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APPLIED ANATOMY OF THE BLADDER Arterial supply

APPLIED ANATOMY OF THE BLADDER Arterial supply

/uni25CF Superior vesical artery (from the umbilical artery , which arises from the internal iliac artery). /uni25CF Inferior vesical artery (directly from the anterior division of the internal iliac artery , or in females from the vaginal artery , which arises from the internal iliac artery). /uni25CF Division of the contralateral superior vesical pedicle can aid cephalad mobilisation of the bladder for psoas hitch procedures.

APPLIED EMBRYOLOGY OF THE BLADDER

APPLIED EMBRYOLOGY OF THE BLADDER

The bladder originally develops from the cloaca, the endodermis-lined hindgut structure that is the common open ing for the urinary , genital and gastrointestinal tracts. Between weeks 4 and 7 of gestation, the cloaca is partitioned into a ventral urogenital tract (the primitive urogenital sinus) and a dorsal anorectal tract by the urorectal septum (Figur e 83.3 The portion of the primitive urogenital sinus that lies above the entry point of the mesonephric ducts becomes the vesicoure thral canal, which gives rise to the bladder and pelvic urethra, whereas the portion of the urogenital sinus caudal to this entry point forms the bulbar and penile urethra in males and the vaginal vestibule in females. Initially , the superior end of the lumen of the b ladder is continuous with the allantois, a sac-like structure that is responsible for embryonic nutrition and waste John Hutch , 1922–1972, American urologist, described paraureteric bladder diverticula in 1961. Heinrich Wilhelm Gottfried Waldeyer-Hartz , 1836–1921, Professor of Pathological Anatomy divides the retrorectal space into superior and inferior compartments; it was described by Waldeyer in 1899. allantois is obliterated and becomes a fibrous cord, the urachus, which runs within the umbilical cord and drains the fetal urinary bladder. As the bladder descends into the pelvis during development, this fibrous cord elong ates. Postnatally , this obliterated fibrous cord extends from the apex of the bladder to the umbilicus as the median umbilical ligament. The urachus acts as a landmark during radical cystectomy and can be traced to the apex of the bladder; identification of this structure can prevent early entry into a high-riding bladder, and the urachus is then removed en bloc with the bladder specimen. -

APPLIED PHARMACOLOGY OF THE BLADDER

APPLIED PHARMACOLOGY OF THE BLADDER

The two predominant neurotransmitters controlling LUT function are acetylcholine and noradrenaline (norepinephrine). Acetylcholine from the somatic nervous system causes contrac tion of striated muscle by activating nicotinic receptors, Onuf ’s nucleus refers to a group of motor neurones located in the anterior horn of the sacral (predominantly S2) spinal cord; it is named after Bronislaw Onuf-Onufrowicz, 1863–1928, who discovered this group of cells in 1899. /uni25CF /uni25CF β /uni25CF /uni25CF α whereas acetylcholine from parasympathetic nerves causes detrusor smooth muscle contraction by activating muscarinic receptors. Noradrenaline is released from the sympathetic nervous system and activates β -adrenergic receptors on the 3 - smooth muscle of the detrusor to cause relaxation and α -adrenergic receptors on the smooth muscle of the bladder 1 base and urethra to cause contraction. The underlying second-messenger mechanisms by which smooth muscle contraction and relaxation occur are shown in Figure 83.1 . Smooth muscle contraction is dependent on calcium influx causing contraction via actin and myosin. Smooth muscle relaxation is dependent on calcium e ffl ux back into the sarco - plasmic reticulum, mediated by the cyclic adenosine mono - phosphate and cyclic guanosine monophosphate pathways. The commonly used agents, their principal actions and their side e ff ects are summarised in Table 83.2 .

outlet Contraction of Relaxation of 3-adrenergic smooth muscle of detrusor smooth (bladder) urethra and bladder muscle base Inhibition of -adrenergic 1 parasympathetic (urethra and ganglia, thereby bladder neck) inhibiting detrusor contraction None Contraction of the detrusor smooth muscle of muscle bladder) Contraction of Afferent sensory external urethral nerves (stretch, (striated muscle sphincter temperature and of external pain) urethral sphincter)

Antimuscarinics

Antimuscarinics

There are five types of muscarinic receptor, M1–5, in various organs in the human body (heart, brain, salivary glands, eye, . smooth muscle). Although the M2 receptor is most abundant in the bladder, it is the M3 receptor that binds acetylcholine to cause detrusor contraction. The most widely used pharma - cological agents to improve bladder function in patients with - neurogenic LUT dysfunction or idiopathic overactive bladder are muscarinic receptor antagonists (antimuscarinics). Mechanism of action Antimuscarinics reduce bladder storage pressures, treat detru - sor overactivity and improve overactive bladder symptoms. Although commonly thought to exert their e ff ect during the voiding phase by antagonising acetylcholine released from parasympathetic nerves and thereby reducing detrusor - contraction, they are now thought to exert their primary e ff ects α α /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF β /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF α /uni25CF /uni25CF in the storage phase of the micturition cycle. During the storage phase they act as antagonists of acetylcholine released from the urothelium, thereby decreasing activity in bladder a ff erent nerves and suppressing involuntary detrusor contractions and the sensation of urgency .

-agonist -receptor 1 (+) G-protein (+) PLC IP PIP 2 3 SR DG (+) 2+ Ca PKC 2+ Contraction Ca Smooth muscle contraction Figure 83.1 Second-messenger systems involved in (a) smooth muscle contraction and phate; cAMP , cyclic AMP; DG, diacylglycerol; IP , inositol 1,4,5-trisphosphate; PIP 3 C; PLC, phospholipase C; SR, sarcoplasmic reticulum. TABLE 83.2 Actions and side effects of commonly used pharmacological agents for the bladder. Class Examples Action Antimuscarinics Solifenacin Reduce bladder storage pressure Oxybutynin Reduce detrusor overactivity Tolterodine Improve functional bladder capacity Fesoterodine Darifenacin Trospium Mirabegron Reduce bladder storage pressure -agonists 3 Vibegron Reduce detrusor overactivity Improve functional bladder capacity Tamsulosin Reduce bladder outlet resistance -antagonists 1 from bladder neck and proximal/ Alfuzosin prostatic urethra Doxazosin

BLADDER CANCER

BLADDER CANCER

Bladder cancer is a highly prevalent disease with 540 /uni00A0 000 cases worldwide and 188 /uni00A0 000 deaths reported in 2015. Risk factors - for developing bladder cancer are shown in Table 83.16 .

BLADDER STONES

BLADDER STONES

Bladder stones account for 5% of all urinary tract stone disease. They can be classified as primary (without underlying urinary tract pathology) or secondary (due to underlying renal tract pathology). Primary bladder stones are commonly seen in children in the developing world and are due to nutritional deficiency in vitamins A and B6, magnesium and phosphate and a reduced protein–carbohydrate ratio. Secondary bladder stones are most commonly related to urinary stasis from elevated postvoid residual volume due to bladder outlet obstruction (Table 83.11). Sven Ivar Seldinger , 1921–1998, Swedish radiologist, introduced this technique in 1953. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Three-stage guidewire TABLE 83.11 Aetiology of bladder stones. Primary Nutritional de /f_i ciency Secondary Urinary stasis Bladder outlet obstruction (elevated Detrusor underactivity residual Bladder augmentation or substitution volume) Neurogenic lower urinary tract dysfunction Foreign body Suture or mesh from previous prolapse/ continence/pelvic surgery Stone from upper tract Indwelling catheter or ureteric stent Migrated intrauterine devices Infection Drugs Indinavir Triamterene

BLADDER TRAUMA

BLADDER TRAUMA

Bladder trauma can be classified as iatrogenic or non-iatrogenic (blunt or penetrating). Of non-iatrogenic causes, abdominal trauma and pelvic fracture are the most common, with blad der injury reported in 10% of cases. Iatrogenic injury is most commonly the result of TURBT , anti-incontinence surgery or pelvic surgery (e.g. hysterectomy , caesarean section, colorectal surgery). Rar ely , spontaneous rupture can occur after bladder augmentation without any history of trauma. This is due to overdistension in those with limited bladder sensation (e.g. SCI), and often presents with vague abdominal pain, fever or sepsis. A high index of suspicion of bladder rupture in patients with a history of bladder augmentation is required.

Bladder exstrophy

Bladder exstrophy

Bladder exstrophy is a congenital disorder in which failure of development of the lower abdominal wall leads to an abdominal wall defect through which the bladder is exposed (Figure 83.9). Diastasis of the pubic symphysis and an Hermann Johann Pfannenstiel , 1862–1909, gynaecologist, Breslau, Germany (now Wroc ł aw , Poland), described this ‘bikini-line’ suprapubic horizontal incision in 1900. anterior opening of the urethra (epispadias) can coexist. The - condition forms part of a spectrum of conditions ranging from epispadias to bladder exstrophy or to more severe cloacal exstroph y , the so-called exstrophy–epispadias complex. During - development, mesenchymal ingrowth between the ectodermal , and endodermal layers of the cloacal membrane leads to - formation of the lower abdominal wall muscles and pelvic bones. However, failure of this mesodermal ingrowth leads to premature rupture of the cloacal membrane and results in epispadias, bladder exstrophy or cloacal exstrophy depending on the developmental stage at which rupture occurs. The incidence of bladder exstrophy is approximately 1 in 46 /uni00A0 000 live births, with a male-to-female ratio of 2.3:1. Clinical features Exstrophy of the bladder can be associated with a spectrum of anomalies a ff ecting the external genitalia, urinary system, bony pelvis, abdominal wall, rectum and anus. Male external genitalia Shortened penis due to diastasis of the pubic symphysis resulting in wide separation of the crural attachments, and congenital deficiency of the corporeal tissue. Female external genitalia /uni25CF Shortened, stenotic and anteriorly displaced vagina. /uni25CF Bifid clitoris.

Figure 83.8 Bladder diverticulum following excision.

Urinary system /uni25CF Incompetent bladder neck continence mechanism. /uni25CF VUR due to lack of obliquity of the vesicoureteric junc tion. Bony pelvis /uni25CF Widening (diastasis) of the pubic bones due to malrotation of the innominate bones, leading to a waddling gait. /uni25CF External rotation of the anterior and posterior segments of the bony pelvis, leading to outward rotation of the lower limbs. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF James Parkinson , 1755–1824, general practitioner of Shoreditch, London, UK, published /uni25CF Low-set umbilicus with a triangular-shaped fascial defect of the lower abdominal wall. /uni25CF Higher incidence of indirect inguinal hernia owing to the lack of oblique muscle fibres of the inguinal canal and large internal and external inguinal rings. Anorectum /uni25CF Shortened perineum and anteriorly displaced anus. /uni25CF Imperforate anus (absence of the normal anal opening) or rectal stenosis. /uni25CF Rectal prolapse. Surgical treatment of bladder exstrophy The aim of surgical treatment is to preserve renal function, achieve urinary continence and create functional and cosmet - ically acceptable external genitalia. The modern staged repair of exstrophy consists of bilateral iliac osteotomies with bladder exstrophy and abdominal wall closure in the neonatal period, followed by epispadias repair and phallic reconstruction at 6 /uni00A0 months to 1 year of age, and finally bladder neck recon - struction and bilateral ureteric reimplantation (to treat VUR) at age 5–7 years. More

recently , complete primary repair of exstrophy in the neonatal period has been advocated in an attempt to optimise outcomes with few er procedures and without a formal bladder neck closure procedure.

Figure 83.9 Bladder exstrophy. (Reproduced with permission from Wein AJ, Kavoussi LR, Partin AW, Peters CA. Campbell-Walsh urol ogy , 11th edn. Philadelphia, PA: Elsevier, 2016: 2424.) TABLE 83.3 Common congenital and acquired causes of neurogenic lower urinary tract dysfunction. Congenital Acquired Neural tube defects Sacral agenesis Anorectal malformations (e.g. VACTERL syndrome) Central nervous system tumours Transverse myelitis VACTERL, vertebral defects, anal atresia, cardiac defects, tracheo-oesophageal /f_i stula, renal anomalies and limb abnormalities.

Bladder pain syndrome interstitial cystitis

Bladder pain syndrome/interstitial cystitis

Bladder pain syndrome (BPS) is a chronic condition char acterised by pelvic pain or pressure that is perceived to be originating from the bladder, accompanied by one or more urinary symptoms, including frequency , urgency and nocturia. The diagnosis is made once other confusable diseases that Guy Hunner , 1868–1957, American surgeon, first described the characteristic inflammatory lesions of the bladder in 1915. interstitial cystitis (IC) is often used interchangeably with BPS but represents a distinctive bladder organ-specific phenotype - with characteristic cystoscopic and histopa thological features as opposed to the systemic phenotype of BPS. The precise aetiology of BPS/IC is unknown. Clinical features - Patients present with disabling bladder or pelvic pain, urinary urgency and severe urinary frequency and nocturia. Those with BPS may have other associated chronic medical condi - tions (e.g. fibromyalgia, irritable bowel syndrome, migraines). Investigation Urine analysis and culture, testing for sexually transmitted infections and urine cytology should be performed to exclude an infective or malignant cause for symptoms. Pelvic imaging should be performed if an alternative diagnosis, such as endo - metriosis, is suspected. Cystoscopy should be performed to exclude other pathology and also to aid accurate phenotyping of BPS (bladder capacity , presence of Hunner lesions). The aim of phenotyping is to separate those with clear bladder pathol - ogy (small capacity , Hunner lesions, chronic inflammation) from those with anatomically normal bladders as treatment - options vary . In those with Hunner lesions, bladder biopsy shows a chronic pancystitis, often with marked infiltra tion with lymphocytes and macrophages and mast cell infiltration. The ‘INPUT’ classification system allows patients’ symptoms to be described in five di ff erent clinical domains in order to guide multimodal therapy: Infection, Neurological/systemic, Psychosocial, Ulcers and Tenderness of muscles. The aim of the evaluation is to assess the relative contribution of each of these factors to the patient’s symptoms. Treatment Treatment of BPS/IC consists of conservative, pharmacolog - ical, intravesical and surgical options. Conservative Patient education about the chronicity of the condition, behavioural modification (timed voiding, bladder training), stress reduction, dietary alteration (avoidance of ca ff eine and spicy and acidic foods) and physical therapy should be the initial management for all patients. Pharmacological Several pharmacological therapies have been studied for BPS/IC, all with variable e ffi cacy . Neuropathic analgesics (e.g. amitriptyline, pregabalin) are used for those with a significant pain component, whereas antimuscarinics and β -agonists 3 are used for frequency , nocturia and urgency symptoms. Oral pentosan polysulphate (Elmiron), a glycosaminoglycan (GAG) - layer replacement treatment, has demonstrated e ffi cacy in pain and urinary symptoms but recent reports of ophthalmic adverse events with long-term exposure may limit its use. Evidence for other oral therapies (e.g. antihistamines and immunosuppressants) is mixed. Direct instillation of GAG layer replacement therapies are thought to repair the defective GAG layer, which may be part of the pathophysiology of BPS/IC. Although the evidence base is weak, they are widely used

with satisfactory outcomes in some patients without the side e ff ects seen with oral therapies. Intravesical ‘cocktails’ with combinations of alkalinised local anaesthetic, steroid and GAG layer therapies may be useful for acute flares. Surgical Several surgical options have been studied. For those with Hunner lesions, fulguration or laser to these lesions can be beneficial. Cystodistension as a treatment has variable evidence for success. Minimally invasive treatment with intravesical BTX-A or SNS should be o ff ered after the above measures have failed. If these options fail to improve symptoms major surgical reconstruction can be considered in selected cases. This is more suitable for those with clear evidence of bladder pathology (small capacity , fibrotic, ulcerated bladder). The aim is to increase the capacity of the bladder or divert the urinary stream, with options including bladder augmentation cystoplasty , cystoplasty with or without subtrigonal resection or urinary diversion with or without cystectomy . It is generally thought that cystectomy with orthotopic bladder reconstruc tion or ileal conduit urinary diversion is the best option as any form of bladder preservation risks ongoing pain in the remnant bladder segment with requirement for secondar y cystectomy in up to 65%.

Bladder

Bladder

- The urinary bladder is a hollow muscular organ that consists of three principal layers: lamina propria, smooth muscle and urothelium. The lamina propria contains a rich plexus of vessels, nerves and lymphatics. The detrusor is made up of a complex haphazard arrangement of smooth muscle, which acts as functional syncytium, and elastic connective tissue, which gives the bladder its viscoelastic properties. The urothe lium is an active layer that not only acts as a barrier to protect underlying stroma from irritant urinary toxins and bacteria but also has a role in a ff erent signalling within the bladder; defects in the urothelial lining are thought to lead to several chronic benign bladder conditions . The bladder is made up of the bladder body (the area above the level of the ureteric orifices), the bladder base/trigone (the area below the level of the ureteric orifices) and the bladder neck smooth muscle.

Nervous system Origin Course Neurotransmitter Receptor Action on bladder Action on bladder Hypogastric Noradrenaline Sympathetic T10–L2 nerve (norepinephrine) (thoracolumbar cord) Pelvic nerve Acetylcholine M3 (smooth Parasympathetic S2–4 (sacral cord ‘spinal micturition centre’) Somatic S2–4 (sacral cord Pudendal Acetylcholine Nicotinic ‘Onuf’s nucleus’) nerve

BLADDER

The LUT consists of the bladder and urethra, and its two functions are urinary storage and urinary emptying. These functions depend on coordinated activity between the smooth and striated muscles of the bladder and the outlet (consisting of the bladder neck, urethral smooth muscle, external urethral sphincter and pelvic floor muscles), which is mediated by a complex of neural circuits in the central and peripheral nervous systems.

CHRONIC INFLAMMATORY CONDITIONS OF THE BLADDER

CHRONIC INFLAMMATORY CONDITIONS OF THE BLADDER

Chronic inflammatory conditions of the bladder are of multi factorial aetiology but present with a similar clinical picture of urinary frequency , urgency and pain, with or without haematuria (Table 83.14). The principles of management are to exclude a malignant cause for the symptoms, to preserve upper tract function and to improve symptoms and quality of life. /uni25CF /uni25CF /uni25CF /uni25CF

TABLE 83.14 Common chronic in /f_l ammatory bladder diseases. Idiopathic Interstitial cystitis Infective Chronic bacterial UTI Fastidious organisms and parasites (Chlamydia , Gonorrhoea , tuberculosis, schistosomiasis) Radiation therapy Radiation cystitis Drugs Ketamine cystitis Cyclophosphamide cystitis Iatrogenic BCG cystitis Autoimmune Lupus cystitis BCG, bacillus Calmette–Guérin; UTI, urinary tract infection.

CONGENITAL BLADDER ANOMALIES

CONGENITAL BLADDER ANOMALIES

Most congenital bladder anomalies can be detected on antena - - tal ultrasound after 10–13 weeks’ gestation, when the bladder should be visualised in the majority of cases.

Catheterisation

Catheterisation

The immediate treatment for urinary retention of any cause is urethral catheterisation. Other indications for catheterisa - tion are shown in Table 83.10 . In chronic urinary retention, patients may have a postobstructive diuresis producing >200 /uni00A0 mL of urine per hour for thr ee consecutive hours. If this is the case, patients should be managed with strict fluid balance monitoring, postural blood pressure checks to detect postural hypotension and daily serum electrolyte monitoring /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF and occasionally may require intravenous fluid replacement to match the loss if the patient is unable to take enough orally .

Drainage Urinary retention (acute and chronic) Fluid management/monitoring in critically unwell patients Palliative management for urinary incontinence where other measures have failed or are unsuitable Following urological surgery to allow healing of the bladder or urethra Therapeutic drug Non-muscle-invasive bladder cancer (e.g. delivery mitomycin C, gemcitabine, BCG) Chronic cystitis such as UTI and interstitial cystitis (e.g. GAG-layer replacement therapies, antibiotics) Diagnostic Micturating cystourethrogram Urodynamics To obtain a catheter specimen of urine for analysis BCG, bacillus Calmette–Guérin; GAG, glycosaminoglycan; UTI, urinary tract infection.

Classification

Classification

Bladder injuries can be either extraperitoneal (the peritoneum is intact and urine extravasates into the retropubic space but not into the peritoneal cavity), intraperitoneal (the peritoneum over the bladder is injured and urine extravasates into the peri toneal cavity) or mixed (Table 83.18). Intraperitoneal ruptures are associated with a risk of urinary peritonitis and ileus, and so are more significant than extraperitoneal ruptures.

Clinical features

Clinical features

/uni25CF May be asymptomatic. /uni25CF Haematuria. /uni25CF Dysuria. /uni25CF Frequency and urgency . /uni25CF Suprapubic pain. /uni25CF Hesitancy and intermittency .

Figure 83.27 Smooth uric acid bladder stones. Figure 83.28 Radiograph showing a vesical calculus (no contrast has been used).

Clinical features

The classic symptoms of UTI include dysuria, suprapubic pain, urinary frequency and urgency . Patients may also pres ent with haematuria, loin pain, fevers, nausea and vomiting. A physical examination should be performed to check for a palpable bladder, for renal angle tenderness and for evidence of pelvic organ prolapse, urethral diverticulum and atrophic vaginitis. Theodor Albrecht Edwin Klebs , 1834–1913, Professor of Bacteriology successively at Prague, Czechoslovakia, Zurich, Switzerland, and the Rush Medical College, Chicago, IL, USA. Igor Tamm , 1922–1995, American virologist. Frank Horsfall , 1906–1971, American microbiologist, together with Igor Tamm first purified the Tamm–Horsfall protein in 1952. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF β /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF

mechanisms. Bacterial virulence factors Host defence mechanisms Adherence mechanisms Commensal organisms (/f_i mbrial and a /f_i mbrial) (lactobacilli) Immune evasion Mechanical integrity of (lipopolysaccharide O, mucous membranes capsule K) Antibacterial secretions (lysozyme, lactoferrin, IgA) Anti-IgA proteases, toxin production, -lactamase Antegrade /f_l ow of urine causing /f_l ushing effect Resistance to antimicrobial bactericidal activity (alteration Tamm–Horsfall protein of antimicrobial binding sites) (binds to bacterial adhesion molecules) Iron acquisition Composition of urine (low pH, high urea) Immune system integrity IgA, immunoglobulin A.

Clinical features

If iatrogenic, the injury may be recognised at the time. If perforation during transurethral surgery is noted, the proce dure should be stopped, haemostasis should be achieved and the patient should be catheterised. In cases of trauma, patients typically present with suprapubic pain, di ffi culty or inability to pass urine, haematuria and abdominal distension.

Composition

Composition

Primary endemic bladder stones in children are usually composed of ammonium urate and calcium oxalate. Second - ary stones due to bladder outlet obstruction are typically smooth and yellow-brown in colour and are composed of uric acid. Infection-related stones tend to be triple phosphate (magnesium ammonium phosphate) and are white in colour (Figure 83.27).

Congenital and acquired bladder

Congenital and acquired bladder

-

Congenital neuropathic bladder

Congenital neuropathic bladder

Neurogenic lower urinary tract dysfunction (NLUTD) refers to the spectrum of bladder dysfunction that can arise from congenital or acquired abnormalities of those parts of the nervous system that are responsible for normal bladder func - tion (Table 83.3). The most common congenital cause of NLUTD is - abnormal development of the spinal canal (neural tube defects). The neural tube develops in early gestation (closure of the spinal canal is complete by day 35) and maternal folic acid deficiency is one of the primar y risk factors for incomplete closure. Spina bifida, the most common neural tube defect, ranges in severity from mild (spina bifida occulta), in which there is only mild separation of the spinal vertebrae but no neurological involvement, to severe (myelomeningocele), in An essay on the shaking palsy in 1817.

-

Central nervous system tumours In /f_l ammatory/infective conditions of the central nervous system (encephalitis, transverse myelitis) Vascular conditions affecting the central nervous system (infarct, haemorrhage) Spinal cord injury Neurodegenerative and demyelinating diseases (e.g. multiple sclerosis, Parkinson’s disease) Other encephalopathy (e.g. cerebral palsy) Iatrogenic – pelvic/spinal/cerebral surgery Lesions of the peripheral nervous system (e.g. diabetes)

and exposed onto the skin of the lower back. Clinical features The clinical features of myelomeningocele can be variable, depending on which nerves have been everted in the menin gocele sac. Infants will have a visible cutaneous abnormality overlying the lower spine. In cases of spina bifida occulta, any suspicion of lower spinal cutaneous abnormality warrants further investigation with spinal ultrasound or MRI. In some cases, cutaneous lesions may be absent and, as the child grows with increasing age, tethering of the cord (fixation of the lower spinal cord due to scarring from surgery , lipoma or deep skin dimples, leading to stretching of the cord with growth of the child) can lead to the development of symptoms. Therefore, spinal investigation should be considered in any infant present ing with bladder or bowel dysfunction, failure to toilet train or lower extremity weakness, as this may be a sign of occult spinal dysraphism. An associated Arnold–Chiari malformation with hydro cephalus is commonly seen with myelomeningocele, resulting in developmental brain abnormalities. Infants may develop urinary infections, dribbling of urine and incomplete bladder emptying. End-stage renal disease (ESRD) is the commonest cause of death in infants with spina bifida and so ear ly identification, surveillance and treatment of those at risk for ESRD is the cornerstone of management. Investigation Myelomeningocele requires surgical closure of the spinal defect immediately after birth, and so urological investigations are delayed until the patient has recovered from surgery . /uni25CF Renal tract ultrasound and postvoid residual urine mea surement are required. /uni25CF Video urodynamics should be performed as

soon as feasi ble (usually in the first 2–3 months of life) to assess bladder function. /uni25CF In the presence of VUR, a dimercaptosuccinic acid (DMSA) renal scan is recommended at 3 months to pro vide accurate measurement of renal function. /uni25CF In those with risk factors for renal deterioration (hydro nephrosis, elevated postvoid residual, poor compliance, detrusor overactivity and DSD) early treatment should be initiated. All infants should undergo lifelong surveillance, initially with 6-monthly renal tract ultrasound and post void residual, and yearly urodynamics f or the first 2 years to detect any deterioration in renal drainage and bladder function. Treatment Bladder management aims to prevent deterioration in renal function. This is dependent on achieving low-pressure storage and voiding, with complete bladder emptying. Julius Arnold , 1835–1915, Professor of Pathological Anatomy , the University of Heidelberg, Heidelberg, Germany , described this condition in 1894. Hans Chiari , 1851–1916, Austrian, Professor of Pathological Anatomy , Strasbourg, Germany (Strasbourg was returned to France in 1918 after the end of the First World War), gave his account of this condition in 1891. The Arnold–Chiari malformation refers to a structural defect in the cerebellum characterised by ventral herniation of the cerebellar tonsils through the foramen magnum of the skull. findings (Table 83.4). The management of incomplete blad - der emptying, high-pressure storage, detrusor overactivity and high-pressure voiding is centred ar ound clean intermittent self-catheterisation (CISC) in combination with antimuscarinic - therapy to reduce bladder pressure. If this fails , then intraves - ical botulinum toxin A (BTX-A) is injected into the bladder wall. Augmentation enterocystoplasty , in which the bladder is bivalved and enlarged using a segment of ileum, is reserved f or those with ongoing risk factors for renal deterioration despite the above treatments. Patients, or parents, who are unable to perform urethral CISC should be considered for a continent urinary diversion with appendicovesicostomy , in which the appendix is used as a channel to connect the bladder with the skin of the umbilicus through which the patient can perform - self-catheterisation. These management options are discussed in more detail later in this chapter. Disorders that cause NLUTD often also lead to neuro - pathic bowel dysfunction (constipation or faecal incontinence) - and so this should also be addressed in all children presenting with NLUTD. Summary box 83.2 Congenital neuropathic bladder /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF - /uni25CF /uni25CF - -

The pattern of NLUTD depends on the site, severity and type of neurological lesion The bladder and sphincter may be overactive, normoactive or underactive The combination of an overactive bladder and overactive sphincter represents the highest risk of renal deterioration An overactive bladder is treated with pharmacotherapy, intravesical BTX-A, sacral neuromodulation or augmentation enterocystoplasty An underactive bladder is managed primarily with CISC, or if urethral catheterisation cannot be performed, then appendicovesicostomy An overactive sphincter is primarily treated with intrasphincteric BTX-A or sacral neuromodulation Bladder and bowel dysfunction often coexist and should be addressed together

Enuresis

Enuresis

Enuresis, or bedwetting, describes urinary incontinence during sleep in any child over the age of 5 years, in the absence of - congenital or acquired neurological disorders. Monosymp - tomatic enuresis (MSE) is defined as enuresis without any other urinary symptoms; primary MSE describes those who have never achieved night-time continence, whereas secondary MSE refers to those who de velop enuresis after a dry period of at least 6 months. Enuresis with any daytime LUTS is defined as non-monosymptomatic enuresis (NMSE). By 15 years of age 1–2% will su ff er from enuresis and the prevalence in adults is 0.5%. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF β /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF β /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Investigation Three underlying pathophysiological mechanisms are predominantly implicated in enuresis and should be evaluated clinically . 1 Nocturnal detrusor overactivity and reduced nocturnal bladder capacity . Up to half of all children overactivity or reduced functional capacity , in the absence of nocturnal polyuria. Patients should be investigated ini - tially with a bladder diary to assess daytime and night-time frequency and incontinence episodes, as well as to assess functional capacity . Urodynamics should be reserved for those who fail initial therapy; if detrusor overactivity is present then patients should be managed with antim usca - rinics or β -agonists. 3 2 Nocturnal polyuria . Increased nocturnal urine pro - duction (defined as a nocturnal urine output exceeding 130% of expected bladder capacity for age), which may be due to increased intake or underlying medical conditions, should be identified on a bladder diary and investiga ted further if present (e.g. diabetes insipidus, obstructive sleep apnoea). 3 Arousal and sleep disorders . Children with enure - sis are typically unable to wake from sleep to void, and it is thought that arousal disorders may account for part of the pathogenesis of this condition. Evaluation by a sleep specialist should be consider ed as part of the management strategy for children in whom sleep disorders are suspected. Treatment The treatment of enuresis consists initially of behavioural management techniques. These include fluid modification (night-time fluid restriction, reducing sugary , ca ff einated and fizzy drink intake), bedwetting alarms, star charts and rewards systems, and maintaining regular bowel habits. If this fails to improve symptoms and the child is experi - encing distress from these symptoms, pharmacological therapy should be considered. Desmopressin, a synthetic analogue of antidiuretic hormone, is best suited for those with nocturnal polyuria with normal bladder function, wher eas antimusca - rinics and β -agonists should be considered for those with low 3 functional capacity or those who have failed to respond to des - mopressin.

tract dysfunction. Storage-phase Treatment options disorder CISC Low compliance/ detrusor Overnight catheter drainage overactivity/low Pharmacological therapy a capacity Antimuscarinic -agonist 3 Minimally invasive therapy Intravesical BTX-A Surgical therapy Augmentation cystoplasty Urinary diversion Low outlet Bladder neck bulking agent injection resistance Bladder neck sling or bladder neck reconstruction Arti /f_i cial urinary sphincter Voiding-phase disorder Detrusor–sphincter CISC a

dyssynergia Overnight catheter drainage Pharmacological therapy Antimuscarinic -agonist 3 Minimally invasive therapy Intravesical and intrasphincteric BTX-A Neuromodulation Surgical therapy Augmentation cystoplasty Urinary diversion Detrusor CISC underactivity Overnight catheter drainage Neuromodulation BTX-A, botulinum toxin A; CISC, clean intermittent self

catheterisation. a Risk of renal function deterioration. (a) (b) Figure 83.10 Urachal anomalies. (a) Normal; (b) patent urachus;

Epidemiology

Epidemiology

Urinary incontinence is highly prevalent, a ff ecting 25–45% of men and women, and this increases with age. In men, the most common cause of SUI is radical prostatectomy for prostate cancer, with prevalence estimates of 5% at 24–36 months after surgery . Continence is primarily dependent on normal bladder compli ance, an intact urethral sphincter, strong urethral support by the pelvic floor and a leakproof mucosal seal. Compliance is the ability of the bladder to expand in vol ume without any significant rise in pressure, and during the normal storage phase the bladder pressure remains lo maximum capacity is reached. This enables normal renal drainage and is dependent on the viscoelastic properties of the bladder wall (low collagen le vels). However, detrusor over activity during the storage phase, bladder muscle hypertrophy or increased levels of bladder wall collagen (e.g. due to fibro sis) can all reduce compliance and lead to incontinence and deterioration in renal drainage. This may occur as a result of pelvic surgery , irradiation, neurological conditions, chronic inflammatory b ladder conditions leading to bladder fibrosis or longstanding bladder outlet obstruction. Deficiencies in the active urethral sphincter mechanism, the urethral mucosal seal and the pelvic floor support contrib ute to varying degrees of SUI. Hypermobility of the bladder base and proximal urethra due to laxity of the usual supporting ‘hammock’, consisting of endopelvic and pubocervical fascia attac hed to the ATFP and levator ani, is thought to lead to dis placement of the urethra out of the pelvis. As a result, during stress manoeuvres the raised intra-abdominal pressure is not transmitted to the urethra and so incontinence occurs. Laxity of the vaginal wall and pubourethral ligaments is thought to contribute to this deficiency in urethral support. These theories are the basis for retropubic suspension and mid-urethral sling procedures to treat SUI. Intrinsic sphincter deficiency occurs when the normal submucosal vascularity of the urethra and sphincter muscle tone are deficient. This may occur as a result of previous surgery , causing fibrosis, irradiation and nerve injury , or loss of oestrogenisation. Hypermobility and intrinsic sphincter deficiency exist on a spectrum and most women with SUI have elements of both. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - When evaluating patients with urinary incontinence, the history should ascertain whether symptoms are predominantly SUI, UUI or MUI, and should assess their impact on the patient’s - quality of life. Predisposing and exacerbating factors should be treated where possible (Table 83.5). The body mass index w until (BMI) should be noted and patients advised to lose weight if this is elevated. Abdominal examination to identify a palpable bladder suggestive of chronic urinary retention, and vaginal - examination to assess pelvic floor tone and oestrogenisation status and to identify pelvic organ prolapse, should also be - performed. Neurological examination to assess anal tone and sensation and lower limb function will aid identification of a neurological lesion.

TABLE 83.5 Predisposing and exacerbating factors for urinary incontinence (UI) Predisposing Exacerbating Familial (increased risk in Age those with family history of Obesity UI) Increased intra-abdominal pressure (chronic cough, Congenital or acquired straining due to constipation,

anatomical abnormalities (e.g. exercise) ectopic ureter, urinary tract /f_i stulae, urethral diverticulum) Cognitive impairment Neurological conditions (e.g. Restricted mobility spina bi /f_i da, spinal cord Urinary tract infection injury, Parkinson’s disease, Drugs (e.g. diuretics) stroke, multiple sclerosis) Menopause causing atrophic vaginitis Pregnancy and childbirth Pelvic surgery Fluid intake (e.g. excess caffeine) Pelvic radiotherapy Chronic in /f_l ammatory conditions resulting in bladder /f_i brosis (tuberculous cystitis, ketamine cystitis, interstitial cystitis)

Extraperitoneal injury

Extraperitoneal injury

The management of extraperitoneal rupture consists of urethral catheterisation with free bladder drainage for 10–14 days, followed by a cystogram to ensure that the injury has healed prior to removal of the catheter. If the extraperitoneal injury is iatrogenic and recognised at the time of open or laparoscopic surgery , it can be repaired at the time in two layers with 2/0 Vicryl absorbable suture. If the bladder injury is associated with a pelvic fracture and the patient is undergoing surgery for open fixation, or repair of a rectal or vaginal perforation, the bladder should be repaired at the same time. - - - -

Figure 83.38 Computed tomography showing intraperitoneal bladder injury after transurethral resection of the bladder tumour (arrow point

ing to intraperitoneal urinary extravasation). TABLE 83.18 Grading of bladder trauma. Grade Injury Description I Haematoma Contusion, intramural haematoma Laceration Partial thickness II Laceration Extraperitoneal bladder wall laceration <2 /uni00A0 cm III Laceration Extraperitoneal ≥ 2 /uni00A0 cm or intraperitoneal <2 /uni00A0 cm bladder wall laceration IV Laceration Intraperitoneal bladder wall laceration ≥ 2 /uni00A0 cm V Laceration Laceration extending into the bladder neck or ureteral ori /f_i ce (trigone)

Fascia and ligamentous supports

Fascia and ligamentous supports

/uni25CF At the posterolateral bladder neck, condensations of fascia pass forward medially and laterally to the ureter to join with the prostatic fascia; this fascia needs to be divided during cystectomy . /uni25CF The puboprostatic ligaments are well-defined condensa - tions of the anterior endopelvic fascia; they stretch from the front of the prostate to the periosteum of the pubis and lie lateral to the dorsal vein complex. /uni25CF The urachus and obliterated hypogastric arteries, together with the folds of peritoneum overlying them, are called the median and lateral umbilical ligaments. /uni25CF Condensations of fascia also occur around the superior and inferior vascular pedicles. /uni25CF The pelvic floor organs are supported by the pelvic floor muscles, which predominantly consist of the levator ani group of muscles. The muscles are covered by endopel - vic fascia, which attaches the vagina to the pelvic sidewall and is thickened laterally as the arcus tendineus fascia pel - vis (ATFP). The ATFP lies medial to the obturator inter - n us and is an important landmark into which sutures are placed for pelvic or gan prolapse surgery .

Grading and staging

Grading and staging

Bladder cancer is graded as well di ff erentiated (G1), moderately di ff erentiated (G2) and poorly di ff erentiated (G3). Stages Tis, Ta and T1 are non-muscle-invasive (NMIBC) and stages T2, T3 and T4 are muscle-invasive (MIBC) or locally advanced (Figure 83.34). Approximately 70% of tumours are NMIBC at presentation, whereas 30% are MIBC or metastatic. -

HAEMATURIA

HAEMATURIA

Haematuria is the presence of blood in the urine. It can be clas sified as visible (VH, or macroscopic) and non-visible (NVH, microscopic or dipstick). Microscopic haematuria is defined as the presence of red blood cells (RBCs) on microscopic exam ination of the urine and is variably defined as three or more or five or mor e RBCs per high-power field. A few RBCs can be found in the urine of healthy people, especially after rigorous exercise, sexual intercourse or from menstrual contamina with an upper limit of 1 million RBCs per 24 hours considered normal. Overall, 30–60% of patients with NVH are found to have an underlying cause, depending upon the age and risk factors of the population studied and the type of investigation performed, but the rate of malignancy is around 5% for those with NVH compared with almost 20% for those with VH. Both VH and NVH can arise from anywhere in the renal tract, including renal parenchyma, renal pelvis, ureter, bladder, prostate and urethra. Certain diseases outside the renal tract may also lead to haematuria (T able 83.15). There is a lack of consensus between national guidelines regarding who should be investigated for haematuria. How ever, all patients should hav e a digital rectal examination to evaluate prostate size and consistency , urine culture to ex infection and urine cytology to aid diagnosis of urothelial malignancy in those at higher risk (smokers, occupational his tory , family history , elderly). Serum estimated glomerular fil tration rate (eGFR) should be assessed, and prostate-specific antigen (PSA) testing should be discussed in men with a 10- to 15-year life expectancy to assess prostate cancer risk. Those with visible haematuria should under go evaluation of the LUT with cystoscopy and upper urinary tract with CT urogram. Patients with NVH should have the urine microscopy repeated on three occasions, and only be investigated if the haematuria is persistent. Patients with NVH who are over 40 years old should also undergo evaluation with flexible cystoscopy and renal tract imaging (ultrasound or CT urogram), but investi gations could be rationalised to flexible cystoscopy and renal Summary box 83.7 Haematuria /uni25CF Eduard Heinrich Henoch , 1820–1910, Professor of Diseases of Children, Berlin, Germany , described this form of purpura in 1868. Johann Lucas Schönlein , 1793–1864, Professor of Medicine, Berlin, Germany , published his description of this form of purpura in 1837. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF tion, /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF clude - - tract ultrasound in younger patients deemed to be at low risk of urothelial malignancy . In those with NVH and proteinuria, where the above urological investigations are negative, nephro - logical causes should be sought.

Patients with haematuria require upper tract investigation with CT

urogram and lower tract investigation with cystoscopy Site Cause Kidney Cancer (renal cell, urothelial, squamous cell, adenocarcinoma) Stones Infection Trauma Cystic diseases (e.g. medullary sponge kidney, polycystic kidney disease) Vascular disorder (e.g. vascular malformations, renal vein thrombosis) Nephrological causes (IgA nephropathy, glomerulonephritis, vasculitis, Henoch– Schönlein purpura) Papillary necrosis Ureter Cancer (urothelial) Stones Infection

Trauma Benign diseases (PUJ obstruction, stricture) Cancer (urothelial, squamous cell, Bladder adenocarcinoma) Stones Infection (bacterial, TB, schistosomiasis) Trauma Chronic in /f_l ammatory conditions (IC, radiation cystitis, ketamine cystitis, cyclophosphamide cystitis) Prostate Cancer Benign prostatic enlargement Infection Medical Bleeding disorders (e.g. sickle cell, thrombophilia) Anticoagulation therapy Iatrogenic Urethral instrumentation Nephrostomy IC, interstitial cystitis; IgA,

immunoglobulin A; PUJ, pelviureteric junc

tion; TB, tuberculosis.

INNERVATION OF THE BLADDER

INNERVATION OF THE BLADDER

The lower urinary tract (LUT) is innervated by sympathetic, parasympathetic and somatic a ï¬€ erent and e ï¬€ erent nerves, under higher control from the cerebral cortex and pontine micturition centre (PMC). The actions of the spinal and peripheral nerves on the LUT are summarised in Table 83.1 Coordination of the micturition process is performed by the PMC in the brainstem and is under the control of higher centres in the cortex, thalamus and hypothalamus, which play an important role in delaying voiding until it is socially con venient.

Intraperitoneal injury

Intraperitoneal injury

Intraperitoneal injuries usually require open surgical repair to reduce the risks of urinary contamination of the peritoneal space. If the injury is small without significant fluid extravasa - tion, a period of catheterisation can be attempted in clinically well patients, but close monitoring is required and a cystogram at 2 weeks should confirm complete healing prior to removal of the catheter. If it has not healed, open re pair will be required. Summary box 83.9 Bladder trauma /uni25CF /uni25CF /uni25CF

Bladder trauma can be extraperitoneal or intraperitoneal Extraperitoneal injury can be managed with indwelling catheterisation for 10–14 days Intraperitoneal injury most often requires laparotomy and repair of the bladder defect

Abrams P , Andersson KE, Birder L et al . Fourth International Consultation on Incontinence Recommendations of the International Scientific Committee: evaluation and treatment of urinary incontinence, pelvic organ prolapse, and fecal incontinence. Neurourol Urodyn 2010; 29 (1): 213–40. Babjuk M, Burger M, Compérat EM. European Association of Urology guidelines on non-muscle-invasive bladder cancer (TaT1 and carcinoma in situ) – 2019 update. Eur Urol 2019; 76 : 639–57. Bonkat G, Bartoletti R, Bruyere F et al . EAU guidelines on urological infections . Available from https://uroweb.org/wp-content/uploads/ EAU-Guidelines-on-Urological-Infections-2018-large-text.pdf (accessed 29 August 2020). micturition. Nat Rev Neurosci 2008; 9 (6): 453–66. Malde S, Palmisani S, Al-Kaisy A, Sahai A. Guideline of guidelines: bladder pain syndrome. BJU Int 2018; 122 (5): 729–43. Partin AW , Peters CA, Kavoussi LR, Dmochowksi RR, Wein AJ. Campbell–Walsh–Wein urology , 12th edn. Philadelphia, PA: Elsevier, 2021. Witjes JA, Bruins HM, Cathomas R et al . European Association of Urology guidelines on muscle-invasive and metastatic bladder cancer: summary of the 2020 guidelines. Eur Urol 2021; 79 (1): 82–104.

Introduction

Introduction

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Investigation

Investigation

The diagnosis is made on imaging (ultrasound, computed tomography [CT], magnetic resonance imaging [MRI] or cystogram) or through direct vision at cystoscopy (Figure 83.7). Bladder outlet obstruction should be confirmed with urody - namics if suspected. Investigation

Investigation should aim to identify predisposing or exacerbat - - ing factors for urinary incontinence, as well as any features that may have a detrimental outcome on treatment (e.g. BMI). The following investigations are recommended: 1 Urinalysis – to identify UTI. - 2 Flow rate and postvoid residual measurement – to identify voiding dysfunction or urinary retention. 3 Three-day bladder diary – to assess daytime and night - time frequency episodes, polyuria (the production of >2.8 litres of urine in 24 hours in adults), nocturnal polyuria (>20–33% of urine production occurs at night), functional capacity (based on voided volumes) and inconti - nence episode frequency . The following in vestigations should be considered if con - servative and pharmacological measures have failed to improve symptoms, if there is suspicion of underlying anatomical or neurological pathology based on the history and examination or in cases of recurrent urinar y incontinence after previous surgery . 1 CT urogram or MRI will identify an ectopic ureter or ure - terovaginal or vesicovaginal fistula (VVF). MRI should be performed if there is suspicion of a urethral diverticulum. 2 Cystourethroscopy in cases where fistula or iatrogenic bladder or urethral pathology is suspected (e.g. following previous incontinence surgery). 3 Urodynamics/video urodynamics – this test is used to assess the pressure–volume relationship of the bladder during the storage and voiding phases of the micturition cycle. In the investigation of urinary incontinence, uro - dynamics is recommended in the following situations: /uni25CF MUI (SUI with UUI or overactive bladder symptoms); /uni25CF suspicion of voiding dysfunction or neurological LUT dysfunction; /uni25CF previous failed anti-incontinence surgery; /uni25CF prior to invasive treatment. Urodynamics (filling and voiding cystometry) Urodynamic investigation is used to measure the detrusor pres - sure during filling and voiding. The detrusor pressure cannot be measured directly and so it is derived from subtracting the intra-abdominal pressure from the intravesical pressure (Figure 83.12). /uni25CF Technique . A 6Fr or 8Fr transurethral pressure- measuring catheter is inserted into the bladder to measure the intravesical pressure (p), and another is inserted into ves the rectum (or vagina) to measure the intra-abdominal pressure (p). The detrusor pressure (p) can then be abd det derived by subtracting intra-abdominal pressure from intravesical pressure. /uni25CF Storage phase . The bladder is filled with saline through the transurethral catheter at a steady rate (usually 50 /uni00A0 mL/min in non-neurogenic patients) and the following observations are recorded: cystometric capacity , compliance of the bladder (the relationship between the change in bladder volume and the change in detrusor pressure), bladder sensations and the presence of phasic rises in detrusor pressure (detrusor overactivity) (Figure 83.13). Any incontinence associated with phasic rises in detrusor pressure represents UUI. Stress tests (e.g. cough) are also performed throughout the filling phase and any leakage associated with increases in intra-abdominal pressure, in the absence of an increase in detrusor pressure, represents SUI. /uni25CF Voiding phase . When the patient has

reached a strong desire to pass urine, bladder filling is stopped and the pa tient is asked to void. During voiding, a high detrusor pres sure with corresponding low flow rate represents bladder outlet obstruction (Figure 83.14). Several nomograms to identify bladder outlet obstruction based on pressure–flow criteria exist for men and women. Jerry G Blaivas , b. 1943, American urologist. Carl A Olsson , contemporary , American urologist, with Jerry G Blavias described the radiographic classification of stress urinary incontinence in 1988. /uni25CF Video urodynamics . The addition of fluoroscopic im - aging enables the diagnosis of VUR and anatomical ab - normalities (bladder or urethral diverticula or fistulae) and can classify the type of SUI (Table 83.6). In this case, the bladder is filled with radiographic contrast. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF -

500 375 250 125 Millilitres 0 75 O 2 50 cmH 25 0 72 O 48 2 24 cmH 0 69 O 2 46 23 cmH 0 Figure 83.12 Urodynamic trace showing normal bladder storage and voiding. The red line represents intra-abdominal pressure (blue line intravesical pressure (p). The subtracted pressure – the detrusor pressure (ves no rise in p , which represents a normal bladder storage phase. abd, intra-abdominal; det, detrusor; MCC, maximum cystometric capacity; det p1, pressure 1; p2, pressure 2; ves, intravesical. 345 348 p det det -2 p1 ves 44 p2 abd 45 p) and the abd p) – is shown by the orange line. During /f_i lling, there is det TABLE 83.6 Blaivas–Olsson classi /f_i cation of stress urinary incontinence. Type At rest On stress Rotational descent, 0 Bladder neck closed bladder neck open, but no Situated above the leak demonstrated superior margin of the pubic symphysis I Descent less than 2 /uni00A0 cm, Bladder neck closed bladder neck open and Situated above the leak seen. No cystocele inferior margin of the pubic symphysis IIA Descent more than 2 /uni00A0 cm, Bladder neck closed bladder neck open and Situated above the leak seen. Cystocele seen inferior margin of the pubic symphysis IIB May or may not be further Bladder neck closed descent, but bladder neck Situated below the opens and leak seen inferior margin of the pubic symphysis 3 Obvious gravitational Bladder neck and incontinence in the proximal urethra open at absence of signi /f_i cant rest (in the absence of a mobility detrusor contraction) Positions at rest and on stress refer to the bladder neck and proximal urethra.

Cg Cg 500 375 250 125 72 19 0 75 50 25 12 4 0 72 48 36 35 24 0 69 46 32 23 23 0 Figure 83.13 Urodynamic trace showing detrusor overactivity (phasic rises in intravesical pressure [a corresponding rise in intra-abdominal pressure [p]). abd, intra-abdominal; Cg, cough; det, detrusor; dlp, detrusor leak point; do, detrusor abd overactivity; MCC, maximum cystometric capacity; p1, pressure 1; p2, pressure 2; ves, intravesical. MCC Values 348 Millilitres O 2 cmH -3 47 O 2 cmH 49 O 2 cmH mL/s 0 Figure 83.14 Urodynamic trace showing bladder outlet obstruction (very high intravesical pressure [low /f_l ow rate represented by the lowest trace). abd, intra-abdominal; det, detrusor; MCC, maximum cystometric capacity; p1, pressure 1; p2, pressure 2; ves, intravesical. Cg do MCC 232 234 214 194 137 p det 59 det 23 9 3 -1 90 p1 ves 49 40 32 31 p2 abd 32 31 30 29 26 p] and detrusor pressure [p] without ves det Values 348 p det det 5 p1 ves p2 53 abd 0 p] and detrusor pressure [p] with the ves det

Conservative The recommended initial treatment for urinary incontinence consists of lifestyle interventions (weight loss, fluid modifi cation, smoking cessation, treat constipation), behavioural therapy (timed voiding and bladder training) and pelvic floor muscle training. Pharmacological therapy Pharmacotherapy with antimuscarinics and β -agonists is the 3 mainstay of management. Antimuscarinics should be used with caution in the elderly or in those who take multiple medi

cations with antimuscarinic activity owing to the association with dementia with long-term use. The aim of pharmacological treatment of SUI is to increase the urethral closure pressure by increasing smooth and stria muscle tone. Duloxetine, a selective serotonin and noradrena line reuptake inhibitor, has been shown to increase sphincteric muscle activity and therefore improve urinary incontinence. However, pharmacological trea tment for SUI is less commonly used than for OAB because of high discontinuation rates and reports of serious adverse events, such as mental health disor ders and suicide, with duloxetine. Invasive treatment of stress urinary incontinence in women Several surgical options for the treatment of SUI are available, and the choice of therapy depends upon the pathophysiology of the incontinence (relative degree of hypermobility and intrinsic sphincter deficiency) and individual patient prefer ences. Intraurethral injection therapy The least invasive surgical option for the treatment of SUI is the injection of bulking agent into the urethral submucosa to improve the urethral closure mechanism and hence restore continence. This is particularly beneficial for those with a greater degree of intrinsic sphincter deficiency (type 3 SUI) rather than those with predominant hypermobility . The procedure can be performed as an outpatient and involves injecting bulking agent into di ff erent aspects of the urethra, at the level of the bladder neck or mid-urethra, order to obtain visual urethral coaptation (Figure 83.15 Although less invasive than other surgical treatments, suc cess rates are generally lower, with patient-reported improve ment rates of approximately 60% in the short term; repeat injections are often required. Synthetic mid-urethral sling The synthetic mid-urethral sling has been the most commonly performed surgical procedure for SUI over the past three decades. Its less invasive nature compared with the autologous sling and retropubic suspension procedures in addition to its relative ease of insertion have contributed to its popularity . Synthetic slings are made of type 1 macroporous polypropylene Anders Retzius , 1796–1860, Swedish anatomist. - - ted - mesh and can be inserted through the retropubic or transob - - turator routes. A 1-cm incision in the anterior vaginal wall at the level of the mid-urethra is made, and the paraurethral space is dissected bilaterally to the pubic bone. With an empty blad - der, the sling is placed through the retropubic space, exiting on the lower abdominal wall appr oximately 2–3 /uni00A0 cm lateral to the midline bilaterally . Cystoscopy should be performed after sling placement to ensur e that the mesh has not perforated the - bladder or urethra. The sling is placed without tension. The transobturator sling is placed in a similar manner, but the sling is placed through the obturator foramen, exiting in the groin crease at the level of the clitoris. Success rates are high with an almost 90% cure rate at 17-year follow-up, and e ffi cacy is similar between retropu - bic and transobturator slings. Howev er, increasing concerns about serious long-term mesh-related complications (erosion; chronic pelvic, groin, perineal and vaginal pain; sexual dys - function) ha ve led surgeons in many countries to abandon the use of these devices. Retropubic suspension procedures in). Colposuspension aims to restore the bladder neck and proxi - - mal urethra to their normal intra-abdominal position, thereby - allowing equal pressure transmission to the bladder and prox - imal urethra at times of raised intra-abdominal pressure, and so impro ve SUI by augmenting the urethral closure pressure. It is recommended for those with demonstrable hypermobility and concomitant cystocele. The procedur e is traditionally performed through a Pfannenstiel incision, although minimally invasive approaches (laparoscopic, robot assisted) have been described in recent years in an attempt to reduce morbidity . The procedur e involves dissection of the retropubic space (space of Retzius), the relatively avascular space between the pubic symphysis and

Figure 83.15 Urethral mucosal coaptation following intraurethral injection of bulking agent. The urethral mucosa can be seen to be bulging at the site of bulking agent injection, thereby occluding

the urethral lumen.

bladder. The vaginal fascia at the level of the bladder neck and proximal urethra is exposed bilaterally and two to four non absorbable sutures are placed 2–3 /uni00A0 cm lateral to the bladder neck (proximal suture) and proximal urethra (distal suture) on each side. T hese sutures are then attached to the iliopectineal ligament (Cooper’s ligament) and the knots tied gently in order to achieve elevation without tension (Figure 83.16). Cystoscopy should be performed following the procedure to ensure that the sutures have not been passed through the bladder. The Burch colposuspension has good long-term e ffi cacy with cure rates of 70–90% at 5-year follow-up. Howe ver, risks of posterior pelvic organ prolapse are higher than with sling procedures. Autologous fascial sling The autologous pubovaginal sling aims to improve SUI by adding strength to the mid-urethral posterior supporting ‘hammock’. It is an e ff ective and durable treatment for SUI and can be inserted ‘tension-free’, similar to the synthetic mid-urethral sling, or can be inserted under tension in order to provide a compressive e ff ect on the urethra for those with intrinsic sphincter deficiency . As this sling is not a foreign body , there is very low risk of urethral erosion. Most commonly , a strip of rectus fascia (8 /uni00A0×/uni00A0 2 /uni00A0 cm) is har vested through a small Pfannenstiel incision, although if this cannot be used then fascia lata can be harvested from the leg using a fascial stripper. The sling is then attached to a 0 polydioxanone (PDS) suture on each side (Figure 83.17 passed into the retr opubic space in a similar manner to the synthetic mid-urethral sling. In patients with a greater degree of intrinsic sphincter deficiency the sling can be inserted under a greater degree of tension. Sir Astley Paston Cooper , 1768–1841, surgeon, Guy’s Hospital, London, UK, described the ligament that runs on the pectineal line of the pubic bone in 1804. John C Burch , 1900–1977, gynaecologist, V anderbilt University , Nashville, TN, USA. - Success rates of 75% at 10-year follow-up are reported. The pubovaginal sling is a suitable option for those who have failed previous SUI surgery , irradiated patients or those with urethral fibrosis from previous urethral pathology (e.g. urethral diverticulum, fistula, mesh erosion). However, there is a higher risk of voiding dysfunction requiring CISC with this technique, as well as new onset OAB symptoms. Artificial urinary sphincter - The artificial urinary sphincter (AUS) is considered for women with severe recurrent SUI due to intrinsic sphincter deficiency that has failed to improve after previous surgical intervention. This three-piece device consists of a cu ff that is) and placed around the urethra at the level of the bladder neck, a pressure-regulating balloon placed in the extraperitoneal space and a control pump placed in the labia majora (Figure 83.18). Recent advances in minimally invasive surgery have reduced the morbidity associated with the traditional open approach,

Figure 83.16 Burch colposuspension showing the suture position from the vaginal fascia (arrow) to the iliopectineal (Cooper’s) ligament (star). Figure 83.17 Autologous rectus fascial sling harvested and attached to non-absorbable sutures (‘sling on a string’). Cuff Pressure regulating reservoir Control pump Figure 83.18 The AMS 800 Arti /f_i cial Urinary Sphincter.

and safety in randomised trials. Evidence of e ffi cacy is limited to case series from specialist centres. One of the largest series of 376 AUS implantations reported a cure rate of 85.6% at a mean of 9.6 years of follo w-up, but this was at the expense of a 10-year revision rate of 30%. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Invasive treatment of stress urinary incontinence in men Post-prostatectomy incontinence is the commonest cause of SUI in men. Several factors may contribute

(Table 83.7 Pharmacological treatment with duloxetine has a limited role; the mainstay of treatment is surgical, with the male sub urethral sling or the AUS. The male transobturator suburethral sling is recommended for men with mild to moderate SUI and those w ho have not had prior radiotherapy . In cases of severe SUI or recurrent SUI or in those who have had prior radiotherap y , the AUS is the gold standard treatment. The male sling repositions the bulb of the urethra in a retrourethral position, providing additional support to the existing sphincter without causing obstruction. The AUS is occlusive. Both approaches require a midline perineal incision through the bulbospongiosus muscle. For placement of the male suburethral sling, a helical trocar is used to pass the polypropylene mesh through the obturator foramina bilaterally . Placement of the AUS requires circumfer ential mobilisation of the proximal bulbar urethra for insertion of the cu ff , and a separate inguinal incision for insertion of the pressure-regulating balloon into the extraperitoneal space (Figure 83.19). The control pump is connected and inserted into a subdartos pouch in the scrotum through this inguinal incision. T he transobturator suburethral sling has reported cure rates of 66% at 3-year follow-up whereas the AUS has long term satisfaction rates of 80–90%. Reoperation with the AUS is r equired in 26% in the long term for infection, erosion or mechanical failur e. Invasive treatment of overactive bladder and urgency urinary incontinence If pharmacological therapy for idiopathic or neurogenic OAB fails, intravesical injection of BTX-A and sacral nerve stimu lation (SNS) have demonstrated high e ffi cacy in randomised). - trials. For those who prefer a less invasive alternative, percuta - neous tibial nerve stimulation (PTNS) may be useful. Failure to respond to these minimally invasive treatments often leads to surgical treatment with augmentation enterocystoplasty or urinary diversion for end-stage incontinence. Intravesical injection of botulinum toxin A BTX-A is a neurotoxin produced by the anaerobic bacterium - Clostridium botulinum . There are seven subtypes (A–G) and type A is most clinically useful because of its longer duration of action. It is thought to have both a ff erent and e ff erent mech - anisms of action. The procedure is performed under local anaesthesia with a flexible cystoscope. Risks include UTI and voiding dysfunction requiring CISC in approximately 10%. Furthermore, symp - egen - toms typically return after around 6 months owing to r - eration of new nerve terminals, so the treatment needs to be repeated at regular intervals. High-quality randomised trials have demonstrated the e ffi - thic OAB and neurogenic detru - cacy of BTX-A for both idiopa sor overactivity , with success rates of approximately 60–90%. Sacral nerve stimulation This treatment involves implantation of an electrical pulse generator to stimulate the S3 sacral nerve root, thereby - improving OAB symptoms. The mechanism of action is not

TABLE 83.7 Factors contributing to post-prostatectomy incontinence. Patient factors Surgical factors BMI Fibrosis Age Urethral stricture Prostate size Technique of prostatectomy (non-nerve-sparing) Membranous urethra length Pre-existing LUTS Laxity of posterior support Previous TURP Neurovascular bundle damage Previous radiotherapy Devascularisation BMI, body mass index; LUTS, lower urinary tract symptom; TURP , transurethral resection of the prostate. Figure 83.19 Perineal approach demonstrating exposure of the corpus spongiosum.

completely understood but it is thought that a ff erent stimu lation modulates reflex pathways involved in the micturition cycle. The procedure is typically performed in two stages. The first, known as the ‘first stage tined lead’, involves insertion of a lead with f our electrodes percutaneously , under fluoro scopic guidance, to the S3 foramen via a posterior approach. Electrical cur rent is then applied to achieve stimulation of the sacral nerve, and correct lead

placement is identified by the typical motor responses of plantarflexion of the g reat toe and inward movement of the intergluteal fold due to contraction of the levator ani (anal reflex). This lead is then kept in place for approximately 2 weeks; if clinical e ffi cacy (based on at least 50% improvement in bladder diary parameters) has been achieved then the second stage involves insertion of a perma nent implant (Figure 83.20). E ffi cacy rates of 70–80% have been reported with this technique for both idiopathic OAB and NLUTD, and there is no significant di ff erence in terms of e ffi cacy between BTX-A and SNS. Complications include lead migration, device infec tion and implant site pain. Percutaneous tibial nerve stimulation PTNS is a minimally invasive form of peripheral neuromod ulation that is recommended for patients who are unsuitable is thought to improve symptoms of idiopathic OAB through stimulation of the sacral plexus S2–4, indirectly via the tibial nerve. Success rates are lower than those reported for BTX-A and SNS (approximately 50%), and patients require weekly treatment sessions over 12 weeks. However, there are no signif - icant side e ff ects related to this treatment. Augmentation enterocystoplasty Augmentation enterocystoplasty is reserved for those with high-pressure detrusor overactivity , poor compliance and reduced bladder capacity who have failed to respond to the above treatments. It is recommended for patients with both idiopathic and neurogenic bladder dysfunction. The aim is to create a low-pressure reservoir with increased functional capacity , thereby preserving renal function. The procedure can be performed through a Pfannenstiel or lower midline abdominal incision with an extraperitoneal approach to mobilise the bladder from the peritoneum. The bladder is then bivalved in the coronal plane to a point 1 /uni00A0 cm anterior to the ureteric orifices bilaterally . Although several gastrointestinal segments have been used, ileocystoplasty is the most common. A 25-cm segment of ileum is isolated, opened along its antimesenteric border and attached to the bivalved bladder, thereby increasing the bladder capac - ity (Figure 83.21). A suprapubic catheter is placed, and the patient undergoes a cystogram 3 weeks after surgery to ensure that the enterocystoplasty segment has completely healed prior to catheter removal. Augmentation cystoplasty is an e ff ective option with conti - nence and satisfaction rates of over 90%, as well as considerable improvements in urodynamic parameters (detrusor overactiv - ity , compliance, maximum detrusor pressure). Howev er, this is a major surgical undertaking with risks of UTI, need for - CISC, metabolic disturbances, mucus and stone formation, spontaneous perforation and possibly a small long-term risk of malignancy . - - - -

Figure 83.20 Radiograph demonstrating correct lead placement of the tined lead in the S3 foramen and the implant in the buttock. Figure 83.21

Augmentation ileocystoplasty. One of the ureteric ori

/f_i ces can be seen with the interureteric bar (arrow).

In those with so-called ‘end-stage’ incontinence that has failed to respond to the above measures, urinary diversion remains a last resort to improve quality of life. Ileal conduit urinary diversion, with or without cystectomy , is most commonly performed. When performed for benign indications, overall revision rates approach 40% (incisional or parastomal hernia, stomal complications, ureteroileal anastomosis revision or secondary cystectomy for refractory pyocystis). Summary box 83.3 Urinary incontinence /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Urinary incontinence should be classi /f_i ed as UUI, SUI or MUI Initial management for urinary incontinence of any type is with behavioural modi /f_i cation, bladder training and pelvic /f_l oor muscle training OAB and UUI can be managed in a stepwise manner with pharmacotherapy, intravesical BTX-A, SNS or augmentation enterocystoplasty SUI in women is initially managed with bulking agents, mid- urethral slings (synthetic or autologous) or colposuspension SUI in men is managed with the suburethral sling or AUS Urinary diversion is a last-resort option for those with end- stage urinary incontinence Suprapontine lesion • History : predominantly storage problems • Ultrasound • Urodynamics Spinal (infrapontine–suprasacral) lesion • History : both storage and voiding problems • Ultrasound • Urodynamics dyssynergia Sacral/infrasacral lesion • History : predominantly voiding symptoms • Ultrasound • Urodynamics acontractile detrusor Figure 83.22 Characteristic lower urinary tract disorders arising from neurological disease. (Reproduced with permission Fowler CJ, Kessler TM. Lower urinary tract dysfunction in the neurological patient: clinical assessment and management. 14 (7): 720–32.)

Investigation

Plain radiograph of the bladder, renal tract ultrasound or CT will confirm the diagnosis and provide an estimation of size to aid treatment planning (Figure 83.28). Investigation

Initial investigation is with urinalysis (a dipstick test checks for the presence of red cells, white cells and nitrites), urine - microscopy , culture and sensitivity , flow rate assessment and measurement of postvoid residual volume. In those with rUTI, renal tract ultrasound to exclude anatomical pathology should be performed; cystoscopy is reserved for those with atypical symptoms, haematuria or other features that raise suspicion of underlying pathology (e.g. bladder cancer, stones, urinary tract fistula). Current methods of microbiological UTI diagnosis are based on the identification of a ‘significant’ pure growth 3 of a known uropathogen in the urine. A cut-o ff of ≥ 10 colony-forming units/mL is commonly used to diagnose acute uncomplicated cystitis in women, but it is becoming increasingly clear that patients can develop symptoms of UTI with much lower concentrations of urinary bacteria; the Pseudo - minimum concentration required to cause UTI or rUTI has not yet been defined. - Investigation

Urine Urine should be cultured and examined cytologically for malignant cells. An increasing number of urinary biomarkers based on panels of epigenetic markers are being studied, but none has been shown to surpass the accuracy of cystoscopy and so are not routinely used. Cross-sectional imaging: CT urography and MRI of the bladder CT urogram is the gold standard evaluation for upper tract disease (including hydronephrosis) and assessment of nodal metastases (Figure 83.35). MRI of the bladder can be useful in staging of the primary tumour. Imaging should ideally be performed prior to transurethral resection of the bladder tumour (TURBT) as false-positive T3 disease can be diagnosed if cross-sectional imaging is carried out too soon after TURBT . CT of the chest should be performed in confirmed bladder cancer cases for complete staging. Cystourethroscopy Flexible cystourethroscopy under local anaesthetic is the main stay of diagnosis and should always be performed on patients with haematuria (Figure 83.36). The diagnostic accuracy of conventional ‘white’ light cystoscopy has been improved optical enhancement techniques such as narrow-band imaging (NBI) and photodynamic ‘blue’ light cystoscopy (PDD), which relies on the photosensitiser hexaminolevulinate. These tech niques are recommended in patients with a high suspicion of cancer and negative initial findings, or in those with positive cytology but negative ‘white’ light cystoscopy . Investigation

Retrograde cystogram or CT cystogram confirms the diag nosis and identifies whether the injury is intraperitoneal or extraperitoneal (Figure 83.38). Postdrainage views should be obtained as a small amount of contrast extravasation may be missed with a full bladder. With intraperitoneal perforation, contrast is seen to outline loops of bo wel.

Ketamine cystitis

Ketamine cystitis

Ketamine is an N -methyl-d-aspartate (NMDA) antagonist. It has been used for decades as an anaesthetic agent but has because of its euphoric and psychedelic e ff ects. As a result of long-term ketamine abuse, up to 30% develop the condition of ketamine cystitis – a chronic inflammatory bladder condition ladder with characterised b y a small, contracted, inflamed b ureteric stricture and hydronephrosis in advanced cases. The severity of the inflammatory e ff ect is related to the duration of abuse. The clinical presentation is very similar to that of BPS/ IC and investigation with cystoscopy , bladder biopsy and CT urogram should be performed to exclude other inflammatory or infective conditions (e.g . TB, schistosomiasis) and to evaluate the e ff ect on the upper urinary tract (Figure 83.33). - - - Initial management is centred around the cessation of ket - amine use as surgical intervention should not be performed in those continuing to use ketamine. If patients have upper - tract obstruction due to ureteric involvement in the inflamma - - tory process, renal drainage with stent or nephrostom y will be required as a temporising measure to preserve renal function. Management of LUTS and pain follows the same pathway of oral and intravesical therapies as for BPS/IC, although regimes for analgesia consisting of co-codamol, amitriptyline - and buprenorphine patches have proved particularly beneficial in this condition. Surgical approaches are similar to those for the other chronic inflammatory conditions, namely augmentation enterocystoplasty , supratrigonal cystectomy and ureteric reim - plantation, total cystectomy with orthotopic neobladder, het - erotopic neobladder with appendicovesicostomy or urinary diversion with or without a cystectomy . However, the rate of perioperative complications with major reconstructive surgery in this popula tion is high; those with upper tract involvement at presentation are likely be at higher risk of postoperative com - plications.

(a) (b) Figure 83.33 Computed tomography showing a thickened bladder (a) and bilateral hydroureteronephrosis (b) secondary to ketamine cystitis.

Chronic inflammatory conditions of the bladder /uni25CF /uni25CF /uni25CF

Patients with chronic in /f_l ammatory bladder conditions should have a bladder biopsy to identify a treatable cause (e.g. TB) Upper tracts should be evaluated with cross-sectional imaging to identify renal obstruction due to a high-pressure bladder Management is aimed at symptomatic improvement and maintaining low bladder pressure

Learning objectives

Learning objectives

To describe: The anatomical, embryological and pharmacological • features of the bladder The physiology of micturition and the neurological basis of • lower urinary tract function The clinical features, investigations and principles of • management of congenital anomalies of the bladder The clinical features, investigation and principles of • management of urinary incontinence The principles of management of acute and chronic • retention of urine The indications and technique of urethral and suprapubic • bladder catheterisation The causes, investigations and principles of management • of bladder diverticula and bladder stones

Lymphatics

Lymphatics

/uni25CF Internal iliac, hypogastric, obturator and external iliac chain of nodes. /uni25CF Pelvic lymphadenectomy for bladder cancer should in clude complete clearance of all these nodes.

The clinical features, investigations and principles of • management of urinary tract infections and chronic in /f_l ammatory bladder diseases The causes, investigations and principles of management • of haematuria The clinical features, investigations and principles of • management of bladder cancer The clinical features, investigations and principles of • management of bladder trauma To describe and distinguish: Based on the clinical presentation, the types of bladder • dysfunction associated with diseases of the central nervous system

Muscle-invasive bladder cancer

Muscle-invasive bladder cancer

The two primary radical treatment options for MIBC are radi cal cystectomy with urinary diversion or chemoradiotherapy . Whichever modality is employed, 5-year survival rates are approximately 60%. There is a move towar ds primary surgical treatment in most centres. The use of systemic chemotherapy given before (neoadjuvant) radical cystectomy has been shown to improve survival by about 5–7%. Newer immunotherapy approaches are being evaluated in the neoadjuvant and adjuvant setting; in particular, immune-checkpoint inhibitors with antibodies targeting the programmed cell death ligand 1 (PDL1) pathway are demonstrating promising results. Radical cystectomy and ileal conduit urinary diversion Those with poor bladder function, significant haematuria, upper tract obstruction, widespread CIS or factors that a ff ect successful radiotherapy (e.g. bilateral hip replacements, inflam matory bowel disease) are more suitable for radical cystectomy and pelvic lymphadenectomy . This is major surgery with a perioperative morbidity rate of up to 50%, and so patients Charles Pierre Denonvilliers , 1808–1872, Professor of Anatomy and later of Surgery , Paris, France. Denonvilliers' fascia is the fascial layer that separates the prostate and bladder from the rectum. Eugene Bricker , 1908–2000, American surgeon, described the separate anastomosis of each ureter to the ileal segment. David Mitchell Wallace , 1913–1992, urologist, St Peter's Hospital, London, UK, described the anastomosis of both ureters together followed by anastomosis to the ileal segment in one plate in 1966. surgery . Alternativ e drainage for urine is necessary after removal of the bladder. The standard procedur e is to perform an ileal conduit diversion. Male patients should be counselled about the onset of erectile dysfunction and anejaculation after the operation, although in some cases the nerv e supply for erec - - tile function can be preserved through careful dissection; they should also be told about alternative forms of urinary diver - sion, which include continent urinary diversions and orthot - opic bladder replacement. P atients should be seen by a stoma care therapist, who will ensure that the correct stoma site is chosen, avoiding skin creases to prevent leakage from the ileostomy . The abdomen is opened through a low er midline incision from the umbilicus to pubic symphysis. The liver and the retro- peritoneum are checked for evidence of metastases and the operability of the bladder is assessed. A bilateral pelvic lymph - adenectomy is perf ormed, removing external iliac nodes, internal iliac nodes and the nodes in the obturator fossae. The vessels passing to the bladder from the side wall of the pelvis are ligated and divided; the ureter s are then divided. The pos - terior ligaments extending from the pararectal area to the back of the bladder are ligated and divided, and the layer poste - - rior to Denonvilliers' fascia is opened. The endopelvic fascia is then divided on each side and the puboprostatic ligaments are divided. The dor sal venous complex is divided, and the ure - thra is then mobilised and divided. The ligaments lateral to the prostate are divided and the b ladder is removed. In women, the uterus and anterior vaginal wall need to be included.

Women must be counselled about the loss of ovarian and uterine func - tion. Laparoscopic and robotic cystectom y are increasingly becoming the standard of care with the aim of minimising - perioperative morbidity , but evidence for superiority of these techniques over the traditional open approach is awaited. An isola ted loop of ileum is then prepared on its own mes - entery , and continuity of the small bowel restored. The ureters are then implanted into the bowel either separately (Bricker) or as one plate (Wallace) and the ileostomy is created. Meticu - lous care must be taken to close all mesenteric windows, thus avoiding inter nal hernias (Figure 83.37). Alternative techniques of urinary diversion Alternative forms of diversion are most suitable for highly motivated patients with adequate renal and liver function who wish to avoid an external collection device. Orthotopic bladder An orthotopic bladder is the creation of a pouch (typically using small or large bowel) that is then anastomosed to the patient’s - urethra. Contraindications to an orthotopic bladder include widespread CIS and tumour in the prostatic urethra. Many di ff erent types of orthotopic bladder have been described but a large-capacity , low-pressure reservoir. A pouch is made from 57 /uni00A0 cm of detubularised ileum. The ureters are implanted into a proximal ‘chimney’ that acts as an antireflux mechanism, and the pouch is anastomosed to the urethra (Figure 83.37 Patients can void by relaxing the pelvic floor and straining, but CISC may be required to completely empty the pouch in 15–30%. Continent cutaneous diversion (heterotopic bladder substitute) For those who require urethrectomy , or in whom the urethra is non-functional, a continent cutaneous diversion can be performed. A Studer pouch can be made as described above. The appendix, or a separate section of ileum if the appendix is not available, is then anastomosed from the bladder to the umbilicus or right iliac fossa. The patient can then intermit tently catheterise this channel to drain the pouch. Ureterosigmoidostomy (Mainz II pouch) This option is popular in developing countries as there is no requirement for an external appliance or catheters. Patients require good anal sphincteric function. The sigmoid colon is detubularised and refashioned into a pouch into which the ureters are inserted (Figure 83.37). However, high rates of ascending UTI and increased risk of malignancy (associated with mixing of faecal and urinary streams), bowel frequency and urge incontinence have limited its use. Complications of urinary diversion The ileal conduit urinary diversion has the lowest rate of complications of all forms of urinary diversion. Risks include ureteroileal leak or stricture (5%), stomal complications such as stenosis or hernia (20%), upper tract dilatation (30%), recurrent UTIs and rarely metabolic complications (hyperchloraemic metabolic acidosis). Summary box 83.8 Bladder cancer /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Urs Studer , contemporary , Swiss urologist, described a neobladder pouch that is made of ileum. The Mainz II pouch describes the formation of a low-pressure sigmoid colon pouch into which both ureters are anastomosed. It is named after the city where the inventors of this technique worked.). - Complications of orthotopic or continent pouches include ureteroileal leak or stricture, urinary leak from the pouch, stone formation, UTIs, metabolic complications (hyperchloraemic metabolic acidosis) and rarely adenocarcinoma (5%). Stenosis and incontinence are the main complications of appendico - vesicostomy and up to 50% may require some form of revision surgery to the channel over a 5-year period. Radical external beam radiotherapy Radical radiotherapy is an option for very elderly or unfit patients who are unsuitable for radical cystectomy . Typically , treatment with 66 /uni00A0 Gy is administered in 30 fractions over 6 weeks. However, long-term urinary and bowel side e ff ects can impair quality of life and there is a risk of secondary malig - nancy and fistula.

The commonest presenting symptom of bladder cancer is haematuria Smoking and occupational exposure to certain chemicals are the commonest risk factors Bladder cancer can be non-muscle-invasive or muscle-invasive The management of NMIBC is TURBT, followed by intravesical mitomycin C or BCG depending on the risk strati /f_i cation The management of MIBC is neoadjuvant chemotherapy followed by radical cystectomy Options for urinary diversion include ileal conduit, orthotopic

bladder substitute, heterotopic bladder substitute or ureterosigmoidostomy The choice of diversion is dependent on patient factors, tumour factors and surgeon experience (b) (c) Figure 83.37 Techniques of urinary diversion of urine. (a) Ileal conduit; the ureters are spatulated and anastomosed to ileum; (b) ureterosig

moidostomy; (c) ileal neobladder with an antire /f_l ux long afferent limb.

chemotherapy and then chemoradiotherapy is being studied as an option for those with very localised MIBC, but this remains an option for only a very select group.

Non-muscle-invasive bladder cancer

Non-muscle-invasive bladder cancer

The aim of managing patients with NMIBC is to reduce the risk of tumour recurrence and progression to MIBC. Transurethral resection The initial management of bladder tumours consists of TURBT for accurate staging purposes. This is performed with a rigid cystoscope under general anaesthesia. A biman ual examination should be performed prior to resection to determine whether a mass is palpable and, if so, whether it is mobile or fixed. This should be repeated following resec tion. For lar ge tumours, a standard fractionated resection of the entire tumour, including the tumour base with deep muscle, is performed. For smaller (<3 /uni00A0 cm) solitary tumours, en bloc resection of the entire tumour can be performed and may reduce the risk of tumour recurrence by preventing the spread of tumour cells and subsequent implantation across the bladder. The following details should be recorded to enable accurate risk stratification: the size of the primary tumour, the number of tumours, the nodular or papillary features, concern for the presence of CIS and completeness of visual resection. - by - - - Mapping biopsies from the trigone, bladder dome and the right, left, anterior and posterior bladder wall should be taken if CIS is suspected.

Figure 83.35 Computed tomography showing a large right-sided bladder tumour. Figure 83.36 Cystoscopic appearance of a papillary bladder tumour.

Pathogenesis

Pathogenesis

The most common route of infection is ascending UTI; contamination of the vaginal and periurethral area with uropathogenic organisms originating from the gastrointestinal tract leads to adherence and migration of bacteria into the urethra and bladder. Once in the bladder, adherence of the bacteria to the urothelium triggers a process of bacterial inter nalisation into the urothelial cell and the subsequent formation of intracellular bacterial communities (IBCs) and quiescent intracellular reservoirs (QIRs), which may remain viable for months and act as a source of rUTI. These IBCs and QIRs act in a similar way to a biofilm, protecting bacteria from the host immune response and from the action of antimicrobial agents. Less common routes of infection include haematogenous spread (seen with Staphylococcus aureus and fungal infections) or direct infection from retroperitoneal abscess or inflammatory bowel disease. The commonest organisms implicated in uncomplicated UTI are Escherichia coli (85%), Staphylococcus saprophyticus, Enterococcus faecalis, Proteus and Klebsiella . Complicated UTIs are caused by E. coli (50%), enterococci , S. aureus and monas . Successful infection depends on the bacterial virulence rel ative to the host defence mechanisms. The common bacterial virulence factors and host defence mechanisms are shown in Table 83.13 .

Pathology

Pathology

The commonest type of bladder cancer is transitional cell (urothelial) carcinoma (Table 83.17). Squamous cell carcinoma occurs secondary to chronic inflammation (e.g. indwelling catheter, stone, schistosomiasis), and primary adenocarcinoma usually originates in the urachus (dome of the bladder) or in /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF those with bowel in the urinary tract (augmentation entero cystoplasty , bladder exstrophy repair). Histological variants (e.g. micropapillary , sarcomatoid, plasmacytoid, nested variant) can coexist with urothelial carcinoma and generally signify aggressive tumours with poorer prognosis than pure ur othelial carcinoma.

Smoking 2–5 times increased risk Occupational Tanner exposure Rubber (aromatic Paint and dyes hydrocarbons) Gas and tar Hairdressers Plumbers Painters Environmental Arsenic in drinking water carcinogens Chronic Indwelling catheter in /f_l ammation of Stones bladder Schistosomiasis (predisposes to squamous cell carcinoma) Recurrent infections leading to keratinising squamous metaplasia Drugs Phenacetin Cyclophosphamide Pelvic radiotherapy Staging of urachal tumours There is no AJCC staging system for tumours arising in urachal remnants, but they may be staged according to several proposed systems Urothelium Lamina propria Muscularis propria Perivesical fat Tis Ta T1 Staging of diverticula Muscularis propria is T3 absent; thus, there is no T2 Figur e 83.34 Staging of the primary tumour in bladder cancer. (Repr al . Staging of bladder cancer. Histopathology 2019; 74 (1): 112–34.) cancer. Type Frequency Transitional cell carcinoma

> 90% Squamous cell carcinoma 1–7% Adenocarcinoma 2% Rare: Melanoma, lymphoma, sarcoma, small cell <1% carcinoma, phaeochromocytoma Metastatic adenocarcinoma (colorectal, prostate, <1% kidney, ovary)

Postoperative mitomycin C instillation

Postoperative mitomycin C instillation

Approximately 30% of patients with NMIBC will experience early recurrence following initial TURBT and so an immediate bladder. This has been shown to reduce the risk of tumour recurrence by 12%. Risk stratification Based on the final histological grade and stage, the patient can be risk stratified. Those with multifocal high-grade T1 tumours with CIS are at highest risk of disease recurrence and progres sion to MIBC. For these patients, early radical cystectomy should be discussed as they are at high risk of tumour progression. An alternative is intravesical bacillus Calmette–Guérin (BCG) to reduce the risk of tumour progression. T hose with solitary low-grade Ta tumours have the lowest risk of recurrence and progression and so the management of this group consists of regular cystoscopic surveillance alone. For intermediate-risk tumours, a 6-week course of intravesical mitomycin C can be considered to reduce the risk of recurrence. Repeat TURBT For those with high-grade or T1 tumours, a repeat TURBT 2–6 weeks after initial TURBT should be performed to identify any residual tumour. Upstaging to MIBC is found in up to 40%. Intravesical BCG For high-risk tumours, intravesical treatment with immunother apy (BCG) has been shown to reduce the risk of progression to MIBC. The treatment is given weekly for 6 weeks, followed by a 3-weekly treatment ever y 6 months for 3 years. Side e ff ects include transient fever, dysuria, rarely BCG sepsis and BCG cystitis necessitating cystectomy .

Presentation

Presentation

Patients most commonly present with painless haematuria (in 85%). Storage LUTS of frequency , urgency , dysuria and recurrent UTI may be present. Rarely , patients may present

Urinary bladder staging Tis: urothelial carcinoma in situ Ta: non-invasive papillary urothelial carcinoma T1: invasive into lamina Tis propria Ta T1 T2a: invasive into inner T2a half of muscularis propria T2b T2b: invasive into outer T3a half of muscularis propria T3b T3a: microscopic invasion in perivesical soft tissue T4 T3b: macroscopic invasion in perivesical soft tissue T4: invasion into adjacent organs Discontinuous involvement of urethra is assigned a separate urethra stage per the urethral staging system oduced with permission from Magers MJ, Lopez-Beltran A, Montir oni R et

oedema in advanced cases.

Radiation cystitis

Radiation cystitis

Radiation cystitis is a common complication of pelvic radio therapy with incidence rates ranging from 23% to 80%. Radia tion treatment causes endothelial cell damage and perivascular fibrosis, resulting in ischaemia and obliterative endarteritis. Haematuria is more pronounced than that seen in BPS/IC. The end stage is a small, fibrotic b ladder with poor compliance and a risk of upper tract compromise, as for other chronic inflammatory bladder diseases. Emergency admission with haematuria requires resuscita tion, catheterisation and bladder washout and blood transfu sion as required. Cystoscopic management with fulguration or laser to bleeding vessels should be performed initially to stop bleeding. Intravesical GAG layer replacement therapies can be considered, and hyperbaric oxygen therap y has shown benefit in severe, refractory cases of haemorrhagic cystitis. Radiolog ical arterial embolisation can also be considered for refractory cases, but ischaemic complications occur in 10–63% (e.g. skin or bladder necr osis, gluteal paresis, perineal or buttock pain). Finally , urinary diversion with or without cystectomy can be performed for end-stage cases, but perioperative morbidity is almost 50% and mortality is 16%.

Special cases

Special cases

Genitourinary tuberculosis Genitourinary tuberculosis (GU-TB) caused by Mycobacterium tuberculosis can a ff ect any part of the urinary tract. Renal calci fication and ureteric strictures are typical in the upper urinary tract. In the bladder, initial manifestations include a red, oedematous bladder wall with ulceration and visible tubercles (yellow lesions with a red halo). This typically starts around the ureteric orifices and trigone . As the disease progresses, fibrosis and contraction of the bladder occur (‘thimble bladder’), as well as calcification and fistula formation (Figure 83.31 Patients may present with fevers, weight loss, night sweats, UTI symptoms or haematuria. Investigation may reveal a ster ile pyuria, and cystoscopy with biopsy will confirm the diagno sis. Three early mor ning urine samples for acid-fast bacilli or polymerase chain reaction of the urine can be used for diag nosis of TB infection. Cross-sectional imaging with a CT uro gram should be performed to evaluate the kidneys and ureter as they are likely to also be a ff ected. Treatment consists of antituberculous therapy with isonia zid, rifampicin, pyrazinamide and ethambutol. Severe bladder disease may r equire surgical treatment following completion of antituberculous therapy . Options include augmentation enterocystoplasty , cystectomy and orthotopic bladder substi tution, or ileal conduit urinary diversion. However, the choice of treatment will depend upon the concomitant upper tract involvement and patient preferences. Schistosomiasis Parasitic infection with the trematode Schistosoma haematobium endemic in Egypt, parts of Africa, Israel, Syria, Saudi Arabia, Iran, Iraq and the shores of China’s great lakes. The parasite penetrates the skin and travels to the liver (as schistosomules), where it matures. Adult trematodes migrate to vesical veins and lay eggs (containing miracidium larvae), which leave the body by penetrating the bladder and entering the urine. The active phase is when eggs are actively being laid, whereas the inactive phase is when the adult has died but there is an ongoing reac - tion to the remaining eggs. At the time of infection, a local inflammatory response leads to irritation of the skin (‘swimmer’s itch’). Acute fever (Katayama fever) may ensue at the onset of egg laying (3 weeks to 4 months after infection) with fever, lymphadenopathy , sple - nomegaly and eosinophilia. When the eggs are deposited in the bladder, the typical bladder symptoms of intermittent painless haematuria and terminal dysuria occur. Chronic infection can lead to a small, contracted, fibrotic bladder similar to that seen with tuberculous cystitis. Patients ar e at increased risk of devel - oping ureteric strictures, urethral strictures and squamous cell carcinoma of the bladder. Investigation with midday (to coincide with the time of maximum egg shedding) urine microscopy may show charac - teristic eg gs with terminal spines (Figure 83.32). Cystoscopy may show characteristic ‘sand y patches’ at the trigone (due to calcified dead ova with degeneration of the overlying urothe - lium), ulcera tion or papillomas. In advanced cases, carcinoma may be present. Bladder or rectal biopsies may identify eggs. -). - - - - s - -

Figure 83.32 Schistosoma haematobium eggs with terminal spines. (Reproduced with permission from Ray D, Nelson TA, Fu CL et al . Transcriptional pro /f_i ling of the bladder in urogenital schistosomiasis reveals pathways of in /f_l ammatory /f_i brosis and urothelial compromise. PLoS

Negl Trop Dis 2012; 6 (11): e1912.)

identified, serology (enzyme-linked immunosorbent assay [ELISA]) has high sensitivity and specificity . A CT urogram should be performed to assess for obstructive uropathy second ary to a scarred, contracted bladder. Treatment is with praziquantel 20 /uni00A0 mg/kg in two divided doses 4–6 hours apart. A small, contracted bladder may require reconstruction with augmentation enterocystoplasty , cystectomy and orthotopic bladder substitution, or ileal con duit urinary diversion. Summary box 83.5 Urinary tract infections /uni25CF /uni25CF

UTIs can be classi /f_i ed as uncomplicated or complicated Patients should undergo evaluation for TB and schistosomiasis if suspected based on the history or presence of persistent sterile pyuria

Spinal cord injury

Spinal cord injury

SCI often results in significant LUT dysfunction with a high risk of UTI/sepsis, renal function deterioration, renal and bladder calculi and autonomic dysreflexia. Urinary tract complications and renal failure were the leading causes of death in this population, but thorough evaluation and early definitive management have considerably improved urinary tract outcomes in patients with SCI. Immediately after SCI, a period of ‘spinal shock’ occurs in which there is a marked reduction in all spinal reflex activ ity below the level of the lesion. This results in an areflexic and acontractile b ladder; urinary retention lasts 6–12 weeks in complete SCI but may be shorter in incomplete lesions. Patients are managed with indwelling catheterisation or CISC during this phase . Return of function is characterised by spas ticity (detrusor overactivity and DSD) and should be managed as described under Urinary incontinence, Treatment Autonomic dysreflexia Autonomic dysreflexia is a sudden and exaggerated autonomic (primarily sympathetic) response to various stimuli in patients with SCI or spinal dysfunction above the cord level of T6–8 (the sympathetic outflow). Stimuli below the level of the lesion (commonly a distended bladder or rectum) lead to symptoms of headache, hypertension (varying from mild headache to seizures or cerebral haemorrhage) and flushing or sweating of the face and body above the level of the lesion. Autonomic dysreflexia is thought to occur as a result of an unopposed sympathetic response to noxious stimuli. Noci ceptive a ff erents elicit refle x sympathetic outflow , resulting in piloerection, sweating and arteriolar vasoconstriction, leading to hypertension. The peripheral vasoconstriction activates nating in the central nervous system to counteract the sympa - thetic outflow . The resulting vagal outflow causes bradycardia, but this parasympathetic signal is unable to transmit below the level of the spinal cord lesion. This leads to brad ycardia, vaso - dilatation and flushing above the level of the lesion, but hyper - tension and pale, cold skin due to ongoing vasoconstriction below the level of the lesion. Initial treatment involves sitting the patient upright, remov - ing any constricting clothing and identifying and removing the source of stimulation (urinary retention, blocked catheter, loaded rectum). Regular blood pressure monitoring should be performed; if the systolic blood pressure remains elevated (>150 /uni00A0 mmHg), patients should be treated with fast-acting anti - hypertensives such as sublingual nifedipine or glyceryl trini - trate. -

Suprapubic catheterisation

Suprapubic catheterisation

Suprapubic catheterisation (SPC) carries a small but significant risk of bowel injury , especially in those who have undergone - previous abdominal or pelvic surgery , and so should be performed under ultrasound and cystoscopic guidance. SPC should be considered for those who require long-term catheter - isation, as it is often more comfortable with no risk of urethral trauma compared with long-term urethral catheterisation, as well as for those in whom urethral catheterisation is not suitable or possible. Contraindications to SPC insertion include known or suspected bladder carcinoma, abdominal wall infection at

(c) (d) (b) standard urethral catheter with an in /f_l atable (d) coudé tip catheter.

the site of insertion, uncorrected coagulopathy or the presence of a vascular graft in the suprapubic region. The procedure requires infiltration of local anaesthesia (1% lidocaine) to the skin and fascia of the suprapubic region, two fingerbreadths above the pubic symphysis in the midline. This procedure should only be performed in those with a palpable bladder. A Seldinger technique of insertion is the safest for percutaneous insertion (Figure 83.26). A long needle is inserted in a perpendicular direction into the bladder and aspiration of clear urine confirms correct entry . A guidewire is placed through the needle, and the needle removed. A small skin incision at the site of the guidewire allows a trocar to pass over the guidewire and into the bladder, thereby dilating the tract. Through this trocar, a 16Fr catheter is placed and the balloon inflated. The trocar is removed and the catheter bag attached. If the bladder cannot be confidently identified, or in those with extensive abdominal or pelvic surgery , an open cystotomy should be performed to safely enter the bladder and ensure that there is no bowel in the path of the catheter.

18G hypodermic needle – 12 cm long Dilator and peelable sheath Low-profile silicone Foley catheter Balloon capacity 5–10 mL Set includes 2 × 10-mL syringe and a scalpel Figure 83.26 A suprapubic ‘Seldinger’ catheter kit.

Surgical treatment of bladder diverticula

Surgical treatment of bladder diverticula

Congenital bladder diverticula should only be treated if symptomatic, or if there is concern regarding malignant transformation. Bladder outlet obstruction should be excluded by flow rate or urodynamic studies prior to any surgical inter - vention, and this should be treated prior to consideration of bladder diverticulectomy . Even large diverticula do not require there is no evidence of bladder outlet obstruction treatment if and the patient is asymptomatic with none of the above complicating factors. Surgical excision of a bladder diverticulum can be per formed through an open approach (Pfannenstiel or low midline abdominal incision) or through minimally invasive (laparoscopic or robotic) techniques. Depending on the prox imity of the neck of the diverticulum to the ureteric orifice patients should be counselled about the possible need for con epair can be performed comitant ureteric reimplantation. R purely extravesically , or through a combined extravesical and h (Figure 83.8). intravesical approac Summary box 83.1 Bladder diverticula /uni25CF /uni25CF /uni25CF

Figure 83.7 Bladder diverticulum seen cystoscopically. Can be congenital or acquired Complications include infection, stones, renal obstruction and rarely malignancy Bladder outlet obstruction should be identi /f_i ed and, if present, treated prior to bladder diverticulectomy

TRACT DYSFUNCTION

TRACT DYSFUNCTION

NLUTD refers to bladder and/or urethral sphincteric disorders that result from neurological lesions. Discrete neurological lesions will a ff ect LUT storage and voiding in a consistent manner depending on the site of the lesion, the nature of the lesion (destructive, inflammatory , irritative) and the extent of the lesion (complete or incomplete). Neurological lesions can be classified based on location as suprapontine, spinal (infrapontine–suprasacral) and sacral/infrasacral. Each has characteristic clinical and urodynamic features (Figure 83.22). Suprapontine lesions (e.g. cerebrovascular accident, Parkinson’s disease, brain injury) lead to storage LUTS owing to loss of inhibition from higher brain centres. This results in neurogenic detrusor overactivity , but since local sacral micturition refluxes are preserved the voiding phase is intact. Spinal lesions (e.g. spinal cord injury [SCI], myelitis, disc herniation) can have the most serious impact as they can lead to both detrusor overactivity and sphincteric overactivity (DSD), resulting in high-pressure voiding, which risks deterioration in renal function. Furthermore, patients with spinal lesions above the T6 spinal cord level are at risk of developing autonomic dysreflexia (see Autonomic dysrefl exia). Sacral and infrasacral lesions are relatively safe as they result in low-pressure underactive bladder and/or sphincter function. Patients predominantly

Over

active : insignificant PVR urine volume : detrusor overactivity Normoactive Over

active : PVR urine volume usually raised : detrusor overactivity,

detrusor–sphincter Overactive : PVR urine volume raised Under

Under- : hypocontractile or active active Normoactive Underactive from Panicker JN, Lancet Neurol 2015;

bladder emptying) and SUI. The aims of treatment in NLUTD are to: 1 preserve renal function by ensuring low-pressure storage and voiding; 2 achieve continence; 3 prevent UTI. Treatment of detrusor overactivity and poor compliance in order to attain low-pressure storage is achieved through the conservative, medical and sur gical treatment pathways outlined above for the treatment of UUI. Treatment of sphincteric overactivity or DSD to attain low-pressure voiding is typically achieved by CISC, although sphincteric overactivity can be treated with intrasphincteric BTX-A, sacral neuromodulation, SNS or direct sphincterotomy . If these methods are ine ff ective or unsuitable for the patient, continent diversion (appendico vesicostomy) can be performed to prevent high-pressure voiding. Detrusor underactivity is managed with CISC and sphincteric underactivity is treated as described above for SUI. Several factors should be considered when managing patients with NLUTD . These relate to patient factors (body habitus, hand function, motivation and level of compliance, mental status, values and preferences, support network) and neurological disease factors (prognosis).

The micturition cycle

The micturition cycle

The key characteristics of the two phases of the micturition cycle are (Figure 83.2): 1 Urinary storage (filling): /uni25CF low pressure (normal compliance) – dependent on viscoelastic properties of the bladder wall and lack of parasympathetic input to the detrusor; /uni25CF normal sensation (absence of pain or urgency); tivity); /uni25CF a closed bladder outlet to enable continence – depen - dent on the sympathetic reflex, which increases outlet resistance (α -adrenergic stimulation), inhibits detrusor contractility (through inhibitory e ff ect on parasym - pathetic gang lia) and reduces bladder smooth muscle tension (β -adrenergic stimulation). 3 2 Urinary emptying (voiding): /uni25CF coordinated detrusor contraction of appropriate strength and duration to enable complete bladder emptying – dependent on inhibition of the spinal sym - pathetic reflexes and activation of parasympathetic e ff erent pathways to the bladder; /uni25CF relaxation of the bladder neck and external urethral sphincter – dependent on inhibition of spinal sympa - thetic reflexes. Disorders of the LUT can therefore be related to failure to store urine (due to the bladder or the outlet) or failure to empty (due to the bladder or the outlet).

(a) Pontine storage centre (+)

- Contracts bladder outlet – Inhibits detrusor R Pelvic Bladder nerve Pudendal External nerve urethral sphincter Figure 83.2 The micturition cycle and its neurological control. system and pudendal nerve activity leads to contraction of the bladder neck and external sphincter, and relaxation of the detrusor muscle. /uni00A0 (b) Urine voiding re /f_l exes. During voiding, the pontine micturition centre stimulates detrusor contraction through activation of the parasympa thetic out /f_l ow to the bladder, with inhibition of sympathetic out /f_l ow to the bladder neck and external urethral sphincter. PAG, periaqueductal grey; R, receptor. (Reproduced with permission from Fowler CJ, Grif /f_i ths D, de Groat WC. The neural control of micturition. 2008; 9 (6): 453–66.) PAG (b) Pontine micturition centre Hypogastric nerve (–) Pelvic nerve R (+) Bladder (–) (+) (a) Urine storage re /f_l exes. During bladder /f_i lling, increased sympathetic nervous

Treatment

Treatment

The cause of the stone should be sought and treated; this may include bladder outlet obstruction, incomplete bladder emptying in patients with neurogenic bladder dysfunction or the presence of a foreign body that should be excised simulta neously (Figure 83.29). The majority of stones can be managed endoscopically with a stone punch, ultrasound lithotripsy or holmium laser lithotripsy (Figure 83.30). In those in whom urethral access is not possible (e.g. reconstructed LUT), percutaneous cystolitho - tomy can be performed using a similar technique to percuta - neous nephrolithotomy . The open approach (open cystolithotomy) is reserved for those with very large bladder stones that cannot be treated with endoscopic means.

Figure 83.29 Stone on a vaginal sling that had eroded into the bladder. Figure 83.30 An endoscopic ultrasound probe, which is used to fragment bladder or kidney stones.

Treatment

Acute UTI should be treated with an appropriate antimicro - bial agent based on local antibiograms and resistance patterns. Following treatment of an acute UTI, non-antimicrobial measures to prevent future UTI should be considered (e.g. increasing fluid intake , probiotics, methenamine hippurate, - d-mannose, vaginal oestrogen therapy for postmenopausal women). Episodes of rUTI can be treated with antimicro - bial prophylaxis, postcoital antimicrobial use for those with sex-linked infections or intermittent self-start antimicrobial therapy .

Figure 83.31 Retrograde cystography showing a small ‘thimble’ bladder due to tuberculosis.

Types of catheter

Types of catheter

Catheters can be classified based on their size (French scale), number of channels or composition (latex-coated or silicone) (Figure 83.25). Size The French scale refers to the external diameter of the cathe ter, and 1Fr is 0.3 /uni00A0 mm in diameter. The standard catheter size for uncomplicated urethral catheterisation in women is 12Fr or 14Fr, and in men 16Fr. In patients with urethral stricture, a smaller size catheter should be consider ed. In cases of haema turia with ‘clot retention’, a 22Fr catheter is typically used to aid drainage of thick clots. Coudé tip catheters have a curved tip to allow easier passage past an enlarged prostate. Number of channels Single-channel catheters consist of a single drainage channel only and are used for CISC as these catheters do not require an Frederic Eugene Basil Foley , 1891–1966, urologist, Anker Hospital, St Paul, MN, USA. of two channels – a drainage channel and a channel for inflation of a balloon at the tip, which allows the catheter to be retained in the bladder. This is the standard catheter used for uncomplicated cases of urinary retention. The ‘three-way’ catheter has an additional channel for bladder irrigation and is used for patients with haematuria and ‘clot retention’ and following transurethral prostate or bladder surgery .

(a) (b) Figure 83.25 Types of urethral catheter. (a) Single-lumen catheter used for self-catheterisation; balloon retention mechanism; (c) a three-way catheter with an extra channel for irrigation;

URINARY INCONTINENCE

URINARY INCONTINENCE

Urinary incontinence refers to the involuntary leakage of urine. It can be classified into several di ff erent subtypes based on the circumstances leading to episodes of leakage. /uni25CF Stress urinary incontinence (SUI) refers to invol - untary leakage on e ff ort or exertion, or on sneezing or coughing. /uni25CF Urgency urinary incontinence (UUI) refers to in - voluntary leakage accompanied by or immediately preced - - ed by urgency (a sudden compelling desire to pass urine which is di ffi cult to defer). /uni25CF Mixed urinary incontinence (MUI) refers to invol - untary leakage associated with urgency and also with exer - - tion, e ff ort, sneezing or coughing. /uni25CF Continuous urinary incontinence refers to the continuous involuntary loss of urine (this warrants investi - gation for anatomical pathology such as ectopic ureter or fistula from the ureter, bladder or urethra to the vagina). - /uni25CF Nocturnal enuresis refers to involuntary leakage during sleep. /uni25CF Incontinence associated with chronic urinary retention refers to leakage in conditions where the blad - der does not empty completely . /uni25CF Functional incontinence refers to leakage that results from an inability to reach the toilet because of cognitive, functional or mobility impairments in the presence of an intact LUT system. /uni25CF Overactive bladder (OAB) refers to symptoms of urgency with or without UUI, usually with frequency and nocturia. It can be neurogenic (secondary to a neurological condition) or idiopathic (without identifiable cause). Urinary incontinence can occur in isolation, but more commonly is associated with other LUTS. The International Continence Society classifies LUTS based on the phase of the micturition cycle in which they occur, as storage (frequency , urgency , nocturia), v oiding (hesitancy , slow stream, inter - mittency , straining to void, splitting of the stream, terminal dribble) and post micturition (feeling of incomplete emptying, postmicturition dribble).

URINARY RETENTION

URINARY RETENTION

Urinary retention is defined as the inability to pass urine despite persistent e ff ort. It can be classified as acute (painful inability to pass urine with relief of pain on catheterisation) or chronic (painless, elevated residual volume after passing urine). The causes of urinary retention in men and women are given in Table 83.9 . Heinrich Martius , 1885–1965, German surgeon, described a labial flap of bulbocavernosus muscle (Martius modified labial fat pad) in 1928. Clare Juliet Fowler , contemporary , Professor of Uroneurology , National Hospital for Neurology and Neurosurgery , London, UK. /uni25CF Summary box 83.4 Urinary tract fistulae /uni25CF /uni25CF /uni25CF /uni25CF

Women Urethral Stricture Diverticulum Meatal stenosis Carcinoma Prolapse Extrinsic compression Paraurethral cyst Pelvic mass (e.g. large /f_i broids) Gynaecological malignancy Iatrogenic Anti-incontinence surgery Urethral reconstruction Idiopathic high-tone non-relaxing external urethral sphincter (Fowler’s syndrome) Dysfunctional voiding Primary bladder neck obstruction The principles of surgical repair of VVF are: Adequate exposure of the /f_i stula tract Tension-free, watertight, multilayer closure with non- overlapping suture lines Interposition with a well-vascularised /f_l ap Urinary tract drainage postoperatively to allow healing

URINARY TRACT FISTULAE

URINARY TRACT FISTULAE

A fistula is an abnormal or surgically made passage between a hollow or tubular organ and the body surface, or between two hollow or tubular organs. The most common cause of urinary tract fistulae in less economically developed countries is traumatic labour; in well-resourced countries, fistulae are most commonly iatrogenic, resulting from pelvic surgery (hysterectomy accounts for the majority) or radiotherapy , or they can be due to advanced pelvic malignancy , inflammatory conditions (e.g. tuberculosis [TB]), congenital disorders or foreign body erosion.

URINARY TRACT INFECTION

URINARY TRACT INFECTION

UTI is the inflammatory response of the urothelium to bacte - rial invasion, usually associated with bacteriuria (the presence of bacteria in the urine) and pyuria (the presence of white blood cells in the urine). Pyuria in the absence of bacteriuria (sterile pyuria) indicates an inflammatory response that may still be related to UTI or that ma y be a response to another pathology such as fastidious organisms (e.g. TB, gonorrhoea), in situ (CIS) bladder stones or other inflammatory carcinoma uncomplicated (occur - conditions. UTIs can be classified as ring in a healthy patient with a structurally and functionally normal urinary tract) or complicated (occurring in a patient with an anatomical or functional urinary tract abnormality , in an immunocompromised patient or with more virulent or resistant bacteria). Factors that suggest a complicated UTI are shown in Table 83.12 . An isolated UTI is one in which there - has been an interval of at least 6 months between infections. A recurrent UTI (rUTI) is defined as ≥ 2 episodes in 6 months or ≥ 3 episodes in 12 months. Infections can also be classified based on their site (urethritis, prostatitis, cystitis, pyelonephri - tis). This chapter focuses on cystitis. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Half of all women have been estimated to experience a UTI in their lifetime, and up to 50% of these will have recur rent infection within the following 6-month period.

tract infection. Patient Functional or anatomical abnormality of the factors urinary tract Sex (male gender) Age (postmenopausal) Pregnant Immunosuppressed (e.g. diabetes, transplant, steroids) Indwelling catheter Bacterial Increased virulence (hospital-acquired infection) factors Antimicrobial resistance (recent antibiotic use)

Urachal anomalies

Urachal anomalies

Urachal anomalies are often detected after birth with symptoms of umbilical discharge or bleeding (Figure 83.10). However, asymptomatic urachal anomalies may be incidentally found on abdominal imaging in adults. There are four principal

(c) (d) (e) (c) urachal cyst; (d) urachal sinus; (e) urachal diverticulum.

eration of the urachus, resulting in a connection between the bladder and umbilicus. A urachal cyst occurs when a portion of the urachus does not obliterate but there is no connection between the bladder and umbilicus. A urachal sin us occurs when the urachus fails to obliterate close to the umbilicus, resulting in a blind-ending tract from the umbilicus into the urachus. A urachal diverticulum occurs when the urachus fails to obliterate close to the bladder, resulting in a blind-ending tract from the bladder into the urachus. Clinical features Depending on the anomaly , symptoms include: /uni25CF umbilical discharge or bleeding; /uni25CF enlarged or oedematous umbilicus; /uni25CF lower abdominal pain; /uni25CF UTI; /uni25CF haematuria. Investigation In children, ultrasound or micturating cystography will demon strate a patent urachus. In adolescents and adults, MRI will clearly demonstrate the anomaly (Figure 83.11). If patients present with UTIs or haematuria, thorough investigation with r enal tract imaging, cystoscopy and postvoid residual measure ment should be performed to exclude other more common causes for these symptoms. Treatment Urachal anomalies have a small risk of malignant trans formation to adenocarcinoma, with high mortality rates. Complete surgical excision is therefore recommended for both performed through open or minimally invasive (laparoscopic or robotic) approaches. Cystoscopy and insertion of a small catheter into the patent urachal tract can aid identification during surgery . The principle is to excise the urachus with a wide bladder cu ff . The bladder is then closed in two layers. The urachus can be circumscribed and removed at the umbilicus, leaving the umbilicus intact for optimal cosmesis.

Figure 83.11 Magnetic resonance imaging scan showing a urachal cyst (arrow).

Urethral catheterisation

Urethral catheterisation

1 Aseptic technique – handwashing, sterile gloves, sterile catheter pack. 2 Clean urethral meatus with antiseptic solution. 3 Instil lidocaine gel into the urethra and hold for 2–3 min - utes. In men, the penis should be held perpendicular and taught. In women, the labia should be held apart to pro - vide adequate exposure of the urethral meatus. 4 The catheter should be inserted as far as the ‘hilt’ of the catheter and should pass freely (the type of catheter is dependent on the indication). If there is any resistance catheterisation should be stopped and assistance sought. The patient may require a coudé tip catheter if the obstruction is thought to be a large prostate, or a cystos - copy to negotiate a false passage or stricture. 5 The position in the bladder is confirmed with the drain - age of clear urine and the balloon should be inflated with 10 /uni00A0 mL of sterile water. The catheter bag should then be attached. 6 Details regarding the type and size of catheter, and resid - ual volume, should be clearly recorded. -

Venous drainage

Venous drainage

V esical plexuses on the lateral and inferior surfaces of the bladder drain into the internal iliac vein (the prostatic plexus in males and the vaginal plexus in females are continuous with the vesical plexus).

Vesicovaginal fistulae

Vesicovaginal fistulae

VVF is the most common urinary tract fistula. In developing countries, obstetric fistula account for the majority of cases. Lack of adequate prenatal care, younger age at first marriage, short stature, low socioeconomic status and illiteracy are risk factors for developing obstetric fistulae. These fistulae are due - to prolonged obstructed labour resulting in ischaemic pressure necrosis to the anterior vaginal wall, bladder and urethra, and large areas of the bladder neck and urethra may be involved. Concomitant rectovaginal fistulae may also be present, making these fistulae very complex to manage. In developed countries, - iatrogenic VVF most commonly occurs after hysterectomy , usually thought to be due to an unrecognised bladder injury . near the vaginal cu ff . Other mechanisms include diathermy injury resulting in delayed tissue necrosis or a suture placed through the bladder and vaginal wall during closure of the vaginal cu ff . Abdominal hysterectomy is three times more likely to result in fistula than vaginal hysterectomy , although the overall rate of VVF after hysterectomy is low at 0.1–4%. Clinical features The most common presenting symptom is constant urinary leak from the vagina. This may be intermittent in cases of very small fistulae, and so other causes of urinary incontinence (SUI, UUI) must be excluded. Post-hysterectomy VVF may be - recognised in the first few days after surgery , or 1–3 weeks later after catheter removal. Post-irradiation VVF may not manifest until years later. Physical examination may demonstrate the fistula site, typically on the anterior vaginal wall at the vaginal cu ff , and leakage of urine may be seen. Instillation of blue dye into the bladder may aid visual identification of the fistula site. Investigation 1 Cross-sectional imaging with CT urogram, MRI with gadolinium contrast or cystogram will aid diagnosis of the fistulous tract and exclude concomitant ureteric injury (Figure 83.23). 2 Cystoscopy , bilateral retrograde ureteropyelography and examination under anaesthesia should be performed to assess the fistula site, location, size, proximity to ure teric orifices, vaginal size, depth and mobility . This will aid surgical planning and help to determine whether an abdominal or vaginal approach will be most suitable. Fur thermore, biopsy of the fistula tract to exclude recurrent malignancy can be performed in cases of prior history of pelvic malignancy . 3 ‘Three-swab’ test . This investigation can be per formed in cases where fistula is suspected but cannot be identified on the investigations above. Three numbered gauze swabs are placed into the vagina, with swab number 1 placed most pro ximally , swab number 2 in the middle and swab number 3 most distal in the vagina. A blue dye is then instilled into the bladder through a catheter, and the catheter removed. Blue staining of swab 1 or 2 suggests VVF whereas blue staining of swab 3 suggests a urethra– /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF vaginal fistula or SUI. If swab 1 is wet but not stained blue, - this suggests the presence of a ureterovaginal fistula. Treatment Conservative treatment with urethral catheter drainage is - rarely successful, but in certain situations (e.g. very small VVF in the absence of radiotherapy) may be warranted for an initial 2- to 6-week period. If this fails to heal the fistula surgical treatment is required. The approach can be vaginal, trans- abdominal (open or minimally invasive) or a combination of both (Figure 83.24). Principles of surgical repair are shown in Table 83.8 . Vaginal repair relies on adequate exposure of the fistulous opening. The fistula is circumscribed, and the

bladder

Figure 83.23 Computed tomography scan showing a vesicovaginal /f_i stula from the posterior wall of the bladder to the vagina. Figure 83.24 Transabdominal approach showing a vesicovaginal /f_i stula (VVF) on the posterior wall (forceps in VVF). White ureteric catheters have been inserted into the ureteric ori /f_i ces and there is a catheter in the urethra. TABLE 83.8 Principles of surgical repair of a vesicovaginal /f_i stula. Adequate exposure of the /f_i stula tract and debridement of ischaemic tissue Adequate separation of involved organs Watertight closure, multilayer closure, tension-free, non- overlapping suture lines Use of well-vascularised tissue /f_l aps (omentum, peritoneum, Martius labial fat pad) Adequate postoperative urinary drainage Treatment and prevention of infection Meticulous haemostasis

/uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF β /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF side is adequately separated from the vaginal side. The bladder is closed in two layers and a Martius labial fat pad is then harvested to cover the fistula. The vaginal wall is then closed, and a labial drain is placed for 1–2 days. Abdominal repair is typically performed transvesically . The bladder is opened, and the fistulous site identified and circumscribed to separate it from the vaginal wall. If the ureteric orifices are in close proximity , then ureteric catheters are placed intraoperatively . The vaginal wall is closed and then the bladder is closed in two layers after insertion of omentum or peritoneum between the fistula margins. The patient is discharged with a urethral catheter in situ ; this is typically removed 3 weeks later after a pericatheter urethrogram confirms absence of leak.

Mechanism Men Anatomical bladder outlet Benign prostatic obstruction obstruction Malignant enlargement of prostate Bladder neck obstruction Urethral stricture Urethral rupture (e.g. following pelvic fracture) Functional bladder outlet Idiopathic high-tone non-relaxing external obstruction urethral sphincter Detrusor underactivity Pelvic surgery Peripheral neuropathy Diabetic cystopathy Secondary to longstanding bladder outlet obstruction Neurological disease Detrusor–sphincter dyssynergia (any cause of suprasacral spinal cord disease) causing obstructed voiding Sacral nerve lesion or cauda equina causing detrusor underactivity Drugs Antimuscarinics or -agonists 3 Sympathomimetic drugs Anaesthetic agents Opioids Transient causes Following spinal or general anaesthesia Pain Faecal impaction due to constipation Blood clot secondary to haematuria

cycle

cycle

Storage phase The storage phase of the micturition cycle requires relaxation of the detrusor to ensure low-pressure filling, and contraction of the smooth and striated muscle of the bladder neck, urethra and external urethral sphincter to ensure continence. The higher centres in the cortex receive low-intensity a ff erent signals during bladder filling, which in turn induces the PMC to inhibit micturition by inhibiting parasympathetic innervation (result ing in detrusor relaxation) and activating somatic innervation (resulting in closure of the bladder outlet). Glutamate is the principal e ff erent neurotransmitter involved in activating the pudendal nerve through Onuf ’s n ucleus. Detrusor relaxation and bladder outlet contraction are achieved through sympa thetic β - noradrenergic activity , resulting in direct relaxation 3 of the detrusor smooth muscle; inhibition of parasympathetic ganglia, resulting in indirect relaxation of the detrusor smooth muscle; and α - noradrenergic activity , resulting in contraction 1 of the smooth muscle of the bladder neck and urethra. Further more, somatic cholinergic activity results in contraction of the striated external urethral sphincter. Voiding phase The voiding phase of the micturition cycle requires coordi nated detrusor contraction and relaxation of the bladder outlet to ensure complete bladder emptying. When the desire to void is strong enough, the higher centres in the cortex receive high-intensity a ff erent signals from the bladder, which in turn switc hes the PMC to ‘voiding’ mode. The PMC then activates micturition by activating parasympathetic nerves and inhibiting somatic nerves (by cessation of the glutamate e ff ect on Onuf ’s nucleus). This is achieved through parasympathetic cholinergic activity via M3 receptors, resulting in detrusor contraction, and central inhibition of somatic and sympathetic nerves, resulting in relaxation of the bladder outlet.

diverticula

diverticula

Bladder diverticula can be congenital or acquired (secondary to infravesical bladder outlet obstruction). Acquired bladder diverticula are most commonly seen in adult men with benign - prostatic obstruction; acquired diverticula can less commonly be seen in children with infravesical obstruction (e.g. secondary to posterior urethral valves) or in children with neurogenic bladder associated with detrusor–sphincter dyssynergia (DSD). Primary congenital bladder diverticula develop as a herni - ation of bladder mucosa through a congenital muscular defect between the intravesical ureter and the roof of the ureteral hiatus, the so-called ‘Hutch’ diverticulum. Congenital divertic - ula are therefore typically located in the vicinity of the ureteric orifice and may be associated with vesicoureteric reflux (VUR), both of which are thought to occur as a r esult of inadequate development of the musculature of the bladder wall or Wald - eyer’s fascia around the portion of the intravesical ureter, at -). - , Berlin, Germany . Waldeyer’s fascia is a layer of fascia that

Allantois Future bladder Urorectal septum Urogenital sinus Cloaca Anorectal canal Figure 83.3 Partitioning of the cloaca to form the urogenital sinus and anorectal canal. (Redrawn with permission from Wein AJ, Kavoussi LR, Partin AW, Peters CA. Campbell-Walsh

urology , 11th edn. Phila

delphia, PA: Elsevier, 2016: 2834.)

the embryological junction of the ureteric bud and urogenital sinus. As opposed to acquired diverticula, intravesical pressures in those with congenital diverticula are not elevated and so the bladder is generally thin walled, without trabeculation or mul tiple diverticula. In adults with acquired diverticula, the intra vesical pressures are often elevated, and the bladder is thick walled with trabeculations and multiple diverticula. The raised intravesical pressure causes the lining between the inner layer of hypertrophied muscle to protrude, f orming multiple sac cules. If a saccule is forced through the bladder wall, it becomes a diverticulum. The diverticulum is made up of mucosa with very few , if any , muscle fibre cov erings. As a result, it does not contract to empty and therefore holds residual urine, which can lead to the complications described below (Figure 83.4 Clinical features Small congenital bladder diverticula are often asymptomatic. Haematuria (due to infection, stone or tumour) is a symptom in about 30%. Common symptoms or complications include: /uni25CF recurrent urinary tract infections (UTIs); /uni25CF bladder stones (Figure 83.5); /uni25CF urinary retention – due to extrinsic compression of the bladder outlet by a large diverticulum; /uni25CF hydronephrosis – due to extrinsic ureteral compression by a large diverticulum; /uni25CF neoplasm (Figure 83.6) – account for 1% of all bladder tumours; the lack of a muscular layer to the diverticulum a ff ects pathological staging of bladder tumours as there is, by definition, no T2 stage. Therefore, any invasion beyond the lamina propria in a bladder diverticular tumour should be staged as T3. - - -).

Figure 83.4 Cystogram showing a large bladder diverticulum. Figure 83.5 Magnetic resonance imaging scan showing a large left- sided bladder diverticulum containing stones (arrow). Figure 83.6 Computed tomography scan

showing a bladder divertic

ulum containing soft-tissue material consistent with tumour (arrow).

α -adrenoceptor antagonists

α -adrenoceptor antagonists

1 The α -adrenoceptor antagonists are commonly used to 1 improve voiding lower urinary tract symptoms (LUTS) in men. Mechanism of action The α -receptors are densely prevalent at the bladder base, 1 bladder neck and proximal urethra in males, and antagonism of these receptors inhibits sympathetic-mediated contraction of the bladder outlet, thereby reducing outlet resistance.

β -adrenoceptor agonists

β -adrenoceptor agonists

3 β -adrenoceptor agonists are relatively new pharmacological 3 agents that are used for the treatment of overactive bladder and neurogenic LUT dysfunction. Mechanism of action Similar to antimuscarinics, the β -agonists are thought to 3 exert their principal e ff ects during the storage phase of the β β /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF micturition cycle by reducing bladder a ff erent activity . It is also thought that activation of β -adrenoceptors may downregulate 3 acetylcholine release, resulting in inhibition of parasympa - thetic activity .

-agonist -receptor (+) G-protein (+) Adenylyl cyclase ATP cAMP (+) 2+ Ca Actin and myosin SR Smooth muscle relaxation (b) smooth muscle relaxation. ATP , adenosine triphos

, phosphatidylinositol 4,5-bisphosphate; PKC, protein kinase 2 Common side effects Dry mouth Dry eyes Blurred vision Constipation Cognitive decline Dizziness (postural hypotension) Cardiac arrhythmia Increased blood pressure Headaches Urinary tract infection Nasal congestion Dizziness (postural hypotension) Ejaculatory dysfunction Headache Nasal congestion