrostomy drainage of the ureter following perforation of the ureter during ureteroscopy.

surance that they have not. The atmosphere in the operating theatre will be tense and it is important to keep your cool, so that you can go about confirming or excluding a ureteric injury, and repairing it if present, in a systematic and sensible fashion.

Optimise the conditions. Ensure that the area of interest is adequately exposed by packing the bowel out of the way if this

has not been done. Different surgeons use different retractors. If you are not happy with the type of retractor being used, ask for one that you like (we prefer a Bookwalter retractor). There may be continued bleeding, and the area of interest may be an inch under urine or blood. You cannot adequately examine a ureter or repair it under such conditions. Control any bleeding. It is better to do this now, before a potential repair, than afterward where the presence of the reconstruction may make access to bleeding vessels difficult. Make sure the operating lights provide adequate light. Ask for a headlamp if you are not happy with the light from the overhead operating lights. Ask the anaesthetist to give some intravenous antibiotics, fluids, and blood as required.

The options for examining the ureters are several. Remember, bilateral injuries can occur, particularly following hysterectomy, and therefore you should examine both ureters. You may look directly at the ureters, administer intravenous or intraureteric methylene blue and look for extravasation of dye, do an on-table IVU, or perform retrograde ureterography.

Direct Inspection of the Ureter

This is a good way of inspecting the ureter for injury, but a considerable length of ureter may have to be exposed in order to establish that it has not been injured, and for the lower ureter this exposure is more difficult than for the upper ureter.

Extravasation After Injection of Methylene Blue into the Ureter or Collecting System

Direct injection of diluted methylene blue into an exposed segment of normal ureter (or directly into the renal pelvis if this has been exposed) can be used to demonstrate the integrity of the ureter, leakage of dye from a more distant section of ureter confirming the presence of an injury. Be careful, however, not to spill any of the dye, because if this occurs it stains the surrounding tissues, making it impossible to see a leak. There will be no leak of dye in a ligation injury so the methylene blue method will 'miss' such injuries.

On-Table Intravenous Urography and Retrograde Ureterography

The conditions for performing on-table x-rays are not always ideal. The patient may be on an operating table through which x-rays cannot pass! The hospital portable x-ray C-arm may be in use or the radiographer may be busy elsewhere. In a shocked patient, possibly with a ureter obstructed by a ligature, contrast may not be excreted from the affected kidney in sufficient con-

centration to allow interpretation of the IVU. The results of on-table intravenous urography have been reported in the context of renal trauma management (Morey et al. 1999). While not strictly comparable with iatrogenic ureteric injury, the results from the San Francisco General Hospital experience demonstrate the difficulty of determining the presence or absence of ureteric injuries using intravenous urography. In 50 patients undergoing on-table IVU, complete radiologic demonstration of one or both ureters was possible in only 36% of cases.

The technique of on-table IVU has been discussed elsewhere (p. 58).

Retrograde ureterography can be performed via an incision made in the bladder, or via a cystoscope. This is a very accurate method of establishing the presence or absence of a ureteric injury, and the contralateral ureter can easily be examined using this technique to exclude a bilateral injury. However, similar logistical problems can be encountered to those with on-table IVU. We use a 4 or 6 cm ureteric catheter, with a hole at the distal end but no side-holes, so that contrast flows up the ureter rather than leaking out of the ureter and into the bladder.

Making the Diagnosis Postoperatively

When a ureteric injury is suspected some days or weeks postoperatively, on the basis of the symptoms and signs discussed above, an IVU or retrograde ureterogram should be done. Ultrasonography may demonstrate hydronephrosis, but hydronephrosis may be absent when urine is leaking out of a transected ureter into the retroperitoneum or peritoneal cavity. The IVU usually shows an obstructed ureter (Fig. 5.7). Occasionally contrast can be seen leaking from the site of injury (Fig. 5.8). It is our policy to perform *bilateral* retrograde ureterography in all cases immediately prior to operation for repair of a ureteric injury (if not done already) in order to determine whether the contralateral ureter has been injured or not.

When to Do the Repair

In general terms, the best time to repair the ureter is as soon as the injury has been diagnosed. This is certainly the case when the injury has been recognized intraoperatively. However, there are situations where delayed repair is a better option, and in these situations temporary urine drainage will need to be achieved until definitive repair can be carried out.

Definitive ureteric repair is best delayed when (a) the patient's condition is such that they would not tolerate a procedure under



FIGURE 5.7. A ureter obstructed by a distal injury. The ureter had been ligated during hysterectomy.



FIGURE 5.8. Leak of contrast from the ureter in a case of distal injury, as demonstrated by retrograde ureterography. Note the drain tube in situ adjacent to the ureteric injury which has been demonstrated by leakage of contrast from the ureter.

general anaesthetic, which is likely to last an hour or more; or (b) there is evidence of active infection at the site of proposed ureteric repair. Thus, if there is an infected urinoma, this should be drained radiologically, intravenous antibiotics given, and ureteric repair delayed until the patient is apyrexial.

Traditional teaching held that surgical repair should be delayed when the injury was diagnosed between roughly day 7 and day 14 after ureteric injury, because this period was believed to represent the time during which oedema and inflammation at the site of injury was maximal. However, favourable outcomes have been demonstrated after early repair (after 7 days) and the time of the original injury is nowadays seen as a less important determinant of time of definitive repair. Blandy and colleagues (1991) reported favourable results of repair (by Boari flap) of

iatrogenic ureteric injuries in 43 patients, 28 (65%) of whom underwent definitive repair within 6 weeks of injury.

Delayed Treatment—Temporizing Procedures

Temporary urine drainage may be achieved by placement of a percutaneous nephrostomy, and if there is a significant urinoma demonstrated by CT or ultrasound, this can be drained percutaneously by a radiologist. If the patient is too unstable for definitive repair, you may insert a nephrostomy on the operating table (by opening the renal pelvis and inserting it from inside out). However, this can take a considerable amount of time, which you may not have in a shocked patient. In such cases, tie the ureter off at the site of the leakage with a long, nonabsorbable suture. This allows dilatation of the ureter so your interventional radiologist can subsequently place a nephrostomy tube under x-ray control a day or so later. The nonabsorbable suture allows easier identification of the ureter when you later come back for definitive repair.

Definitive Treatment

The options include:

- JJ stenting
- Primary closure of partial transection of the ureter
- Direct ureter to ureter anastomosis (primary ureteroureterostomy)
- Reimplantation of the ureter into the bladder (ureteroneocystostomy), either using a psoas hitch or a Boari flap
- Transureteroureterostomy
- Autotransplantation of the kidney into the pelvis
- Replacement of the ureter with ileum
- Permanent cutaneous ureterostomy
- Nephrectomy

JJ Stenting

For some injuries, JJ stenting may be adequate for definitive treatment, particularly where the injury does not involve the entire circumference of the ureter and continuity, therefore, is maintained across the region of the ureteric injury. In situations where a ligature has been applied around the ureter, and this has been immediately recognised such that viability of the ureter has probably not been compromised, the ligature should be removed and a JJ stent should be placed (cystoscopically if this is feasible or, if not, by opening the bladder). If, however, there has been a

delay in recognition of a ligature injury to the ureter, it is probably safer to remove the affected segment of ureter and perform a ureteroureterostomy (Assimos et al. 1994). Generally speaking the stent is maintained in position for somewhere between 3 to 6 weeks. At the time of stent removal a retrograde ureterogram can be done to confirm that there is no persistent leakage of contrast from the original site of injury, and to see if there is evidence of ureteric stricturing (Fig. 5.9).

For other injuries, in general terms, the type of treatment depends on the level of ureteric injury. It has been traditional teaching that the blood supply to the distal ureter is somewhat tenous, and for injuries at this level (below the takeoff of the internal iliac artery) reimplantation directly into the bladder via a psoas hitch or Boari flap is recommended. The approach to repair at different levels of ureteric injury is summarised in Figure 5.10.

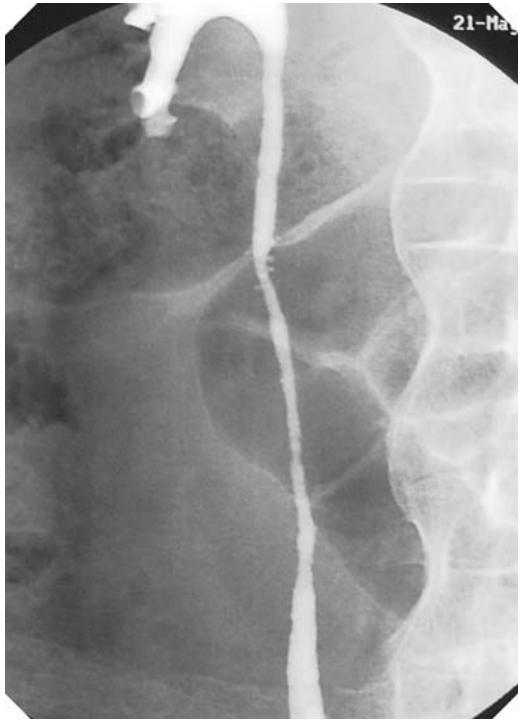


FIGURE 5.9. A retrograde ureterogram post–stent removal.

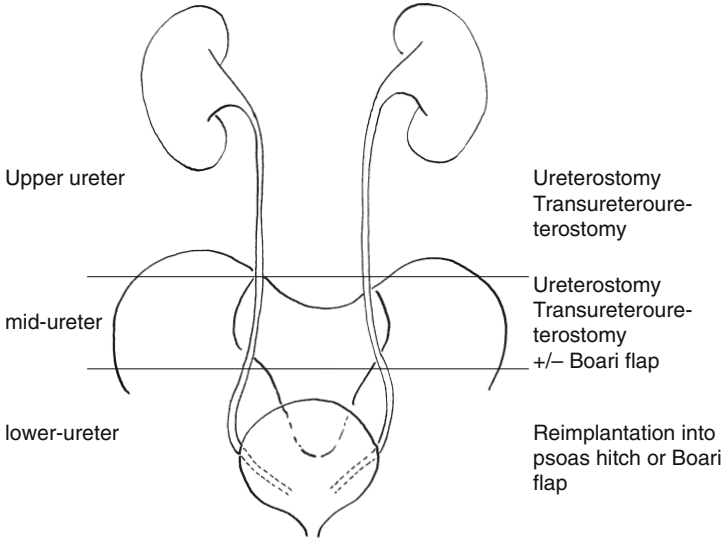


FIGURE 5.10. Surgical techniques for repair of ureteric injuries at different levels of the ureter.

Factors other than the level of injury are important in determining the type of repair. Blast injuries characteristically cause considerable collateral damage to the ureter and surrounding tissues, which may not be apparent at the time of surgery. Delayed necrosis can occur in such apparently normal looking ureters. Simple ureterostomy may therefore be inappropriate in such cases, and debridement of a considerable length of ureter, followed by reimplantation into a Boari flap, may be necessary.

General Principles of Ureteric Repair

- The ends of the ureter should be debrided, so that the edges to be anastomosed are bleeding freely.
- The anastomosis should be tension free.
- For complete transection, the ends of the ureter should be spatulated, to allow a wide anastomosis to be done.
- A stent should be placed across the repair.
- Mucosa-to-mucosa anastomosis should be done, to achieve a watertight closure.
- A drain should be placed around the site of anastomosis.

Primary Closure of Partial Transection of the Ureter

A partial transection of the ureter may be repaired over a JJ stent, as long as the injury has not been caused by a gunshot wound (in which case there may well be a blast effect causing more extensive necrosis than is immediately apparent at the time of surgery; such injuries are better managed by excising the affected segment of ureter and performing a primary ureteroureterostomy). Mobilise the ends of the ureter to allow a tension free anastomosis to be done. Pass a guidewire into the renal pelvis and pass the stent up into the renal pelvis. To introduce the stent into the lower ureter, remove the guidewire and place it in a side hole of the stent, so as to straighten the end of the stent so that it may be introduced into the distal end of the ureter (Fig. 5.11). We find it easier to place the guidewire through a side hole in the *middle* of the stent, because this makes it easier to disengage the wire from the stent. The stent may be pulled out of the bladder as the guidewire is withdrawn if the latter has been placed through a side hole near the *end* of the stent. Thread the stent and guidewire down the ureter and into the bladder. We instill some diluted methylene blue into the bladder via catheter and fill the bladder with saline, clamping the catheter so that the bladder can be distended. When the JJ stent reaches the bladder and the guidewire is withdrawn, blue fluid refluxes up the stent and this confirms that the distal end of the stent is in the bladder. We use 4/0 Vicryl (i.e., absorbable suture material) to close the hole in the ureter. Place a drain down to the site of the repair.

Primary Ureteroureterostomy

This is anastomosis of one end of the ureter to the other end. The essential factor for successful anastomosis is the absence of tension. If the defect between the ends of the ureter is of a length where a tension-free anastomosis would not be possible, then reimplantation into the bladder via a psoas hitch or Boari flap will be needed. The technique for anastomosis of the two ends of the ureter is the same as for partial transections, other than the fact that the two ends of the ureter should be spatulated to allow a wide-bore anastomosis.

Ureteroneocystostomy: Reimplantation of the Ureter into the Bladder, Either Using a Psoas Hitch or a Boari Flap

Identify the end of the proximal ureter. If the injury has been recognised intraoperatively, the end will usually be easily identifiable. If, however, there has been a delay in recognising the

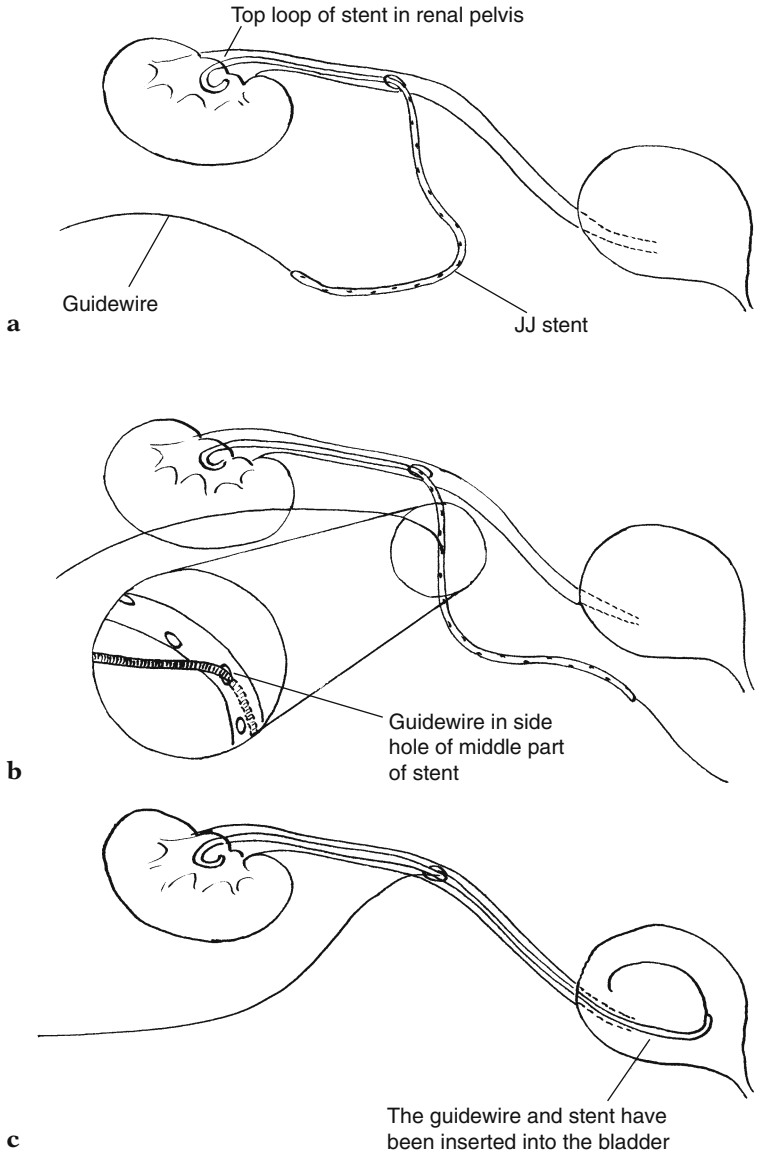


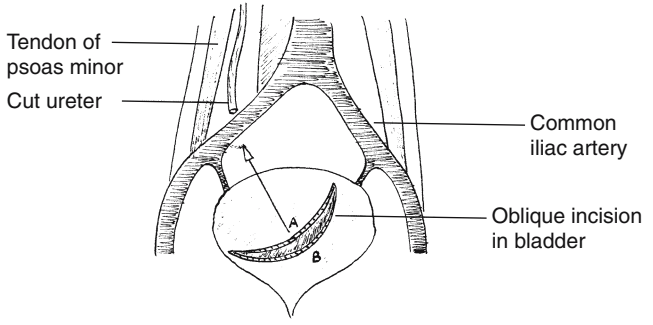
FIGURE 5.11. Technique for introducing a stent into the lower ureter. a: The end hole of the JJ stent is passed over the guidewire, which has been placed in the renal pelvis. The guidewire is withdrawn while holding the stent in place. b: Inserting the guidewire into a side hole halfway along the length of the JJ stent makes it easier to disengage. c: The distal end of the guidewire, with the stent, is then passed down the ureter and into the bladder. The guidewire is then removed.

injury, the end of the ureter may be encased in a mass of fibrous and oedematous tissue. In such cases, trace the ureter down as far as you can, and transect it as it enters the area of fibrosis. Place a stay suture through the end of the ureter.

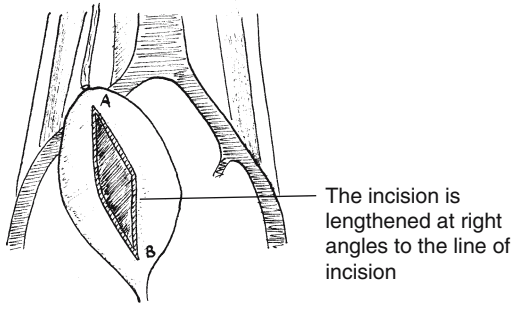
The defect between the bladder and the proximal end of the ureter may be bridged using either a psoas hitch or a Boari flap. A Boari flap is generally able to bridge a greater defect than a psoas hitch, and therefore you must decide before you start to make an incision in the bladder whether you are going to employ a psoas hitch or a Boari flap. It is easier to assess the length of bladder flap or hitch that needs to be created by 'inflating' the bladder with a few hundred millilitres of water (we use water because we make the incision in the bladder with diathermy; saline would prevent the diathermy from cutting). Use a sterile giving set attached to a 1L bag of water. So you can control the inflow and outflow yourself. Mark out the site of the incision in the distended bladder, using a marker pen if you find this easier, and apply stay sutures around the edges of the incision; these sutures make it easier to manipulate the tissues, and they create less tissue damage than using forceps. Measure the defect and make sure you can bridge it, with a few centimeters to spare, with your proposed method (psoas hitch or Boari flap). Remember, if you prefer to reimplant the ureter in a nonrefluxing fashion, you will need an extra 3 cm or so of length, to allow the ureter to be tunneled into the bladder.

Psoas Hitch (Turner-Warwick and Worth 1969)

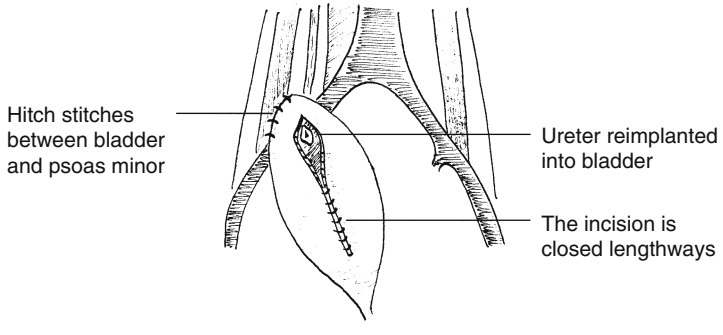
A psoas hitch is fashioned by making an incision in the bladder that lies at right angles to the long axis of the ureter, and this incision is opened out in the same axis as the ureter (Fig. 5.12a). This essentially lengthens the bladder, allowing it to reach the ureter, which can be anastomsed to the bladder without tension. Place two stay sutures on either side of the planned incision (Fig. 5.12b). As the incision is made, intermittently pull the stay suture apart until you have produced an incision that is long enough to breach the defect. Alternatively, place two fingers inside the bladder and elevate the bladder toward the cut ureter. To achieve an adequate length of bladder, you may well have to divide the contralateral superior vesical vessels. The psoas hitch will need to reach well above the iliac vessels so that it can be anchored to the psoas minor tendon (or psoas major tendon if the former is absent) and to achieve this length the incision in the bladder may have to comprise as much as 50% of the circumference of the bladder.



a



b



c

FIGURE 5.12. a: Oblique incision, which is opened at right angles to the line of incision. b: Creating the psoas hitch. c: Placing the hitch stitches.

Hitch stitches are used to anchor the bladder to the psoas minor tendon (Fig. 5.12c). They take tension off of the ureterovesical anastomosis and also prevent tension at this site developing as the bladder fills and empties. We place the hitch stitches (2/0 Vicryl) that will anchor the bladder to the tendon of psoas minor at this time, first so that we can be sure we have achieved an adequate length of bladder for tension-free ureter-to-bladder anastomosis, and second so that we can perform the anastomosis in a position that will avoid kinking the ureter. We clip, but do not tie, the stitches yet, because as Turner-Warwick and Worth (1969) suggested, 'Having sited the position of the hitch-sutures, it is often easier to create the ureteric tunnel before actually anchoring the bladder.' When placing the hitch stitches be careful not to place the sutures too deeply, as it is possible to hit the genitofemoral nerve (which lies on psoas major) and even the femoral nerve (which exits laterally from the psoas major).

Create a hole or a tunnel through which the ureter will be anastomosed to the bladder. Draw the ureter through the tunnel in the bladder. The ureter may be either anastomosed to the bladder in a refluxing fashion or tunnelled through the muscle of the bladder to produce a nonrefluxing anastomosis. In the former situation, place a right-angled forceps on the outside of the bladder at the site of intended reimplantation, cut onto the tip of the forceps, and simply draw the end of the ureter (by the stay suture) into the bladder. Spatulate the end of the ureter on its anterior surface using a Potts scissors. Perform the anastomosis over a JJ stent. Place the first suture through all layers of the posterior wall of the ureter and take a deep bite of the bladder. The remaining sutures may be mucosa to mucosa only.

For a nonrefluxing anastomosis, create a submucosal tunnel in the wall of the bladder. It is easier to do this by starting inside the bladder with a pair of McIndoe or Addson's scissors. Make a small cut in the mucosa of the bladder, and then tunnel under the mucosa with the tips of the scissors, rapidly opening and closing the tips to create the tunnel. After 2 cm or so (allowing a tunnel length to ureteric diameter ratio of approximately 3 : 1), turn the scissors over, and cut onto their ends with diathermy so that the scissors may exit the bladder. Exchange them for a Robert's forceps, which is used to grasp the suture in the end of the ureter. Anastomose the ureter to the bladder in the same way as for the refluxing anastomosis.

The defect in the bladder is then closed, in the same axis as the ureter. Place a drain down to the site of bladder closure and leave the catheter in the bladder for 2 weeks.

Boari Flap

Place stay sutures in the inflated bladder, around the edges of the flap (Fig. 5.13a). The flap will receive all its blood supply from its base and therefore it should be at least 4 cm wide and with a length-to-width ratio of no more than 3 : 1. Fold the flap backward. If more length is required, small transverse incisions can be made in the side of the flap; by pulling lengthways, these can lengthen the flap (Fig. 5.13b). Remember, if you prefer to reimplant the ureter in a nonrefluxing fashion, you will need an extra 3 cm or so of length. Perform the reimplantation as described above and then close the bladder. We find this easier to do by starting at the ureter end, folding the sides of the flap toward each other in the form of a tube. Complete the bladder closure, place a drain down to the site of bladder closure, and leave the catheter in the bladder for 2 weeks.

Transureteroureterostomy (Fig. 5.14)

A transureteroureterostomy is used where the bladder cannot be mobilised or is of small volume (e.g., post-radiotherapy), such that a psoas hitch or Boari flap cannot be made without tension at the ureterovesical anastomosis. The damaged ureter is swung over to the normal ureter and the two are anastomosed together.

First check that the 'recipient' ureter has not been injured. Perform an on-table retrograde ureterogram. There must be an adequate length of ureter to swing over to the opposite ureter. Remember, just above the pelvic brim the ureters are the closest together of any point throughout their course (6 or 7 cm apart), and therefore at this point the least amount of mobilisation will be required.

Ideally the caecum should be mobilised to avoid having to tunnel the ureter through the retroperitoneum, which runs the risk of angulating or constricting the ureter. The 'donor' ureter (the cut ureter) may be brought over to the opposite ureter below or above the inferior mesenteric artery, but if brought below, be careful that it does not make an acute angle beneath the artery, as it will be obstructed. Make a longitudinal incision in the recipient ureter that is slightly longer than the diameter of the donor ureter. By cutting the end of the donor ureter obliquely (Fig. 5.14), you can increase its length slightly and this may help reduce the chances of postoperative obstruction.

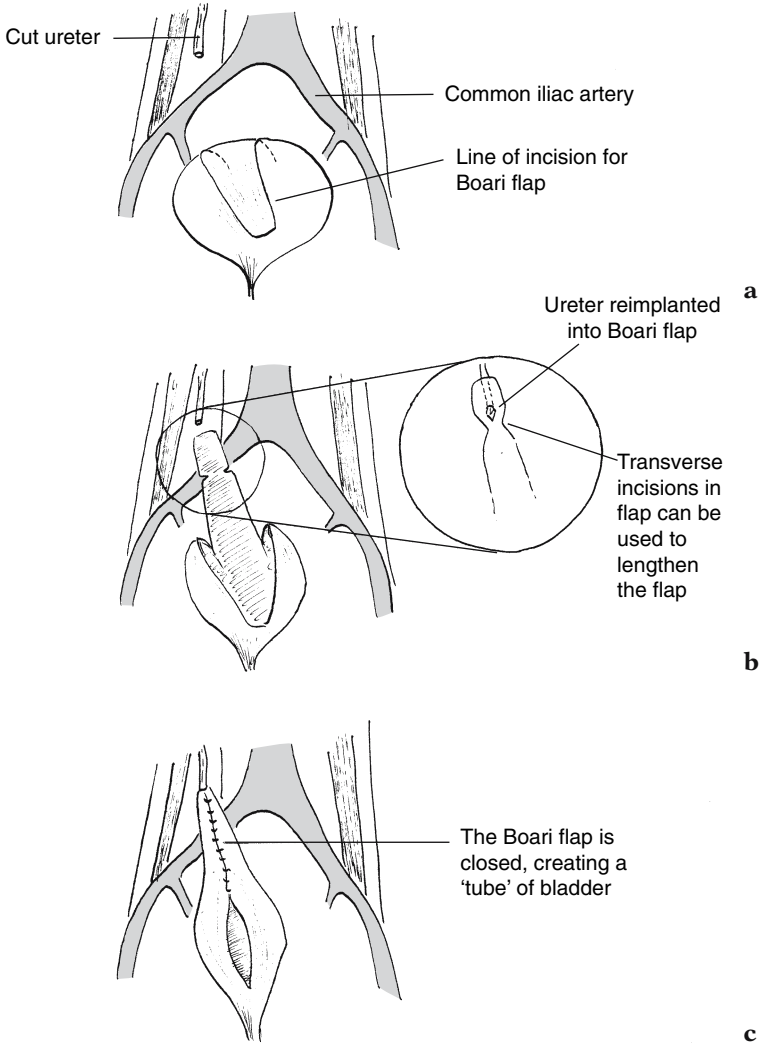


FIGURE 5.13. a: Creating a Boari flap. b: Lengthening the Boari flap. c: Closing the Boari flap.

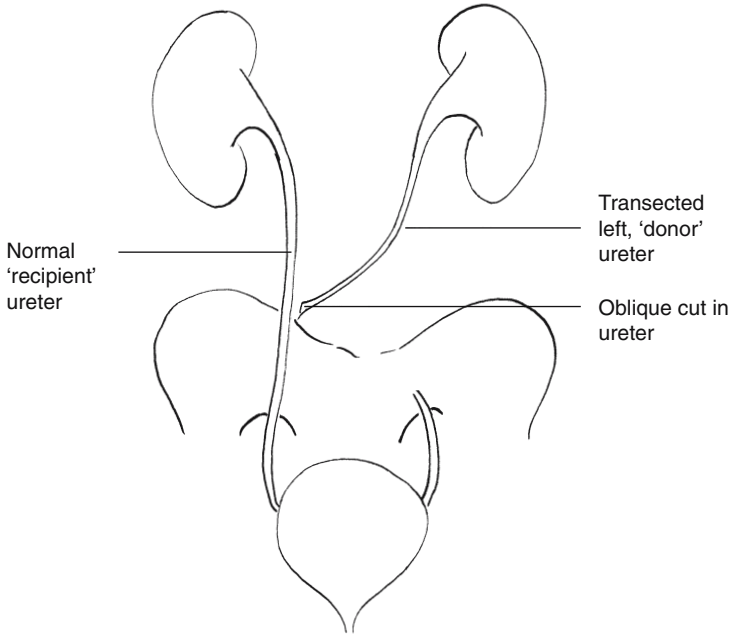


FIGURE 5.14. A transureteroureterostomy.

Place a 4/0 Vicryl suture from outside to inside at the top end of the recipient ureter and then pass it from inside to outside of the donor ureter. Do the same at the bottom end. Complete the back wall of the anastomosis from inside the ureter, and the front wall from the outside. Before completing the anastomosis, place a JJ stent passing from the donor ureter, across the anastomosis, and down into the recipient ureter, and complete the anastomosis. There is usually not enough space to place a second stent all the way along the recipient ureter. Place a drain down to the site of the anastomosis.

Alternative Procedures for Managing Ureteric Injuries

Alternative procedures, where the segment of damaged ureter is very long, include autotransplantation of the kidney into the pelvis and replacement of the ureter with ileum. Specialised surgical texts may be consulted for details on how to perform such procedures. Very occasionally ureteric injuries may be managed

by a permanent cutaneous ureterostomy, where the patient's life expectancy is very limited.

When a ureter has been injured in a patient who has undergone a vascular graft procedure, e.g., an aortobifemoral graft, the traditional teaching advocated nephrectomy because of the potential for graft infection as a consequence of infection of urine which might leak from the site of a ureteric anastomosis. However, renal failure is a significant cause of death after aneurysm repair, particularly in the context of emergency (ruptured) aneurysm repair. Preservation of as much functioning renal tissue as possible, therefore, is clearly desirable in such patients, and this would tend to sway one away from nephrectomy. McAninch (2002) recommends repair of the ureteric injury, with nephrectomy being performed only in those cases where a urine leak develops postoperatively (as evidenced by continuing drainage of urine from the drain placed at the site of the ureteric anastomosis).

PELVIC FRACTURES AND INJURIES TO THE URINARY SYSTEM

Nowadays, pelvic fractures are usually due to run-over or crush injuries, where massive force is applied to the pelvis. Not suprisingly, associated head, chest, intra-abdominal (spleen, liver, mesentery of bowel), pelvic (bladder, urethra, vagina, rectum), and genital injuries are common. These, along with massive blood loss from the pelvic fracture itself, account for the substantial (20%) mortality after pelvic fracture. Bleeding occurs from the fractured bone surfaces, tears in large pelvic veins and small pelvic arteries, as well as from chest and abdominal injuries. Injuries to large arteries are rare, but blood loss from small arterial and venous injury can be massive. A large pelvic haematoma is common and this may track up into the retroperitoneum.

Pelvic fractures are often occult. Patients with run-over or crush injuries should be screened with an anteroposterior x-ray of the pelvis. The initial assessment of patients with pelvic fractures includes checking the patient's vital signs, a neurovascular examination of the lower limb (the lumbosacral plexus and peripheral nerves may be damaged), and examination for associated injuries to the head, chest, abdomen, and perineum.

Is the Fracture Stable or Unstable?

It is useful for the non-orthopaedic specialist to have some understanding of the nomenclature used to describe pelvic fractures

and the associated injuries that one can anticipate. Most pelvic fractures can withstand normal physiological forces and are therefore stable. Fractures that cannot withstand normal physiological forces are unstable. Early identification of an unstable pelvic fracture is important. First, its presence suggests a greater degree of trauma to the pelvis and increases the likelihood of serious associated injuries, which should be looked for and treated if found. Second, fixation of an unstable fracture reduces blood loss, mortality, hospital stay, leg length discrepancy, and long-term disability (Latenser et al. 1991, Leung et al. 1992) and makes nursing care easier (turning, sitting, early mobilisation) and lowers the need for analgesic consumption.

Pelvic stability is maintained by a series of ligaments. Anterior and much stronger posterior sacroiliac ligaments stabilise the sacroiliac joints. The sacrum and the ischium are stabilised by sacrotuberous ligaments and in front of this the sacrospinous ligaments. The sacrospinous ligaments resist external rotation of the hemipelvis and the sacrotuberous ligaments resist rotational and shearing forces in the vertical plane. The two pubic bones are joined by a cartilaginous symphysis (Fig. 5.15).

Types of Pelvic Fracture

The Tile classification system of pelvic ring disruptions includes stable fractures (type A), horizontally unstable fractures (B), and vertically unstable fractures (C) (Table 5.2) (Tile 1984).

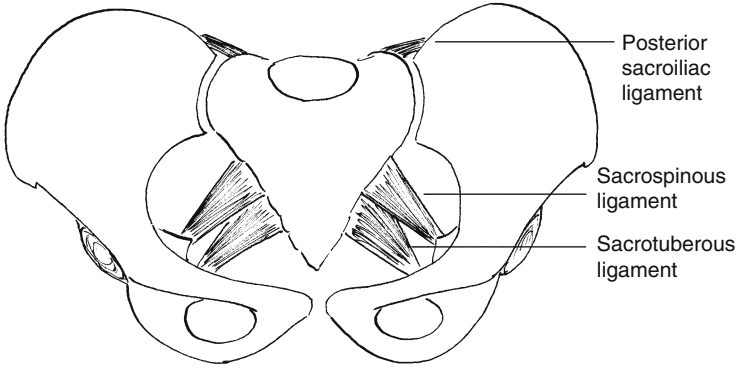


FIGURE 5.15. The position of the ligaments that stabilise the pelvis.

TABLE 5.2. The Tile classification system of pelvic ring fractures

Type A—stable	A1: Fracture of pelvis not involving the pelvic ring A2: Minimal displacement of pelvic ring with no instability
Type B—rotationally (horizontally) unstable	B1: Open book B2: Closed book; lateral compression: ipsilateral fracture B3: Closed book; lateral compression: contralateral fracture (bucket handle fracture)
Type C—rotationally (horizontally) and vertically unstable	C1: Unilateral C2: Bilateral C3: With acetabular fracture

Approximately 70% of unstable pelvic fractures are type B2 and B3, 10% to 20% of unstable fractures are of the open-book type (B1), and 10% to 20% are of type C. External or internal fixation is used to stabilise unstable fractures.

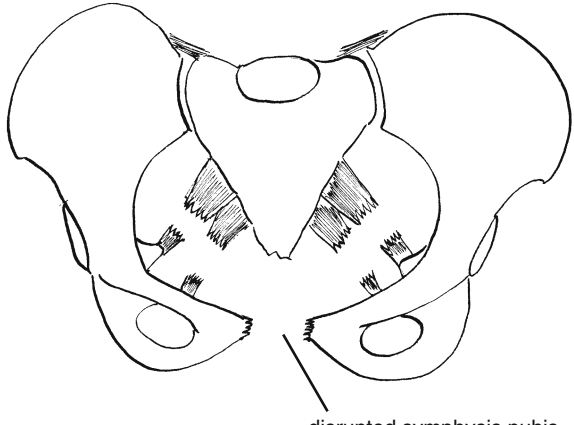
The open-book pelvic fracture (B1 in the Tile classification) is shown in Figure 5.16. If the symphysis pubis is disrupted (by >2.5 cm) in combination with the anterior sacroiliac ligament and the sacrospinous ligament, the affected half of the pelvis is free to open outward, like a book, and this fracture is thus called the 'open-book' fracture (horizontal instability). This type of fracture is caused by an anteroposterior compression injury. In this type of fracture there is a dramatic rise in pelvic volume and this stretches vessels, nerves, and organs, such as the bladder, resulting in damage to these structures.

The closed-book pelvic fracture (B2 or B3 in the Tile classification) is shown in Figure 5.17. When a lateral compression force is applied to the pelvis, a so-called closed-book injury occurs. The pubic rami may fracture and overlap and the ilium and sacral wings may be compressed and fractured. Nerves and vessels are not stretched, but the urethra is more likely to be damaged in this type of injury than in an anteroposterior compression fracture (Zingg et al. 1990).

Thus, the type of force applied to the pelvis and the subsequent radiological appearance give some indication of the likelihood of associated bladder and urethral injuries, the open-book



a



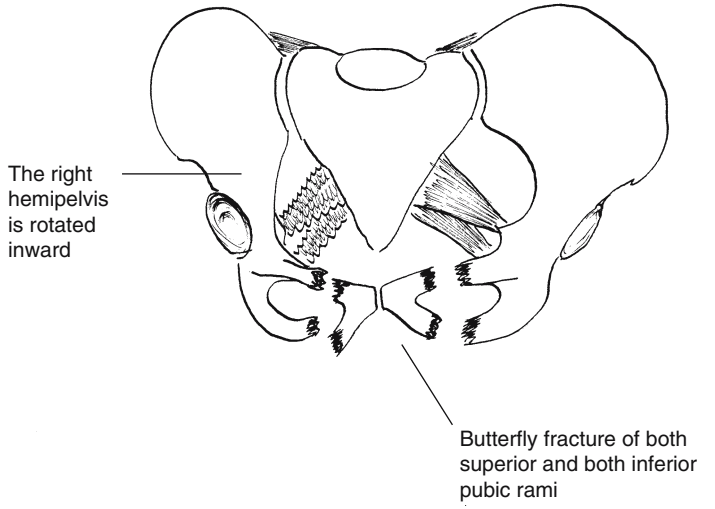
b

FIGURE 5.16. Open-book—B1—pelvic fracture. a: Plain x-ray. The bladder neck in this case had been cut by the fractured bone. b: Ligaments disrupted in an open-book fracture.



a

FIGURE 5.17. Closed-book pelvic fracture B2. a: These are the images obtained at the time of retrograde urethrography, which shows complete disruption of the posterior urethra (contrast does not progress beyond the bulbar urethra). During the process of fracturing, the overlapping bones of the fractured pubis have sheared through the urethra. b: Ligaments and bones disrupted in a closed-book fracture.



b

FIGURE 5.17. *Continued*

fracture being more likely to be associated with a bladder injury and the closed-book fracture with a urethral injury.

In the vertically unstable pelvic fracture (C in the Tile classification), if a sacrotuberous ligament and a posterior sacroiliac ligament are torn, the affected hemipelvis can move upward and posteriorly with respect to the sacrum (vertical instability). A fracture of the transverse process of L5 vertebra is a sign that such a fracture has occurred (i.e., it is a sign of vertical pelvic instability). Again, vessels and nerves can be damaged.

Radiologic Determination of Stability

This is based on inlet and outlet views of the pelvis, the x-ray beam being angled accordingly. These views demonstrate anteroposterior (inlet view) and vertical (outlet) displacement of the pelvic ring. CT can provide better definition of sacral, sacroiliac, and acetabular fractures and dislocations.

The degree of displacement of bone fragments, which on plain x-rays may not look too severe, usually looks much worse on CT (Fig. 5.18). Remember, the degree of bone displacement will have been more pronounced when the injury was actually taking place, and thus while the bone position you see on CT or plain x-ray represents the final position of displacement, the

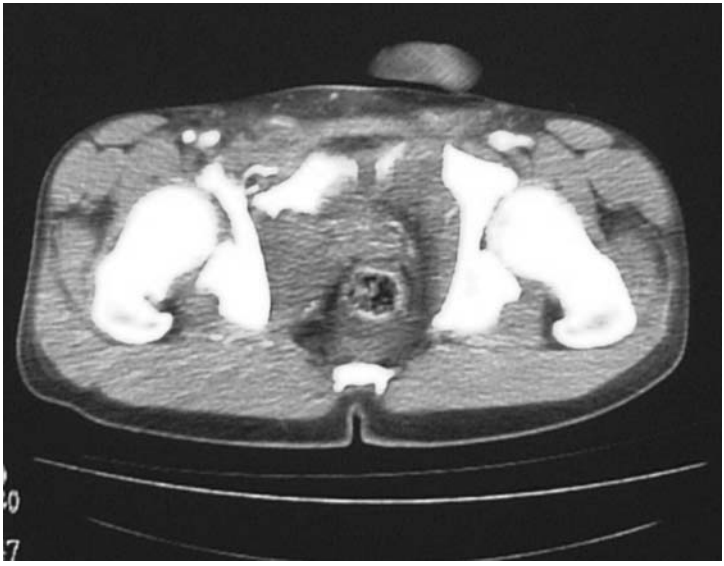


FIGURE 5.18. This is a computed tomography (CT) scan of the same case as in Figure 5.16 (open-book fracture). The degree of displacement of bone fragments looks much worse on CT.

fractured bones will have moved a greater distance during the process of fracturing. With this in mind, it is not difficult to imagine that soft tissues might have been injured. In the case of the bladder; for example, this occurs either by shearing forces that fracture the bone and literally tear the bladder apart by virtue of its fascial attachments to the pelvis or as a consequence of a direct scissors like action of sharp edges of the fractured bone. The bladder's location immediately behind the pubic bone makes it a vulnerable target, and whenever a patient with a pelvic fracture is seen, you should assume they have a bladder and/or urethral injury until proven otherwise. Not surprisingly, thin-walled pelvic veins can be torn by these same shearing or cutting forces.

Bladder Injuries Associated with Pelvic Fractures

Approximately 10% of males with a pelvic fracture and a slightly lower percentage of females will have an associated bladder injury. Of those bladder ruptures that are due to external blunt trauma

(as opposed to perforation during, for example, bladder tumour resection), approximately 85% are associated with a pelvic fracture, 10% with a fracture of the proximal femur, and 5% have no associated bony injury, the force applied to the abdomen in these latter cases having been sufficient to burst the bladder, but not to fracture any bones (e.g., the patient intoxicated by alcohol whose full bladder is already under tension and therefore ruptures when he falls down stairs or is hit by a car). Bladder injuries are often associated with anteroposterior pelvic compression fractures (rather than lateral compression fractures) (Zingg et al. 1990). Approximately 60% of traumatic blunt bladder ruptures are extraperitoneal, 30% are intraperitoneal, and 10% are a combination of extraperitoneal and intraperitoneal (Peters 1989).

Thus, while the majority of patients with pelvic fractures do not have bladder injuries, a substantial minority (1 in 10) do, and almost half of these are intraperitoneal. Missing a diagnosis of bladder perforation in this situation can have disastrous consequence, and for this reason all patients who have a pelvic fracture should undergo a urethrogram and a cystogram. Up to 10% of patients with bladder rupture may have no macroscopic haematuria and a further 10% may have only microscopic haematuria (Cass and Luxenberg 1987). Absence of haematuria is more common in patients with intraperitoneal bladder rupture, than in those with extraperitoneal perforation.

Combined Bladder and Posterior Urethral Injuries Following Pelvic Fracture

If the bladder has been ruptured by a blunt injury causing a pelvic fracture, have a high index of suspicion for an associated urethral injury. About one third of patients with a traumatic blunt bladder rupture have associated injuries to other urinary structures, most commonly the urethra. Approximately 5% to 10% of patients with a pelvic fracture and bladder rupture also have a posterior urethral rupture (Cass and Luxenberg 1987). In a series of pelvic fractures, Cass (1988) found bladder ruptures in 6%, urethral rupture in 2%, and combined bladder and urethral rupture in 0.5%.

Urethral Injuries Associated with Pelvic Fractures

The posterior urethra (essentially the membranous urethra) is injured with roughly the same frequency as the bladder in subjects who sustain a pelvic fracture, occurring in between 5% and 15% of such cases. The great majority of posterior urethral injuries occur in association with pelvic fractures and approxi-

mately 10% to 20% of patients with a posterior urethral injury have an associated bladder rupture (Cass et al. 1984) (specifically bilateral pubic rami fractures and especially those with sacroiliac joint displacement; Koraitim 1996). Very occasionally a posterior urethral injury can occur in the absence of an associated pelvic fracture following blunt trauma to the perineum.

Not surprisingly (because it is longer), the male urethra is more likely to be injured than is the female urethra. Signs that the urethra may have been injured include blood at the meatus, gross haematuria, and perineal or scrotal bruising. Approximately 40% to 50% of patients with a pelvic fracture and urethral injury have blood at the external meatus (Cass 1984, Lowe et al. 1988). In the remaining patients the urethral injury is not apparent until an attempt has been made to pass a urethral catheter and has failed. A so-called high-riding prostate occurs when the prostate and bladder become detached from the membranous urethra and reach a higher than normal position. A large pelvic haematoma develops and pushes the bladder upward. When one performs a rectal examination in a patient with a high-riding prostate, it may be felt just at the tip of your finger or may not be felt at all. The associated pelvic haematoma may also make it impossible to feel the prostate, so the patient may be thought to have a high-riding prostate when in fact it is in a normal position and vice versa. Thus, the presence of a high-riding prostate is an unreliable sign (Elliott and Barrett 1997). A digital rectal examination may be more important as a way of establishing whether there is an associated rectal injury, in which case blood may be seen on the examining finger when it is withdrawn. However, the absence of blood on the examining finger cannot be taken as a guarantee that the rectum is intact.

Abdominal and Pelvic Imaging in Pelvic Fracture, and What to Do If Imaging Cannot or Has Not Been Done

The radiologic workup in patients with a pelvic fracture usually includes an abdominal and pelvic CT scan, a retrograde urethrogram (to exclude or confirm a urethral injury), and, if the urethra is intact, a retrograde cystogram to assess the integrity of the bladder. The cystogram usually demonstrates the presence of a bladder perforation. The abdominal CT scan allows associated injuries to abdominal viscera to be assessed.

In some hospitals retrograde urethrography is performed only in patients with blood at the meatus while others perform this investigation in all patients with pelvic fractures where the pubic rami have been disrupted. If there is no blood present at

the meatus, a gentle attempt at urethral catheterisation may be made. It has been suggested that this could convert a partial urethral rupture into a complete rupture. However, McAninch (2002) has stated, 'We and others have not seen any evidence that this can convert an incomplete into a complete transection . . . and we usually make one gentle attempt to place a urethral catheter in suspected urethral disruption' (see also Jackson and Williams 1974, Kotkin and Koch 1996). If any resistance is encountered, stop, and obtain a retrograde urethrogram. If the retrograde urethrogram demonstrates a normal urethra, proceed with another attempt at catheterisation, using plenty of lubricant. If there is a urethral rupture, most centres recommend insertion of a suprapubic catheter via a formal open approach, to allow inspection of the bladder (and repair of injuries if present) at the same time that the suprapubic catheter is placed. Radiological inspection of the bladder is not possible in such cases because the urethral rupture will have prevented performance of a cystogram. Direct inspection of the bladder is required to determine the presence/absence of a bladder injury.

Suprapubic Catheterisation Versus Open Suprapubic Cystostomy in Patients with Posterior Urethral Disruption

Why go to the trouble of taking the patient to the operating theatre, exposing the bladder, opening it, and inserting a catheter, when a suprapubic catheter could easily be passed percutaneously in the emergency department? There are several reasons for recommending open suprapubic cystostomy for catheter placement over percutaneous suprapubic catheterisation:

1. Opening the bladder affords the opportunity of inspecting the bladder for evidence of a rupture (extraperitoneal or intraperitoneal) and of a bladder neck injury. If such an injury is found, it can be repaired.
2. The bladder is often pushed upward by the pelvic haematoma that follows any serious pelvic fracture. It can be difficult, even for the experienced urologist, to locate the bladder for safe suprapubic puncture. The catheter can inadvertently be inserted into the pelvic haematoma. At best, it will clearly be in the wrong position and bladder drainage will not have been achieved; at worst, infection can be introduced into the pelvic haematoma, with disastrous consequences.
3. A catheter of adequate size should be inserted into the bladder. As there is likely to be some bleeding from the bladder

in the days following placement of the catheter, if too small a catheter has been used, it could become blocked by clots. Formal open placement of a suprapubic catheter allows a larger catheter to be placed in the bladder than is possible through a percutaneous trocar, where the maximum catheter size is 14 Ch.

In practice, however, infection of metal plates is rarely seen, and it has been suggested that as long as the bladder is approached from a high-enough position (so as to avoid the pelvic haematoma) a percutaneous suprapubic catheter may be safely placed (McAninch 2002). Certainly, if the patient is unstable, a percutaneous suprapubic catheter should be inserted, rather than the patient undergoing a general anaesthetic just for insertion of a suprapubic catheter. Once the patient has been stabilised, a cystogram can be done to exclude a bladder injury.

How to Perform a Retrograde Urethrogram

The contrast agent used varies from hospital to hospital. We use Urografin 150 (sodium amidotrizoate and meglumine amidotrizoate), but other contrast agents can be used. A small (e.g., 12 or 14 Ch) catheter is placed in the fossa navicularis of the penis (approximately 1–2 cm from the external meatus). To prevent contrast spilling out of the urethra and to hold the catheter in place, either inflate the catheter balloon with 2 mL of water or apply a penile clamp to the end of the penis. Ideally continuous screening (fluoroscopy) should be done as contrast is gently instilled until the entire length of the urethra has been demonstrated. Alternatively, static images may be taken at intervals. Remember, as the urethra passes through the pelvic floor (the membranous urethra) there is a normal narrowing, and similarly the prostatic urethra is narrower than the bulbar urethra (Fig. 5.19).

How to Perform a Retrograde Cystogram

Retrograde cystography is the gold standard radiographic technique for demonstrating bladder ruptures. It will not miss a perforation, as long as

- the bladder is adequately filled;
- a postdrainage image is taken once the bladder has been emptied of contrast.

Both aspects of the technique are important. If the bladder is not properly expanded with contrast, a perforation may be obscured

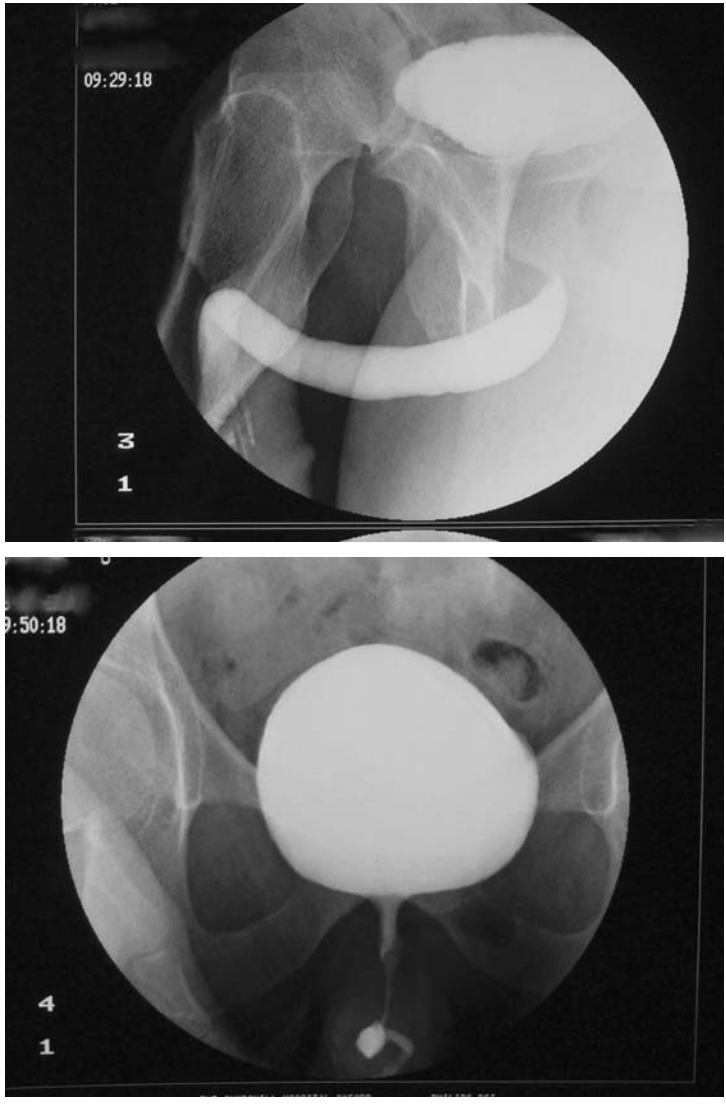


FIGURE 5.19. A normal urethrogram. a: Lateral projection. b: Antero-posterior projection.

by a 'plug' of omentum or small bowel temporarily sealing the hole (false-negative cystograms have been reported, when volumes of 250 mL or less were used for the cystogram; Cass and Luxenberg 1987). Conversely, a posterior perforation can sometimes be obscured by a mass of contrast filling the bladder and the leak of contrast only becomes apparent as a 'whisper' of contrast outside the bladder when the bladder has been emptied (approximately 10% of bladder perforations are diagnosed on the postdrainage film).

Pass a small (e.g., 12 or 14 Ch) catheter into the bladder and, using gravity, instill approximately 400 mL of contrast (in children, 60 mL plus 30 mL per year of age up to a maximum of 400 mL) into the bladder. Again, we use Urografin 150. Images may be taken fluoroscopically or several static images can be taken as the bladder is filled and then emptied.

Alternatively, a CT cystogram can be done. If the patient is going to have a CT scan done anyway (and it usually is done), it is simpler to image the bladder with CT than fluoroscopically (the patient would have to be moved to another room in the radiology department to allow this to be done). Diluted contrast should be used if a CT cystogram is to be done because undiluted contrast is so dense that it produces poorer images. The key point in CT cystography is to instill the contrast *retrogradely* through a catheter inserted into the bladder—CT cystography using intravascularly administered contrast can miss bladder perforations. Haas et al. (1999) found that retrograde cystography successfully diagnosed all of 15 cases of bladder rupture due to blunt trauma, but spiral CT with intravenous contrast and catheter clamping to distend the bladder successfully diagnosed only nine of these 15 ruptures. CT correctly diagnosed four of five (80%) intraperitoneal ruptures and 6 of 11 (55%) extraperitoneal ruptures.

Problems Imaging the Bladder in Patients with Urethral Rupture

Ten percent to 20% of patients with a posterior urethral rupture also have a bladder rupture (Cass et al. 1984), and 5% to 10% of patients with a pelvic fracture and bladder rupture also have a posterior urethral rupture (Cass and Luxenberg 1987). This presents a dilemma because the urethral rupture makes it difficult, radiologically, to diagnose a bladder injury. A catheter cannot be negotiated past the urethral rupture into the bladder to allow a cystogram to be done, and contrast administered during the urethrogram may not reach the bladder in sufficient quantities to diagnose a bladder rupture, or it may extravasate around the

bladder and obscure a perforation. A CT cystogram can be done by taking delayed films in the CT scanner, relying on the intravenously administered contrast to define the bladder. However, as discussed above, these images are not as accurate at diagnosing or excluding a bladder rupture when compared with instillation of contrast into the bladder by the *retrograde* route (retrograde cystography). Furthermore, these patients are usually very unwell and are often transferred rapidly to the operating room for treatment of the pelvic fracture and associated injuries. In this situation there often simply isn't time to wait for contrast administered intravenously to work its way into the bladder to allow a CT cystogram to be done.

Where a cystogram cannot be done because of a urethral rupture, the patient should be transferred to the operating theatre so that a suprapubic catheter can be inserted by a formal open approach—an open suprapubic cystostomy (if there is a urethral injury this will usually be left alone and definitive repair carried out at a later date when the patient's condition is stable). By making the incision in the bladder somewhat larger than is necessary for placement of a suprapubic catheter, the bladder may be inspected to see if there is a perforation, and if so, it can be repaired. Rarely, fragments of bone may be seen poking through the wall of the bladder, and these can be removed with bone forceps before the bladder is repaired. It is better to open the bladder and find that it has not been injured than to allow urine from a missed perforation to pour into the pelvis of a patient with a large haematoma and fractured bone, with the obvious risk of subsequent pelvic sepsis.

Occasionally one is called to the operating room to see a pelvic fracture patient who is already undergoing pelvic fixation or surgery for other injuries. A urethrogram has not been, or cannot be done, and the orthopaedic team has tried, but failed, to pass a catheter urethrally. It is reasonable for the more experienced urologist to make a single attempt to pass a catheter, but if this fails, assume the patient has a urethral rupture. In the ideal world a urethrogram followed by a cystogram would be done on the operating table to establish whether the urethra and bladder are intact or injured. But the world is not ideal. There may be lots of metal work in the way (obscuring that bit of the urethra you're interested in). The patient may not be ideally positioned for a urethrogram. Trying to reposition a patient who is draped in sterile towels and who has just undergone pelvic fixation is never easy. Finally, just to make life even more difficult the radiographer may have been called away to another case and will be

busy for hours! One can vainly struggle to do a urethrogram, and sometimes you will be lucky and the images will be good enough for interpretation. More often than not, the exercise proves a frustrating failure. If faced with this situation, the other (simpler) option is to place a suprapubic catheter via a formal open cystotomy, and to inspect the bladder as you do so for perforations. Get a urethrogram a few days later. The bladder will often already have been exposed (for fixation of the pelvis). You will know for sure that the bladder is not perforated (and will have repaired it, if it is), and the patients will have adequate drainage of their bladder. Leaving a posterior urethral injury, if present, for subsequent repair is entirely reasonable.

An additional advantage of opening the bladder is that this allows retrograde ureterography to be performed or ureteric stents or catheters to be placed if the ureters have not been adequately visualised on preoperative imaging. Inadequate visualisation of the ureters occurs frequently since in the trauma situation the IVU is often not a complete examination, but is limited to just one or two images, such that the ureter may not be completely opacified. Such limited IVUs will miss a substantial number of ureteric injuries (Presti and Carroll 1996). Indeed, in a series of 50 patients undergoing single-shot intraoperative IVU, the renal collecting system and ureter were not visualised at all in 35% of cases and in only 36% of cases was ureteral detail seen on one or both sides (Morey et al. 1999). In many trauma centres the IVU has been completely replaced by the abdominal and pelvic CT scan, which provides less precise imaging of the ureters than does an IVU or retrograde ureterogram. An abdominal x-ray taken 10 to 15 minutes after administration of contrast for the CT scan can visualise the ureters, but for the same reasons that a limited IVU may not visualise the entire length of the ureter, so too may it be difficult with such an x-ray to confidently exclude a ureteric injury. As for on-table urethrography, performing retrograde ureterography on the operating table is easier said than done in the trauma situation. If in doubt, assume that there might be a ureteric injury and place ureteric stents or catheters.

BLADDER INJURIES

Situations in Which the Bladder May Be Injured

Transurethral resection of bladder tumour (TURBT)
Cystoscopic bladder biopsy
Transurethral resection of prostate (TURP)

Cystolitholapaxy
Penetrating trauma to the lower abdomen or back
Caesarean section, especially as an emergency
Blunt pelvic trauma—in association with pelvic fracture or 'minor' trauma in the inebriated patient
Total hip replacement (very rare)
Rapid deceleration injury—seat belt injury with full bladder in the absence of a pelvic fracture
Spontaneous rupture after bladder augmentation

Types of Perforation

Bladder perforations are categorised as extraperitoneal or intraperitoneal. In an intraperitoneal perforation, the peritoneum overlying the bladder, has been breached along with the wall of the bladder, allowing urine to escape into the peritoneal cavity. In an extraperitoneal perforation, the peritoneum is intact and urine escapes into the space around the bladder, but not into the peritoneal cavity. For a perforation to be intraperitoneal, it must occur in that part of the bladder that is covered by peritoneum, and the injury must, of course, be deep enough to make a hole all the way through the muscular wall of the bladder, the surrounding perivesical fat, and the peritoneum.

Making the Diagnosis

As with urological injuries in general, if you know the potential scenarios in which a bladder injury can occur, you are halfway there in terms of making a diagnosis. From the nature of the injury, which makes you suspect a possible bladder injury, you can arrange appropriate imaging studies to confirm your suspicions. Thus, the history is all-important in making the diagnosis.

The need to perform diagnostic tests depends on the clinical situation. In the case of iatrogenic injury (e.g., after a TURBT), the patient is usually anaesthetised and diagnosis is usually obvious on visual inspection alone. No diagnostic tests are required. In other situations, e.g., the drunk patient who has suffered apparently minor trauma such as a fall, the classic triad of symptoms and signs that are suggestive of a bladder rupture is suprapubic pain and tenderness, difficulty or inability in passing urine, and haematuria (or there may be just one or two of the symptoms or signs of the 'triad'). Additional signs may include abdominal distention and absent bowel sounds, occurring as a consequence of an ileus caused by urine being present in the peritoneal cavity. In these non-atrogenic causes, the great majority of patients (>95%) will have macroscopic haematuria or 'heavy'

microscopic haematuria. However, remember, the absence of macroscopic haematuria does not necessarily mean the absence of a bladder injury. Have a low threshold for arranging imaging studies.

Imaging Studies

As discussed above, there are two main ways of imaging the bladder—conventional retrograde cystography or CT cystography. Whatever method is used, several points of technique are worth emphasising. First, the bladder must be adequately distended with contrast. If only 100 mL or so of contrast is instilled into the bladder, a clot, omentum, or small bowel may continue to 'plug' the perforation, which therefore may not be diagnosed. Use at least 400 mL of contrast in an adult and 60 mL plus 30 mL per year of age in children up to a maximum of 400 mL in children. Second, images must be obtained after the contrast agent has been completely drained from the bladder (a postdrainage film). A whisper of contrast from a posterior perforation may be obscured by a bladder distended with contrast.

In extraperitoneal perforations, extravasation of contrast is limited to the immediate area surrounding the bladder (Fig. 5.20). In intraperitoneal perforations, loops of bowel may be outlined by the contrast (Fig. 5.21).

Extraperitoneal and Intraperitoneal Perforation During Resection of a Bladder Tumour (TURBT)

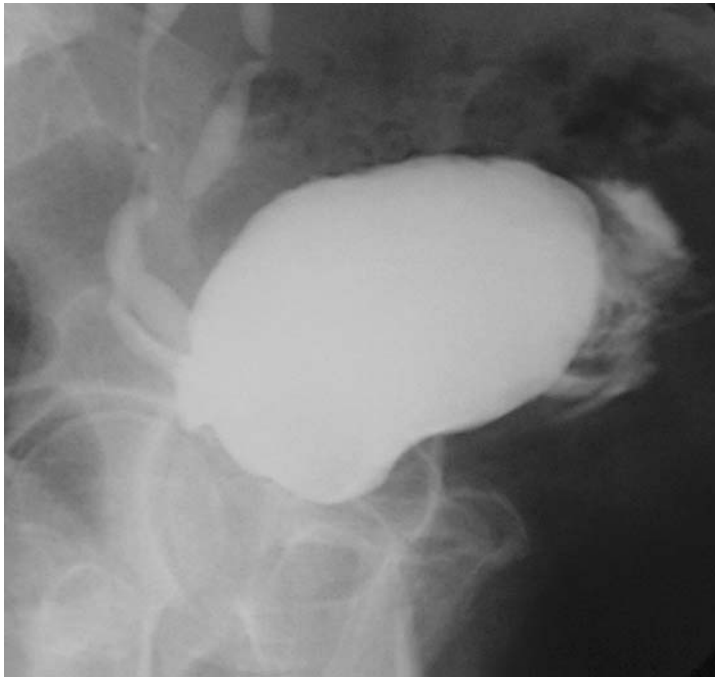
When a bladder cancer is being resected, its location will determine the likelihood of a perforation being extraperitoneal or intraperitoneal. A perforation at the neck of the bladder or on the trigone is not adjacent to the peritoneal cavity, and therefore such a perforation cannot be intraperitoneal. However, when a tumour is located in the dome of the bladder, immediately beneath which is the peritoneum, it is quite possible for an intraperitoneal perforation to occur.

Small perforations into the perivesical tissues are not uncommon when resecting small tumours of the bladder. Perivesical fat is seen. As long as you have secured good haemostasis and all the irrigating fluid (if you use this) is being recovered, no additional steps are required except that perhaps one should leave the catheter in for 4 days rather than 2. You may decide to irrigate the bladder with irrigating fluid. Alternatively, allow the patient's own urine output to wash out the bladder (the urine output can be increased by giving a low dose—20–40 mg—of intravenous frusemide).



a

FIGURE 5.20. In an extraperitoneal perforation, extravasation of contrast is limited to the immediate area surrounding the bladder. a: On the anteroposterior (AP) views the leak is not obvious. b: On the lateral views an anterior leak is obvious. Note the two ureters posteriorly (the patient refluxes contrast up both ureters).



b

FIGURE 5.20. *Continued*

Trainees are sometimes uncertain whether a perforation is extraperitoneal or intraperitoneal. Establishing this can sometimes be difficult, because *both* can cause marked distention of the lower abdomen—an intraperitoneal perforation by allowing escape of irrigating solution directly into the abdominal cavity, and an extraperitoneal perforation by expanding the retroperitoneal space, with fluid then diffusing directly into the peritoneal cavity. The fact that a suspected intraperitoneal perforation was actually extraperitoneal becomes apparent only at laparotomy when no hole can be found in the bladder! However, in such cases where there is marked abdominal distention, whether the perforation is extraperitoneal or intraperitoneal is in many senses academic. The important thing is to explore the abdomen, principally to drain the large amount of fluid that can compromise respiration in an elderly patient by splinting the diaphragm, but also to



a

FIGURE 5.21. In an intraperitoneal perforations, loops of bowel may be outlined by the contrast. There was an associated left ureteric injury managed by JJ stenting. a: On initial bladder filling no leak is seen. b: Small bowel loops are outlined by contrast as more contrast is instilled into the bladder.



b

FIGURE 5.21. *Continued*

check that loops of bowel adjacent to the site of perforation have not been injured at the same time. Failing to make the diagnosis of an intraperitoneal perforation, particularly if bowel has been injured, is a worse situation to be in than performing a laparotomy for a suspected intraperitoneal perforation, but then finding that the perforation was 'only' extraperitoneal.

The diagnosis of an intraperitoneal perforation is obvious if you can actually see loops of bowel as you are looking through the resectoscope. The telltale sign of the Ellik evacuator not sucking back can occur with both intraperitoneal and extraperitoneal perforation, and this therefore tells you that something is wrong, rather than what is wrong.

When there is marked abdominal distention, or where it is obvious that the perforation has been made right through into the peritoneum or, as is often the case, the perforation is obscured and accompanied by haemorrhage, then it is necessary to explore the abdomen.

The bladder is approached through a Pfannenstiel incision or lower midline abdominal incision, opened between stay sutures, the clot evacuated, the bleeding controlled, and the hole sewn up. The peritoneum is opened if not already done so. This allows you to see if there is any blood-stained fluid inside. Adjacent loops of small and large bowel should be pulled out and diathermy damage looked for. A hole in the small bowel is closed in its transverse axis. A hole in the colon should be protected with a temporary loop-colostomy.

The Delayed Diagnosis of Iatrogenic Bladder Perforation

Bladder perforation during surgery (TURBT, TURP, open pelvic surgery, etc.) may not initially be recognised until after the patient has returned to the ward. All may initially appear to be well until the patient develops a fever and an ileus. After open abdominal or pelvic surgery, a drain may have been left and there may be persistent output of fluid (urine) from this. The creatinine level in this fluid will be greater than that in serum. Imaging studies may show a pelvic or abdominal fluid collection. In this situation, if the patient has undergone gynaecological or bowel surgery, imaging studies should be done to determine whether there is an associated ureteric injury as well as a bladder one (see below).

Bladder Perforation During TURP

This occurred in 0.25% of cases in a large audit of complications occurring after TURP (Neal 1997). In practice, danger only arises

from perforations where large prostatic veins are opened and a large volume of fluid escapes into the circulation; it is rare for escape of fluid into the retropubic space to cause any trouble. However, occasionally fluid introduced with the Ellik evacuator does not suck back, or a change in the character of the respiration and a coldness and swelling of the suprapubic tissues may suggest that there has been a massive loss of fluid.

Stop the resection. If there is significant abdominal distention make the decision to proceed with open drainage of the retropubic space. Make a Pfannenstiel incision. Expose the bladder, open it between stay sutures, and evacuate the clot. Complete the prostatectomy (if it is not already complete) by enucleating the remaining adenoma with the finger. Get exact haemostasis by sutures, and if you can see the hole in the capsule, close it with a stitch. Only when all the bleeding is controlled should you close the wound with a suprapubic and urethral catheter and a drain to the retropubic space.

Bladder Perforation Following Pelvic Fractures

This has been discussed previously. If the bladder injury is extraperitoneal and there are no other associated injuries to the urethra, kidneys, or intraabdominal viscera, then bladder drainage with a urethral catheter for approximately 2 weeks is all that is necessary. A cystogram can be done 10 to 14 days later to confirm that the perforation has healed. If it has not, it is reasonable to wait for another week or so, and in most cases a further cystogram will demonstrate no further leak. Occasionally there will be persistent leakage of contrast and this can be an indication that a piece of bone is poking into the bladder. Exploration and repair should be carried out.

There are situations in which an extraperitoneal bladder perforation should undergo suture repair:

- If you have opened the bladder to place a catheter, because, for example, there is an associated urethral injury, there seems little point in not repairing an extraperitoneal rupture if one is found.
- If imaging, such as a CT scan, has demonstrated a bone spike protruding into the bladder, the bladder should be opened, the bone removed, and the defect in the bladder sutured.
- Rectal perforation in association with a bladder rupture is uncommon, but again where there is a rectal injury, an extraperitoneal bladder rupture should be repaired because of the high risk of severe pelvic sepsis.

- Occasionally, where conservative management of an extraperitoneal bladder rupture with catheter drainage is started, there may be persistent bleeding from the edges of the bladder injury. This can cause troublesome clot retention and is another indication for formal open repair of such injuries.
- Finally, where the patient is undergoing open fixation of a pelvic fracture, urine leaking from the bladder while the bladder rupture heals spontaneously can potentially cause infection of the metal plate. Simultaneous repair of an extraperitoneal rupture may reduce the likelihood of this occurring.

Surgical repair of an extraperitoneal bladder rupture also allows an accurate assessment to be made of the integrity of the bladder neck. Similarly, vaginal injuries can also be repaired at the same time. The easiest way to repair an extraperitoneal bladder injury is by opening the bladder at the dome, and performing the repair from inside the bladder. This is the most 'direct' approach and it avoids the need to mobilise the posterior wall of the bladder. An associated bladder neck or vaginal injury can also be repaired via such an approach. Attempting such a repair by a vaginal approach is technically difficult because of associated labial oedema or haematoma formation, which makes access to the vagina very difficult.

The key thing with operative management of extraperitoneal bladder ruptures is to inspect the entire surface of the bladder, first to look for other perforations and second to remove any bone fragments that might be poking into the lumen of the bladder. Inspection of the bladder is most easily done by formally opening it between stay sutures and inspecting its interior. Make your incision at the dome, i.e., as far away from the pelvic haematoma as you possibly can so as not to disturb it and precipitate uncontrollable haemorrhage. A urethral or suprapubic catheter (through a separate stab wound) should be inserted for subsequent bladder drainage over the course of the next 2 weeks or so. Some surgeons are happy with a single-layer closure with absorbable sutures, while others feel that a two-layer closure is more secure. In the case of an extraperitoneal tear, a drain runs the potential risk of allowing a site of access for infection of the pelvic haematoma, and many surgeons prefer not to place a drain after repair of an extraperitoneal rupture, as long as adequate bladder drainage with a catheter has been obtained.

Intraperitoneal Injuries

These are often much larger than is suggested by cystography and for this reason are less likely to close spontaneously than are extraperitoneal perforations. A single or two-layered repair is performed with absorbable suture material such as 2/0 Vicryl. An intraperitoneal, perivesical drain should be placed and removed as soon as it has stopped draining significant amounts of urine. A suprapubic or urethral catheter may be used to drain the bladder (Volpe et al. 1999).

Bladder Injury During Caesarean Section

During emergency caesarean section, in the desperate rush to deliver the baby safely, the bladder may be injured. This problem can be avoided by catheterising the bladder to deflate it, and so 'moving' it out of the way of the line of incision in the uterus, but despite this precaution, the bladder is from time to time injured during this procedure. The injury is usually immediately apparent and can be repaired straight away. It may have involved both the anterior and posterior walls of the bladder.

Spontaneous Rupture After Bladder Augmentation

Bladder augmentation, performed either by using a patch of intestine or stomach sutured into the bivalved bladder or by removing a disc of muscle from the dome of the bladder (detrusor myectomy), is most often carried out in patients with neuropathic bladder problems. It is designed to convert the poorly compliant, low-volume bladder into a compliant, high-volume reservoir, thereby improving continence and in those cases with associated hydronephrosis, protecting renal function.

Spontaneous bladder rupture has been reported in approximately 5% to 10% of patients after bladder augmentation (DeFoor et al. 2003). Its occurrence is by no means limited to the first few week or months after augmentation. Indeed, it may occur many years after augmentation. Ileocystoplasties are probably more likely to rupture than are gastrocystoplasties (DeFoor et al. 2003, Shekarriz et al. 2000).

While the majority of reported cases of perforation of augmented bladders occur with no history of preceding trauma, there are a reports of perforation occurring following trauma, e.g., after motor vehicle accidents. It is difficult to know whether the augmented bladder is more likely to rupture after abdominal trauma than is the normal, nonaugmented bladder, as such cases occur with very low frequency. It is, however, difficult to imagine

that a surgical scar is stronger than the 'normal' bladder, and so augmented bladders are very probably at greater risk of spontaneous or traumatic rupture than is the normal, nonaugmented bladder.

These patients usually have underlying conditions such as spina bifida and spinal cord injury, and therefore usually have limited awarness of bladder filling or of pelvic pain. Perforation of an augmented bladder, unless it occurs in a patient with normal sensation, may therefore present a diagnostic challenge because pain, though usually present, is not usually severe enough to make one think that a serious event has occurred. A high index of suspicion is therefore needed to make the diagnosis. The usual presentation is one of abdominal pain that may be vague in onset and nature, fever, or sepsis. The diagnosis may be confirmed by a cystogram, or by CT with contrast instillation into the bladder. However, a normal cystogram or CT does not necessarily exclude a diagnosis of perforation. Where there are clinical sign such as persistent or progressive abdominal distention, one should consider exploratory laparotomy even though imaging studies may be normal. Management usually consists of immediate laparotomy and repair of the perforation, but in cases where there is severe sepsis, this should be managed prior to exploration.

Detrusor Myectomy

This alternative form of bladder augmentation has gained popularity over the last few years because it avoids the complications associated with harvesting a loop of small bowel and of implanting it into the urinary tract. A disc of detrusor muscle is removed from the dome of the bladder, so increasing bladder compliance and thus improving continence. It is perhaps surprising that more of these bladders do not rupture spontaneously when one considers how very thin the bladder is once the muscle of the detrusor has been dissected off of the underlying urothelium and connective tissue. Spontaneous rupture was reported in one of 50 of cases after myectomy in Stohrer's (1997) series.

Surgical Repair of Bladder Injuries

We use a continuous 2/0 Vicryl suture (i.e., absorbable) and close the bladder in two layers. In the first layer, ensure that any bleeding vessels in the cut edge of the bladder are ligated with the suture.

Whenever the urinary tract has been opened and then closed, it is a sensible precaution to leave a drain in place. It is inevitable

that the closure will not be completely watertight, and as a consequence urine will leak through the suture line for a few days. A drain removes this and can prevent the consequences of a urine collection (a urinoma) becoming infected.

POSTERIOR URETHRAL INJURIES

As discussed above, these are essentially an injury that occurs following pelvic fracture, and specifically fracture of the pubic rami. In the emergency situation their management consists of diversion of the flow of urine past the injury, by suprapubic catheterization (see above).

ANTERIOR URETHRAL INJURIES

These injuries are rare. The majority occur as a result of a straddle injury in boys or men. For example, while riding a bicycle and suddenly applying the brakes, the perineum comes into forcible contact with the crossbar of the bicycle. The bulbar urethra is crushed between the crossbar and the pubic bone. Other mechanisms include direct injuries to the penis, penile fractures (Marsh et al. 1999), inflating a catheter balloon in the anterior urethra (Sellett 1971), and penetrating injuries by gunshot wounds.

Making the Diagnosis

In cases with these types of injuries, you should have a high index of suspicion that an anterior urethral injury has occurred. The patient may complain of blood at the end of the penis, difficulty in passing urine, or frank haematuria. A haematoma may develop around the site of the rupture. There may be swelling of the penis as a consequence of extravasation of urine into the periurethral tissues. If Buck's fascia has been ruptured, urine and blood may track into the scrotum, causing swelling and a characteristic 'butterfly-wing' pattern of bruising, which reflects the extent to which the bruising may spread as a consequence of the anatomical attachments of Colles' fascia (see below) (Fig. 5.22).

The Anatomy of 'Butterfly-Wing' Bruising

Beneath the penile skin, the fascia of the penis consists of superficial fascia and deep fascia (Fig. 5.23).

Superficial Fascia

The superficial fascia of the penis (dartos fascia) is continuous with the membranous layer of the superficial fascia of the groin and perineum (Colles' fascia). Colles' fascia in the perineum is

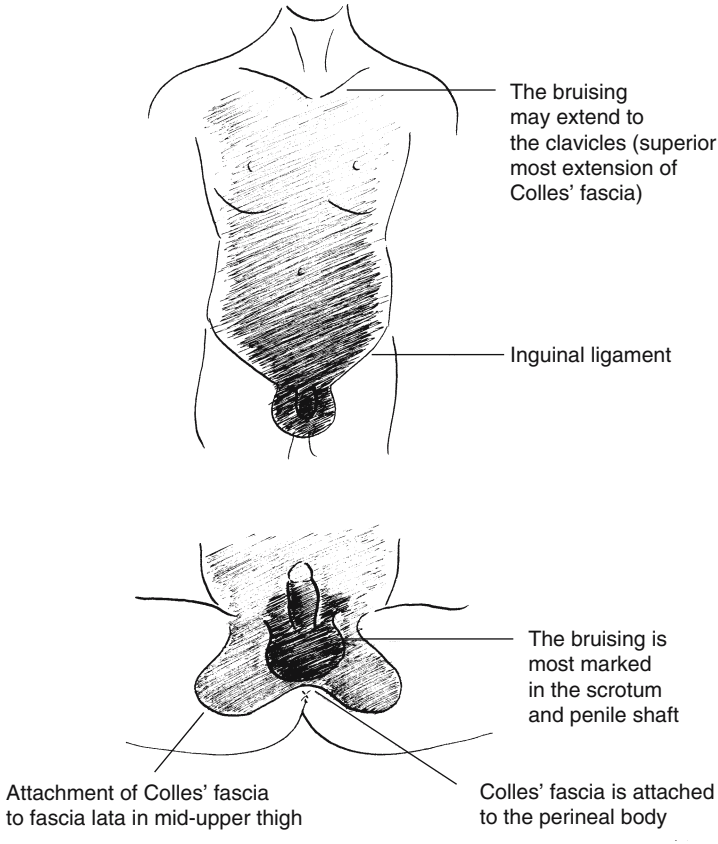


FIGURE 5.22. Butterfly bruising following rupture of Buck's fascia.

the equivalent of Scarpa's fascia in the abdomen (Colles' fascia and the dartos fascia together form the membranous layer of the superficial fascia of the perineum and penis). Beneath the dartos fascia is Buck's fascia (the deep layer of the superficial fascia).

Deep Fascia

Beneath Buck's fascia is the deep fascia of the penis (the tunica albuginea), which covers the two dorsal rods of erectile tissue, the corpora cavernosa, and the ventrally located corpus spongiosum, which surrounds the urethra (Fig. 5.23).

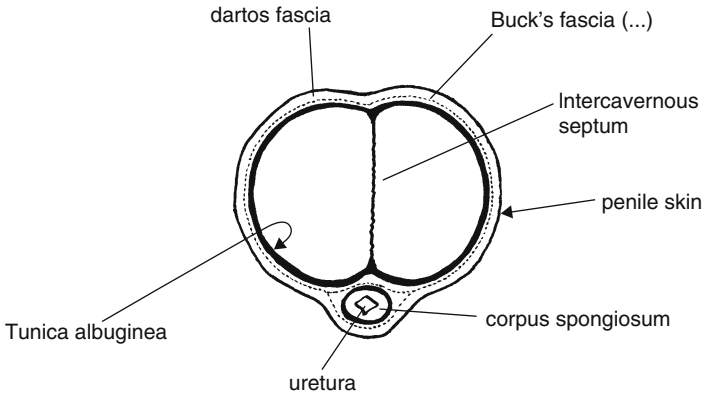


FIGURE 5.23. Fascial layers of the penis.

Attachments of Superficial Fascia of Penis and Perineum

Distally Buck's fascia is firmly attached to the base of the glans (the coronal sulcus), and laterally it is attached to the pubic rami and to the ischial spines and tuberosities. The attachments of Colles' fascia are, inferiorly, the fascia lata (the deep fascia of the thigh) in the upper mid-thigh, posteriorly to the perineal body just in front of the anus, laterally to the inguinal ligaments, and superiorly the coracoclavicular fascia (Fig. 5.22).

If the urethra has ruptured, but Buck's fascia is intact, bruising is confined in a sleeve-like configuration, along the length of the penis, by Buck's fascia. If Buck's fascia has ruptured, the extravasation of blood and thus the subsequent bruising, is limited by the attachments of Colles' fascia (the superficial perineal fascia).

Confirming the Diagnosis

The history, symptoms, and/or clinical signs described above are indications for retrograde urethrography (see Posterior Urethral Injuries, above). The key thing is to position the patient to allow adequate demonstration of the anterior urethra. The patient should lie at a 45-degree oblique angle with the bottom leg flexed at the hip and knee and the top leg completely straight and extended. An anterior urethral rupture is diagnosed when there is extravasation of contrast. If the patient has blood at the meatus, but there is no extravasation of contrast, the patient has a ure-

thral contusion. If there is extravasation of contrast, with contrast also present in the bladder, the patient has a partial rupture of the anterior urethra. If there is no filling of the posterior urethra or bladder, the anterior urethral disruption is complete.

Management of Anterior Urethral Injuries

Anterior Urethral Contusion

A small-gauge urethral catheter (12 Ch in an adult) is passed. It is removed a week or so later.

Partial Rupture of Anterior Urethra

The majority of such injuries can be managed by a period of suprapubic urinary diversion, without the need for subsequent surgery. Most will heal without a functionally significant stricture (Cass and Godec 1978, Pierce 1989), after a few weeks of drainage. If there is a penetrating partial anterior urethral disruption (e.g., knife, gunshot wound), primary (immediate) repair may be carried out, but this depends on the presence of a surgeon experienced in these techniques. There is some evidence that the stricture rate with immediate surgical repair is lower than that associated with realignment of the urethra by urethral catheterisation alone (Husmann et al. 1993).

Suprapubic catheterisation (percutaneously) is preferred over urethral catheterisation because of the concern that a partial rupture can be converted to a complete rupture. If the bladder cannot easily be palpated, such that a suprapubic catheter cannot safely be inserted, then a formal open suprapubic cystostomy (under general anaesthetic) should be performed.

It seems a sensible idea to give these patients a course of a broad-spectrum antibiotic to prevent infection of extravasated urine and blood. A voiding cystogram can be done after 2 weeks to confirm that the urethra has healed, and the suprapubic catheter can then be removed. If there is still extravasation of contrast, the suprapubic catheter can be left in place a little longer.

Seventy percent or more of partial urethral tears heal without stricture formation following a short period of suprapubic catheter drainage alone. The presence of a substantial degree of oedema and of haematoma at the site of injury makes primary closure technically difficult and can convert a short area of urethral injury into a longer one. Attempts to re-establish urethral continuity over sounds can also lead to greater damage and should be avoided. With simple suprapubic catheter drainage, if

a stricture does result, it is usually only 0.5 cm or so long and can be easily managed with optical urethrotomy or anastomotic urethroplasty.

Complete Rupture of Anterior Urethra

Where the anterior urethra has been completely torn across, then if the patient is unstable, as a consequence of other injuries, a suprapubic catheter can be placed and repair delayed until the patient has recovered from the other injuries.

If the patient is stable, the urethra may either be immediately repaired or a suprapubic catheter can be placed with delayed repair. Whether immediate repair is performed, as for partial ruptures, depends on the presence of a surgeon with sufficient experience in dealing with these injuries.

Penetrating Anterior Urethral Injuries

These are uncommon, and result from knife or gunshot wounds. They are generally managed by surgical debridement and repair (Gomez et al. 1993).

TESTICULAR INJURIES

Causes and Pathophysiology

The majority of testicular injuries in civilian practice are blunt injuries occurring during sports, motor vehicle accidents, or as a consequence of assaults. Very rarely these injuries are self-inflicted. The testicles are forced against the pubis or the thigh. Bleeding can occur into the parenchyma of the testis, and if the force is sufficient, the tunica albuginea of the testis, the tough fibrous coat surrounding the parenchyma, can rupture, allowing extrusion of seminiferous tubules.

Penetrating testicular injuries occur as a consequence of gunshot wounds, knife wounds, and from bomb blasts. Associated limb (e.g., femoral vessel), perineal (penis, urethra, rectum), pelvic, abdominal, and chest wounds may occur.

Where bleeding is confined by the parietal layer of the tunica vaginalis, a *haematocele* is said to exist (Fig. 5.24). Intraparenchymal (intratesticular) haemorrhage and bleeding beneath the parietal layer of tunica vaginalis cause the testis to enlarge slightly. The seemingly minor degree of swelling hides the fact that such a testis may be under great pressure as a consequence of the intratesticular haemorrhage. This can subsequently lead to ischaemia, necrosis, and atrophy of the testis (McDermot and Gray 1989).

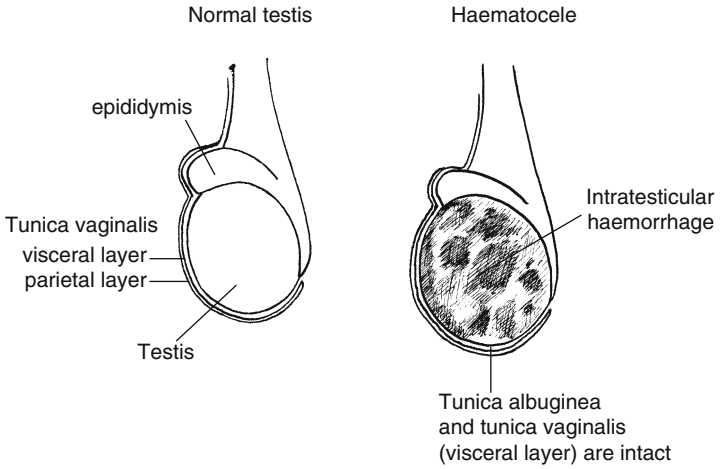


FIGURE 5.24. A haematocele within the testis.

Usually, however, a force that is sufficient to rupture the tunica albuginea will also usually rupture the parietal layer of the tunica vaginalis. Seminiferous tubules and blood extrude into the layers of the scrotum and a substantial *haematoma* may develop (Fig. 5.25).

Examination

The patient is usually in severe pain and may have nausea and vomiting. The testis may be surrounded by haematoma and therefore may not be palpable. If it is possible to palpate the testis, it is usually very tender. The degree of scrotal swelling does not always correlate with the presence of testicular rupture, since as stated above in some cases bleeding from the ruptured testis may be confined (tamponaded) by the parietal layer of the tunica vaginalis and the testis may be only slightly enlarged. The slightly enlarged testis, following trauma, may be at risk for pressure-induced ischaemia.

The scrotal haematoma resulting from a rupture of the testis and both layers of the tunica (visceral and parietal) can be very large, and the bruising and swelling so caused may as a consequence spread into the inguinal region and lower abdomen.

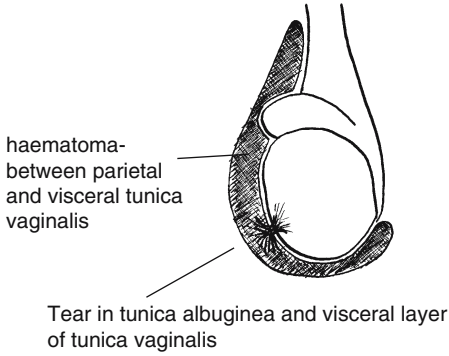


FIGURE 5.25. A haematoma around a ruptured testis.

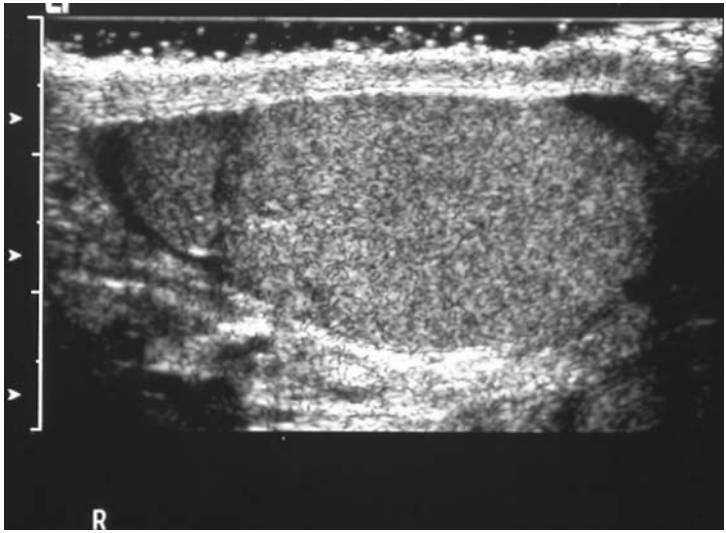


FIGURE 5.26. A normal testicular parenchymal echo pattern.

Testicular Ultrasound in Cases of Blunt Trauma

This helps decide whether or not scrotal exploration and testicular repair is necessary. A normal parenchymal echo pattern (Fig. 5.26) suggests there is no significant testicular injury, i.e., no testicular rupture. The presence of hypoechoic areas within the testis suggests testicular rupture. This is the presence of intra-



FIGURE 5.27. Intraparenchymal haemorrhage within the testis.

parenchymal haemorrhage (Fig. 5.27), the expansion of which may be limited if the tunica albuginea and/or the pariteal layer of the tunica vaginalis are intact (haematocele), or may expand into the scrotum (haematoma). The tear in the tunica may or may not be seen. The absence of a tear in the tunica does not imply the absence of a rupture of the testis.

Indications for Exploration in Scrotal Trauma

Penetrating trauma should be explored, since structures such as the vas deferens may have been severed and can be repaired. Blunt trauma resulting in testicular rupture (altered echo pattern due to intraparenchymal haemorrhage) should also be explored, so that the haematoma can be evacuated, the extruded seminiferous tubules excised, and the tear in the tunica albuginea repaired. We use a 3/0 or 4/0 Vicryl for closure of the tunica albuginea.

PENILE INJURIES

Causes and Mechanisms

These occur as a result of penile amputation (accidental or self-inflicted), knife and gunshot wounds, penile fracture, and other self-inflicted injuries. The diagnosis is usually obvious.

Penile Amputation

If the penis has been retrieved (sometimes in self-inflicted wounds it has been thrown away by the patient), place it in a wet swab inside a plastic bag, which is then placed inside another bag containing ice ('bag in a bag'). (Aboseif et al. 1993) The penis may survive for up to 24 hours if so preserved, though clearly the shorter the ischaemia time, the more likely it will survive. Blood loss can be severe, and resuscitation with intravenous fluids and blood should be used in the shocked patient.

Surgical Reimplantation

The urethra should be repaired first, over a catheter, to provide a stable base for subsequent neurovascular repair. Next close the tunica albuginea of the corpora with a 4/0 absorbable suture (repair of the cavernosal arteries is technically very difficult and does not improve outcome in terms of viability of the penis). Next, the dorsal artery of the penis should be anastomosed (11/0 nylon), followed by the dorsal vein (9/0 nylon) to provide venous drainage, and then the dorsal penile nerve (10/0 nylon). A suprapubic catheter provides additional security in draining the bladder.

Knife and Gunshot Wounds to the Penis

Associated injuries are common (e.g., scrotum, major vessels of the lower limb).

Unless the injury is minor, the majority of such injuries should be managed by primary repair (Bertini and Corriere 1988, Gomez et al. 1993). Remove debris from the wound, e.g., particles of clothing. Obviously, necrotic tissue should be debrided,

but do not be overzealous, as the penis has a very good blood supply that aids subsequent healing. Repair the tunica of the corpora with absorbable or nonabsorbable sutures (with the knots buried). Repair anterior urethral injuries over a catheter with absorbable sutures.

PENILE FRACTURE

Definition

This is the traumatic rupture of the tunica albuginea of the erect penis resulting in rupture of one or both corpora cavernosa. The corpus spongiosum with the contained urethra may also rupture. It most commonly occurs during vigorous sexual intercourse. It may also occur during masturbation, forced bending of the erect penis or any mechanical trauma to the erect penis.

During intercourse the tunica albuginea, normally measuring approximately 2 mm in thickness, thins to about 0.25 mm as the penis expands. It is therefore vulnerable to rupture if the penis is suddenly and forcibly bent. Rupture of both corpora cavernosa can occur, as can that of the corpus spongiosum surrounding the urethra, i.e., urethral rupture.

History

Penile fracture usually occurs during sexual intercourse and in this situation it is thought to occur as a consequence of forcible contact of the erect penis with the female pubis. The patient may report hearing a sudden snap or popping sound, associated with sudden onset of pain in the penis and detumescence of the erection.

Examination

The penis is swollen and bruised (Fig. 5.28). It may be so swollen that it has the appearance of an aubergine. If Buck's fascia has ruptured, then bruising will extend onto the lower abdominal wall, and into the perineum and scrotum. A tender, palpable defect may be felt over the site of the tear in the tunica albuginea. If the urethra is damaged, there will usually be blood at the urethral meatus or dipstick/microscopic haematuria. There may also be macroscopic haematuria, pain on voiding, or urinary retention.

Very occasionally a patient presents with a history of sudden pain during intercourse with bruising and swelling of the penis, but at penile exploration the tunica albuginea is found to be intact. Such cases represent rupture of the dorsal



FIGURE 5.28. Penile fracture. (See this figure in full color in the insert.)

vein of the penis, and all that needs be done is simple ligation of the vein.

Investigations

Dipstick the urine looking specifically for blood. If blood is present, or if the patient complains of pain or difficulty on voiding or inability to void, arrange a retrograde urethrogram to see if the urethra has ruptured. Agrawal et al. (1991) recommend urethrography in all cases of penile rupture and this is also our policy.

Cavernosography, the intracorporeal injection of contrast to demonstrate a fracture and penile ultrasound have been used to confirm the diagnosis where uncertainty exists. Magnetic resonance imaging (MRI) can accurately demonstrate the presence and site of a rupture, but this seems an overly complex way of investigating a condition where the diagnosis is usually obvious from the characteristic history (snapping sound, sudden detumesence, and pain during intercourse) and findings on clinical examination (marked swelling and bruising of the penis).

Treatment

Two broad categories of management are available—conservative and surgical.

Conservative treatment consists of the application of cold compresses to the penis, analgesics, and antiinflammatory drugs

and abstinence from sexual activity for 6 to 8 weeks after the injury to allow healing at the fracture site.

Surgical treatment consists of exposing the fracture site in the tunica albuginea, evacuating the haematoma, and closing the defect in the tunica. The fracture site can be exposed by degloving the penis via a circumcising incision made around the subcoronal sulcus (Fig. 5.29). Alternatively, an incision can be made directly over the defect, assuming that the degree of swelling is not too great to prevent accurate identification of this site. However, if there is a urethral injury, then a degloving injury usually allows better exposure of the urethra for subsequent repair. An alternative is a midline incision extending distally from the midline raphe of the scrotum, along the shaft of the penis. This latter incision, along with a degloving incision, allows excellent exposure of both corpora cavernosa so that an unexpected bilateral injury can be repaired easily, as can a urethral injury, should this have occurred.

The defect in the tunica may be closed with absorbable sutures or by nonabsorbable sutures, burying the knots so that



FIGURE 5.29. The fracture site in the corpora cavernosum has been identified by a degloving incision. (See this figure in full color in the insert.)

the patient is unable to palpate them. Nonabsorbable sutures may possibly be associated with prolonged postoperative pain (Asgari et al. 1996). A urethral catheter is left in place at the end of the procedure since it can be difficult for the patient to void in the immediate postoperative period.

In cases where the urethra has ruptured, this should be repaired at the same time as the tear in the tunica albuginea (Marsh et al. 1999). A spatulated one- or two-layer urethral anastomosis is carried out. The repair is splinted with a urethral catheter, which is left in place for 3 weeks.

There has been a trend away from conservative management of penile fracture toward surgical repair. There are no reported studies where patients have been randomised to conversative versus surgical treatment (and indeed this would be difficult for a condition that presents very infrequently). However, it is generally felt that conservative treatment is associated with a higher rate of complications than is surgical treatment including penile deformity, residual penile mass (presumably scar tissue), prolonged penile pain, and pulsatile cavernosal diverticulum.

Other Penile Injuries

These include bites (from humans or animals), 'zipper' injuries (catching the end of the penis in the zipper of the patient's trousers), injuries as a consequence of inserting the penis into vacuum cleaners, and injuries occurring as a consequence of industrial accidents (e.g., saw or crush injuries).

In general, devitalised tissue should be debrided, but remember that the penis has superb vascularity and aggressive debridement is not necessary. The wound should be carefully cleaned, particularly if there is a bite injury and antibiotics should be prescribed with a broad spectrum (a combination of a cephalosporin and amoxycillin is a reasonable empirical choice, but seek advice from your local microbiology department).

Zipper Injuries

If the penis is still caught in the end of the zipper, lubricate the zip, e.g., with K-Y jelly, and gently attempt to open it. If this fails, the zipper may have to be cut with orthopaedic cutters or the teeth of the zip may be prised apart with a pair of surgical clips on either side of the zipper.

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Chapter 6
Scrotal and Genital Emergencies

John Reynard and Hashim Hashim

TORSION OF THE TESTIS AND TESTICULAR APPENDAGES

During fetal development the testis descends into the inguinal canal and as it does so it pushes in front of it a covering of peritoneum (Fig. 6.1). This covering of peritoneum, which actually forms a tube, is called the processus vaginalis. The testis lies behind this tube of peritoneum and by birth, or shortly afterward, the lumen of the tube becomes obliterated. In the scrotum, the tube of peritoneum is called the tunica vaginalis. The testis essentially is pushed into the tunica vaginalis from behind. The tunica vaginalis, therefore, is actually two layers of peritoneum, which cover the testis everywhere apart from its most posterior surface (Fig. 6.2). The layer of peritoneum that is in direct contact with the testis is called the visceral layer of the tunica vaginalis, and the layer that surrounds this, and actually covers the inner surface of the scrotum, is called the parietal layer of the tunica vaginalis.

In the neonate, the parietal layer of the tunica vaginalis may not have firmly fused with the other layers of the scrotum, and therefore it is possible for the tunica vaginalis and the contained testis to twist within the scrotum. This is called an extravaginal torsion, i.e., the twist occurs *outside* of the two layers of the tunica vaginalis. In boys and men, the parietal layer of the tunica vaginalis has fused with the other layers of the scrotum. Thus, an extravaginal torsion cannot occur.

In most boys and men the testis is covered on its front and sides by the visceral layer of the tunica vaginalis, but its posterior surface is not so covered, and the posterior surface of the testis is therefore in direct contact with, and fused to, the layers of the posterior scrotum. Being fused to the scrotum in this way, the testis cannot twist around (Fig. 6.2). However, in some boys and men, the entire surface of the testis, together with a length of the spermatic cord, is covered with the visceral layer of the tunica vaginalis (Fig. 6.3). In these individuals the testis hangs

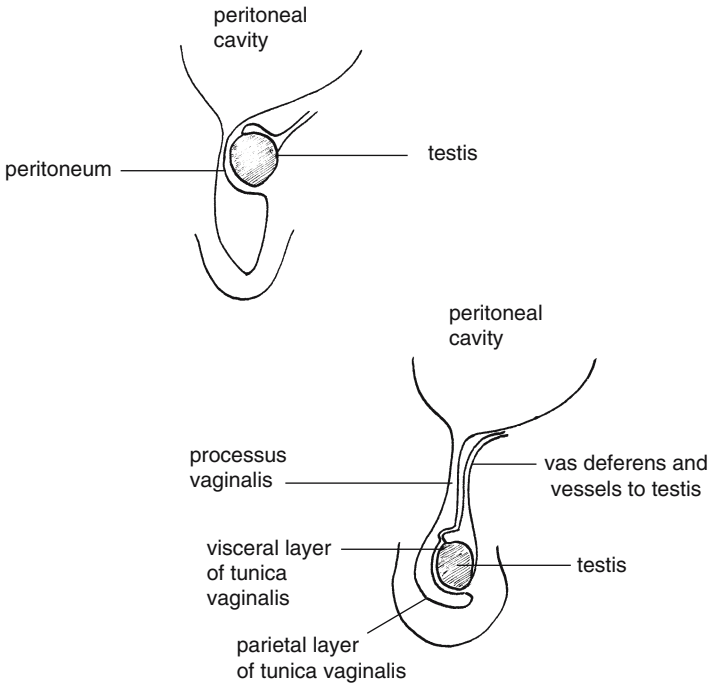


FIGURE 6.1. The testis pushing into the processus vaginalis.

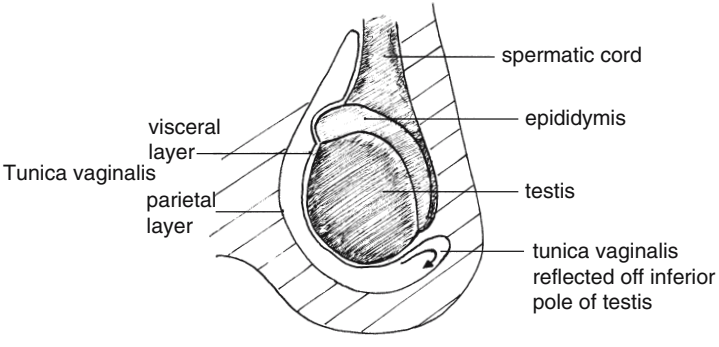


FIGURE 6.2. The posterior surface of the testis is fused to the posterior scrotum.

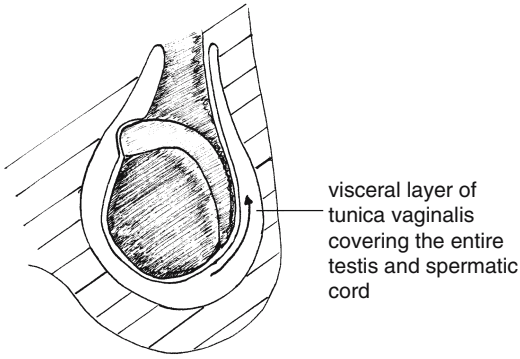


FIGURE 6.3. The entire surface of the testis together with a length of the spermatic cord, is covered with the visceral layer of the tunica vaginalis. This is the bell clapper, and it predisposes to intravaginal torsion of the testis and epididymis.

like the clapper of a bell within the scrotum. It is therefore free to rotate within the scrotum. This is called an intravaginal torsion, i.e., it occurs *between* the two layers of the tunica vaginalis.

Testicular Appendages

Attached to the testis are so-called testicular appendages. These are vestigial and are derived from embryological structures. The appendix testis (also known as a hydatid of Morgagni) is a remnant of the müllerian duct (in the female fetus this develops into the fallopian tubes and upper part of the vagina). In 80% of individuals it is pedunculated (Rolnick et al. 1968) (i.e., it is on a stalk) and is therefore prone to torsion, which can cause pain (mimicking that of a testicular torsion).

The epididymis, vas deferens, and seminal vesicles are derived from the mesonephric (wolffian) duct. An appendix epididymis, not surprisingly, is a derivative of the wolffian duct (more specifically a remnant of a cranial mesonephric tubule) and it is almost always pedunculated. Like an appendix testis, the appendix epididymis may twist and cause scrotal pain.

Definition

A testicular torsion is a twist of the spermatic cord resulting in strangulation of the blood supply to the testis and epididymis. It

may be (a) intravaginal where the testis twists within the tunica vaginalis, the more common type; or (b) extravaginal, the type that occurs in the neonatal or prenatal period.

Testicular torsion occurs most frequently between the ages of 10 and 30, with a peak at the age of 13 to 15 years, but any age group may be affected. The left side is said to be affected more often, and 2% are said to present with torsion of both testes.

Presentation

The presentation is usually one of sudden onset of severe pain in the hemiscrotum, sometimes waking the patient from sleep. It may radiate to the groin or loin, reflecting the embryological origin of the testis and its nerve supply). There may be a history of a blow to the testis in the hours before the acute onset of pain. Some patients report similar episodes occurring in the past, with spontaneous resolution of the pain, suggesting an episode of torsion with spontaneous detorsion. The patient will be in considerable pain, and may have a slight fever. Patients do not like the testis being touched and will find it difficult to walk and to get up on the examination couch, as movement causes pain. The testis is usually swollen, very tender to touch and may appear abnormally tense (if the patient lets you squeeze it!). It may be high-riding (lying at a higher than normal position in the testis) and may be in a horizontal position due to twisting of the cord. The testis may feel hard and there may be scrotal wall erythema.

The cremasteric reflex may be lost, although the presence or absence of this reflex should not be taken as reliable evidence either that the patient has a torsion or does not (Nelson et al. 2003). The cremasteric reflex may be elicited by stroking the finger along the inside of the thigh, which results in upward movement of the ipsilateral testis.

Differential Diagnosis

This includes epididymo-orchitis, torsion of a testicular appendage, and causes of flank pain with radiation into the groin and testis. From time to time the pain of a ureteric stone may be localised to the ipsilateral testis, but when the testis is palpated, the patient has no tenderness. In such cases a computed tomography urogram (CTU) confirms the presence of a stone.

Clinically, the pain of a twisted appendix testis or appendix epididymis can be difficult to distinguish from that of a testicular torsion. Sometimes, though, a little boy presents with scrotal pain and the area of tenderness on examination of the scrotum is confined almost to a single spot, which can be localised by the

tip of the examining finger (Fig. 6.4). We have never felt comfortable relying on this sign to exclude a testicular torsion and have always explored such cases.

Investigations

Both colour Doppler ultrasound and radionuclide scanning can be used to diagnose testicular torsion (Al Mufti et al. 1995). Colour Doppler ultrasound shows reduced arterial blood flow in the testicular artery. Radionuclide scanning shows decreased uptake of the radioisotope in the affected testis, an indication of absent blood flow to that testis (Melloul et al. 1995). Useful though these tests may be, they are not readily available in many hospitals. In the case of Doppler ultrasound scanning, the testis may be too tender to allow the pressure of the ultrasound probe to be applied.

Surgical Management

The mainstay of investigation and treatment of a suspected case of testicular torsion remains, in many hospitals, scrotal exploration. This should be undertaken as a matter of urgency. Delay in relieving the twisted testis can result in permanent ischaemic damage to the testis with subsequent atrophy, loss of hormone production, and loss of sperm production. Furthermore, as the testis undergoes necrosis, the blood–testis barrier breaks down and an autoimmune reaction has been shown to develop (sympathetic orchidopathia), in animal models (Cerasaro et al. 1984, Wallace et al. 1982). Whether this occurs in humans to the extent that spermatogenesis is impaired is uncertain (Anderson and

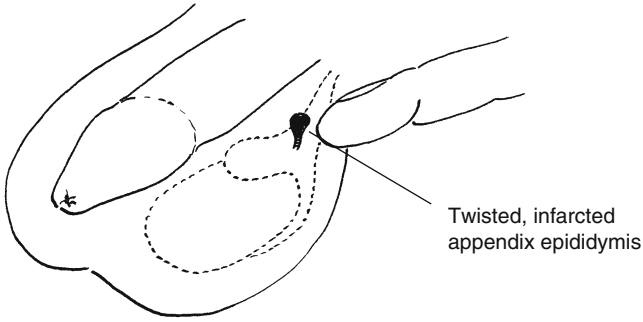


FIGURE 6.4. Point tenderness in a case of a twisted appendix epididymis.

Williamson 1986). The autoantibodies so produced can then damage the contralateral testis, thereby impairing hormone production and spermatogenesis of this side as well. A delay in relieving the torsion of more than 6 hours increases the risk that ischaemic necrosis will take place.

Warn the patient (and if he is a child, warn his parents) that if the testis is found to be dead at exploration, the best thing to do is to remove it. This is done to reduce the likelihood of an autoimmune reaction affecting the normal contralateral testis, but also because this provides the best pain relief and prevents the potential complication of infection of the necrotic tissue (which could lead to subsequent abscess formation).

Under general anaesthesia, the scrotum is explored. We use a midline incision, since this allows access to both sides so that they may both be 'fixed' within the scrotum. In some cases the testis may already be black and obviously necrotic. The spermatic cord should be ligated with a transfixion stitch of an absorbable material and the testis should be removed. If the testis has twisted and appears to be viable, untwist it and wait for it to 'pink up.' Give it the benefit of the doubt. Wait 10 minutes, placing the testis in a warm swab. You can use this timing to fix the other side. If, after 10 minutes, the viability of the testis is in doubt, make a small cut with the tip of a scalpel. If the testis bleeds actively, it should be salvaged (close the small wound with an absorbable suture).

There is some controversy surrounding the best technique for fixation. Some surgeons fix the testis within the scrotum with suture material, inserted at two or three points. Those who recommend three-point fixation do so because they argue it reduces the risk of retorsion (Phipps 1987, Thurston and Whitaker 1983). Some use absorbable sutures and others nonabsorbable sutures. Those who use the latter argue that absorbable sutures may disappear, exposing the patient to the risk of subsequent retorsion. Indeed, in a literature review Kuntze et al. (1985) found that 15 of 16 patients with recurrent torsion had undergone previous orchidopexy using absorbable suture material, and they recommended the use of 2/0 or 3/0 nonabsorbable suture material. Those who use absorbable sutures argue that the fibrous reaction around the absorbable sutures used to fix the testis will prevent retorsion and that the patient may be able to feel nonabsorbable sutures, which can be uncomfortable (though this should not occur if the sutures are placed medially, i.e., into the septum between the two testes). If you use suture fixation, these should pass through the visceral layer of the tunica vaginalis cov-

ering the testis, through the tough tunica albuginea of the testis, and then through the parietal layer of the tunica vaginalis, which lines the inner surface of the scrotum. We find it easier to clip each suture, and to tie them only after all three have been placed. Tying them after each has been placed can make it difficult to insert the next suture.

Other surgeons have argued that the testis should be fixed within a dartos pouch (Frank and O'Brien 2002). The rationale behind this form of fixation is that suture fixation breaches the blood–testis barrier, thereby exposing both testes to the risk of sympathetic orchidopathia. Dartos pouch fixation should, in theory, avoid this potential risk. In a review of 387 patients who had undergone unilateral or bilateral orchidopexy, Coughlin et al. (1998) reported that the use of testicular suture material was strongly associated with infertility. Concerns have also been expressed about a possible increased cancer risk in testes that have been suture fixed (Frank and O'Brien 2002).

Many surgeons continue to use suture fixation, and indeed operative surgery textbooks still describe this technique for use in testicular fixation for torsion (Hinman 1998).

If you use dartos pouch fixation, open the tunica vaginalis, bring the testis out, and untwist it. Develop a dartos pouch in the scrotum by holding the skin with forceps, and dissecting with scissors between the skin and the underlying dartos muscle. Once you have started to develop this space, it can be enlarged by inserting your two index fingers and pulling them apart. Place the testis in this pouch. A few absorbable sutures may be used to attach the cord near the testis to the inside of the dartos pouch. This can help to prevent retorsion of the testes (which we have seen in testes that have been placed in a dartos pouch). The dartos may then be closed over the testis and the skin can be closed in a separate layer.

Whatever technique you use, remember to fix *both* testes since the bell-clapper abnormality, which predisposes to torsion, can occur bilaterally.

If we find an appendix testis or appendix epididymis at the time of scrotal exploration, whether there is a testicular torsion or not, we remove it (with diathermy or by ligating it with a small suture), so that it cannot twist in the future, which would necessitate repeat scrotal exploration.

If we find that the testis is not twisted, then we assume that the testis had undergone torsion, but had untwisted once the patient had been anaesthetised, or that the diagnosis could be epididymo-orchitis. If there was free fluid surrounding the testis,

we take a swab and send it for culture. We fix the testis and the contralateral testis as a prophylactic measure.

PRIAPISM

Definition

Persistent erection of the penis for more than 4 hours that is not related or accompanied by sexual desire. There are two main types: *ischaemic* (veno-occlusive, low flow), and *nonischaemic* (arterial, high flow). It can affect any age, but the two main age groups affected are 5- to 10-year-old boys and 20- to 50-year-old men. There is a third type of priapism called *stuttering priapism*, which is an intermittent recurrent form of ischaemic priapism.

History

Ask the patient about these four main points:

- Duration of erection >4 hours?
- Is it painful or not? Pain implies ischaemia due to low flow; absence of pain implies high flow priapism with no ischaemia.
- Previous history and treatment of priapism?
- Identify any predisposing factors

Causes

Idiopathic drugs:
  Antihypertensives
  Anticoagulants, e.g., heparin, warfarin
  Antidepressants, e.g., paroxetine, fluoxetine
  Alcohol
  Recreational drugs, e.g., Marijuana, cocaine
  Intracavernous injections of vasoactive drugs, e.g., alprostadil, papaverine
Trauma:
  Pelvic
  Genital
  Perineal, e.g., straddle injury
Neurological:
  Seizure
  Cerebrovascular accident
  Lumbar disc disease
  Spinal cord injury
Haematological disease:
  Sickle cell disease
  Thalassaemia
  Thrombophilia
  Leukaemia
  Myeloma

Tumours:
- Bladder cancer
- Prostate cancer
- Metastatic renal cancer

Miscellaneous:
- Amyloid
- Carbon monoxide poisoning
- Total parenteral nutrition
- Rabies
- Black widow spider bites
- Malaria
- Fabry's disease

Examination

Look for the following:

- Rigid corpora cavernosa
- The corpus spongiosum and glans penis are usually flaccid.

Investigations

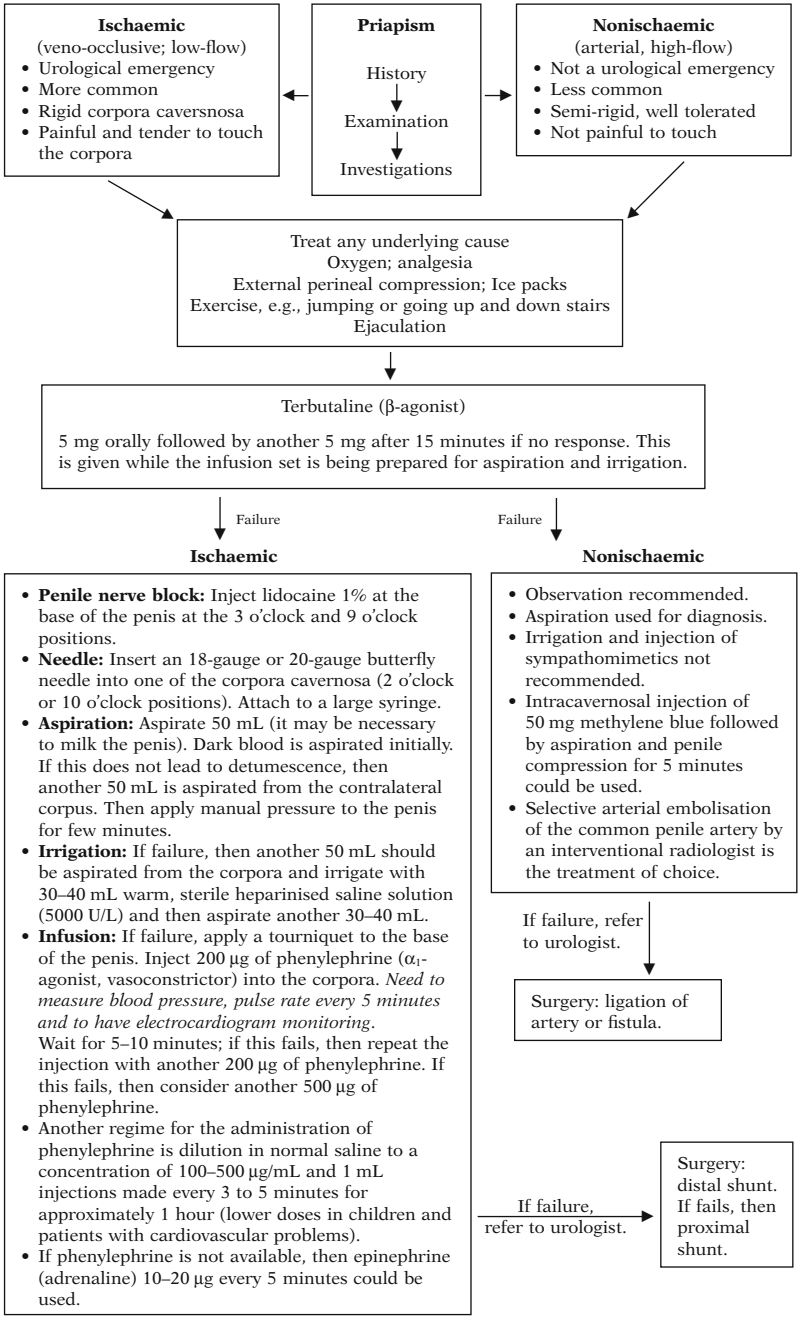
- Full blood count (white cell count and differential, reticulocyte count)
- Haemoglobin electrophoresis for sickle cell test
- Urinalysis including urine toxicology
- Blood gases taken from either corpora, using a blood gas syringe to aspirate blood, will help in differentiating between low-flow (dark blood; pH <7.25 (acidosis); pO_2 <30 mm Hg (hypoxia); pCO_2 >60 mm Hg (hypercapnia)) and high-flow priapism (bright red blood similar to arterial blood at room temperature; pH = 7.4; pO_2 >90 mm Hg; pCO_2 <40 mm Hg)
- Colour flow duplex ultrasonography in cavernosal arteries: ischaemic (inflow low or nonexistent) versus nonischaemic (inflow normal to high). This investigation may not be available at all hours.
- Penile pudendal arteriography may be done, but is not readily available at all hours.

Treatment

Treatment depends on the type of priapism. Conservative treatment should first be tried, and if it fails then it is followed by medical treatment and then by minimally invasive treatment and then by surgical treatment (Table 6.1).

Note: It is important to warn all patients with priapism of the possibility of impotence. It should be recorded in the notes and clearly written on the discharge instruction sheet.

TABLE 6.1. Treatment algorithm for priapism (*Hashim Hashim*)



PARAPHIMOSIS

Definition

This is a condition in which the foreskin is retracted from over the glans of the penis, and cannot then be pulled back over the glans into its normal anatomical position. Essentially the foreskin becomes trapped behind the glans of the penis. It can affect males at any age, but it occurs most commonly in teenagers or young men. It also occurs in elderly men who have had the foreskin retracted during catheterisation, but not been returned to its normal position after catheterisation. It can occur in an otherwise normal foreskin, which if left in the retracted position may become oedematous to the point where it cannot be reduced. Occasionally a phimotic foreskin (a tight foreskin that is difficult to retract off the glans) is retracted, and it is then impossible for it to be put back in its normal position.

History

Ask the patient if he is normally able to retract the foreskin (suggesting an otherwise normal foreskin if he can and a phimotic one if he cannot).

Examination

Paraphimosis is usually painful. The foreskin is oedematous. It may become so engorged with oedema fluid that the appearance can be very confusing for those who have never seen it. Occasionally in a paraphimosis that has been present for several days, a small area of ulceration of the foreskin may have developed, which those unfamiliar with the condition may misinterpret as a malignant or infective process.

Treatment

There are several options. The patient will probably already have tried the application of pressure to the oedematous foreskin in an attempt to reduce it, and usually the attending doctor does the same, sometimes successfully reducing the foreskin, but more often than not failing to do so.

The 'iced-glove' method: Apply topical lignocaine (lidocaine) gel to the glans and foreskin. Wait for 5 minutes so you achieve anaesthesia of the area. Place ice and water in a rubber glove and tie a knot in the cuff of the glove to prevent the contents from pouring out. Also tie off the four fingers of the glove. Place the thumb of the glove over the penis so that the penis lies within it

and in contact with the ice and water. This may reduce the swelling and allow reduction of the foreskin.

Granulated sugar has been used to reduce the oedema (by an osmotic effect). The sugar may be placed in a condom or glove applied over the end of the penis. The process of reduction may take several hours (Kerwat et al. 1998).

Hyaluronidase injections have been used (1 mL; 150 U/cc), injected via a 25-gauge hypodermic needle into the prepuce. This breaks down hyaluronic acid and decreases the oedema.

The Dundee technique (Reynard and Barua 1999): Give the patient a broad-spectrum antibiotic such as 500 mg of ciprofloxacin by mouth. Apply a ring block to the base of the penis using a 26-gauge needle. Use 10 mL of 1% plain lignocaine or 10 to 20 mL of 0.5% plain bupivacaine (Marcaine) to the skin at the base of the penis. Wait for 5 minutes. Touch the skin of the prepuce to check that the penis has been anaesthetised. Try pricking the skin of the penis with a sterile needle and ask the patient if he can feel it to make sure it is well anaesthetised. Occasionally adequate anaesthesia is not achieved and the patient will require a general anaesthetic. In children we have tended to use general anaesthesia. Clean the skin of the foreskin and the glans with cleaning solution. Using a 25-gauge needle make approximately 20 punctures into the oedematous foreskin. Firmly squeeze the foreskin. This forces the oedema fluid out of the foreskin (Fig. 6.5). Small 'jets' of oedema fluid will be seen. Once the foreskin has been decompressed, it can usually be returned to its normal position. We discharge the patient on a 7-day course of ciprofloxacin as a prophylactic measure and recommend daily baths with careful cleaning of the glans and skin with soap and water. The patient should be advised to dry the foreskin carefully and return it to its normal position afterward.

Since we first used the Dundee technique in 1996, we have not had to perform a dorsal slit in any patient (Reynard and Barua 1999). We have used this method of reduction in cases where the paraphimosis had been present for a week. Approximately one third of patients underwent elective circumcision for an underlying phimosis.

If this method fails to reduce the paraphimosis, then recourse to the traditional surgical treatment of a dorsal slit is required, usually under general anaesthetic or ring block. Make an incision in the tight band of constricting tissue. Pull the foreskin back over the glan, checking that it can move easily over the glans. If you make a longitudinal incision, this may be closed transversely, so essentially lengthening the circumference of the



FIGURE 6.5. A case of paraphimosis undergoing reduction by the Dundee technique. (See this figure in full color in the insert.)

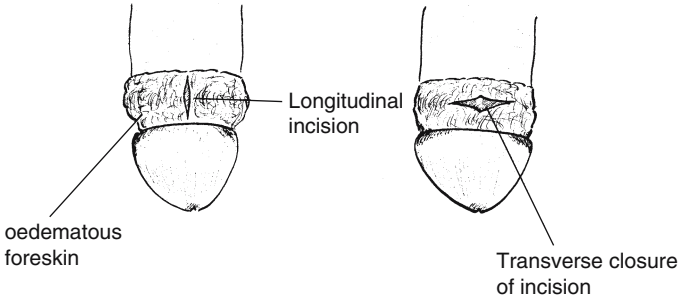


FIGURE 6.6. A dorsal slit with the longitudinal incision closed transversely.

foreskin, and hopefully preventing further recurrences of the paraphimosis (Fig. 6.6).

If, having had a dorsal slit, the patient is concerned about the cosmetic appearance, or if the underlying cause of the paraphimosis was a phimosis, then he may undergo circumcision at a

later date. We have avoided immediate circumcision in such cases, because the gross distortion of the normal anatomy of the foreskin can make circumcision difficult and lead to a less than perfect cosmetic result.

FOREIGN BODIES IN THE URETHRA AND ATTACHED TO THE PENIS

All manner of foreign bodies have been inserted into the urethra and bladder either voluntarily, by accident, or as a consequence of assault (van Ophoven and deKernion 2000). Most 'find' their way into the urethra or bladder in the search for sexual gratification. Occasionally elderly patients with dementia insert objects into their urethra and from time to time catheters and endoscopic equipment (e.g., the insulated tip of a resectoscope) may be 'lost' within the urethra or bladder.

History

Patients may present either acutely or months or even years after the object was inserted. They may complain of pain on voiding or suprapubic pain, they may report episodes of haematuria, or may present in urinary retention. The patient may volunteer that they have inserted something into the urethra, but sometimes no such history is forthcoming.

Examination and Investigations

The object may be protruding from the urethral meatus or you may be able to feel it within the urethra. A plain x-ray of the pelvis and genitalia may locate the foreign body if it is radiopaque. Alternatively, an ultrasound can locate the object. If no foreign body is seen ascending, urethrography or flexible cystoscopy can be used to identify its presence and location.

Treatment

Removing the foreign body can be a challenge. Occasionally it may be voided spontaneously, but more often than not you have to go in after it. Attempts may be made to remove it using a flexible cystoscope if it is smooth and small enough to be grasped in a stone basket or grabbed with forceps, but the latter usually cannot apply enough purchase on the object to allow it to be drawn all of the way out of urethra. It may be possible to retrieve the object under general anaesthetic using a rigid cystoscope or wider-bore resectoscope. If this fails, then open cystostomy will be required. If the object is made of glass, such as a thermometer, then it may be safer to avoid the attempt to remove it per the

urethra because of the danger that it might break and damage the urethra or even become lodged within the urethra. A formal open cystostomy may be safer for retrieval of glass objects.

If the foreign body is lying within the urethra and it cannot be pulled out or pushed back into the bladder (to be retrieved by rigid cystoscopy or open cystostomy), a urethrostomy will have to be performed in order to extract it.

Foreign bodies that have been attached to the penis, such as rings, may be particularly difficult to remove, especially if they are made of steel. The object may have become obscured from view by penile swelling, in which case the overlying tissues will have to be divided to allow the object to be seen. A technique for removing rings from fingers has been adopted for those stuck on the penis. A silk suture is passed underneath the ring, and the remainder of the suture is then bound tightly around the glans. The proximal end of the suture is then lifted and unwound from the penis, and as this is done the encircling object may be gently pushed distally over the glans, which has been wrapped in the suture. Alternatively, files, saws, or strong bone-cutting forceps may be required to remove the object. If it is made of steel, a high-speed drill, such as a dentist's drill, may be needed to cut it off. These drills can generate a substantial amount of heat as they cut through the metal, and the penis will need to be cooled as the procedure is carried out.

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Chapter 7

Postoperative Emergencies After Urological Surgery

Hashim Hashim and John Reynard

SHOCK DUE TO BLOOD LOSS

Shock is defined as inadequate organ perfusion and tissue oxygenation. The causes are hypovolaemia, cardiogenic, septic, anaphylactic, and neurogenic. The commonest cause of hypovolaemic shock is haemorrhage. Haemorrhage is an acute loss of circulating blood volume.

Following surgery, it is important to recognise the presence of shock early, identify the cause, and treat it promptly. Haemorrhagic shock may be categorised into four classes:

- Class I: up to 750 mL of blood loss (15% of blood volume); normal pulse rate (PR), respiratory rate (RR), blood pressure, urine output, and mental status.
- Class II: 750 to 1500 mL (15–30% of blood volume), PR >100; decreased pulse pressure due to increased diastolic pressure; RR 20 to 30; urinary output 20 to 30 mL/h; mildly anxious.
- Class III: 1500 to 2000 mL (30–40% of blood volume); PR >120; decreased blood pressure and pulse pressure due to decreased systolic pressure; RR 30 to 40; urine output 5 to 15 mL/h; anxious and confused.
- Class IV: >2000 mL (>40% of blood volume); PR >140; decreased pulse pressure and blood pressure; RR >35; urine output <5 mL/h; lethargic. The skin will feel cold and clammy.

Look at the trend in the vital signs in the hours preceding the development of shock. Examine the heart and lungs and check for capillary refill. A diagnosis of shock is based on the interpretation of clinical signs. Important parameters are the pulse rate, blood pressure, respiratory rate, urine output, and mental status. Changes in these parameters give an idea about the degree of hypoperfusion of vital organs (brain, kidneys) and therefore of the degree of bleeding.

Bleeding may be observed through a wound or drain, but the absence of blood in drains should not be taken as a sign of absent bleeding (drains can be blocked by clots). If the patient has undergone abdominal surgery, then intraabdominal bleeding may cause abdominal distention.

Treatment

- Remember ABC (airway, breathing, and circulation). Give the patient 100% oxygen to improve tissue oxygenation.
- Perform an electrocardiogram (ECG) and put the patient on a cardiac monitor.
- Insert two short and wide intravenous cannulae in the antecubital fossa, e.g., 16 gauge. If you cannot establish peripheral venous access due to vascular shutdown, either insert a central venous line or perform a short saphenous vein cutdown.
- Infuse 1 L of warm Hartmann's solution or if severe haemorrhage then start a colloid instead, e.g., gelofusin. Aim for a urinary output of 0.5 mL/kg/h and try to maintain the blood pressure.
- Take blood samples for full blood count (FBC), coagulation screen, urea and electrolytes, and cardiac enzymes.
- Cross-match six units of blood. There may already be blood in the bank, depending on the operation the patient had. Patients undergoing intermediate or major urological operations will at least have a group and save sample. If there is a delay in the arrival of the blood products, transfuse with O-negative blood. You should be familiar with the location of the blood bank. It takes about 1 hour to provide cross-matched blood and 10 minutes for type-specific blood.
- Do arterial blood gases to check for metabolic acidosis.
- If the patient does not stabilise or the situation deteriorates, then you will need to take the patient back to the operating room to stop the bleeding.

ANAPHYLAXIS AFTER ADMINISTRATION OF INTRAVENOUS CONTRAST OR ANTIBIOTICS

Anaphylaxis is usually encountered by urologists in the context of drug administration, e.g., antibiotics or following intravenous injection of an iodine-based contrast medium during intravenous urography (IVU). It is a type I hypersensitivity reaction mediated by immunoglobulin E (IgE) or IgG and the release of histamine, and can lead to severe shock and death. Early recognition of its symptoms and signs is therefore very important.

Symptoms

- Itching and erythema due to urticaria and a cutaneous rash
- Shortness of breath due to angio-oedema or pulmonary oedema
- Feeling faint and unconsciousness due to cardiovascular collapse
- Wheezing or stridor due to bronchospasm
- Abdominal pain

Signs

- Swelling of soft tissues including generalised oedema, e.g., lips, eyelids
- Cyanosis
- Cold peripheries
- Pallor
- Diarrhoea and vomiting

These signs and symptoms arise as a consequence of mediators of anaphylaxis acting on smooth muscle cells producing bronchospasm, vasodilation, increased capillary permeability, and secretion of exocrine glands.

Examination

- Look for soft tissue swelling.
- Measure blood pressure (BP), which may be reduced.
- Check the pulse for tachycardia.
- Check oxygen saturation with a pulse oximeter.
- Check for reduced capillary refill (>2 seconds) by pressing on the nail bed.
- Listen to the chest for wheeziness, breath sounds, and heart sounds.

Investigations

The diagnosis is essentially clinical.

Treatment

- Follow Advanced Life Support guidelines (ABC). Secure airway first if the patient has collapsed and start cardiac massage if pulseless.
- Stop the cause, e.g., i.v. infusion.
- If there is compromise to the airway, then the anaesthetist needs to be called for intubation and transfer the patient to the intensive care unit (ICU).
- Administer 100% oxygen.

- Obtain i.v. access in the antecubital fossa with a 'short and fat' venflon, e.g., 16 gauge.
- Obtain an ECG and place the patient on a cardiac monitor.
- Run intravenous normal saline into the drip. Use a colloid, e.g., gelofusin if the BP has dropped.
- Administer 0.5 mL of 1 : 1000 epinephrine i.m. or 3 to 5 mL of 1 : 10,000 epinephrine i.m. Repeat every 10 minutes until improvement. If that fails, then a slow infusion of norepinephrine could be started instead, especially if 2 L of colloid have gone in without any help. If still no improvement, then give hydrocortisone 100 mg i.v., especially if there is bronchospasm. If the patient has angio-oedema or itching, then give an antihistamine, e.g., chlorpheniramine 10 mg i.v. This can also be combined with ranitidine 50 mg i.v., as a combination of H_1 and H_2 antagonist seems to be better.
- Other treatments that could be tried include inhaled β_2-agonist, e.g., salbutamol 5 mg, if there is severe bronchospasm that has not responded to other treatment.
- If mild anaphylaxis, then there is no need for the patient to be admitted to the ICU and will need to be observed for at least 2 hours. However, if severe anaphylaxis, the patient may need inotropic support and ICU admission will be necessary.
- Following recovery, refer patients for skin patch testing and radioimmunoassays for specific IgE to see if they are allergic to anything else. You should also explain to the patients what happened and they should carry a card with them at all times saying they have an allergy to a certain drug or contrast media. If they are susceptible to being exposed to the allergen, then they should be instructed to carry i.m. epinephrine (EpiPen) with them.

To help avoid anaphylaxis you should always ask patients before giving them any medication or intravenous contrast if they have any allergies at all including drug allergies and to document that clearly in the case notes.

SCROTAL SWELLING AFTER SCROTAL SURGERY

Occasionally a large scrotal haematoma can develop after scrotal surgery such as vasectomy, hydrocele repair, or orchidectomy. This occurs in approximately 2% of cases (Kendrick et al. 1987). If the haematoma is large, surgical drainage is best carried out. It can be difficult to identify the bleeding vessel. Leave a small drain to prevent reaccumulation of the haematoma.

WOUND DEHISCENCE LEADING TO BURST ABDOMEN

Definition

This is the disruption of the apposed surfaces of a wound resulting in the breakdown of skin and deeper musculoaponeurotic layers exposing the viscera (Dickenson and Leaper 1999). It typically occurs in the first week postoperatively (Fig. 7.1).

Factors predisposing to wound dehiscence are patient-related and surgeon-related. Patient-related factors include obesity, diabetes, immunosuppression, malnutrition, malignancy, sepsis, and emergency operation. These factors favour the occurrence of wound infection and dehiscence. Other factors include coughing and straining postoperatively, which increase intraabdominal pressure and put extra tension on the sutures.

Surgeon-related factors: tying sutures too tightly can result in the suture cutting through fascial layers. There is a higher rate of wound dehiscence where suture length is less than 4× the length of the wound (Jenkins's rule).

Diagnosis

Daily wound examination may show signs of wound infection, which predisposes to wound dehiscence. Signs of impending



FIGURE 7.1. A wound dehiscence following cystectomy. The patient has an ileal conduit adjacent to the extruded abdominal contents. (See this figure in full color in the insert.)

wound dehiscence are skin breakdown and discharge of serosanguinous 'pink' fluid from the wound. You may be called to the ward because the patient's abdomen has suddenly burst, exposing the small and large bowel.

The abdominal contents should be covered with a sterile dressing, and the patient should be returned to the operating room to allow wound closure. Give intravenous analgesia, e.g., 5 mg morphine with 50 mg of intravenous cyclizine. Reassure patients and explain what has just happened and that you will need to take them back to the operating room for wound closure. At operation, wash the wound thoroughly with warm saline and debride any nonviable tissue. Resuture the wound with interrupted monofilament nonabsorbable sutures. Place the sutures 1 cm apart with a fair margin from the wound edge. The size of the suture depends on the site of the wound. The key thing is to include all the layers, including peritoneum. The sutures should remain in situ for 2 to 3 weeks. If there is evidence of sepsis, then antibiotics should be given.

POSTCIRCUMCISION BLEEDING

Bleeding following circumcision is most likely to be from the frenular artery on the ventral surface of the penis. If local pressure does not stop the bleeding (and if it is from the frenular artery it usually won't), take the patient to the operating room and either under ring block local anaesthesia or general anaesthetic, suture-ligature the bleeding vessel. Be careful not to place the suture through the urethra!

Not infrequently, a crust of coagulated blood develops around the circumference of the penis after circumcision. As blood oxidises it turns black, and this appearance can be mistaken for necrosis of the end of the penis. Reassurance of the patient (and the referring doctor!) is all that is needed.

BLOCKED CATHETER POST–TRANSURETHRAL RESECTION OF THE PROSTATE (TURP) AND CLOT RETENTION

In the U.K. National Prostatectomy Audit (Neal 1997) bleeding severe enough to require return to the operating room was reported in 0.6% of cases. However, not all patients need to return to the operating room. In many cases the bleeding can be controlled in the recovery room or on the ward.

Cross-match blood and other blood products [platelets and fresh frozen plasma (FFP) if a large transfusion is anticipated], and give plasma expanders, if the patient shows cardiovascular compromise, while awaiting the blood.

If the catheter has blocked, take a 50-mL bladder syringe and flush the outflow channel of the catheter. Immediately aspirate urine in an attempt to suck out clots contained within the bladder. If urine flow is reestablished, continue to irrigate the bladder, while applying traction on the catheter so that the balloon will tamponade any bleeding vessels at the bladder neck (these may have been the source of the bleeding). Inflate the balloon of the catheter to a total of 50 mL of water (a 30-mL balloon easily accommodates this volume) to maximise this effect. Applying pressure in this way for 20 minutes can stop the bleeding. If bleeding continues despite traction, or recurs after a period of traction, it is usually best to take the patient back to the operating room to establish where the bleeding is coming from and to control it with diathermy. This also provides the best way of removing large clots from the bladder (by using the Ellik evacuator and a large-bore resectoscope).

The same approach should be used for clot retention due to other sources of heavy haematuria. The bleeding is usually more easily controlled than with post-TURP bleeding.

EXTRAPERITONEAL PERFORATION DURING TURP

See Chapter 5.

THE TRANSURETHRAL RESECTION (TUR) SYNDROME

In the National Prostatectomy Audit (Neal 1997), the TUR syndrome occurred in 0.5% of cases. It is characterised by a number of symptoms and signs that may be present in variable degree depending on the severity of the condition. These include confusion, nausea, vomiting, hypertension, bradycardia, and visual disturbances.

The diagnosis of the TUR syndrome calls for a high degree of awareness on the part of the urological team. It may be ushered in with restlessness and hypertension, and rapidly proceed to what appears to be a grand mal seizure. If the patient is under spinal anaesthesia and is therefore awake during the procedure, he may report visual disturbances such as flashing lights. This can be a very helpful warning that significant amounts of glycine (and therefore fluid) are being absorbed and that corrective measures should be started. One of the authors was once explaining this feature of TUR syndrome to a junior anaesthetic colleague when the patient suddenly complained of flashing lights. The operation was quickly brought to a conclusion and the patient responded rapidly to intravenous frusemide

and fluid restriction, without going on to develop the more serious manifestations of advanced TUR syndrome.

Dilutional hyponatraemia is the most important and serious factor leading to the symptoms and signs. The serum sodium usually has to fall to below 125 mmol/L before the patient becomes unwell. The hypertension is due to fluid overload. Visual disturbances may be due to the fact that glycine is a neurotransmitter in the retina.

Definitive Treatment of the TUR Syndrome

Send a sample of blood to the lab for sodium measurement, and give 20 to 40 mg of intravenous frusemide to off-load the excess fluid that has been absorbed.

DISPLACED CATHETER POST–RADICAL PROSTATECTOMY

Urethral catheters are left in situ post–radical prostatectomy for a variable time depending on the surgeon who performs the operation. Some surgeons leave a catheter for 3 weeks and others for just 1 week. Thus, if a catheter falls out a week after surgery, the patient may well void successfully, and in this situation no further action need be taken.

If, however, the catheter inadvertently falls out the day after surgery, we would make a gentle attempt to replace it with a 12-Ch catheter that has been well lubricated. If this fails, we would pass a fleixble cystoscope, under local anaesthetic, into the bulbar urethra and attempt to pass a guidewire into the bladder, over which a catheter can then safely be passed. If this is not possible, another option is to hope that the patient voids spontaneously, and does not leak urine at the site of the anastomosis. An ascending urethrogram may provide reassurance that there is no leak of contrast and that the anastomosis is watertight. If there is a leak or the patient is unable to void, a suprapubic catheter could be placed, either percutaneously or under general anaesthetic via an open cystostomy.

COMPARTMENT SYNDROME OF THE LOWER LIMB ASSOCIATED WITH THE LITHOTOMY POSITION

Lower limb compartment syndrome (LLCS) is the development of an increased tissue pressure within the closed osteofascial compartment of the leg, which reduces perfusion of the leg leading to ischaemia of the muscles and nerves. If prolonged, it leads to permanent loss of function in the affected muscles and nerves. In the context of urological surgery LLCS is specifically associated with the lithotomy position and is said to occur with

a frequency on the order of 1 in 3500 (Halliwell et al. 1998). Thus, it is rare, but because the consequences of missing the diagnosis of LLCS are devastating, it is important to appreciate its predisposing factors, presentation, and subsequent management.

The leg has four osteofascial compartments, which are bordered by nonelastic fascia and bone. Normal resting tissue pressure in the anterior compartment of the leg ranges between 3 and 22 mm Hg.

Mechanisms

Any factor that induces ischaemia in the leg can lead to a compartment syndrome. Ischaemia disrupts the integrity of the vascular endothelium, leading to fluid shifts into the extracellular tissue space with a consequent rise in tissue pressure. The lithotomy position causes ischaemia in the leg by the following mechanisms:

1. Reduction in hydrostatic perfusion pressure. Every 1-cm elevation of the limb above the heart reduces mean arteriolar pressure by 1 mm Hg and causes a measurable reduction in ankle–brachial pressure index. This reduction in perfusion pressure is compounded by the head-down position.
2. Calf compression. This can occlude both venous drainage and arterial flow.
3. Knee and hip flexion can compress blood vessels.
4. Dorsiflexion of the foot causes an increase in pressure within the calf.

As compartment pressure rises, the lumen of arterioles is eventually occluded. A vicious cycle of ischaemia sets in. When the limb is returned to the supine position, a reperfusion injury can cause a further rise in compartment pressure.

The major factor determining the likelihood of development of a LLCS is time spent in the lithotomy position. The exaggerated lithotomy position is more likely to lead to a LLCS than is a lower lithotomy position. Hypotension, hypovolaemia, and peripheral vascular disease all predispose to development of the compartment syndrome. Young, large men with an increased muscle bulk may be at greater risk of a compartment syndrome because of tighter, less compliant compartments in the leg.

Presentation and Treatment

The classic presentation is with pain in the leg and paraesthesia. Passive stretching of the affected muscles causes worsening of

the pain. The pain may be out of all proportion to the physical signs. The skin may be pink and the pulse may still be present. It is possible to measure compartment pressures, but the equipment for doing this and expertise in recording and interpreting the pressures so measured are unlikely to be available in many cases. A high index of suspicion, therefore, is required to make a clinical diagnosis.

The mainstay of treatment is decompression of the affected compartment by a fasciotomy. Ideally such a procedure should be carried out by an expert (orthopaedic, vascular, or plastic surgeon), but if this is unlikely to be available at very short notice, the urologist will have to proceed with fasciotomy, relying on his anatomical knowledge to avoid damage to structures such as the common peroneal nerve.

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Chapter 8
Ureteric Colic in Pregnancy

John Reynard

While hypercalciuria and uric acid excretion increase in pregnancy (predisposing to stone formation), so too do urinary citrate and magnesium levels (protecting against stone formation). The net effect is that the incidence of ureteric colic is the same as in nonpregnant women (Coe et al. 1978). Depending on what series you read, somewhere between 1 in 1500 to 1 in 2500 pregnancies are complicated by ureteric stones. The great majority of ureteric stones in pregnant women occur during the second and third trimesters (Stothers and Lee 1992). The development of a ureteric stone during pregnancy is an important event, not only because it results in pain, the cause of which can be difficult to establish and to distinguish from other causes, but also because it can be difficult to treat and because it is associated with a significant risk of preterm labour (Hendricks et al. 1991).

THE HYDRONEPHROSIS OF PREGNANCY

In 90% of pregnancies the kidneys are hydronephrotic and this develops from approximately week 6 to week 10 of gestation. It has usually resolved within 2 months of birth (Peake et al. 1983). The hydronephrosis of pregnancy is due to a combination of the smooth muscle relaxant effect of progesterone and to mechanical obstruction from the enlarging fetus and uterus, which compress the ureter (hydronephrosis is said not to occur in pelvic kidneys or those transplanted into ileal conduits, nor does it occur in quadripeds such as dogs and cats where the uterus is dependent and thus 'falls' away from the ureter; Robert 1976).

The hydronephrosis of pregnancy poses diagnostic difficulties in women presenting with flank pain thought to be due to a renal or ureteric stone. Because of the desire to avoid using ionising radiation in pregnant women, renal ultrasonography is often used as the initial imaging technique in those presenting with flank pain. In the nonpregnant patient, the presence of hydronephrosis is taken as surrogate evidence of ureteric

obstruction. Because hydronephrosis is a normal finding in the majority of pregancies, its presence *cannot* be taken as a sign of a possible ureteric stone. Ultrasound is an unreliable way of diagnosing the presence of stones in pregnant (and in nonpregnant) women. In a series of pregnant women, ultrasound had a sensitivity of 34% (i.e., it misses 66% of stones) and a specificity of 86% for detecting an abnormality in the presence of a stone (i.e., false-positive rate of 14%) (Stothers and Lee 1992).

PRESENTATION OF STONES IN PREGNANCY

Flank pain is the usual presentation, with or without haematuria (macroscopic or microscopic). Differential diagnoses include placental abruption, appendicitis, and pyelonephritis, to name but a few.

WHAT IMAGING STUDY SHOULD BE USED TO ESTABLISH THE DIAGNOSIS OF A URETERIC STONE IN PREGNANCY?

Exposure of the fetus to ionising radiation can cause fetal malformations, malignancies in later life (leukaemia), and mutagenic effects (damage to genes causing inherited disease in the offspring of the fetus). Fetal radiation doses during various procedures are shown in Table 8.1.

Radiation doses of <100 mGy are very unlikely to have an adverse effect on the fetus (Hellawell et al. 2002). In the United States, the National Council on Radiation Protection (NCRP) has stated, 'Fetal risk is considered to be negligible at <50 mGy when compared to the other risks of pregnancy, and the risk of malformations is significantly increased above control levels at doses >150 mGy' (NCRP 1997). The American College of Obstetricians and Gynecologists (ACOG) has stated, 'X-ray exposure to

TABLE 8.1. Fetal radiation dose after various radiological investigations

Procedure	Fetal dose mGy (mean)	Risk of inducing fetal cancer (up to age 15 years)
KUB x-ray	1.4	—
IVU 6 shot	1.7	1 in 10,000
IVU 3 shot		
CT—abdominal	8	
CT—pelvic	25	
Fluoroscopy for JJ stent insertion	0.4	1 in 42,000

CT, computed tomography; IVU, intravenous urogram; JJ stent; KUB, kidney and urinary bladder.

<50 mGy has not been associated with an increase in fetal anomalies or pregnancy loss' (ACOG 1995).

While these recommended maximum radiation levels are well above those occuring during even computed tomography (CT) scanning, and a dose of 50 mGy or less is regarded as safe, understandably there is a concern that any radiation dose exposes the fetus to some risk. For this reason every effort should be made to limit exposure of the fetus to radiation, to use alternative imaging tests where possible, and to minimise radiation exposure during treatment by JJ stent insertion or ureteroscopy. However, the pregnant woman may be reassured that the risk to her unborn child as a consequence of radiation exposure is likely to be minimal.

Investigations or treatment that involve exposure to ionizing radiation should not be withheld because of an unjustified fear of damaging the fetus. The risks associated with irradiating the fetus have to be balanced against the risks of missing the diagnosis of a stone obstructing the ureter and the difficulties and potential dangers of performing JJ stent insertion or ureteroscopy without the use of any (ionising radiation) imaging. While ureteroscopy can be performed without fluoroscopy (Rittenberg and Bagley 1988), most urologists nowadays perform the majority of their ureteroscopic work under fluoroscopic control, and may feel uncomfortable doing otherwise in a case that, as it involves a pregnant woman and an unborn baby, is already high risk. It is worth remembering that the radiation dose during fluoroscopy for JJ stent placement is very low (on the order of 0.4 mGy, and up to a maximum of 0.8 mGy) and that the dose used to assist ureteroscopy is likely to be little more than this.

Plain Radiography and Intravenous Urography (IVU)

These studies have limitations in pregnancy. First, the fetal skeleton and the enlarged uterus may obscure ureteric stones, so the imaging study may not be diagnostic. Second, there may be delayed excretion of contrast as a consequence of the physiological dilatation of the kidney. It can be difficult, if not impossible, to differentiate this 'physiological' delay from that due to an obstructing stone. Third, there is also the theoretical risk of fetal toxicity from the contrast material, though none has been reported.

Ultrasound

As stated above, ultrasound is an unreliable way of diagnosing the presence of stones in pregnant women. Jets of urine expelled

by normal peristalsis of the nonobstructed ureter can be seen on ultrasound scanning (Fig. 8.1), and the absence of such ureteric jets is said to have a high sensitivity and specificity for diagnosing obstructing stones (Doyle et al. 1995), though others have reported that ureteric jets may be absent in asymptomatic pregnant women (Burke and Washowich 1998).

Computed Tomography Urography (CTU)

Although CT urography is a very accurate method for detecting ureteric stones and the radiation dose is below 50 mGy, most radiologist and urologists do not recommend this form of imaging in pregnant women. Magnetic resonance urography (see below) provides an alternative form of imaging in this difficult group of patients.

Magnetic Resonance Urography (MRU)

The American College of Obstetricians and Gynecologists and the U.S. National Council on Radiation Protection state, 'Although



FIGURE 8.1. Jets of urine expelled by normal peristalsis of the nonobstructed ureter can be seen on ultrasound scanning or on computed tomography (CT) (as shown here). CT should be avoided if at all possible in pregnancy.

there is no evidence to suggest that the embryo is sensitive to magnetic and radiofrequency at the intensities encountered in MRI, it might be prudent to exclude pregnant women during the first trimester' (ACOG 1995, NCRP 1997). Given this advice, therefore, MRU can potentially be used during the second and third trimesters, but not during the first trimester.

MRU involves no ionising radiation and can be done with the administration of contrast (Fig. 8.2). It is very accurate, with one group reporting a sensitivity for detecting ureteric stones of 100% (Roy et al. 1996). However, MRU is expensive, and not readily available in most hospitals, particularly after 5 o'clock. As MR scanners become more widespread, it is likely that this imaging modality will be used increasingly to establish a diagnosis in pregnant women with flank pain.

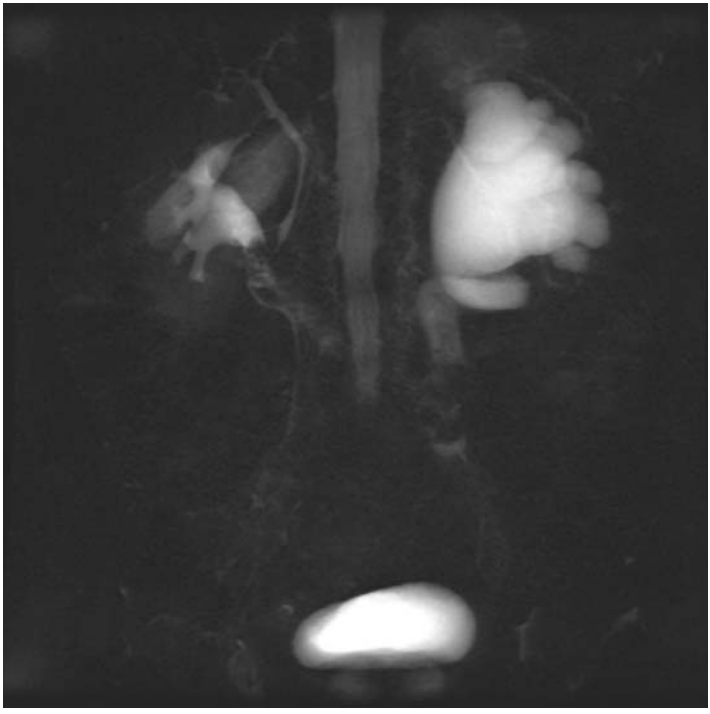


FIGURE 8.2. Magnetic resonance urography.

MANAGEMENT OF URETERIC STONES IN PREGNANT WOMEN

The majority (70–80%) of ureteric stones in pregnant women pass spontaneously (Stothers et al. 1992. Of those that do not pass and require temporizing treatment with nephrostomy tube drainage or JJ stents, many pass spontaneously postpartum. Opiate-based analgesics are used for pain relief and oral and intravenous fluids for hydration. Nonsteroidal antiinflammatory drugs (NSAIDs) should be avoided because they can cause premature closure of the ductus arteriosus by blocking prostaglandin synthesis.

The indications for intervention are essentially the same as in nonpregnant patients and include pain refractory to analgesics, suspected urinary sepsis (high fever, high white count), high-grade obstruction, and obstruction in a solitary kidney.

Options for intervention are JJ stent urinary diversion, nephrostomy urinary diversion, or ureteroscopic stone removal. Which option you use depends on how advanced the pregnancy is, and on local facilities and expertise. Management of cases requiring active intervention should aim to minimize radiation exposure to the fetus, and to minimize the risk of miscarriage and preterm labour. General anaesthesia can precipitate preterm labour (Duncan et al. 1986), and with this in mind many urologists and obstetricians err on the side of temporizing options such as nephrostomy tube drainage or JJ stent placement, rather than on operative treatment in the form of ureteroscopic stone removal.

Nephrostomy urinary diversion is widely available (Fig. 8.3), can be done rapidly, provides good pain relief, drains infected urine if present, and has a low risk of inducing miscarriage or preterm labour (Kavoussi et al. 1992). These advantages must be weighed against the fact that there is a small risk (in the order of 1%) of heavy bleeding, requiring embolisation and/or blood transfusion during nephrostomy insertion, and of septicaemic shock occurring after insertion (2–4%; Ho and Cowan 2002, Ramchandani 2001) (see Chapter 10). Furthermore, the nephrostomy tube may be required for some months, particularly when it is inserted at a relatively early stage in the pregnancy. It can be uncomfortable, may block or become infected, and may need to be changed several times during the remaining pregnancy.

JJ stents overcome some of the problems of nephrostomy tube drainage. They can be placed under local anaesthetic or with light sedation with low doses of pethidine and diazemuls using either ultrasound guidance or limited periods of fluoroscopy (Hellawell et al. 2002, Stothers et al. 1992) (see Chapter 10). They



FIGURE 8.3. Nephrostomy urinary diversion.

are an effective way of managing the pain of obstructing stones. They may be a more comfortable form of urinary diversion than percutaneous tube drainage, though many patients develop 'stent symptoms' (frequency, urgency, and bladder pain), which can be so bothersome that in some cases the stent has to be removed (Hellawell et al. 2002).

In two series totalling 20 pregnant women who underwent JJ stent placement (all under local anaesthetic or with sedoanalgesia), at between 6 to 36 weeks' gestation (mean 31 weeks), there were no cases of premature labour (Hellawell et al. 2002, Stothers et al. 1992).

The hypercalciuria of pregnancy may make stent encrustation and blockage more likely, and as a consequence it has been suggested that stents should be changed every 6 to 8 weeks to prevent the occurrence of blockage from encrustation (Kavoussi et al. 1992). However, in a contemporary series where stent insertion was performed at an average of 28 weeks of gestation for obstructing ureteric stones, stent replacement was not required in any patient (Hellawell et al. 2002), and in a slightly older series, only 1 of 13 stents required replacement because of ongoing pain (presumably indicating obstruction) (Stothers et al. 1992). It may well be, therefore, that regular stent changes, at least when using contemporary stents, are not required. Avoiding the need to change JJ stents is clearly desirable, as this is technically more challenging than replacing a percutaneous nephrostomy tube (though the difficulty of placement and replacement depend on the availability of local expertise). Therefore, one might be more inclined to recommend nephrostomy tube drainage in very early pregnancy, rather than a JJ stent where frequent changes of the latter might, at least in theory, be required throughout the remaining pregnancy (Denstedt and Razvi 1992).

JJ stents have been reported to become obstructed by mechanical impingement of the fetal head (Hellawell et al. 2002) and they may migrate down the ureter and into the bladder and subsequently be voided per urethra as a consequence of the dilatation of the ureter that is normally a feature of pregnancy (Stothers et al. 1992).

Ureteroscopic stone extraction can be performed in pregnancy, but again its use depends on available expertise. Distortion of the distal third of the ureter during the latter stages of pregnancy makes rigid ureteroscopy technically more challenging, as does the presence of a large stone (European Association of Urology 2001). For these reasons the less experienced ureteroscopist may decide that nephrostomy tube drainage or a JJ stent is a better option later on in pregnancy, with subsequent ureteroscopic treatment being used if the stone fails to pass within a few weeks of delivery. In solitary kidneys nephrostomy tube drainage or a JJ stent may also be safer options rather than attempting

ureteroscopic stone extraction under the difficult conditions of late pregnancy.

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Chapter 9

Management of Urological Neoplastic Conditions Presenting as Emergencies

John Reynard and Hashim Hashim

TESTICULAR CANCER

Approximately 10% of cases of testicular cancer present with metastatic disease in the retroperitoneum (retroperitoneal node involvement causing back pain), chest (breathlessness, cough), and neck (enlarged cervical nodes, tracheal compression, and deviation). Spread to the central nervous system or involvement of peripheral nerves can result in neurological manifestations (Fig. 9.1). While most such cases present directly to oncologists, from time to time the urologist is the first port of call. Such cases should be referred to the oncologists as a matter of urgency for high-dose chemotherapy.

MALIGNANT URETERIC OBSTRUCTION

The ureters enter the bladder just a few centimeters from the bladder neck, and it is not difficult to see how a locally advanced prostate or bladder cancer can obstruct them (Clarke 2003) (Fig. 9.2). Similarly, the cervix in women is very closely related to the lower ureters (which is why the latter may be damaged during hysterectomy) and locally advanced cervical cancer can cause lower ureteric obstruction, as can a locally advanced rectal cancer in both sexes (Soper et al. 1988). Other malignancies (colon, stomach, lymphoma, breast, bronchus) can metastasize to pelvic and retroperitoneal lymph nodes, causing unilateral or bilateral malignant ureteric obstruction. In unilateral obstruction with a normally functioning contralateral kidney, the obstruction proceeds silently. In bilateral obstruction, oliguria, leading later to anuria and finally renal failure, is the mode of presentation.

The emergency presentation is usually one of a patient with acute renal failure, who may or may not be known to have cancer. Patients present with a rising creatinine and symptoms



FIGURE 9.1. Advanced testicular malignancy with nodal metastases in the neck causing tracheal deviation.

of renal failure including malaise, nausea, vomiting, and in some cases marked oliguria or anuria as the locally advanced or nodal metastases obstruct their ureters. This presentation is sometimes mistaken for urinary retention, particularly if the patient has some lower abdominal pain. However, when the bladder is catheterised it contains only a small volume of urine and the high creatinine level does not fall. In the case of prostate cancer, digital rectal examination (DRE) reveals a firm (craggy) prostate that has extended laterally. A locally advanced rectal cancer may be felt on DRE, and in women vaginal examination may reveal a hard, craggy mass arising from the cervix.

In terms of clinical examination, it is advisable to perform a DRE in both men and women. Vaginal examination should be done in women as should examination of the breasts. General abdominal examination may reveal other evidence of malignant disease. Look for cervical and axillary lymph nodes. Measure the serum creatinine. A renal ultrasound reveals bilateral hydronephrosis, with an empty bladder. An abdominal computed

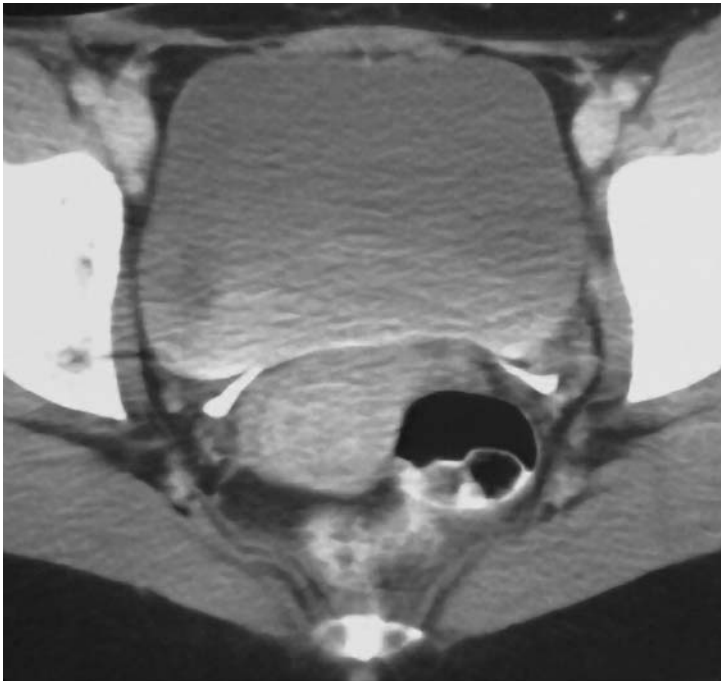


FIGURE 9.2. A computed tomography (CT) scan of the bladder showing the ureters entering posteriorly (outlined with contrast). The ureters enter the bladder just a few centimeters from the bladder neck and can easily be obstructed by locally advanced prostate cancer.

tomography (CT) scan may demonstrate evidence of retroperitoneal and pelvic lymphadenopathy.

Emergency Treatment

In cases of prostate cancer high-dose dexamethsone has been shown to result in an improvement in urine output and reduction in serum creatinine within 24 to 48 hours (Hamdy and Williams 1995). Give an 8-mg intravenous bolus followed by 4 mg i.v. every 6 hours for 3 days, switching to oral dexamathasone thereafter. A reducing regimen can be used over the course of the next month.

Where the patient is uraemic or has a rising serum potassium, more urgent treatment may be required. This can be in the form of percutaneous nephrostomy tube drainage, or if the patient is too unwell for this, acute haemodialysis.

In our experience attempts at retrograde JJ stent placement in the acute situation usually fail (it is impossible to pass a guidewire past the area of ureteric obstruction). A nephrostomy tube allows subsequent antegrade JJ stenting, and this may become the definitive management method, with the stents being changed every few months. In the case of prostate cancer, hormone treatment should be started (if not already done so), in the form of emergency orchidectomy or with antiandrogen blockade followed by a luteinizing hormone–releasing hormone (LHRH) agonist.

There are clearly issues related to the long-term prognosis of such patients. Patients with cervical and prostate cancer can survive for many months after presenting with ureteric obstruction, whereas the prognosis in patients with ureteric obstruction due to other cancers tends to be considerably shorter. Fallon and colleagues (1980) reported a median survival in prostate cancer patients treated with nephrostomy drainage for bilateral ureteric obstruction of 7 months post–nephrostomy insertion, and 55% of patients survived for over 1 year. For cervical cancer patients the average survival was 18 months. Bladder cancer patients did poorly, with a median survival of just 4 months after nephrostomy drainage.

SPINAL CORD COMPRESSION IN PATIENTS WITH UROLOGICAL DISEASE

While cord compression is a relatively uncommon presentation in patients with malignant disease, it can have a devastating impact on quality of life. Urologists should be aware of the presentation and management of cord compression, particularly since prostate cancer is the second most common cause of malignant spinal cord compression. Local extension of a vertebral metastasis compresses the spinal cord, leading to venous obstruction and oedema (at this stage, steroids can decrease the oedema and reverse the neurological symptoms and prevent further progression). The majority of cases involve the thoracic or lumbar spine; the cervical spine is infrequently involved.

All too often patients with spinal cord compression have warning symptoms and signs, the significance of which is not appreciated until irreversible damage to the spinal cord has occurred. Patients are then condemned to spend their remaining

months of life in a wheelchair. In a recent review of 24 patients presenting with cord compression due to metastatic prostate cancer (Tazi et al. 2003), 79% had thoracic or lumbar back pain severe enough to require opiate pain relief, on average for 60 days (and ranging from 10 to 840 days) before they finally presented with neurological symptoms such as paralysis. Occasionally cord compression is the first presenting event in a patient with metastatic prostate cancer.

Back pain is the most common early presenting symptom. It is usually gradual in onset and progresses slowly but relentlessly. The pain may be localised to the area of vertebral metastasis, but may also involve adjacent spinal nerve roots, causing radicular pain. Interscapular pain that wakes the patient at night is characteristic of a metastatic deposit. Associated symptoms suggestive of a neurological cause for the pain include pins and needles, weakness in the arms (cervical cord) or legs (lumbosacral spine), urinary symptoms such as hesitancy and a poor urinary flow, constipation, loss of erections, and seemingly bizarre symptoms such as loss of sensation of orgasm or absent ejaculation. From time to time the patient may present in urinary retention. It is all too easy to assume that this is due to malignant prostatic obstruction if other neurological symptoms and signs are not sought.

The physical sign of spinal cord compression is a sensory level, but this tends to occur late in the course of cord compression. Remember, however, that a normal neurological examination does not exclude a diagnosis of cord compression. If, on the basis of the patient's symptoms you suspect cord compression, arrange for a magnetic resonance imaging (MRI) without delay.

Imaging in Suspected Cord Compression

While plain x-rays of the cervical, thoracic, and lumbar spine can show vertebral metastases in over 80% of symptomatic patients, MRI allows accurate identification and localisation of metastases and is the imaging modality of choice.

Treatment

In the majority of patients initial treatment consists of pain relief, cortiscosteroids, and androgen deprivation (if not already started), followed by radiotherapy.

Dexamethasone is the steroid of choice (Greenberg et al. 1980, Sorensen et al. 1994). It reduces vasogenic oedema. Very high doses may be required (100 mg bolus of i.v. dexamethasone,

followed by doses every 6 hours of between 4 to 24 mg). Androgen deprivation therapy may be in the form of either radical orchidectomy (which produces a rapid response) or maximal androgen blockade with an antiandrogen combined with an LHRH agonist.

Surgical decompression (laminectomy) is used in patients with a life expectancy of >6 months who have had previous radiotherapy at the involved site, for those whose neurology deteriorates during radiotherapy, or for those who have a cord compression of unknown histology.

Prognosis

Patients who are still able to walk by the time they receive treatment have a high chance (70–90%) of remaining ambulatory after treatment. Of those patients who present with complete paralysis prior to onset of treatment, only 20% to 40% will regain the ability to walk (Tazi et al. 2003). Of those presenting with urianry retention prior to onset of treatment, only 40% will regain normal voiding after treatment.

The mean survival of ambulatory patients is longer (on the order of 18 months) compared with those presenting with paraplegia (approximately 4 months) (Smith et al. 1993). Those patients who have not received androgen deprivation prior to the onset of cord compression survive for longer when compared with those who are already on hormone treatment at the time of presentation with cord compression (Huddart et al. 1997, Tazi et al. 2003).

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Chapter 10
Common Emergency Urological Procedures

John Reynard and Nigel Cowan

URETHRAL CATHETERISATION

Indications

Indications for urethral catheterisation include relief of urinary retention; prevention of urinary retention—a period of postoperative catheterisation is common employed after many operations where limited mobility makes normal voiding difficult; monitoring of urine output, e.g., postoperatively; prevention of damage to the bladder during caesarean section; bladder drainage following surgery to the bladder, prostate, or urethra, e.g., transurethral resection of the prostate (TURP), transurethral resection of bladder tumour (TURBT), open bladder stone removal, radical prostatectomy; and bladder drainage following injuries to the bladder.

Technique

Explain the need for and method of catheterisation to the patient. Use the smallest catheter—in practical terms usually a 12 Ch, with a 10-mL balloon. For longer catheterisation periods (weeks) use a Silastic catheter to limit tissue reaction, thereby reducing risk of a catheter-induced urethral stricture. If you suspect clot retention (a history of haematuria prior to the episode of retention), use a three-way catheter (20 Ch or greater) to allow evacuation of clots and bladder irrigation to prevent subsequent catheter blockage.

The technique is aseptic. One gloved hand is sterile, the other is 'dirty'. The dirty hand holds the penis or separates the labia to allow cleansing of the urethral meatus; this hand should not touch the catheter. Use sterile water or sterile cleaning solution to 'prep' the skin around the meatus.

Apply lubricant jelly to the urethra. Traditionally this contains local anaesthetic [e.g., 2% lignocaine (lidocaine)], which takes between 3 and 5 minutes to work. However, a randomised,

placebo-controlled trial showed that 2% lignocaine was no more effective for pain relief than anaesthetic-free lubricant (Birch et al. 1994), suggesting that it is the lubricant action that prevents urethral pain. If using local anaesthetic lubricant, warn the patient that it may 'sting.' Local anaesthetic lubricant is contraindicated in patients with allergies to local anaesthetics and in those with urethral trauma, where there is a (theoretical) risk of complications arising from systemic absorption of lignocaine. When instilling the lubricant jelly, do so gently, as a sudden, forceful depression of plunger of syringe can rupture the urethra! In males, 'milk' the gel toward the posterior urethra, while squeezing the meatus to prevent it from coming back out of the meatus.

Insert the catheter using the sterile hand, until flow of urine confirms it is in the bladder. Failure of urine flow may indicate that the catheter balloon is in the urethra. Intraurethral inflation of the balloon can rupture the urethra. If no urine flows, attempt aspiration of urine using a 50-mL bladder syringe (lubricant gel can occlude eye-holes of catheter). Absence of urine flow indicates either that the catheter is not in the bladder or, if the indication for the catheterisation is retention, that the diagnosis is wrong (there will usually be a few millilitres of urine in the bladder even in cases where the absence of micturition is due to oliguria or anuria, so complete absence of urine flow usually indicates the catheter is not in the bladder). If the catheter will not pass into the bladder, and you are sure that the patient is in retention, proceed with suprapubic catheterisation.

SUPRAPUBIC CATHETERISATION

Indications

Indications are failed urethral catheterisation in urinary retention; preferred site for long-term catheters.

Long-term *urethral* catheters commonly lead to acquired hypospadias in males (ventral splitting of glans penis) and a patulous urethra in females (leading to frequent balloon expulsion and bypassing of urine around the catheter). Hence, the suprapubic site is preferred for long-term catheters.

Contraindications

Suprapubic catheterisation is best avoided in (1) patients with clot retention, the cause of which may be an underlying bladder cancer (the cancer could be spread along the catheter track to

involve the skin); (2) patients with lower midline incisions (bowel may be 'stuck' to the deep aspect of the scar, leading to the potential for bowel perforation); and (3) pelvic fractures, where the catheter may inadvertently enter the large pelvic haematoma, which always accompanies severe pelvic fracture. This can lead to infection of the haematoma, and the resulting sepsis can be fatal! Failure to pass a urethral catheter in a patient with a pelvic fracture usually indicates a urethral rupture (confirmed by urethrography) and is an indication for formal open, suprapubic cystotomy.

Technique

Prior to insertion of the trocar, be sure to confirm the diagnosis by (a) abdominal examination (palpate and percuss lower abdomen to confirm bladder is distended), (b) ultrasound (in practice usually not available), and (c) aspiration of urine (using a green needle). Patients with lower abdominal scars may have bowel interposed between the abdominal wall and bladder and this can be perforated if the trocar is inserted near the scar and without prior aspiration of urine! In such cases, ultrasound-guided catheterisation may be sensible.

Use a wide-bore trocar if you anticipate that the catheter will be in place for more than 24 hours (small-bore catheters will block within a few days). Aim to place the catheter about two to three fingerbreadths above the pubis symphysis. Placement too close to the symphysis will result in difficult trocar insertion (the trocar will hit the symphysis). Instill a few millilitres of local anaesthetic into the skin of the intended puncture site and down to the rectus sheath. Confirm the location of bladder by drawing back on the needle to aspirate urine from the bladder. This helps guide the angle of trocar insertion. Make a 1-cm incision with a sharp blade through the skin. Hold the trocar handle in your right hand, and steady the needle end with your left hand (this hand helps prevent insertion too deeply). Push the trocar in the same direction in which you previously aspirated urine. As soon as urine issues from the trocar, withdraw the latter, holding the attached sheath in place. Push the catheter in as far as it will go. Inflate the balloon. Peel away the side of the sheath and remove it.

BLADDER WASHOUT FOR BLOCKED CATHETER

This may be required after TURP or TURBT. Try to avoid the problem by ensuring that the nursing staff is familiar with this potential complication. Nurses should be aware of the importance of keeping the catheter bag empty and ensuring that there is always a sufficient supply of irrigant solution. If the urine collection bag becomes full, urine flow ceases and the catheter can become blocked with clot.

The patient will complain of lower abdominal pain, and the bladder will be distended (dull to percussion and tense to palpation). Look at the irrigation channel of the three-way catheter. There will be no flow of fluid out of the bladder. A small clot may have blocked the catheter or a chip of prostate may have stuck in the eye of the catheter.

Attach a bladder syringe to the end of the catheter and pull back. This may suck out the clot or chip of prostate and flow may restart. If it does not, draw some irrigant up into the syringe until it is about half-full and forcefully inject this fluid into the bladder. This may dislodge (and fragment) a clot that has stuck to the eye of the catheter. If the problem persists, change the catheter. The obstructing chip of prostate may appear on the end of the catheter as it is withdrawn.

If the bladder is full of clot, then it is sometimes possible, by alternating irrigation and sucking back on the syringe, to remove the clot, but if there is a large quantity in the bladder, you may well have to return the patient to the operating room, remove all the clot by reinserting the resectoscope and applying an Ellik evacuator, and then find and cauterise the bleeding vessel that caused the problem in the first place.

The same technique should be used for post-TURBT catheter blockage as for post-TURP catheter blockage. However, beware of applying overvigorous pressure to the bladder following resection of a tumour, since the wall of the bladder will have been weakened at the site of tumour resection and it is possible to perforate the bladder. This is particularly so with the thin bladders of elderly women.

BLOCKED CATHETERS FOLLOWING BLADDER AUGMENTATION OR NEOBLADDER

Again, the suture line of these bladders is weak, and overvigorous irrigation with a bladder syringe can rupture the bladder. Gently fill the bladder with a 100 mL or so of saline, and very gently wash this fluid around the bladder with the syringe. This

can help to dilute a mucus plug allowing spontaneous flow to be reestablished.

JJ STENT INSERTION

Indications in Urological Emergencies

Obstructing ureteric stones
Ureteric injury
Malignant obstruction of the ureter

Preparation of the Patient for JJ Stent Insertion

Oral ciprofloxacin 250 mg; lignocaine gel for urethral anaesthesia and lubrication; sedoanalgesia (diazemuls 2.5–10 mg i.v., pethidine 50–100 mg i.v.). Monitor pulse and oxygen saturation with a pulse oximeter.

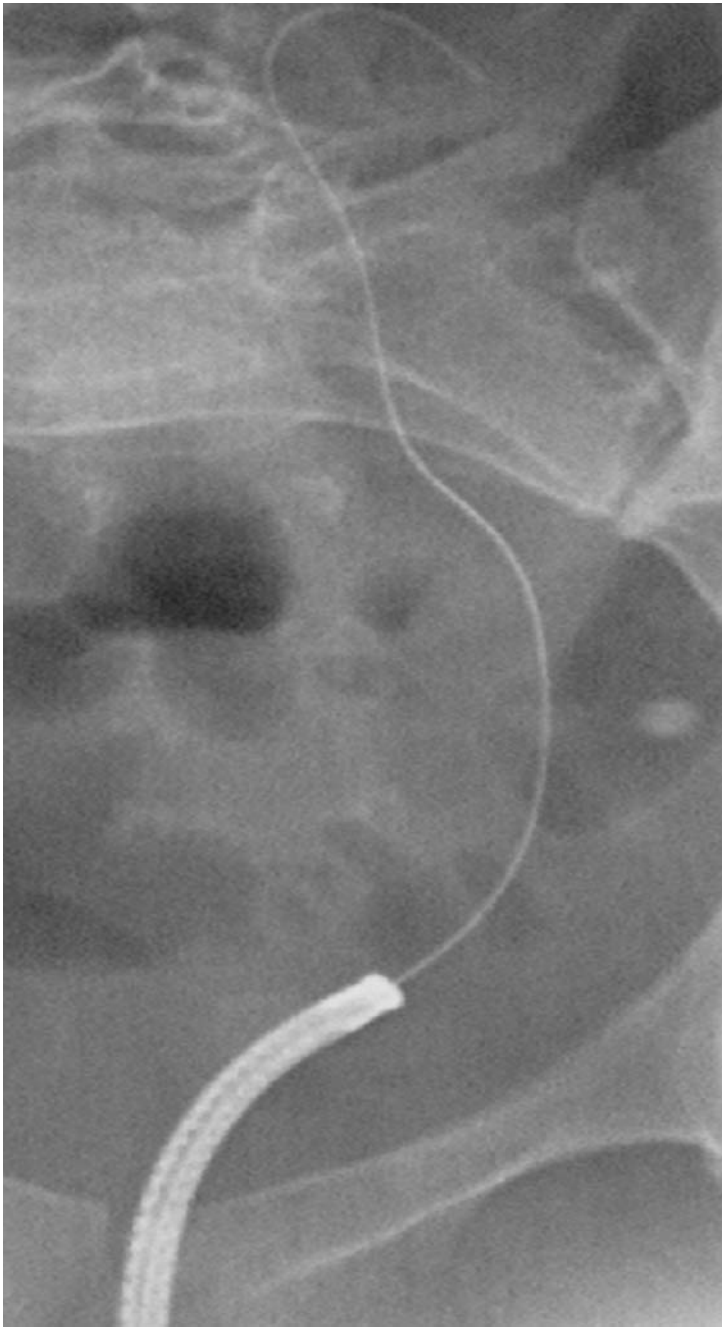
Technique (Hellawell et al. 2002, McFarlane et al. 2001)

A flexible cystoscope is passed into the bladder and rotated through 180 degrees. This allows greater deviation of the end of the cystoscope and makes identification of the ureteric orifice easier. A 0.9-mm hydrophilic guidewire (Terumo Corporation, Japan) is passed into the ureter under direct vision (Fig. 10.1a). The guidewire is manipulated into the renal pelvis using C-arm digital fluoroscopy (Fig. 10.1b). The cystoscope is placed close to the ureteric orifice and its position relative to bony landmarks in the pelvis is recorded by frame grabbing a fluoroscopic image. The flexible cystoscope is then removed and a 4-Ch ureteric catheter is passed over the guidewire, into the renal pelvis. A small quantity of nonionic contrast medium is injected into the renal collecting system, to outline its position and to dilate it. The Terumo guidewire is replaced with an ultrastiff guidewire (Cook UK Ltd., Letchworth, UK), and the 4-Ch ureteric catheter is removed. We use a variety of stent sizes depending on the patient's size (6–8 Ch, 20–26 cm) (Boston Scientific Ltd., St. Albans, UK). The stent is advanced to the renal pelvis under fluoroscopic control, checking that the lower end of the stent is not inadvertently pushed up the ureter by checking the position of the ureteric orifice on the previously frame-grabbed image (Fig. 10.1c). The guidewire is then removed (Fig. 10.1d).



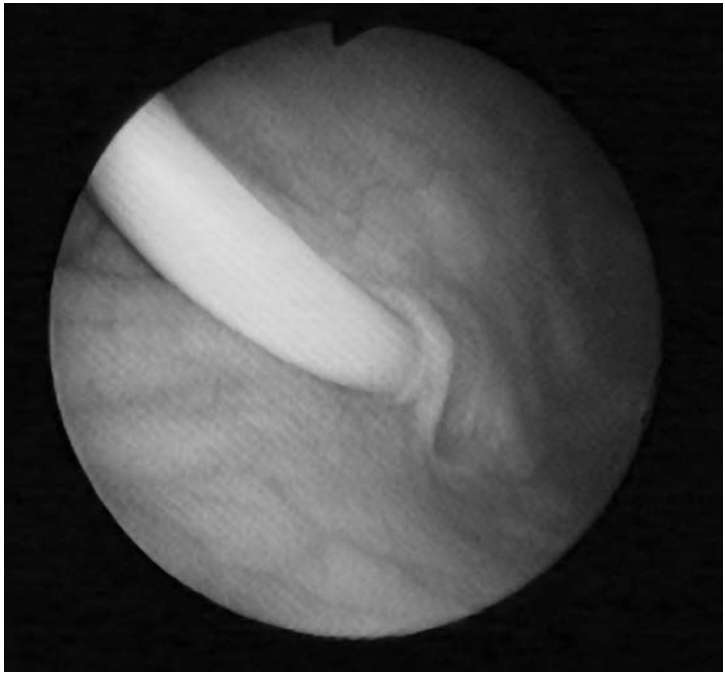
a

FIGURE 10.1. a: A flexible cystoscope has been passed into the bladder and a guidewire is manipulated into the ureter under direct vision. (See this figure in full color in the insert.) b: Under fluoroscopic control, the guidewire is advanced up the ureter and into the renal pelvis. c: The lower end of the stent is seen deployed in the bladder. (See this figure in full color in the insert.) d: Previously instilled contrast medium can be used to confirm that the stent is in the correct position.



b

FIGURE 10.1. *Continued*



c

FIGURE 10.1. *Continued*



d

FIGURE 10.1. *Continued*

PERCUTANEOUS NEPHROSTOMY INSERTION

Indications in Urological Emergencies

Preparation of the Patient for Nephrostomy Insertion

Patients should have their blood clotting checked and serum should be grouped and saved in case heavy bleeding occurs and blood transfusion is required. Verbal consent should be taken and the discussion about risks documented in the patient's notes (see Complications, below).

Technique

This procedure is performed under local anaesthetic with or without sedation, and with antibiotic cover (depending on urine culture; cefuroxime and gentamicin if no culture result is available). The patient lies prone. A nephrostomy needle is inserted into the renal pelvis and contrast is instilled to outline the collecting system of the kidney (Fig. 10.2a). A guidewire is passed into the renal pelvis (Fig. 10.2b), and over this the nephrostomy tube is advanced (Fig. 10.2c).

Complications

These will depend on how experienced the radiologist is and on how many nephrostomies he or she inserts per year. The complication rate of dedicated uroradiologists is lower than that which is generally regarded as acceptable (Ramchandani et al. 2001). Quoted complication rates should be those relevant to your hospital.

In the U.K., acceptable complication rates are haemorrhage requiring embolisation or surgery 1%, septic shock 4%, damage to adjacent organs <1%, and failure to drain the kidney approximately 5% (Ramchandani et al. 2001), but some series report complication rates that are below these (Ho and Cowan 2001).

Failure to Deflate Catheter Balloon for Removal of a Urethral Catheter

From time to time an inflated catheter balloon will not deflate when the time comes for removal of the catheter. No amount of drawing back on the balloon channel with a syringe will make the balloon go down, and attempts to burst the balloon by inflating the balloon with air or flushing the balloon inflation channel with water fail to work.



a

FIGURE 10.2 a: Nephrostomy insertion. A needle has been inserted into the renal pelvis and contrast has been instilled. b: A guidewire has been passed into the renal pelvis. c: The nephrostomy tube is advanced over the guidewire into the renal pelvis.



b

FIGURE 10.2. *Continued*



c

FIGURE 10.2. *Continued*

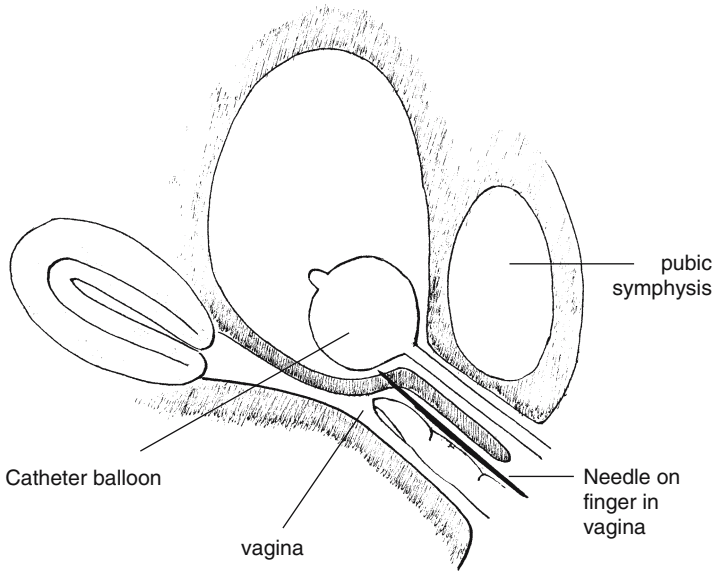


FIGURE 10.3. Technique for bursting a catheter balloon in a woman.

A little patience is required. Leave a 10-mL syringe firmly inserted in the balloon channel and come back an hour or so later. Sometimes, for no apparent reason, the balloon will have deflated and the catheter will be lying in the bed, having fallen out.

If this does not work, and the patient is female, then it is quite easy to burst the balloon using a needle introduced alongside your finger into the vagina (Fig. 10.3). Ask the patient to lie on her back, place a needle on your finger, apply copious lubrication, and gently insert the finger into the vagina. Pull down on the catheter with your other hand (or ask an assistant to do so), until you can feel the balloon of the catheter sitting at the bladder neck. By pulling the balloon onto the needle (which should be advanced a little so it advances just beyond the tip of your finger), the balloon can be deflated.

In male patients, balloon deflation with a needle can also be done, but ultrasound-guided balloon puncture will be required. Either the catheter should be clamped to allow the bladder to fill up, or the bladder can be filled with saline using a bladder syringe. As the bladder is so inflated, the bowel is pushed upward,

out of harm's way, so that the needle can be introduced percutaneously and directly, by ultrasound, toward the balloon of the catheter.

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