

4 UROLOGIC CONSIDERATIONS FOR THE GENERAL SURGEON

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An understanding of the urogenital system is a necessary component of general surgical education. Specifically, surgeons must be aware of the anatomic, physiologic, and pathologic features of urologic processes. Accordingly, in this chapter, we review the key features of genitourinary anatomy and discuss urologic problems that are commonly encountered by the general surgeon, including urologic trauma, iatrogenic injuries to the urogenital system, urologic malignancies, and benign prostatic hyperplasia (BPH).

Anatomic Considerations

KIDNEY AND URETER

The kidneys are obliquely placed on either side of the spine, deep within the retroperitoneum,¹ with the upper poles slightly more medial than the lower poles. On top of each kidney sits an adrenal gland, and these two structures are jointly surrounded by a perinephric fascial layer known as Gerota's fascia (an important anatomic structure that can act as a barrier to contain renal pathologic processes). The anterior and posterior leaves of Gerota's fascia, which extend anterior and posterior to the kidney, become fused around the kidney on three sides: laterally, medially, and superiorly. Inferiorly, however, Gerota's fascia remains an open potential space, containing the ureter and the gonadal vessels on either side. Gerota's fascia is surrounded by retroperitoneal fat.

A clear understanding of the position of the kidneys in the retroperitoneum is vital for surgical exploration. Generally, the right kidney is situated slightly lower than the left kidney, as a consequence of the mass effect of the liver; however, the positions of the kidneys vary somewhat with respiration. Posteriorly, the right kidney and the left have similar relations with adjacent structures. The diaphragm and the pleural reflections cover the upper posterior aspect of each kidney, and the 12th rib crosses the upper pole. On the left side, the 11th rib is directly posterior to the upper aspect of the kidney. The quadratus lumborum and the transversus abdominis aponeurosis cover the posterior surface, and the renal hilum lies against the psoas muscle. Anteriorly, the left kidney is in contact with the adrenal, the spleen, the stomach, the pancreas, the jejunum, and the splenic flexure of the colon [see Figure 1]. The anterior surface of the right kidney is covered to a large extent by the liver. The inferior portion of the right kidney is covered by the hepatic flexure, and the hilum is covered by the duodenum. It is important to recognize the planes between Gerota's fascia, the peritoneum, and the retroperitoneal organs.

Each kidney is supplied by a renal artery and drained by a renal vein. The renal arteries typically branch off the aorta just below the superior mesenteric artery at the level of the second lumbar vertebra, and the renal veins meet the vena cava at the same level. The left renal vein passes in front of the aorta and lies most anteriorly in the renal hilum. Behind the vein lies the renal artery, and the renal pelvis lies most posteriorly. Although, in general, there is one renal artery and one renal vein for each kidney, anatomic variations are common. The most common variation (occurring in 23% to 49% of persons)

is the presence of supernumerary renal arteries arising from the lateral portion of the aorta.²⁻⁷ Whereas there is extensive collateral circulation for the renal veins, the renal arteries are end arteries. Consequently, injury to the main renal artery or an accessory artery typically results in the loss of renal parenchyma. On the right side, the adrenal and gonadal vein drain directly into the vena cava; on the left side, these vessels drain into the renal vein [see Figure 2].

The renal collecting system includes the minor calices, the major calices, the renal pelvis, and the ureter. The ureter connects the renal pelvis to the bladder, and its role is to propel urine toward the bladder by means of peristalsis. The normal adult ureter is 22 to 30 cm long, and it exhibits three distinct narrowings as it travels through the abdomen and the pelvis: one at the ureteropelvic junction, one at the pelvic brim as it crosses over the iliac vessels, and one at the ureterovesical junction. For the purposes of surgical or radiographic description, the ureter is commonly divided into discrete segments. In surgical terms, the portion of the ureter that runs from the renal pelvis to the iliac vessels is the abdominal ureter, and the portion that runs from the iliac vessels down to the bladder is the pelvic ureter. In radiographic terms, the portion of the ureter that runs from the renal pelvis down to the upper border of the sacrum is the proximal ureter, the portion that runs from the upper border of the sacrum to the lower border is the medial ureter, and the portion that runs from the lower border of the sacrum down to the bladder is the distal ureter.

The ureter receives its blood supply from multiple branching vessels along its course. The proximal ureter is supplied medially by branches from the renal artery, the medial ureter is supplied medially by branches from the gonadal artery and the aorta, and the distal ureter is supplied laterally by branches from the common and internal iliac arteries. The arterial vessels then course longitudinally within the periureteral adventitia in an anastomosing plexus that can be extensively mobilized so long as it remains intact.

Posteriorly, the ureter runs along the psoas muscle to the point where the muscle crosses over the iliac vessels near the bifurcation of the common iliac arteries. Anteriorly, the right ureter is in contact with the ascending colon, the cecum, the appendix, the terminal ileum, and the corresponding mesenteries. The left ureter is in contact with the descending colon, the sigmoid colon, and the corresponding mesenteries. It is important to recognize that when these anterior structures are reflected during operation, the ureter tends to adhere to them rather than remain adherent to the psoas muscle. In women, specific attention must be paid to the status of the ureter at the pelvic brim, where it may be in contact with the fallopian tube and the ovary. Attention should also be paid to the uterine arteries, which cross anterior to the ureters near the cervix.

BLADDER

The bladder is a hollow organ that stores and expels urine. The normal adult bladder has a capacity of 400 to 500 ml. When empty, the bladder rests in the pelvis behind the pubic symphysis. As it fills, the bladder rises above the pubic bone, and if it is quite distended, it may be palpated in the lower abdomen.

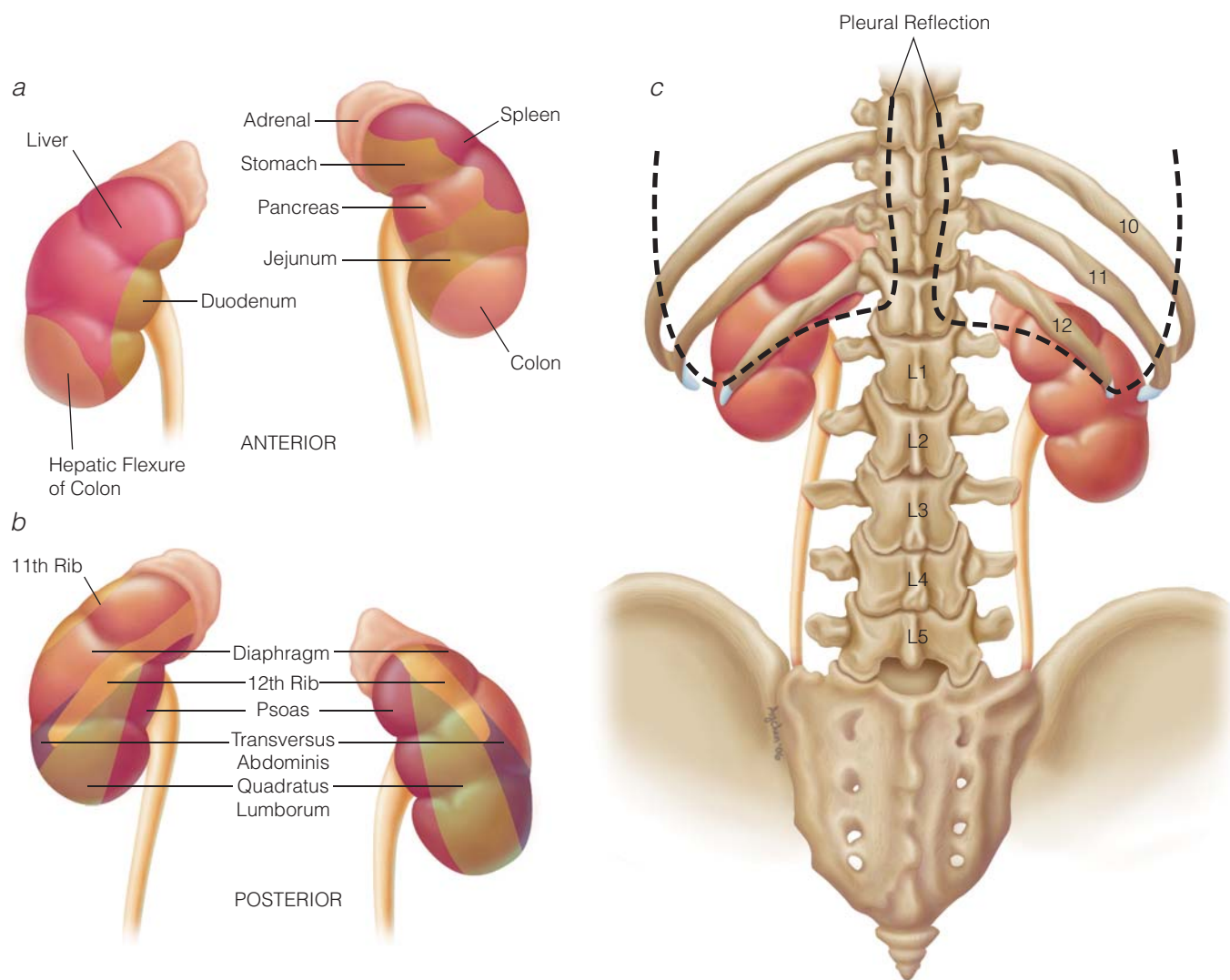


Figure 1 Shown are the anatomic relations of the kidneys (a) to the abdominal organs anteriorly, (b) to the body wall muscles and the ribs posteriorly, and (c) to the pleural reflections and the skeleton posteriorly.

The bladder is anatomically related to many other anatomic structures. Anteriorly, the bladder is tethered to the abdominal wall by the median umbilical ligament (i.e., the obliterated urachus). In rare cases, adenocarcinoma may develop in the urachal remnant; in other cases, the connection may remain patent and predispose to infection or drainage from the umbilicus. The superior aspect of the bladder is covered by a peritoneal layer that sweeps down from the anterior abdominal wall to meet the peritoneum covering either the anterior rectum (in males) or the uterus (in females). Laterally, the bladder is separated from the pelvic side walls by loose connective tissue and both retropubic and perivesical fat. In males, the base of the bladder is adjacent to the seminal vesicles, the ampullae of the vasa deferentia, the distal ureters, and the rectum. In females, the uterus and the vagina are interposed between the bladder and the rectum.

The bladder is very well vascularized and thus may be mobilized or partially resected without compromise of the blood supply. Most of the bladder's blood supply comes from the superior, middle, and inferior vesical arteries, which branch off from the anterior trunk of the internal iliac artery. In females, the bladder is further supplied by branches of the vaginal and uterine arteries.

The veins of the bladder come together to form several plexuses that drain into the internal iliac veins.

PROSTATE AND SEMINAL VESICLES

The prostate and the seminal vesicles produce constituents of the ejaculate. The normal prostate weighs between 18 and 20 g and is composed of glandular elements and fibromuscular stroma. Toward the apex, the prostate is tethered to the pubic bone by the puboprostatic ligaments. Laterally, the prostate is in direct contact with the pubococcygeal portion of the levator ani and is related to its overlying endopelvic fascia. Posteriorly, the prostate is separated from the rectum by the two layers of Denonvilliers' fascia. The prostate can be divided into transitional, central, and peripheral zones, and there is an anterior fibromuscular stroma that extends from the bladder neck to the striated urethral sphincter. The transitional zone is the area that gives rise to BPH, which can cause increased urinary resistance and voiding symptoms. The peripheral zone makes up the bulk of the prostate tissue and is the area where the majority of prostate cancers arise.

The seminal vesicles are about 6 cm long and lie in contact with the base of the bladder. Each seminal vesicle joins the ipsilateral

vas deferens to form the ejaculatory ducts, which exit the prostate at the verumontanum (i.e., the colliculus seminalis).

The prostate and seminal vesicles are supplied by the inferior vesical, internal pudendal, and middle rectal arteries; the seminal vesicles are also supplied by the vesiculodeferential artery, a branch of the superior vesical artery.

PENIS AND URETHRA

The penis is composed of three bodies: the two corpora cavernosa, which are responsible for erectile function, and the corpus spongiosum, which contains the urethra and is contiguous with the glans. Individually, these bodies are covered by a dense fascial layer known as the tunica albuginea; together, they are covered by a thick layer of Buck's fascia. If one or more of the penile bodies is torn, the bleeding will be contained in Buck's fascia, and ecchymosis will be limited to the penile shaft.

The penis is supplied by the common penile artery, which is the terminal branch of the internal pudendal artery. This vessel travels through Alcock's canal and terminates in three branches: the bulbourethral artery, which supplies the urethra, the corpus spongiosum, and the glans; the deep artery, which travels within the corpus cavernosum and supplies the cavernous sinuses; and the dorsal artery, which runs in the neurovascular bundle beneath Buck's fascia and supplies the corpus spongiosum and the urethra.

The male urethra is approximately 20 cm long and can be divided into four anatomic sections: the prostatic urethra, the membranous urethra, the bulbous urethra, and the penile urethra. The

sphincteric mechanism is located in the membranous urethra. The female urethra is approximately 4 cm long and travels from the bladder neck below the pubic symphysis to open anterior to the vagina.

TESTES

The testes have two important functions: spermatogenesis and production of testosterone. Each testis is approximately 20 ml in size and is surrounded by tunica albuginea, which projects inward to form the mediastinum testis.

The testis is supplied by the gonadal or testicular artery, the vasal artery, and the cremasteric artery. The gonadal artery arises from the aorta, just below the renal artery, and courses through the spermatic cord to the testis; the vasal artery is a branch of the superior vesical artery; and the cremasteric artery branches off from the inferior epigastric artery. At the level of the internal ring, these three arteries are in close proximity, and particular care must be taken to keep from injuring them. Venous outflow is provided by the gonadal vein. The right gonadal vein empties into the inferior vena cava below the renal vein, and the left gonadal vein empties directly into the left renal vein.

Urologic Injuries

UROGENITAL TRAUMA IN PATIENTS WITH MULTIPLE INJURIES

Urologic injuries are common when multiple organ systems are damaged, occurring in about 10% of cases of major trauma.

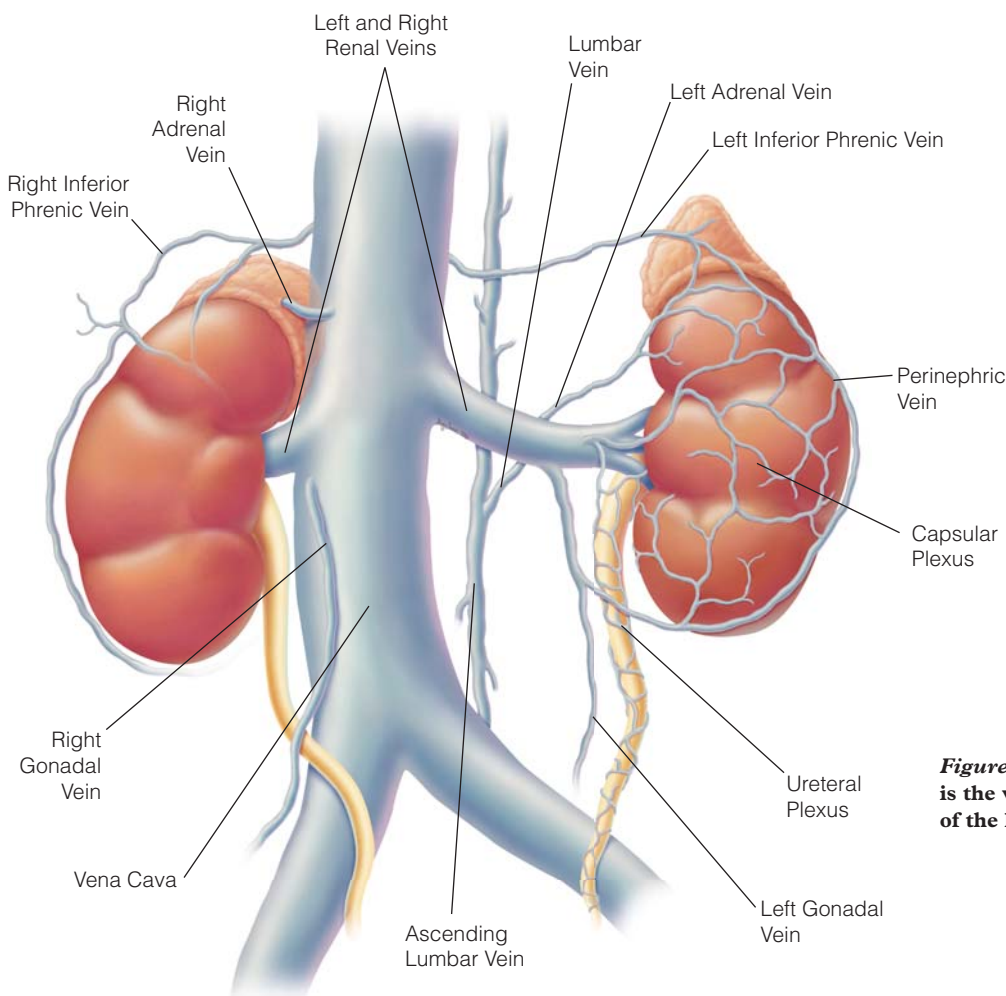


Figure 2 Illustrated is the venous drainage of the kidneys.

Although the majority of injuries to the urogenital tract are not life-threatening, they can result in substantial morbidity if they are not promptly diagnosed and treated. Accordingly, a surgical team treating a patient with multiple injuries must always keep in mind the possibility of concomitant urogenital trauma so that any urologic injuries found can be addressed in a timely manner.

Initial evaluation and resuscitation are generally carried out by an emergency department physician and a trauma surgeon; however, it is important that a urologist be involved as well. Assessment should proceed as outlined by advanced trauma life support (ATLS) protocol. The presence of gross hematuria, flank ecchymosis, rib fractures, pelvic fractures, or blood at the urethral meatus should raise the level of concern for urologic injury and serve as an indication for an expeditious urologic consultation.

Management of urologic trauma is addressed in more detail elsewhere [see 7:11 *Injuries to the Urogenital Tract*].

IATROGENIC INJURY TO UROGENITAL TRACT

Ureter

The majority of injuries to the ureter are iatrogenic. Iatrogenic ureteral injuries may be sustained during any open or laparoscopic procedure in the abdomen or the pelvis and may result from transection, ligation, laceration, resection, crushing, or ischemia. Such injuries are most likely to occur during gynecologic procedures; they also occur during general surgical and urologic procedures and, rarely, during orthopedic⁸ and vascular procedures.⁹ The ureter is most vulnerable to iatrogenic injury at several anatomical locations: at the pelvic brim, where gonadal and ovarian vessels cross; lateral to the uterus, where the ureter crosses under the uterine artery; over the iliac vessels, near the apex of the obturator fossa; and at the insertion of the trigone.

Although most ureteral injuries occur during routine, uncomplicated surgical procedures in which the pelvic anatomy appears normal,¹⁰ the likelihood of such injuries is increased by the presence of conditions that disrupt the architecture of the ureter, such as a large mass, a malignancy, inflammatory disease, endometriosis, a previous operation, irradiation, and congenital abnormalities. Increased blood loss, longer operating times, increased transfusion requirements, and longer hospitalizations are all associated with ureteral injury.^{11,12}

The best way of preventing iatrogenic ureteral injury is to clearly identify the ureter in the surgical field and in those regions where it is most susceptible to injury. Every attempt should be made to limit bleeding and to avoid placing sutures or using the electrocautery in areas where exposure is poor.

Placement of ureteral stents at the beginning of the procedure may facilitate identification of the course of the ureter. A 5-year review of prophylactic ureteral catheterization in patients undergoing colon surgery concluded that whereas stents did not prevent ureteral injuries, they could help surgeons recognize such injuries more quickly.¹³ Accordingly, the authors recommended stenting in patients at high risk for ureteral injury. A subsequent study from another group also recommended the use of stents in doubtful cases, on the grounds that prevention is better than treatment.¹⁴ Other authors, however, have argued that prophylactic ureteral stents are not without risk and have not affected the rate of ureteral injury.¹⁵ Prophylactic ureteral stenting continues to be a controversial practice.

Bladder

The bladder is the genitourinary organ that is most frequently injured during an operation.¹⁶ Fortunately, the bladder is very for-

giving of incidental injuries, and most such injuries are recognized intraoperatively, when they can easily be repaired with a two-layer closure using absorbable sutures. Bladder injury should be suspected when, during an operation, the Foley catheter is exposed, a laceration is noticed, urinary extravasation is present, urine in the Foley catheter becomes bloody, or (in the case of a laparoscopic procedure) gas begins to escape through the Foley catheter. Most patients have an identifiable predisposing factor that complicates the operation. Typically, this factor is previous surgery or inflammation, though bladder injuries resulting from surgery performed vaginally or laparoscopically do not seem to have a predisposing cause. The incidence of laparoscopic bladder injuries reportedly ranges from 0.02% to 0.3%.¹⁷ Foley catheter drainage is maintained for 1 to 2 weeks after repair, and the resulting success rates are quite high.

Urologic Malignancies

RENAL CELL CARCINOMA

It is currently estimated that deaths attributable to cancer of the kidney and the renal pelvis account for approximately 3% of all cancer deaths in the United States, and the annual incidence of renal cell carcinoma (RCC) continues to rise.¹⁸ Moreover, in 22% of patients, despite improvements in imaging and detection, the cancer will have already metastasized at the time of diagnosis.¹⁸ Approximately 85% of kidney cancers originate in the parenchyma; the remaining 15% are transitional cell carcinomas and develop in the collecting system.

In most instances, RCC is diagnosed incidentally during an imaging procedure that is performed to address some other problem. The classic triad of flank pain, hematuria, and a palpable abdominal mass is present in as many as 10% of cases.¹⁹ Because the range of possible presentations is quite wide, RCC is frequently referred to as the “internist’s tumor.” Patients may present with symptoms related to paraneoplastic manifestations, such as cachexia and fever (caused by cytokines), renin-mediated hypertension, nephropathy from immunoglobulin formation, hypercalcemia, cytokine-induced myelosuppression, polycythemia related to erythropoietin production, amyloidosis, or Stauffer syndrome (nonmetastatic hepatic dysfunction). Paraneoplastic symptoms tend to disappear once localized disease is treated.

All patients with hematuria or a renal mass should undergo further evaluation. The presence of gross or microscopic hematuria should prompt contrast evaluation of the kidneys and the ureters, as well as cystoscopy with urine cytology. The presence of a renal mass should prompt evaluation with either triple-phase computed tomography or magnetic resonance imaging with or without gadolinium (if the patient has a contrast allergy or renal insufficiency). No further assessment is required for simple cysts. Renal masses evaluated by means of CT are classified according to the Bosniak system, which correlates the appearance of the mass with the risk of malignancy [see Table 1].²⁰ Bosniak classes I, II, III, and IV roughly correspond to cancer risks of less than 2%, less than 12%, 52%, and greater than 90%, respectively.²¹⁻²⁷ The evaluation of a patient with a renal mass also includes a complete history, a careful physical examination, liver function tests, a comprehensive metabolic panel, and a chest x-ray.

If imaging identifies invasion into the renal vein or a tumor thrombus, further assessment (by means of venography, magnetic resonance angiography or venography, or CT reconstruction) aimed at determining the extent of vena cava involvement is necessary for successful removal of the mass. Renal masses that are

Table 1 Bosniak System for Classification of Renal Cysts²⁰

Category	Cyst Characteristics
I	Benign simple cyst with hairline-thin wall that has no septa, calcifications, or solid components; cyst has density of water and does not enhance
II	Benign cyst that may contain a few hairline-thin septa in which perceived (but not measurable) enhancement may be present; wall or septa may contain fine calcification or short segment of slightly thickened calcification; category includes uniformly high-attenuation lesions < 3 cm ("high-density cysts") that are well margined and do not enhance; no further evaluation required
IIIF*	Cyst that may contain multiple hairline-thin septa or minimal smooth thickening of wall or septa; wall or septa may show perceived enhancement; calcification may be present in wall or septa that may be thick and nodular, but without measurable enhancement; lesions are generally well margined; category includes intrarenal nonenhancing high-attenuation lesions > 3 cm; follow-up studies required to confirm benign status
III	Indeterminate cystic mass with thickened irregular or smooth walls or septa that show measurable enhancement; surgical management required, though some lesions will prove benign (e.g., hemorrhagic cysts, chronically infected cysts, multiloculated cystic nephromas) and others malignant (e.g., cystic and multiloculated cystic RCCs)
IV	Clearly malignant cystic mass that can have all criteria of category III but also contain enhancing soft tissue components adjacent to (but independent of) wall or septa; category includes cystic carcinomas; surgical removal required

*F signifies follow-up.

suspected of harboring cancer on the basis of diagnostic imaging should not routinely undergo biopsy except (1) in cases where there is a possibility of metastasis from another primary cancer or (2) in accordance with the protocol for minimally invasive treatment alternatives (e.g., cryoablation and radiofrequency ablation). Routine biopsies of kidney masses have high false negative rates and tend to yield nondiagnostic results.

Staging is a well-established predictor of prognosis. The TNM staging system developed by the American Joint Committee on Cancer (AJCC) has been widely used for prognostic purposes in RCC patients. In a European study, the 5-year and 10-year disease-specific survival probabilities were 95.3% and 91.4% for TNM stage pT1a RCC, 91.4% and 93.4% for stage pT1b disease, and 81.6% and 75.2% for stage pT2 disease.²⁸ In another study, the 5-year disease-specific survival probabilities were found to be significantly worse for more advanced RCC: 67% for TNM stage III disease and 32% for stage IV disease.²⁹ In the past few years, various other prognostic factors have been validated that are not included in the TNM staging system, including tumor grade, histologic subtype, sarcomatoid features, histologic tumor necrosis, collecting system invasion, and performance status.³⁰ Accordingly, many institutions are now using a more comprehensive staging system for RCC.

For localized RCC, the standard treatment is radical nephrectomy, either open or laparoscopic (hand assisted, retroperitoneal, or transperitoneal). This procedure involves removal of the entire kidney and the fat within Gerota's fascia, which is accomplished with early identification and ligation of the renal artery and the renal vein. To preserve adrenocortical function, it is best to avoid performing unconditional ipsilateral adrenalectomy with radical nephrectomy for RCC: the benefit of routine adrenalectomy in this

setting is extremely limited, especially in patients with small lesions and low-stage disease.³¹ If the tumor is small (generally, < 4 cm) and occupies an anatomically favorable position, it may be removed by means of partial nephrectomy—again, either open or laparoscopic. To prevent bleeding and leakage of urine, care must be taken to ensure that vessels are controlled and the collecting system is closed.

Metastatic RCC responds poorly to standard chemotherapy and radiation therapy. In patients whose disease burden is suitable, whose performance status and general health are adequate, and who are about to undergo immunotherapy with interferon or interleukin-2 (or other trial regimens, as appropriate), cytoreductive nephrectomy should be considered. In selected patients with solitary or limited metastases (confirmed by careful staging investigations), aggressive surgical resection of all disease may prolong survival and should be considered, possibly in conjunction with immunotherapy.³²

BLADDER CANCER

Like RCC, cancer of the urinary bladder currently accounts for approximately 3% of cancer deaths in the United States. The median age at which bladder cancer is diagnosed is 73 years.¹⁸ In the United States, approximately 90% of bladder cancers are transitional cell carcinomas, about 5% are squamous cell carcinomas, and some 2% are adenocarcinomas. Cigarette smoking is the primary environmental risk factor, associated with 50% to 66% of bladder tumors in men and 25% in women.³³ Another risk factor is exposure to carcinogenic aromatic amines, especially benzidine and β-naphthylamine.³⁴ Bladder cancer has been linked to occupational exposure in aromatic amine manufacturing, dyestuff manufacturing, rubber manufacturing, painting, the leather industry, the aluminum industry, and truck driving.³⁵ In Africa and the Middle East, squamous cell carcinoma, associated with *Schistosoma haematobium* infection, is the most common form of bladder cancer.

In 80% to 90% of patients, bladder cancer presents as gross or microscopic hematuria, which commonly is painless and sporadic.³⁶ In some patients, the presenting complaint is a change in voiding habits, such as urgency, increased frequency, painful urination, or difficulty in voiding. Decreased bladder capacity (as a result of a mass effect) and ureteral obstruction are less common presenting symptoms and are suggestive of more advanced disease. Systemic complaints are suggestive of metastatic disease.

The presence of hematuria should prompt a full evaluation consisting of upper urinary tract contrast imaging, cystoscopy, and urine cytology. Cystoscopy is the current standard for detecting bladder masses. Urine cytology has a specificity that approaches 100% and is useful in helping to identify small lesions, lesions that are atypical in appearance, and lesions that are located in the upper urinary tract.³⁷ All suspicious bladder lesions should be resected and sent for pathologic diagnosis. Biopsy specimens should be obtained from the muscle layer to determine whether the tumor has invaded the muscle.

The stage and grade of the tumor are important for determining treatment and are independently correlated with prognosis. As with RCC, the AJCC's TNM system is commonly employed for staging. Bladder tumors that involve the mucosa (Ta) and the lamina propria (T1) are referred to as superficial disease, whereas tumors that extend beyond the lamina propria and invade muscle (T2a) are referred to as invasive disease. Three categories are used for grading, corresponding to well-differentiated disease (grade 1), moderately well differentiated disease (grade 2), and poorly differentiated disease (grade 3). As the stage and grade of the tumor

increase, the prognosis worsens. Carcinoma in situ is a separate disease entity whose presence is associated with a high risk of recurrence and a greater likelihood of disease progression.

Approximately 70% of bladder cancers initially present as superficial disease. Treatment consists of resection and cauterization. If pathologic examination confirms superficial disease, no further metastatic workup is necessary. Surveillance is mandatory, however, because of the high risk of recurrence and progression. Low-grade Ta lesions recur in 50% to 70% of cases and progress in approximately 5% of patients, whereas high-grade T1 lesions recur in more than 80% of cases and progress in 50% of patients within 3 years.³⁸ Patients with high-risk superficial disease—defined as carcinoma in situ; stage T1 lesions; or large, high-grade, recurrent, or multifocal Ta lesions—should receive further treatment with intravesical bacillus Calmette-Guérin (BCG).³⁹

Approximately 25% of bladder cancers are invasive at the time of initial diagnosis. The standard treatment for organ-confined invasive bladder cancer is radical cystectomy with extended lymph node dissection; urine is diverted into a conduit or neobladder created from the large or the small bowel. Disease that extends through the muscle into the fat (T3), node-positive disease, or metastatic disease requires further treatment with chemotherapy after the postoperative period. In older series, if bladder cancer was left untreated, fewer than 15% of patients would survive 2 years.⁴⁰ In a 2001 study of 1,054 patients treated at the University of Southern California, the 5-year survival was 60% to 75% for pT2 tumors, 36% to 58% for pT3 tumors, and 4% to 35% for pT4 or node-positive tumors.⁴¹

PROSTATE CANCER

Cancer of the prostate accounts for approximately 10% of cancer deaths in the United States. It is estimated that prostate cancer will be diagnosed in one of every six men,¹⁸ usually relatively late in life; between 1998 and 2002, the median age at which prostate cancer was diagnosed was 69 years. Over the past 2 decades, the incidence of prostate cancer has risen, partly because of the widespread use of the prostate-specific antigen (PSA) screening test. This rise, however, has not been uniform. Between 1989 and 1992, the incidence of prostate cancer increased by an average of 18% per year, but between 1992 and 1995, it declined by 14% per year; it may now have reached a plateau.⁴²

In the early stages, prostate cancer is generally asymptomatic. Currently, most cases of prostate cancer are detected by an abnormal digital rectal examination or an abnormal PSA test result that prompts prostate biopsy. Routine screening with a PSA test and a digital rectal examination should begin at the age of 40 in men whose life expectancy is greater than 10 years. African-American men and men who have a first-degree relative with prostate cancer are at higher risk for prostate cancer. The generally accepted threshold value for an abnormal PSA level has been 4.0 ng/ml; however, some investigators now question this value, on the grounds that a significant number of men have been found to have cancer with PSA values between 2.6 and 4.0 ng/ml.⁴³⁻⁴⁵ In addition, the rate at which the serum PSA level increases (i.e., PSA velocity) has become a very important factor in the diagnosis of prostate cancer.⁴⁶ For proper interpretation of PSA values, it is important to recognize that the serum PSA level can be influenced by multiple factors besides prostate cancer (including urinary retention, BPH, and prostatitis).

The stage and grade of the tumor play important roles in determining treatment and prognosis. As with RCC and bladder cancer, the most widely accepted staging system is the AJCC's TNM system. The most widely accepted grading system for prostate can-

cer is based on the Gleason score. In this system, the biopsy specimen is examined under low-power magnification, and the most prominent glandular pattern seen is graded on a scale of 1 to 5, with 1 representing the highest degree of differentiation and 5 the lowest. The next most prominent glandular pattern seen is then graded in the same fashion. The two grades are added to yield the Gleason score, which can range from a minimum of 2 (1 + 1) to a maximum of 10 (5 + 5). Cancers with scores of 6 (3 + 3) or lower are considered well differentiated; those with scores of 7 (3 + 4 or 4 + 3) are considered intermediate; and those with scores of 8 (4 + 4) or higher are considered poorly differentiated. The more poorly differentiated the cancer is, the more aggressive it will be and the worse the prognosis will be. In addition to the Gleason score, the clinical stage and the PSA level have important implications for treatment.

There are many options for treating prostate cancer, and there remains some controversy regarding what constitutes the most effective therapy. Typically, treatment is based on the stage and grade of the tumor, the PSA level, and patient characteristics such as general health status, comorbid conditions, age, body habitus, and personal bias. Clinically localized prostate cancer may be treated with curative intent by means of surgery or irradiation (i.e., external-beam radiation therapy or brachytherapy). When the disease is more advanced or the patient is unwilling to undergo operative treatment or radiation therapy, antiandrogen therapies or watchful waiting may be considered.

TESTICULAR CANCER

Testicular cancer accounts for only about 1% to 2% of all malignancies in men; however, it is the most common cancer in males between 15 and 34 years of age.⁴⁷ Moreover, since the middle of the 20th century, the incidence of testicular cancer has been increasing in the United States.⁴⁸ The exact cause of testicular cancer remains unknown. It has been hypothesized that testicular atrophy is a common pathway by which several etiologic factors may be involved in tumor development in the testis.^{49,50} Familial tendency, white race, and a history of cryptorchidism are established risk factors.⁵¹⁻⁵³

The classic presentation of testicular cancer is painless swelling or enlargement of the testis. A 1987 study of 5,172 patients with testicular cancer, however, found that the initial presenting complaints for this condition included a swollen or enlarged testis (58%), a mass in the testis (27%), a painful testis (33%), tender breasts (3%), and a history of trauma (4%).⁵⁴ Acute trauma is not a proven cause of testicular cancer, but it may lead to the awareness of a mass. Examination generally reveals an enlarged testis or a nodule on the testis; it may prove more difficult in a patient with a tender testis or a large hydrocele. Ultrasonography, which has been shown to be highly reliable in differentiating between intratesticular and extratesticular lesions, may be performed to supplement the evaluation.^{55,56} In rare cases, testicular cancer may present with manifestations of systemic disease (e.g., pulmonary complaints or an abdominal mass).

Measurement of serum levels of tumor markers (i.e., human chorionic gonadotropin [hCG], α -fetoprotein [AFP], and lactic dehydrogenase [LDH]) is a well-established component of the management of testicular germ cell tumors and facilitates diagnosis, prognosis, monitoring of treatment, and prediction of relapse. Elevated serum concentrations of these markers are strongly suggestive of cancer, but the absence of such elevated concentrations in a patient with a testicular mass does not rule out cancer. hCG is secreted by the syncytiotrophoblasts present in certain germ cell tumors (i.e., embryonal tumors, teratomas, choriocarcinomas,

and fewer than 20% of seminomas). Its serum half-life is 16 to 24 hours. AFP is synthesized by yolk sac components of germ cell tumors. Its presence is diagnostic of nonseminomatous disease; it is never produced by pure seminomas or choriocarcinomas. AFP concentrations peak between gestational weeks 12 and 14 and start to decline after week 16, falling to undetectable levels after the first year of life. The serum half-life of AFP is 4.5 days. LDH, though not a highly sensitive tumor marker, has proved to be an independent prognostic variable in several large multivariate analyses of testicular germ cell tumors. Serum tumor marker concentrations are followed after radical orchiectomy and should fall in proportion to their respective half-lives.

Treatment is determined by the pathology and stage of the tumor. Most testicular tumors are of germ cell origin and are classified as either seminomatous or nonseminomatous. Nonmetastatic disease can often be treated surgically by means of radical orchiectomy, which is completed through an inguinal incision after vascular control of the spermatic cord has been achieved. Follow-up with diagnostic imaging and measurement of serum tumor marker levels is carried out to look for possible metastatic disease.

Seminomas account for nearly 48% of germ cell tumors and usually present during the fourth and fifth decades.⁵⁷ Initial metastasis is typically to either the periaortic lymph nodes near the renal hilum (for left-side lesions) or the precaval lymph nodes between the aortic bifurcation and the renal pedicle (for right-side lesions). Crossover to the periaortic nodes is common with right-side lesions. Seminomas may also spread hematogenously to the lungs, the liver, the brain, bone, the kidneys, the adrenal glands, the gastrointestinal tract, or the spleen. These lesions are highly radiosensitive, and thus, irradiation is a key part of early-stage treatment. Low-stage disease is treated with radical orchiectomy and radiation therapy, whereas advanced or bulky disease is treated with chemotherapy. Close follow-up is the rule.

Nonseminomatous germ cell tumors tend to be more locally aggressive than seminomas, and they have a high metastatic potential. Overall, the patterns of lymphatic and hematogenous spread are similar to those of seminomatous germ cell tumors; however, choriocarcinoma demonstrates a particularly aggressive natural history, with early hematogenous spread. Nonseminomatous germ cell tumors are treated with surgery, with platinum-based chemotherapy, or with both, depending on the disease stage. Low-stage disease is treated with radical orchiectomy, followed by chemotherapy if tumor marker concentrations do not normalize. If the tumor marker concentrations normalize, subsequent options include surveillance and nerve-sparing retroperitoneal lymph node dissection (RPLND); without additional treatment, 30% of patients will experience relapses. The presence of lymphatic, vascular, scrotal, or spermatic cord invasion increases the likelihood of retroperitoneal lymph node involvement, and RPLND may therefore be preferred in these circumstances.⁵⁸ Disease with minor nodal involvement is treated with radical orchiectomy and RPLND, followed by chemotherapy if RPLND demonstrates the presence of cancer. Disease with more extensive nodal involvement and metastatic disease are treated with chemotherapy. Close follow-up is the rule.

Benign Prostatic Hyperplasia

BPH becomes increasingly common in men as they age: histologic evidence from autopsy studies suggests that the incidence of BPH exceeds 50% in men older than 50 years and exceeds 75% in men older than 70 years.⁵⁹ The prostate is composed of stromal and epithelial components, and either stroma or epithelium (or

both) can give rise to hyperplastic nodules. Typically, nodules occur in the transition zone of the prostate. The etiology of BPH is multifactorial and is not yet completely understood. It is clear, however, that the endocrine system plays an important role and that the levels of testosterone and other hormones are significant factors. A positive family history and early onset of symptoms are associated with an increased risk of progression to more severe BPH. There may be an autosomal dominant or codominant pattern of inheritance.⁶⁰

Patients with BPH may present with obstructive or irritative symptoms, which are collectively referred to as lower urinary tract symptoms. Obstructive symptoms include incomplete emptying, intermittency, a weak stream, and straining; irritative symptoms include urgency, frequency, and nocturia. Although most patients present with one or more of these complaints, some patients may present with urinary tract infections, acute urinary retention, or renal failure. Because lower urinary tract symptoms are not disease specific, one must consider other urologic and medical conditions besides BPH, such as urinary tract infection, stones, prostatitis, strictures, cancer, and neurologic disease.

Patients should be evaluated with a careful history and a thorough physical examination. Symptoms should be quantified by using the International Prostate Symptom Score (I-PSS) questionnaire. This questionnaire consists of seven questions, the answers to which are scored on a scale of 0 to 5 to yield a total score ranging from 0 to 35. A total score of 0 to 7 indicates mild symptoms, a total score of 8 to 19 indicates moderate symptoms, and a total score of 20 to 35 indicates severe symptoms. I-PSS scores can be used to help determine treatment and assess its effectiveness, and their utility in this setting has been reliably validated. A complete urologic examination and a limited neurologic examination should be carried out. Routine PSA screening (for healthy persons of suitable age) and urinalysis should be performed. Whether further examination and testing are required is determined by the initial findings and the course of treatment. Additional evaluation may include measurement of serum creatinine concentrations, cultures, assessment of PSA levels, cystoscopy, biopsy, or urodynamic testing.

Treatment of BPH may involve observation, medical therapy, or surgical management. Symptoms tend to wax and wane, and many patients tolerate their symptoms reasonably well and prefer to avoid therapeutic interventions. Watchful waiting is a viable option for patients who have minimal symptoms and no evidence of renal or bladder dysfunction. Medical therapy focuses on relaxing the bladder neck and shrinking the prostate to facilitate more complete emptying of the bladder. Two classes of medications are used: alpha blockers and 5 α -reductase inhibitors. Alpha blockers are considered first-line treatment for all BPH patients. These medications act on the alpha-adrenergic receptors of the bladder neck and the prostate to help relax and open the channel. Their main side effects are related to their blood pressure-lowering effect. In 2003, the Medical Therapy of Prostatic Symptoms (MTOPS) Research Group published a study advocating combination therapy with an alpha blocker and a 5 α -reductase inhibitor to reduce the risk of overall clinical progression of BPH and the long-term risk of acute urinary retention and need for invasive therapy.⁶¹ Surgical intervention is generally reserved for patients who exhibit acute urinary retention and those who continue to experience significant symptoms despite medical treatment. Currently, surgical treatment is most often performed endoscopically. Prostatic tissue is resected with an electrocautery in the standard fashion, removed via transurethral resection of the prostate, or ablated with a laser. If the

prostate is extremely large (> 80 g), an open or laparoscopic^{62,63} simple prostatectomy may be performed.

Postoperative urinary retention, which occurs in 3.8% to 9% of cases,^{64,65} is a particularly troubling development. It has been associated with preexisting voiding issues, immobility, analgesics, administration of high fluid volumes during operation, and impaired mental status. Treatment is based on eliminating as many of these predisposing factors as possible. After an unsuccessful first voiding

attempt, a single catheterization is successful in resolving postoperative urinary retention in 39% of cases.⁶⁴ Generally, patients are given an alpha blocker first, and a voiding trial is administered once they have become ambulatory and are using less pain medication. Patients with a history of lower urinary tract symptoms should be started on an alpha blocker preoperatively, and administration of the medication should resume as soon as they are capable of tolerating liquids.

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