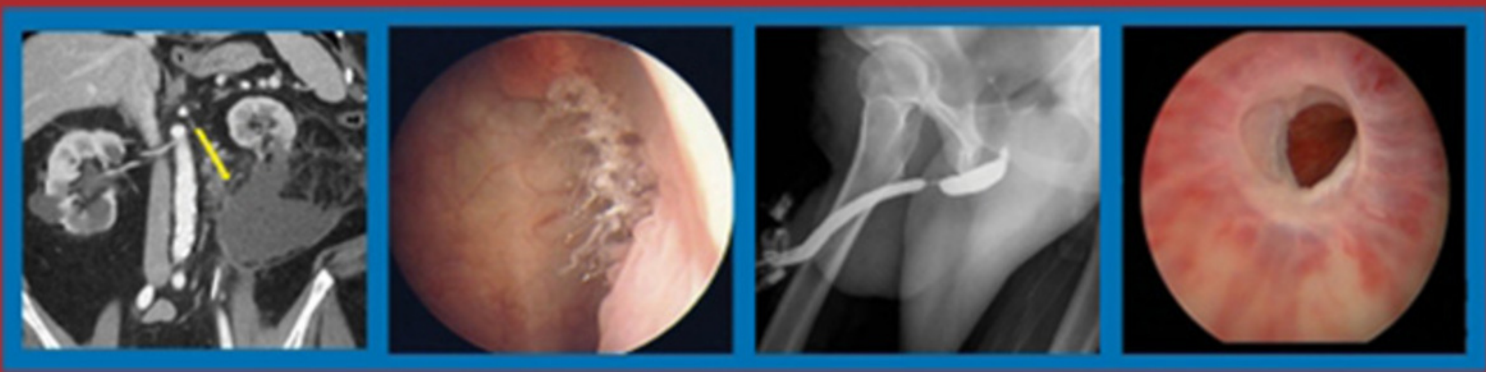
TANEJA'S

COMPLICATIONS of UROLOGIC SURGERY

Diagnosis, Prevention, and Management

FIFTH EDITION



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Preface

Surgery is never perfect, but that should not stop a surgeon's pursuit of perfection.

In many ways, this is the nature of a surgical career – continually refining one's approach and technique through observation, trial and error, and experience. In doing so, excellent outcomes can be achieved with any operation, and when complications arise, early recognition and proper management can allow expeditious recovery.

Optimizing surgical outcomes can be viewed as a lifelong exercise for the surgeon, but for the individual patient, only one outcome matters. Although it is easy for the busy surgeon to consider any operation as routine, it is essential to view each procedure, no matter how routine, as important and carrying potentially life-altering results for the patient. The balance between confidence and neglect is narrow for surgeons, and humility serves the surgeon well in recognizing that no matter how good or experienced the surgeon is, complications are inevitable. Even the operation that appears to go well, in an otherwise healthy patient, can result in complication.

When planning an operation, consideration of unique host factors predisposing the patient to complication is essential. With increasing experience, the surgeon will recognize those cases in which surgery is ill advised. Too often, early in a surgeon's career, complications arise through poor patient selection or preparation, or inadequate attention to risk factors in planning the procedure. Patients with underlying cardiopulmonary compromise, diabetes, coagulopathy, or morbid obesity should be considered at highest risk of complications delaying or preventing recovery. In particular, obese patients carry both the risk of complications due to underlying medical disease and those related to the increased technical difficulty of the procedure. Likewise, re-operative procedures can carry unique technical challenges and risks. When starting out, recognition of these challenges, and consideration of referral to a surgeon experienced with those types of patients, can save a great deal of anguish for surgeon and patient.

While caution is essential, surgeons must also be willing to take on challenging cases when patients are in absolute need of care. Careful preoperative assessment, operative planning, consultation when appropriate, and adherence to fundamental surgical principles allow the best opportunity for a good outcome. When confronted with a complication, the surgeon must refrain from efforts to minimize the problem and use a judicious approach to determine the right course.

First and foremost, complications take a tremendous emotional and physical toll on our patients, and surgeons must remember this when confronted with a complication. For the patient, the process of surgery is one in which control is given completely to the surgeon. The uncertainty of outcome, the loss of control, and the fear of mortality are tremendously stressful for the patient even in the setting of an uncomplicated surgery. When complications arise, these stresses are magnified and patients and their families are often confused, depressed, or angry. Demonstration of sincere empathy and sympathy are critically important. Careful, calm, and comprehensive communication are essential to enable them to understand the nature of the complication, its probable causes and outcomes, and concerns going forward. Specific benchmarks for improvement can allow the patient and family a structured process to mentally cope with the situation. Patients with complications often fear the surgeon will abandon them, and reassurance can go a long way toward maintaining a good relationship.

Physical concerns in the setting of complications relate to the patient's ability to tolerate the stresses and the relative risk of prolonged hospitalization. In patients with preexisting comorbid conditions, careful attention to management of underlying disease processes, particularly those influencing recovery, will help in avoiding secondary complications. Maintaining nutrition, preventing infection, and carefully monitoring fluids and electrolytes are fundamental surgical principles that directly affect recovery from most procedures, but that can easily be forgotten in the heat of stressful complication. Although not all patients recover from complications, the surgeon's primary goal must be to optimize the patient's condition to maximize the patient's odds of recovery.

The balance between action and inaction is a difficult one for surgeons. An underlying desire to make a complication go away often leads to a hasty decision to act quickly through reoperation or intervention. Although sometimes indicated, quick decisions to intervene often result in worsening of the problem or development of secondary complications. At the time of complication, careful diagnostic evaluation to fully understand its nature and extent are critically important before any action is taken. Although stressful for both the patient and the surgeon, sometimes waiting it out is the best course of action.

Over the years, *Complications of Urologic Surgery: Diagnosis, Prevention, and Management* has become a text popular among those in training and those in practice, largely due to its ability to provide information relevant to day-to-day practice. It has been translated in multiple languages, creating access for urologists around the world. In this fifth edition of the text, we have expanded the title to include "Diagnosis", recognizing that the early identification of complications is as important as their management. We have additionally made the content more contemporary, by removing references to operations rarely performed in modern practice, and adding a number of chapters dedicated to complications unique to the emerging standard of minimally invasive surgery and specific procedures commonly employing laparoscopy or robotic-assisted laparoscopy. Recognizing that robotic-assisted surgery is growing in utilization, but remains underutilized, due to cost, around the world, we have retained detailed discussion of the conventional procedures commonly performed in urologic practice, expanding upon the diversity of authors to allow a broad perspective to approach. Finally, for trainees, we have added a new study guide with questions and case presentations to test knowledge and inspire questions in mastering the content of the text. It is our sincere hope that the text will remain a popular, frequently utilized source of information for practicing urologists around the globe.

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We would like to dedicate this book to the many who have so greatly influenced our lives and careers.

To our parents, Vidya Sagar and Sudesh Taneja, and Dinker and Aruna Shah, for setting an example and encouraging us to relentlessly pursue excellence in our education and professional life.

To our mentors, Jean deKernion, Robert Smith, Richard Ehrlich, Dean Assimos, and Herbert Lepor, for teaching us the science and craft of surgery and supporting us in failure or success.

To our families, Uma, Sorab, and Sabina Taneja, and Rupa, Siena, Devon, and Mira Shah, for their patience, love and support in allowing us to pursue the distractions of our academic life.

And, most importantly, to our many patients whose care has taught us so much, whose needs have inspired persistence and innovation, and whose gratitude has motivated us to carry on each day.

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SECTION I

Preoperative Assessment and Perioperative Management

1 Impact of Host Factors and Comorbid Conditions

KATHLEEN F. MCGINLEY and STEPHEN J. FREEDLAND

CHAPTER OUTLINE

Obesity
Obesity and Urologic Malignant Diseases
Prostate Cancer
Kidney Cancer
Bladder Cancer
Obesity and Benign Urologic Conditions
Benign Prostatic Hyperplasia and Lower Urinary Tract Symptoms
Erectile Dysfunction
Stress Urinary Incontinence
Urolithiasis
Malnutrition
Nutritional Status Assessment
Preoperative Management of Malnutrition
Infection and Urosepsis
Quantifying Comorbidity
Conclusion
Chapter 1 Questions and Answers

KEY POINTS

1. With increasing life expectancy in the general population, the prevalence of comorbid conditions such as obesity, heart disease, and diabetes has increased to alarming proportions.
2. Awareness of comorbidities allows the urologist to institute the proper measures to control preexisting diseases to optimize the overall health status of the individual patient, maximize the likelihood of a good outcome, and minimize the risk of a complication.
3. Obesity can directly influence surgical outcome because of certain proposed biologic linkages with urologic malignant diseases.
4. Nutritional status is a key clinical parameter demanding thorough evaluation in the surgical patient to prevent nutrition-related complications.
5. Given that certain host factors predispose the urologic patient to complicated infection, it is necessary to determine the need for antimicrobial prophylaxis preoperatively and to prevent the occurrence of systemic septicemia.

Every urologist would prefer that any patient who has a consultation for a urologic disease would be solely afflicted with the disease for which he or she seeks medical attention, that every surgical patient would be healthy enough to tolerate the proposed surgical intervention to treat the condition, and that complications would occur with only miniscule probability. Unfortunately, this situation is far removed from reality and certainly is becoming less common in current clinical practice in which medical histories, physical examinations, preoperative laboratory examinations, and imaging scans are likely to reveal coexisting medical problems in the urologic patient.

In the present era, with life expectancy ever increasing, the prevalence of comorbid conditions such as obesity, heart disease, and diabetes, which affect urologic diseases and their clinical outcome following management, has congruently reached alarming proportions in the general population. Whether driven by improved medical science, rapid technologic advancement, or an effect of natural selection, men and women are living longer (Fig. 1.1). The medical community recognizes special considerations for elderly patients, and most of these considerations are brought about by medical conditions that are diagnosed in later life and progress with advancing age. In urologic disease entities such as erectile dysfunction in men, pelvic floor disorders in women, and urologic malignant diseases such as prostate and bladder cancer, the predisposition and clinical effects related to advanced age have direct biologic implications for the urologic condition. Moreover, because most of these disease entities are diagnosed in the more mature stages of life, the probability of preexisting medical conditions in these patients at the time of consultation is high.

Notwithstanding the effect of age on comorbid medical conditions in the urologic patient, the past decades have also seen a dramatic rise in the prevalence of disease entities closely linked to harmful lifestyle choices such as smoking and alcohol consumption, unhealthy diets, lack of physical activity, and intravenous drug abuse. These lifestyle choices adversely affect patients of all ages who may seek urologic consultation and who may present with detrimental comorbidities such as childhood obesity, juvenile diabetes, chronic obstructive pulmonary disease, liver disease, and human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS).

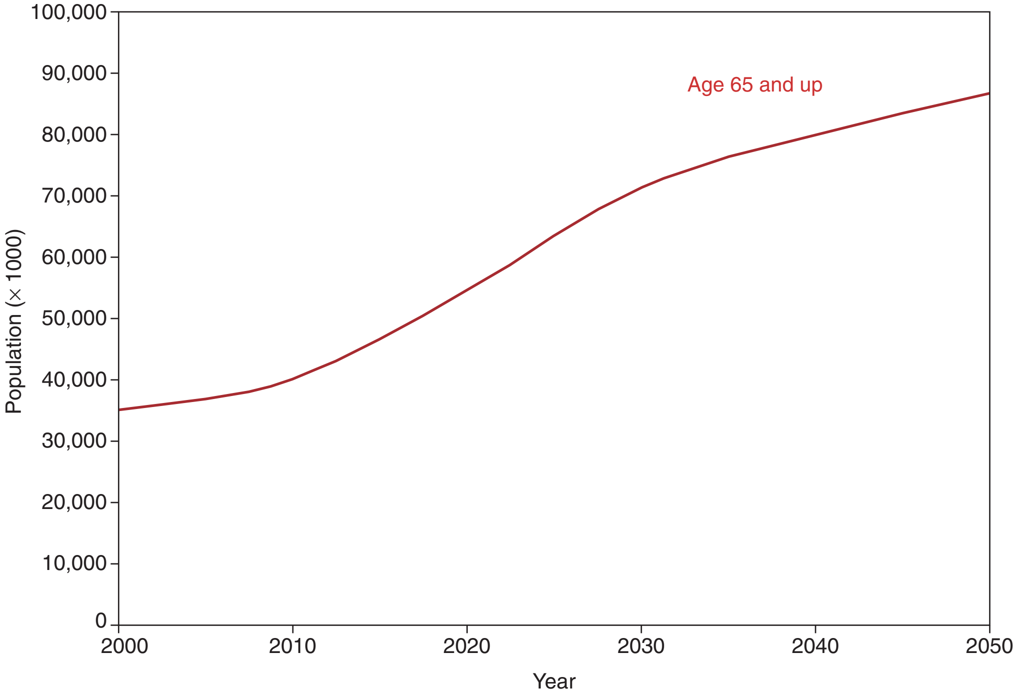

Figure 1.1 Projected population of the United States for adults ≥65 years old (2000–2050). Based on data from the United States Census Bureau (http://www.census.gov/ipc/www/usinterimproj/).

Although biologic links to known urologic diseases may be less apparent, the overall outcome and incidence of complications following surgical intervention are directly affected by coexisting health problems. Indeed, assessing the urologic patient for preexisting comorbidities is of critical importance because host factors play an important role in postoperative complications. Awareness of comorbidities allows the urologist to institute the proper measures to control preexisting diseases to optimize the overall health status of the individual patient, maximize the likelihood of a good outcome, and minimize the risk of a complication. The urologist also can assess the need for ancillary examinations for a more comprehensive evaluation of comorbid conditions more accurately and can determine the need for intraoperative monitoring and specialized intensive postsurgical care. More importantly, comprehensive knowledge of all concurrent illnesses in the urologic patient aids the urologist in deciding whether surgical intervention is the optimal treatment option or whether conservative management may be the best therapeutic alternative.

To serve as an introduction to the succeeding chapters in this section, we tackle host factors that significantly affect the occurrence of nonurologic complications following urologic surgery. We provide an overview of comorbidities in the urologic patient and highlight current prevalent disease entities that influence outcome following definitive surgical management. Comorbidities to which whole chapters are devoted, such as those pertaining to cardiovascular, pulmonary, hematologic, and anesthetic complications, are discussed only briefly here, to leave room for a more detailed discussion of topics of special interest such as obesity that are of major interest in the field of contemporary urology. We also provide insight into clinical tools such as useful comorbidity indices and scoring systems that aim to quantify the severity of comorbidities and predict posttreatment morbidity and mortality.

Obesity

The importance of nutritional status to surgical outcomes and the deleterious effects of obesity are of significant interest in the field of urology. Interest has centered on obesity for two main reasons: First, the prevalence of obesity has been growing at epidemic proportions worldwide,[1] and second scientific evidence suggests a relationship between obesity and multiple urologic conditions including benign prostatic hyperplasia (BPH),[2] urologic malignant diseases, incontinence, erectile dysfunction, and stone disease, to name a few.[3]

Most of the leading causes of death in the United States are linked to obesity, including heart disease, cancer, stroke, and diabetes.[4] Viewed as a growing national health crisis, obesity is the second leading cause of preventable death;[5] obesity not only results in a potentially avoidable toll in human lives but also incurs a substantial cost in health expenditure for the country.[6] Affecting over one-third of all adults in the United States,[7] obesity is further associated with various comorbidities, such as hypertension, sleep apnea, cholecystolithiasis, osteoarthritis, and depression, that may aggravate the overall health status of the overweight or obese patient and may contribute to surgical complications.[8] Childhood obesity is also on the rise and could have undesirable consequences for children and adolescents undergoing pediatric urologic procedures.[9]

Obesity is defined as an excess accumulation of adipose tissue in the body; however, functionally, *overweight* and *obese* are labels used to denote ranges of weight that are in

BODY MASS INDEX (kg/m²)

WEIGHT (lbs)

HEIGHT (ft / in)	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215	220	225	230	235	240	245	250	HEIGHT (cm)
6'2"	14	15	15	16	17	17	18	19	19	20	21	21	22	22	23	24	24	25	26	26	27	28	28	29	30	30	31	31	32	188
6'1"	15	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	28	29	30	30	31	32	32	33	185
6'	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	28	29	30	31	31	32	33	33	34	183
5'11"	15	16	17	17	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	29	29	30	31	31	32	33	33	34	35	180
5'10"	16	17	17	18	19	19	20	21	22	22	23	24	24	25	26	27	27	28	29	29	30	31	32	32	33	34	34	35	36	178
5'9"	16	17	18	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30	31	32	32	33	34	35	35	36	37	175
5'8"	17	17	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30	31	32	33	33	34	35	36	36	37	38	173
5'7"	17	18	19	20	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32	33	34	34	35	36	37	38	38	39	170
5'6"	18	19	19	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32	33	34	35	36	36	37	38	39	40	40	168
5'5"	18	19	20	21	22	22	23	24	25	26	27	27	28	29	30	31	31	32	33	34	35	36	37	37	38	39	40	41	42	165
5'4"	19	20	21	21	22	23	24	25	26	27	27	28	29	30	31	32	33	33	34	35	36	37	38	39	39	40	41	42	43	163
5'3"	19	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	35	36	37	38	39	40	41	42	43	43	44	160
5'2"	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	36	37	37	38	39	40	41	42	43	44	45	46	158
5'1"	21	22	23	24	25	26	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	43	44	45	46	47	155
5'	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	152
WEIGHT (kg)	50	52	54	56	59	61	63	65	68	70	72	74	77	79	81	83	86	88	90	92	95	97	99	101	104	106	108	110	113	

WEIGHT (kg)

UNDERWEIGHT (BMI<18.5 kg/m²) · NORMAL (BMI=18.5–24.9 kg/m²) · OVERWEIGHT (BMI=25–29.9 kg/m²) · MILDLY OBESE (BMI=30–34.9 kg/m²) · MODERATELY OBESE (BMI=35–39.9 kg/m²) · SEVERELY OBESE (BMI≥40 kg/m²)

Figure 1.2 Estimates of body mass index using measured height and weight.

excess of what is generally considered healthy for the given height of a person. Because of its simplicity, body mass index (BMI) is a widely accepted method to assess for obesity. BMI is calculated by dividing the weight (in kilograms) of an individual by the height (in meters) squared.[10] Fig. 1.2 illustrates the standard weight status categories associated with BMI range for adults. Although other anthropometric measurements such as skinfold thickness and midarm circumference may be used for more accurate estimation of body fat, these measurements are not routinely recorded in clinical practice and are of limited availability for retrospective studies.[10]

Fat distribution may also be an important determinant of obesity because individuals with high BMI who have upper body fat distribution (android) have been shown to be at greater risk for comorbidities such as cardiovascular disease, cerebrovascular disease, and hypertension compared with men and women who have lower body fat distribution (gynecoid).[11] Newer studies found better accuracy in gauging obesity with the use of waist circumference and waist-to-hip ratios; however, these parameters are more cumbersome to measure compared with BMI.[12] *Central obesity* correlates with visceral fat accumulation in the abdomen and is diagnosed when the waist-to-hip ratio exceeds 1.0 in men and 0.9 in women. This condition is in contrast to *peripheral obesity*, in which fat accumulation occurs subcutaneously in the gluteofemoral region. However, the distinction is clinically important because central obesity imparts a significantly higher risk of insulin resistance and type 2 diabetes, blood lipid disorders, hypertension, and heart disease compared with peripheral obesity.[13]

The medical consequences of obesity result in part from increased secretion of pathogenic macromolecules from enlarged adipose cells. Increased release of fatty acids from fat cells that are stored in the liver or muscle results in the insulin-resistant state that is commonly seen in obesity. Diabetes ensues as the mounting insulin resistance overwhelms the secretory response of the pancreas.[14] Bioactive cytokines, particularly interleukin-6, released from adipocytes promote the proinflammatory state that is characteristic of obesity. Secretion of prothrombin activator inhibitor-1 from adipose cells, coupled with impaired endothelial function, plays a key role in the hypercoagulable state of obesity and ultimately increases the risk of cardiovascular disease, stroke, and hypertension in obesity. This prothrombotic state is further aggravated directly by increased estrogen levels and is complicated indirectly by decreased antiangiogenic cytokines such as adiponectin.[15] The overall effect of these multiple pathologic consequences of increased fat stores is the risk of shortened life expectancy.[16]

OBESITY AND UROLOGIC MALIGNANT DISEASES

Investigations since the late 1980s have sparked keen interest in the link between obesity and urologic cancer, especially for prostate adenocarcinoma and kidney cancer.[17] Investigators have hypothesized that diet and obesity affect the underlying biologic mechanisms that ultimately lead to carcinogenesis, including promotion of angiogenesis and mitogenesis, increased cellular proliferation, impairment of immune response, increased exposure to oxidative damage by free radicals, and promotion of a proinflammatory state.[18] Obesity can directly influence surgical outcome as a result of these proposed biologic linkages with urologic malignant diseases.

Prostate Cancer

Because obese men with prostate cancer have lower serum prostate-specific antigen (PSA) levels relative to men of normal weight,[19,20] and because physical assessment of the prostate through digital rectal examination is hindered by adiposity, detection of prostate cancer among men with a high BMI may be delayed. Performing a transrectal ultrasound–guided biopsy to establish a tissue diagnosis of prostate cancer can also be more technically difficult in obese men, and because of prostatic enlargement, some cancers may be missed by undersampling.[19] After histopathologic confirmation of prostate cancer, the patient may opt for surgical treatment, but urologists may be reluctant to operate on morbidly obese patients for several reasons. The anesthetic risks pertaining to adequacy of ventilatory support and difficulty in fluid monitoring[21] are further complicated by the increased incidence of comorbid conditions such as hypertension, heart disease, stroke, and diabetes.[14]

If the urologist does perform surgery, adiposity can be a physical hindrance that may curtail adequate exposure of the surgical field, particularly when a retropubic approach is planned for access to the prostate. Among men undergoing an open retropubic prostatectomy, increasing BMI is associated with increasing operative time and increasing intraoperative blood loss.[22] For these reasons, some urologists have advocated that perineal prostatectomy or a robotic-assisted laparoscopic prostatectomy should be favored over an open retropubic approach for treatment of obese men with prostate cancer. However, a study by Fitzsimons and associates[23] suggested that both perineal and open retropubic approaches have comparable outcomes in terms of estimated blood loss and operative time for obese patients. In contrast, Ellimoottil and colleagues[24] found lower transfusion rates among 9108 obese men undergoing robotic-assisted laparoscopic prostatectomy versus an open retropubic approach; however, no difference in perioperative complications between the groups was identified. In a matched pair analysis including 255 patients, Beyer et al.[25] reported lower blood loss, transfusion rates, and fewer 30-day complications among obese men undergoing a robotic-assisted laparoscopic prostatectomy as compared to those undergoing an open retropubic prostatectomy.

Beyond technical issues, obesity may also influence the oncologic outcome among men undergoing radical prostatectomy. First, earlier studies found an increased incidence of positive surgical margins and capsular incision among men with higher BMIs.[26,27] Similarly, men with higher BMIs present with higher-grade tumors and more advance pathologic stages of disease.[28] On postsurgical follow-up, men with an elevated BMI (≥ 30 kg/m^2) are at significantly increased risk of biochemical recurrence relative to men with a lower BMI, as denoted by an elevated postoperative PSA test result (>0.2 ng/mL or two values at 0.2 ng/mL).[26,29,30] More ominously, increased body weight was found to be associated with an increased risk of death from prostate cancer in a large, prospectively studied population.[31] Thus, obesity may well exert a biologic effect on prostate cancer that promotes aggressiveness and disease progression. However, in terms of health-related quality of life after radical prostatectomy, prospective studies have so far failed to demonstrate large differences between mildly obese men and men of normal weight.[32–35] For a more detailed review of obesity and prostate cancer, we recommend the article by Allott and Freedland in *European Urology*.[36]

Kidney Cancer

Obesity, particularly in women, has been shown to be associated with renal cell carcinoma (RCC).[31,37] A high BMI was found to be a strong risk factor for RCC; several underlying mechanisms were suspected, including higher insulin and estrogen levels, hypertension, hypercholesterolemia, and impaired host immune response.[38] Boeing and colleagues[39] examined determinants such as smoking, diet, occupational hazards, beverage consumption, medications, and obesity in a case-control cohort of 277 patients with RCC and 286 matched controls and found that specific dietary patterns associated with obesity, such as consumption of fatty foods and meat products, may explain the higher incidence of RCC in industrialized countries relative to developing countries.[39] Indeed, in a large retrospective study involving 363,992 men, investigators from the National Institutes of Health found that obese men, especially those with a history of tobacco use and elevated systolic blood pressures, have an increased long-term risk for RCC.[40]

As in prostate cancer, open surgical procedures for RCC can be technically difficult in patients with severe adiposity. Thus, wide interest exists in prescribing minimally invasive procedures for obese patients because these approaches have been found to be safe and effective for this subset of patients.[41–43] While BMI was found to be a significant risk factor for major postoperative complications in patients treated with laparoscopic surgery for RCC,[44] more recent literature evaluating robotic-assisted laparoscopic procedures have found no association between obesity and complication rates.[45,46] Finally, with regard to clinical outcome and cancer-specific mortality, overweight and obese patients have a higher risk of death from kidney cancer relative to patients of normal weight.[31]

Bladder Cancer

Compared with prostate and renal cancer, published reports of relationships between bladder cancer and obesity are scarce. In 1994, an epidemiologic study of 514 patients with bladder cancer found that beyond the well-known link with smoking, obesity was also a significant risk factor for bladder cancer.[47] This was substantiated in a recent case control study, in which patients with metabolic syndrome were at a twofold higher risk of bladder cancer.[48] However, a large prospective study of nearly 1 million people found no link between BMI and bladder cancer mortality.[31] With regard to diet, reports on the association between high fat intake and bladder cancer have been conflicting.[49,50]

With respect to surgical outcome for radical cystectomy, abundant reports show not only that obesity contributes to the technical challenge of the operation but also that higher BMI increases the risk of perioperative complications. In a retrospective analysis of 304 consecutive patients who underwent radical cystectomy and urinary diversion for bladder cancer, increased BMI was independently associated with higher estimated blood loss.[51] This finding was later confirmed in a cohort of 498 patients; the investigators concluded that, along with greater blood loss, an increased BMI was also independently associated with prolonged operative time and increased rate of complications.[52] In limited robotic cystectomy case series, increasing BMI was not associated with prolonged operative times or increased blood loss, though it was associated with an increased rate of 90-day re-admission.[53,54]

OBESITY AND BENIGN UROLOGIC CONDITIONS

Several nonmalignant urologic conditions are also unfavorably affected by an increased BMI and morbid obesity.

Benign Prostatic Hyperplasia and Lower Urinary Tract Symptoms

Obesity is a known risk factor for lower urinary tract symptoms (LUTS) and BPH. Indeed, a large-scale, cross-sectional study from the Prostate Study Group of the Austrian Society for Urology found a link between BPH and obesity.[55] The relationship between obesity and LUTS was further confirmed in a report from Johns Hopkins University in Baltimore on 2797 men from the Third National Health and Nutrition Examination Survey.[56] In another confirmatory study, BPH was found to be associated with increased serum insulin levels and abdominal obesity as opposed to BMI itself.[57] The biologic link between obesity and BPH likely has its origin in the association of obesity with hyperinsulinemia and the status of insulin as a direct prostate growth factor.[58]

Erectile Dysfunction

Obesity, particularly central obesity, is a known predictor of erectile dysfunction in men.[59] Both atherosclerosis and diabetes mellitus, which are associated with obesity, play significant roles in the development of erectile dysfunction. Although the underlying cause for erectile dysfunction is thought to be multifactorial, investigators have suggested that obesity increases the risk of erectile dysfunction of vascular origin as a result of the development of chronic vascular disease.[60] Obesity is also known to increase the risk of diabetes. The microvascular complications characteristic of diabetes exert deleterious effects on erectile tissue similar to the pathologic features of diabetic nephropathy, retinopathy, and gastroparesis.[61] Furthermore, weight loss is the only known lifestyle intervention that can improve erectile dysfunction.[62]

Stress Urinary Incontinence

Pelvic floor weakness leading to stress urinary incontinence (SUI) in women is aggravated by increased intraabdominal pressure and is closely associated with truncal obesity. A report examined the association of bladder function with smoking, food consumption, and obesity in 6424 women with SUI and found a strong relationship between SUI and obesity.[63] These findings were confirmed in a questionnaire-based study conducted in Norway involving 27,936 women.[64] The proposed underlying mechanism for the association between high BMI and incontinence is that a high BMI leads to increased intravesical pressures and thus lowers the differential between the detrusor pressure and leak point pressure such that incontinence is more likely to occur.[65] With regard to the perioperative effect of obesity in surgical treatment of SUI, a study involving 250 women who underwent retropubic anti-incontinence procedures revealed that operative time was significantly longer for obese women; however, blood loss and major perioperative complications were similar across BMI groups.[66]

Urolithiasis

Urinary stone formation has been linked to obesity, as illustrated by a report on 527 calcium oxalate stone formers wherein an increased BMI was strongly associated with an elevated risk of stone formation for both men and women.[67] However, a retrospective study of 5492 stone formers revealed that the association between obesity and stone formation was significant only in women.[68] In a study conducted at Duke University, the major metabolic abnormalities found in obese stone formers that were possible contributors to recurrent stone formation were hypocitraturia, gouty diathesis, and hyperuricosuria.[69] An inverse association between pH and body weight suggests that production of excessively acidic urine promotes uric acid nephrolithiasis in obese stone formers.[70]

With respect to urologic procedures to treat stone disease, obesity adversely affects outcome following extracorporeal shock wave lithotripsy (ESWL). In a report examining clinical and radiologic variables associated with poor outcome after ESWL, along with obesity, pelvic ureteral stones, stones >10 mm, and obstruction were independent predictors of unsuccessful outcome.[71] Thus, because of the probability of treatment failure, obese patients, particularly those with a skin-to-stone distance of >10 cm, may be better served by endourologic procedures than by ESWL.[72,73]

Malnutrition

At the opposite end of the nutritional spectrum from overnutrition and obesity is malnutrition. With regard to the surgical patient, malnutrition has been associated with an increased incidence of nosocomial infection, poor wound healing, an increased length of hospital stay, multiorgan dysfunction, and mortality.[74] Various scientific investigations have demonstrated that deterioration of nutritional status has an invariably deleterious effect on surgical outcome. As early as 1932, Cuthbertson reported the association of impaired wound healing with negative nitrogen balance in trauma patients.[75] A more recent prospective study conducted in a cohort of patients who did not have cancer used four clinical parameters to predict perioperative morbidity:

1. Percentage of ideal body weight
2. Preoperative percentage of weight loss
3. Arm muscle circumference
4. Serum albumin.

Results of the study revealed that patients with at least one abnormal clinical parameter had a significant increase in the incidence of major complications and in length of hospital stay relative to patients with normal preoperative parameters.[76] Not only has malnutrition per se been implicated in surgical complications but also certain types of nutrient deficiency, protein malnutrition in particular, may lead to more severe postoperative problems. Relative to protein-calorie malnutrition, which is characterized by a lack of both proteins and carbohydrates, severe protein malnutrition leads to low serum albumin concentration,

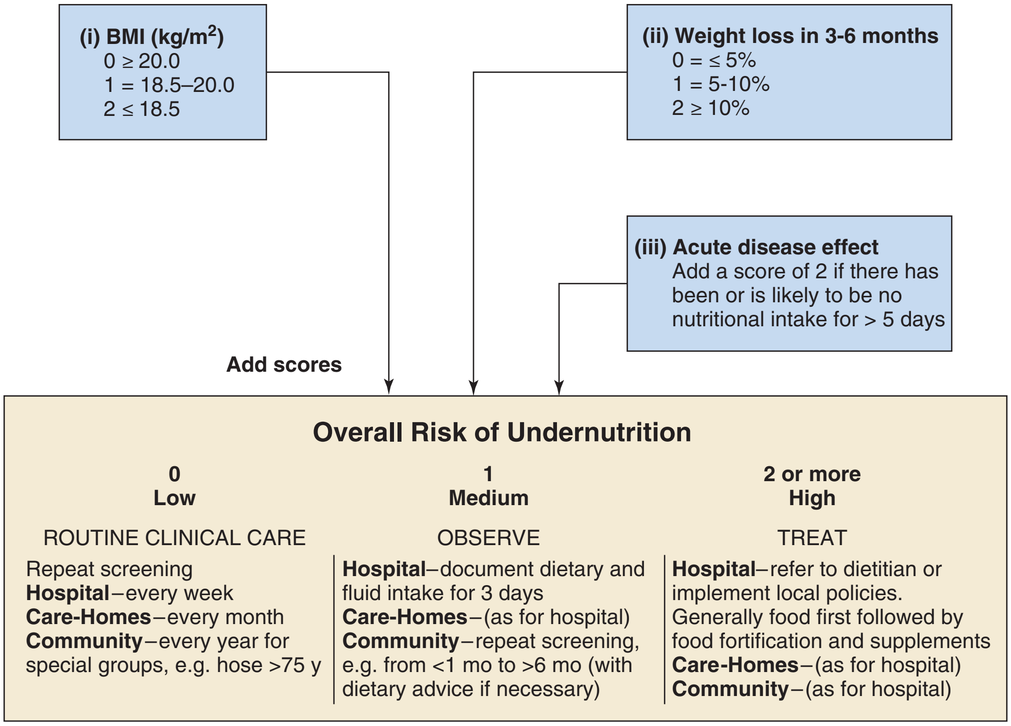

Figure 1.3 Malnutrition Universal Screening Tool. (With permission from Kondrup J, Allison SP, Elia M, Vellas B, Plauth M. ESPEN guidelines for nutrition screening 2002. Clinical nutrition (Edinburgh, Scotland). 2003;22(4):415-21.)

edema, and a high prevalence of acute infections.[77] Thus, it is evident that nutritional status is a key clinical parameter demanding thorough evaluation in the surgical patient to prevent nutrition-related complications.

Nutritional Status Assessment

Traditionally, clinicians relied on anthropometric measurements, which they compared with tables providing ideal weight-for-height estimates to evaluate the nutritional status of patients.[78] Clinicians also determined body mass composition determinants such as lean body mass based on limb skinfold or circumference measurements and used these variables as indicators for adequacy of nutrition. However, problems pertaining to the precision of anthropometric measurements, wide intra-observer and inter-observer variations, and the lack of reliable reference standards have challenged the validity of these methods in ascertaining nutritional health of the surgical patient.[74] These issues surrounding the traditional methods of screening for malnutrition led to an interest in studying serum markers for more accurate determination of preoperative nutritional competence. However, the use of serum markers to diagnose malnutrition is fraught with inaccuracy, as the most commonly used serum markers, albumin and prealbumin, are affected by multiple conditions other than malnutrition, including inflammation, liver disease, and kidney disease.

In the absence of reliable serum markers to judge nutritional status and challenges applying anthropometric measurements, the next best option for nutritional assessment may be screening tools such as the Malnutrition Universal Screening Tool (MUST)[79] or the NRS 2002.[79,80] MUST was designed to be used in the community and factors in BMI, weight loss, and acute disease effect, with referral to a dietitian recommended for a score ≥2 (Fig. 1.3). The NRS 2002, validated for in-hospital use, provides an initial screening based upon a BMI <20.5, recent weight loss, reduced dietary intake, and the presence of severe illness (Tables 1.1 and 1.2). During an inpatient hospitalization, nutritional plans are advised for patients with NRS-2002 scores of ≥3.

An evolving tool for preoperative assessment with bearing upon nutritional status is sarcopenia, a condition characterized by progressive and generalized loss of skeletal muscle mass and strength.[81] Sarcopenia is commonly measured by assessing psoas muscle density and total psoas area on preoperative CT scan.[82] Sarcopenia has been correlated with major postoperative complications in women following radical cystectomy[83] and with infectious complications in both men and women undergoing radical cystectomy.[84] While interesting and objective, measurement of total psoas area and psoas muscle density has not yet transitioned from retrospective research to mainstream clinical practice.

Preoperative Management of Malnutrition

While preoperative nutritional support has been found to improve surgical outcomes in other specialties, limited data exist in the urologic literature. Pilot studies with immunonutrition among bladder cancer patients undergoing radical cystectomy reveal an association of immunonutrition with decreased postoperative complications, including infections and paralytic ileus.[85,86] Larger studies are anticipated.

Applying data from other surgical fields, preoperative nutritional support may be useful for patients with severe malnutrition. In a study of 1085 abdominal surgery patients, patients with a NRS 2002 assessment score of ≥5 who received preoperative enteral or parenteral nutrition had fewer postoperative complications and a shorter length of stay as compared to those patients with severe malnutrition who did not receive preoperative nutritional supplementation.[87] When possible, enteral feeding, rather than parenteral, should be provided, as enteral nutrition is associated with fewer infectious complications and better glycemic control.[88] Among patients with mild-to-moderate malnutrition, there is no proven benefit to preoperative nutritional supplementation.[87,89] In a systematic review and meta-analysis of preoperative nutrition among patients undergoing gastrointestinal surgery, no differences in overall complications, infectious complications, or length of stay were identified among patients receiving preoperative liquid oral supplements as compared to those receiving usual care or dietary advice.[90]

Table 1.1 Initial Screening

1	Is BMI <20.5?	Yes	No
2	Has the patient lost weight within the last 3 months?		
3	Has the patient had a reduced dietary intake in the last week?		
4	Is the patient severely ill? (e.g., in intensive therapy?)		

Yes: If the answer is 'Yes' to any question, the screening in Table 1.2 is performed.
No: If the answer is 'No' to all questions, the patient is re-screened at weekly intervals. If the patient, e.g., is scheduled for a major operation, a preventive nutritional care plan is considered to avoid the associated risk status.

(With permission from Kondrup J, Allison SP, Elia M, Vellas B, Plauth M. ESPEN guidelines for nutrition screening 2002. Clinical nutrition (Edinburgh, Scotland). 2003;22(4):415-21.)

Infection and Urosepsis

Although community-acquired urinary tract infections (UTIs) are very common and are considered relatively easy to treat, complicated UTIs such as those acquired in the hospital setting are a legitimate cause for concern in urology. The term *complicated UTI* connotes infections brought about by a functional or anatomic abnormality in the urinary

Table 1.2 Final Screening

Impaired Nutritional Status		**Severity of Disease (≈ Increase in Requirements)**	
Absent Score 0	Normal nutritional status	Absent Score 0	Normal nutritional requirements
Mild Score 1	Wt loss >5% in 3 mths or food intake below 50–75% of normal requirement in preceding week	Mild Score 1	Hip fracture* Chronic patients, in particular with acute complications: cirrhosis*, COPD*. *Chronic hemodialysis, diabetes, oncology*
Moderate Score 2	Wt loss >5% in 2 mths or BMI 18.5–20.5 + impaired general condition or food intake 25–60% of normal requirement in preceding week	Moderate Score 2	Major abdominal surgery* Stroke* *Severe pnuemonia, hematologic malignancy*
Severe Score 3	Wt loss >5% in 1 mnth (>15% in 3 mths) or BMI <18.5 plus impaired general condition or food intake 0–25% of normal requirement in preceding week	Severe Score 3	Head injury* Bone marrow transplantation* *Intensive care patients (APACHE >10)*
Score	+	Score	= Total Score
Age	If ≥70 years: add 1 to total score above	= age-adjusted total score	

Score ≥3: the patient is nutritionally at risk, and a nutritional care plan is initiated.
Score <3: weekly screening of the patient. If the patient, e.g., is scheduled for a major operation, a preventive nutritional care plan is considered to avoid the associated risk status.

NES 2002 is based on an interpretation of available randomized clinical trials.
*indicates that a trial directly supports the categorization of patients with that diagnosis.
Diagnoses shown in *italics* are based on the prototypes given below.
Nutritional risk is defined by the present nutritional status and risk of impairment of present status, due to increased requirements caused by stream metabolism of the clinical condition.
A nutritional care plan is indicated in all patients who are (1) severely undernourished (score = 3), or (2) severely ill (score = 3), or (3) moderately undernourished + mildly ill (score 2+1), or (4) mildly undernourished + moderately ill (score 1 + 2).
Prototypes for severity of disease
Score = 1: a patient with chronic disease, admitted to hospital due to complications. The patient is weak but out of bed regularly. Protein requirement is increased but can be covered by oral diet or supplements in most cases.
Score = 2: a patient confined to bed due to illness, e.g., following major abdominal surgery. Protein requirement is substantially increased but can be covered, although artificial feeding is required in many cases.
Score = 3: a patient in intensive care with assisted ventilation, etc. Protein requirement is increased and cannot be covered even by artificial feeding. Protein breakdown and nitrogen loss can be significantly attenuated.
(With permission from Kondrup J, Allison SP, Elia M, Vellas B, Plauth M. ESPEN guidelines for nutrition screening 2002. Clinical nutrition (Edinburgh, Scotland). 2003;22(4):415-21.)

tract, but it may also be used to indicate an infection that occurs in a patient with altered defense mechanisms.[91] When an infection previously localized to the urinary tract enters the bloodstream and causes a systemic infection, urosepsis ensues.

Judicious use of prophylactic antibiotics in surgical procedures has served to minimize the incidence of these preventable yet potentially lethal complications in urologic practice.[92] However, the rising incidence of antimicrobial resistance, especially of gram-positive pathogens such as methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE), can lead to treatment failure and life-threatening sepsis.[93] Moreover, the increasing numbers of patients who are immunocompromised either by an underlying disease (e.g., HIV/AIDS) or through concurrent medical therapy (e.g., steroids, chemotherapy)[94] also lead to greater infection risk. These risk factors are particularly relevant when surgery entails instrumentation and manipulation of the urinary tract. Given that certain host factors predispose the urologic patient to complicated infection, it is necessary to determine the need for antimicrobial prophylaxis preoperatively and to prevent the occurrence of systemic septicemia.

Both demographic factors and medical conditions play a role in susceptibility to complicated UTI. Advanced age in a patient should alert the urologist to the possible presence of UTI. The prevalence of UTI increases with age and reaches approximately 3.6% in men ≥70 years old and 7% in women ≥50 years old.[95] As previously discussed, nutritional imbalances leading to obesity and malnutrition could impair cellular immunity and thereby predispose patients to UTI. Preexisting local or systemic infections intuitively are associated with complicated UTI.

Recent antimicrobial use has been linked to complicated UTI, possibly through two mechanisms: (1) antibiotic therapy fails, and the initial infection, either systemic or local, progresses to complicated UTI or frank urosepsis; or (2) antibiotics used to eliminate competing pathogens promote the growth of resistant strains and lead to infection with a more virulent strain.[96] Diabetes mellitus not only increases the incidence of UTI in adults but also contributes to a complicated course despite antibiotic prophylaxis and treatment. This situation is the result of defects in the secretion of urinary cytokines and increased adherence of microorganisms to the uroepithelial cells in diabetic patients.[97]

Not surprisingly, many urologic and medical renal conditions are associated with an increased incidence of complicated UTIs and urosepsis. One of the most consistent contributors to complicated UTI is obstruction of the urinary tract.[98] This underlying mechanism encompasses the following: intrinsic disorders of the kidney, renal pelvis, and ureters (e.g., congenital anomalies including vesicoureteral reflux, renal or ureteral calculi, neoplasms, strictures); extrinsic abnormalities of the upper urinary tract (e.g., aberrant vessels, retroperitoneal hematomas or fibrosis, nonurologic neoplasms); and disorders of the bladder and bladder neck (e.g., BPH, prostate and bladder cancer, cystolithiasis, bladder neck contracture) and urethra (e.g., valves, strictures). Functional impairment of the bladder, as seen in spastic or atonic neurogenic bladders, may have the same consequences as conditions causing physical obstruction.[99] Renal diseases, whether unilateral, bilateral, or segmental, may also complicate UTI and include conditions such as azotemia, polycystic kidney disease, and papillary necrosis, as well as nephropathies brought about by abuse of analgesics such as nonsteroidal antiinflammatory drugs.[100]

Immunosuppressed urologic patients present a unique problem with regard to susceptibility to complicated UTI. Whether impairment of immunologic response was brought about iatrogenically (e.g., patients with cancer who are undergoing chemotherapy, transplant recipients receiving steroids) or was the result of a disease process (e.g., HIV/AIDS, persistent neutropenia or granulocytopenia),[94] avid use of broad-spectrum antibiotics not only for common infections but also for opportunistic organisms should be considered by the urologist for an optimal clinical outcome. Finally, urologic instrumentation leads to an increased probability of introducing microorganisms into an otherwise sterile urinary tract and thus predisposes patients to infections. The same principle applies to urologic procedures in which foreign bodies are purposefully left in the human body (e.g., ureteral stents, penile prostheses).[101] Although intended to elicit only a minimal inflammatory response, any foreign body can serve as a nidus of infection and must be removed promptly when it is determined to be the source of infection or when its presence in the body contributes to a complicated UTI.

Quantifying Comorbidity

In medicine, *comorbidity* is defined as the effect of all other pathologic conditions an individual patient may have other than the primary disease of interest. The very nature of comorbidities, as secondary or lesser diseases of interest, has led to some indifference among practicing clinicians and research investigators regarding the significance of these illnesses in treatment decision making and survival outcomes. Because of the significant correlation between advanced age and increased prevalence of preexisting comorbidities at the time of surgery, physicians have traditionally used age as a surrogate for the effects of concurrent medical conditions, especially in elderly urologic patients.[102] Although no one can discount the value of age in treatment decisions, the use of age as a strict criterion that may deny appropriate curative therapy to healthy older patients is unacceptable and may even have litigious consequences.

The impact of comorbidities is substantial in the field of urology, particularly in urologic oncology. An analysis of 34,294 newly diagnosed cases of cancer in patients from the Netherlands Eindhoven cancer registry showed that, aside from lung cancer (58%) and stomach cancer (53%), the crude prevalence of comorbidities was highest in malignant diseases of the kidney (54%), bladder (53%), and prostate (51%).[103] In terms of prognosis, Post and colleagues[104] acknowledged that comorbidity was the most important prognostic factor for 3-year survival in a population-based study of 1337 patients with localized prostate cancer. In a series of 1023 consecutive radical nephrectomies and nephron-sparing surgical procedures for RCC in Dresden, Germany, comorbidities were closely associated with overall morbidity and mortality.[105] With regard to treatment-related side effects, both peripheral vascular disease and diabetes have been shown to be significant risk factors for development of impotence following external beam radiation for prostate cancer as well as for gastrointestinal and genitourinary toxicities.[106] Thus, comorbidities can affect almost all aspects of urologic disease but most importantly the incidence of posttreatment morbidity and all-cause death.

Up until the late 1980s, the effects of comorbidities were largely unquantifiable and subjective. As a result, certain beliefs and attitudes in clinical practice were based mostly on anecdotal data rather than on appropriate evidence-based information. The need for methods to quantify the effects of comorbidities led to the development of comorbidity scoring systems, which are gaining utility for both research and clinical purposes.

The most extensively studied and most commonly used comorbidity scoring scheme in medicine is the *Charlson Index score*.[107] Dr. Mary E. Charlson, a clinical epidemiologist and methodologist who was interested in improving clinical outcome in both medical and surgical patients, first published the index in 1987 at Cornell University in Ithaca, New York. The Charlson Index is a list of 19 pathologic conditions (Table 1.3). Based on the proportional hazards regression model that Charlson constructed from clinical data, each condition is assigned a weight from 1 to 6. The Charlson Index score is the sum of the weights for all concurrent diseases aside from the primary disease of interest. Thus, for example, in men with prostate cancer, although cancer is generally assigned a score of 2, in this case, men are assigned no points for prostate cancer because it is the primary index disease. In a cohort of 685 patients with breast cancer in the original study, the Charlson Index score showed a strong association of a 2.3-fold increase in the 10-year risk of mortality per 1-point increment in the comorbidity level.

Table 1.3 Weighted Index of Comorbidity [Defined by the Charlson Index]

Assigned Weights for Diseases	Conditions
1	Myocardial infarct Congestive heart failure Peripheral vascular disease Cerebrovascular disease Dementia Chronic pulmonary disease Connective tissue disease Ulcer disease Mild liver disease Diabetes
2	Hemiplegia Moderate or severe renal disease Diabetes with end organ damage Any tumor Leukemia Lymphoma
3	Moderate or severe liver disease
6	Metastatic solid tumor AIDS

Assigned weights for each condition that a patient has. The total equals the score. Example: chronic pulmonary (1) and lymphoma (2) = total score (3).

(With permission from Charlson ME, Pompei P, Ales K, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis. 1987;40:373-83.)

The Charlson Index score provides a simple means to quantify the effect of comorbid illnesses, incorporate the severity of a particular disease (diabetes without complications versus diabetes with end-organ damage), and account for the aggregate effect of multiple concurrent disease processes on clinical outcome, most often mortality. In prostate cancer research, the Charlson Index score has been extensively evaluated as a predictor of both cause-specific mortality and all-cause mortality. Albertsen and colleagues[108] showed that the Charlson Index score provided significant predictive information on cancer-specific and all-cause survival independent of age, Gleason score, or clinical stage in a cohort of 451 patients with Jewett-Whitmore stage A1-B prostate cancer treated with hormonal ablation. In 2002, a competing risk analysis of 751 men who underwent radical prostatectomy for clinically localized prostate cancer at the Mayo Clinic in Rochester, Minnesota, showed that, whereas the Gleason score emerged as the only significant predictor of prostate cancer–specific mortality, both the Charlson Index score and the Gleason score were predictive of overall mortality.[109] Comparable results were reported by other groups who performed similar analyses using Charlson Index scores in prostate cancer outcome studies.[104,110,111] With recent efforts to minimize the overtreatment of men with prostate cancer, the Charlson Index has a critical predictive role. In a retrospective study of 1482 men with nonmetastatic prostate cancer diagnosed from 1997 to 2004, each point increase in Charlson score was associated with a twofold increase in hazard of nonprostate mortality. After stratification by tumor risk, prostate cancer mortality was rare among low-risk and intermediate-risk groups, leading to the recommendation that men with the highest Charlson scores should consider conservative management of low-risk and intermediate-risk tumors.[112,113] In a larger study of 140,553 men aged ≥ 66 years with early-stage prostate cancer who were diagnosed between 1991 and 2007 from the Surveillance, Epidemiology, and End Results-Medicare database, men with Charlson scores ≥ 3 were found to garner no survival benefit from aggressive treatment.[114]

The clinical utility of the Charlson Index score extends to other urologic diseases as well. With regard to bladder cancer, the Charlson Index score was evaluated in predicting adverse pathologic characteristics, cancer-specific death, and overall survival following radical cystectomy.[115] Logistic regression revealed that the Charlson Index score was independently associated with an increased risk of extravesical disease. Cox regression models further revealed that the index was significantly associated with decreased cancer-specific survival. In 302 men undergoing transurethral resection of the prostate (TURP) or simple prostatectomy for BPH, the Charlson Index score correlated with 5-year mortality.[116] Thus, even for nononcologic urologic operations, comorbidity indices have demonstrated power for predicting mortality following surgery.

Other indices of comorbidity are available but have not been as broadly used as the Charlson Index. Three prime examples are the Index of Co-Existent Disease (ICED),[117] the Kaplan-Feinstein Index (KFI),[118] and the Cumulative Illness Rating Scale (CIRS).[119] Similar to the Charlson Index, the

ICED, KFI, and CIRS are designed to measure the impact of concurrent diseases on prognosis. Because of the lack of definitive head-to-head comparisons of the various comorbidity assessments, no clear-cut evidence exists to establish the advantage of one scale over the other.[120] In fact, in a study in which Charlson Index scores were shown to be predictive of 5-year mortality following TURP for BPH, similar analysis using the KFI and ICED demonstrated comparable predictive power.[116]

Conclusion

It is the ultimate goal of every urologist to provide the best possible care for the urologic patient. The projected surge in life expectancy in the new millennium translates into an analogous increase in urologic patients who will potentially present with various comorbid diseases. These patients will require thorough evaluation including the addressing of associated comorbidities to obtain an excellent outcome. The impact of host factors and comorbidities cannot be taken lightly because more and more scientific evidence points to associations of these pretreatment parameters with a heightened risk for undesirable posttreatment complications. In particular, obesity, which is associated with other significant comorbidities and has been found to affect both the urologic disease process and consequent complications, must be investigated comprehensively. Furthermore, adequate assessment of nutritional status to ensure sufficient nutritional support in the surgical patient is also warranted.

Patients with host factors that predispose them to infections may require prophylactic antibiotic coverage and must be closely monitored to anticipate the need for further antimicrobial treatment to prevent urosepsis. Finally, various comorbidity scoring systems are being investigated for their clinical value and may further provide urologists and other clinicians with more accurate predictive models for assessing the risk of complications among patients with urologic diseases. As subsequent chapters in this book delve into more organ-specific, urologic disease–specific, or procedure-specific complications, we encourage the readers to make every effort in taking a broad, encompassing approach when evaluating urologic patients by diligently considering the effects of comorbid conditions in each individual person.

References

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Chapter 1 Questions

1. By 2050, the estimated population of the United States ≥65 years of age will be:
   a. 25 million
   b. 42 million
   c. 67 million
   d. 88 million
   e. 110 million

2. Obesity is associated with increased rates of:
   a. Erectile dysfunction
   b. Stress urinary incontinence
   c. Kidney cancer
   d. Uric acid nephrolithiasis
   e. All of the above

3. What is the calculated BMI of a patient who is 6'0" tall and weighs 221 pounds?
   a. 24.3
   b. 27.8
   c. 30.2
   d. 33.2
   e. 38.6

4. Compared to men of normal weight, obese men have:
   a. Lower PSA levels
   b. Lower PCA3 levels
   c. Lower % free PSA levels
   d. Higher PSA levels
   e. Higher PCA3 levels

5. A validated tool that may be used for the assessment of inpatient nutritional status is:
   a. NRS 2002
   b. MUST
   c. MMSE
   d. Albumin
   e. Prealbumin

6. A 79-year-old male with muscle invasive bladder cancer is scheduled to undergo a radical cystectomy. He has a history of chronic obstructive pulmonary disease, congestive heart failure, obesity, and obstructive sleep apnea. What is his Charlson comorbidity score?
   a. 1
   b. 2
   c. 3
   d. 4
   e. 5

7. Which of the following statements regarding prostate cancer and obesity is *incorrect*?
   a. Obese men with prostate cancer have lower serum prostate-specific antigen levels relative to men of normal weight
   b. Among men undergoing an open retropubic prostatectomy, increasing BMI is associated with increasing operative time and increasing intraoperative blood loss
   c. Mildly obese men undergoing radical prostatectomy have worse health-related quality of life postoperatively than men of normal weight
   d. Men with higher BMIs present with higher-grade tumors and more advance pathologic stages
   e. Increased body weight was found to be associated with an increased risk of death from prostate cancer in a large, prospectively studied population[31]

8. A 67-year-old male just underwent emergent stent placement for a 7 mm proximal right ureteral calculus with associated hydronephrosis, a fever of 101.7°F (38.7°C), and a blood pressure of 86/57. Risk factors in his history that increase his risk for urosepsis include:
   a. Ongoing cisplatin and etoposide chemotherapy for treatment of small cell lung cancer
   b. An indwelling Foley catheter for a recent episode of acute urinary retention
   c. Prednisone taper following a recent diagnosis of herpes zoster ophthalmicus
   d. Obstructing urolithiasis
   e. All of the above

9. In urologic diseases, comorbidity indices have demonstrated power for predicting mortality among patients undergoing:
   a. RPLD for testis cancer
   b. InterStim placement for refractory urinary urgency/frequency
   c. Transobturator sling placement for stress urinary incontinence
   d. Transurethral resection of the prostate for BPH
   e. Percutaneous nephrolithotomy for nephrolithiasis

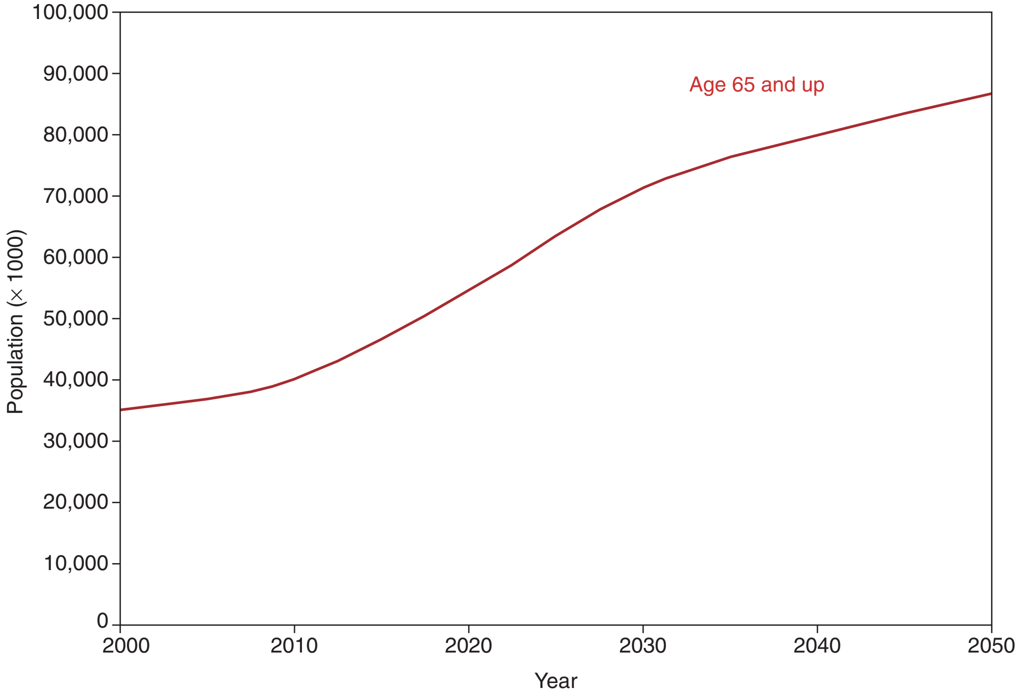
10. The most important prognostic factor for 3-year survival among men with localized prostate cancer is:
    a. Gleason score
    b. PSA doubling time
    c. Comorbidities
    d. Clinical stage
    e. Interval of time between initial treatment and biochemical recurrence

Answers

1. **d.** According to the US Census Bureau, and as illustrated in Case Fig. 1.1, the US population ≥65 years of age is exponentially growing and is estimated to reach approximately 88.5 million by 2050.
2. **e.** In Dr. Mydlo's article "The impact of obesity in urology," he reviewed that obesity is a risk factor for stress urinary incontinence, erectile dysfunction, infertility, renal cell carcinoma, and renal calculi.
3. **c.** Using the formula BMI = Weight (in kilograms)/height (in meters) squared, 100 kg/$(1.82\ \text{m})^2$ = 30.2. Alternatively, using Case Fig. 1.2, a man who is 6'0" tall and weighs 220 pounds has a BMI of 30, falling within the mildly obese group.
4. **a.** In Baillargeon's population-based study of 2779 men without prostate cancer, mean PSA decreased in

a linear fashion with an increase in BMI category, from 1.01 ng/mL in normal-weight men to 0.69 ng/mL in obese men, after adjusting for race/ethnicity and age.

5. **a.** The NRS 2002 is a predictive, validated assessment tool for screening of undernutrition in hospitalized patients. In contrast, the Malnutrition Universal Screening Tool (MUST) is used to detect undernutrition among community-dwelling adults. While widely used as assessments for nutritional status, albumin and prealbumin are affected by many disease processes, including liver disease, kidney disease, and acute inflammation and are therefore unreliable for monitoring nutritional status.
6. **b.** The patient receives 1 point each for his diagnoses of chronic obstructive pulmonary disease and congestive heart failure. As his bladder cancer is our primary disease of interest, the patient receives no points for that diagnosis.
7. **c.** Thus far, prospective studies have failed to demonstrate large differences in postoperative health-related quality of life between mildly obese men and men of normal weight.
8. **e.** One of the most consistent contributors to complicated urinary tract infections is obstruction of the urinary tract, including obstruction due to ureteral calculi. Indwelling catheters are associated with an increased risk of urosepsis. Patients on chemotherapy or immunosuppression due to steroids are also at increased risk for urosepsis.
9. **d.** In Krousel-Wood et al.'s study of 302 men undergoing transurethral resection of the prostate (TURP) or simple prostatectomy for BPH, the Charlson Index score correlated with 5-year mortality.
10. **c.** Post and colleagues found that comorbidity was the most important prognostic factor for 3-year survival in a population-based study of 1337 patients with localized prostate cancer.



Case Figure 1.1

BODY MASS INDEX (kg/m²)

WEIGHT (lbs)

HEIGHT (ft / in)	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215	220	225	230	235	240	245	250	HEIGHT (cm)
6'2"	14	15	15	16	17	17	18	19	19	20	21	21	22	22	23	24	24	25	26	26	27	28	28	29	30	30	31	31	32	188
6'1"	15	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	28	29	30	30	31	32	32	33	185
6'	15	16	16	17	18	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	28	29	30	31	31	32	33	33	34	183
5'11"	15	16	17	17	18	19	20	20	21	22	22	23	24	24	25	26	26	27	28	29	29	30	31	31	32	33	33	34	35	180
5'10"	16	17	17	18	19	19	20	21	22	22	23	24	24	25	26	27	27	28	29	29	30	31	32	32	33	34	34	35	36	178
5'9"	16	17	18	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30	31	32	32	33	34	35	35	36	37	175
5'8"	17	17	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	30	31	32	33	33	34	35	36	36	37	38	173
5'7"	17	18	19	20	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32	33	34	34	35	36	37	38	38	39	170
5'6"	18	19	19	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	32	33	34	35	36	36	37	38	39	40	40	168
5'5"	18	19	20	21	22	22	23	24	25	26	27	27	28	29	30	31	31	32	33	34	35	36	37	37	38	39	40	41	42	165
5'4"	19	20	21	21	22	23	24	25	26	27	27	28	29	30	31	32	33	33	34	35	36	37	38	39	39	40	41	42	43	163
5'3"	19	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	35	36	37	38	39	40	41	42	43	43	44	160
5'2"	20	21	22	23	24	25	26	27	27	28	29	30	31	32	33	34	35	36	37	37	38	39	40	41	42	43	44	45	46	158
5'1"	21	22	23	24	25	26	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	43	44	45	46	47	155
5'	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	152
WEIGHT (kg)	50	52	54	56	59	61	63	65	68	70	72	74	77	79	81	83	86	88	90	92	95	97	99	101	104	106	108	110	113	

WEIGHT (kg)

UNDERWEIGHT (BMI<18.5 kg/m²) · NORMAL (BMI=18.5–24.9 kg/m²) · OVERWEIGHT (BMI=25–29.9 kg/m²) · MILDLY OBESE (BMI=30–34.9 kg/m²) · MODERATELY OBESE (BMI=35–39.9 kg/m²) · SEVERELY OBESE (BMI≥40 kg/m²)

Case Figure 1.2

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2 Preoperative Pulmonary Assessment and Management of Pulmonary Complications

JAMES S. WYSOCK

CHAPTER OUTLINE

Preoperative Pulmonary Assessment
Patient Factors
Cigarette Smoking
Procedure Factors
Surgical Approach and Anatomic Considerations
Anesthesia
Preoperative Patient Evaluation
Preoperative Strategies
Postoperative Strategies
Incentive Spirometry and Deep Breathing Exercises
Early Mobilization
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KEY POINTS

1. Accurate preoperative identification of patients at risk for developing postoperative pulmonary complications optimizes preoperative testing and ideally minimizes occurrence of postoperative complications.
2. Risk factors for respiratory complications stem from patient-related factors as well as from procedure-related factors including surgical site, duration of surgery, and type of anesthesia. Procedure-related factors carry the highest risk for postoperative pulmonary complications.
3. Urologists should identify and inform patients who smoke of the associated risk factors for pulmonary complications as well as for urothelial cancer. Cigarette smoking cessation should be 4 weeks or more prior to surgery to begin to provide benefit.
4. Multiple new preoperative risk calculators are available to assist in assessing risk stratification. The most important patient-related risk factor is age, increasing significantly over the age of 50.
5. Recommendations for patients undergoing major urologic procedures include intermittent pneumatic compression, use of graduated compression stockings, early and frequent ambulation, in addition to pharmacologic venous thromboembolism (VTE) prophylaxis when possible.

Multiple studies evaluating complications following noncardiac surgery demonstrate that postoperative pulmonary complications (PPCs) occur at similar frequency and result in similar rates of morbidity and mortality as cardiovascular complications.[1,2] The reported rates of PPCs range from 5% to 40%, dependent upon procedure risk, with PPC-associated mortality risks ranging from 10% to 25%.[3–7] Data from the National Surgical Quality Improvement Program (NSQIP) demonstrate that PPCs are the most costly of postoperative complications and result in the longest hospital length of stay.[8]

Although there is no exact definition of what constitutes a PPC, most literature includes conditions such as atelectasis, infection (pneumonia, bronchitis), respiratory failure requiring prolonged mechanic respiratory support, acute respiratory distress syndrome (ARDS), and hypoxemia.[7,9] For the purposes of this chapter, we have also included discussion regarding pulmonary embolism and additional surgical complications such as pneumothorax. The economic impact of PPCs has been estimated to account for 3.4 billion dollars in health care costs in the United States.[10] Ultimately, prevention of PPC requires careful assessment of risk factors, both from patient characteristics and procedure-associated factors. Proper identification of these risk factors aids development of strategies to prevent these complications.

Preoperative Pulmonary Assessment

Identifying the primary risk factors associated with PPCs assists with accurate risk stratification prior to surgery and informs strategies to reduce morbidity and mortality. Pulmonary risks factors stem from two sources, preexisting patient characteristics as well as procedure-associated factors.

Table 2.1 Primary Patient Related Risk Factors From the American College of Physicians Guideline

Risk Factor	Odds Ratio for PPC	95% Confidence Interval
Age		
50–59	1.50	1.31–1.71
60–69	2.09	1.65–2.64
70–79	3.04	2.11–4.39
≥80	5.63	4.63–6.85
ASA Score ≥2	4.87	3.34–7.10
Congestive heart failure	2.93	1.02–8.03
Total functional dependence	2.51	1.99–3.15
COPD	2.36	1.90–2.93
Cigarette use	1.40	1.17–1.69

COPD, chronic obstructive pulmonary disease; PPC, postoperative pulmonary complication

(Adapted from Smetana GW, Lawrence VA, Cornell JE, American College of Physicians. Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. Ann Intern Med. 2006;144(8):581-95.)

PATIENT FACTORS

Identifying factors that predispose to the development of PPC requires an understanding of the principal patient-related risk factors as well as an understanding of the degree of risk associated with these factors. The American College of Physicians (ACP) provides guidelines for assessing PPC risk factors for noncardiopulmonary surgery. These guidelines stem from a systematic review of the existing literature performed by Smetana and colleagues.[1,2] This work, published in 2006, remains the largest evidence-based source of information regarding PPC. From these data, the primary patient-related factors associated with increased risk of PPCs and are detailed in Table 2.1.

The most important patient-related risk factors found by this review are patient age and presence of an increased American Society of Anesthesiologist's (ASA) score.[1,11] Age confers an increasing level of risk for PPC for each decade over 50. Ultimately, as age increases beyond 80, the odds ratio increases to 5.63 as compared to patients younger than 50. Given that the population requiring major urologic surgery is often in this age range, this risk factor becomes especially pertinent to consider during urologic preoperative evaluation.[12,13]

The existence of chronic lung diseases such as chronic obstructive pulmonary disease (COPD) warrants special consideration when assessing PPC risk. In fact, the presence of chronic lung disease represents the most common risk factor identified by Smetana and colleagues. However, the degree of this risk factor varies widely upon the degree of pulmonary impairment. Consequently, the presence of preoperative congestive heart failure (CHF) confers a higher risk for PPC than COPD (OR 2.93 vs 2.36, respectively).

Additional significant risk factors for PPC include active cigarette use and decreased overall functional dependence. More recently, data regarding obstructive sleep apnea (OSA) indicate that OSA confers an increased risk for immediate airway management complications as well as for PPC.[14–16] Patients should thus be screened for signs and symptoms of OSA prior to undergoing anesthesia. Several tools for assessing for OSA have been published and assist with assessing level of OSA risk.[17,18]

Interestingly, this systematic review did not find strong evidence demonstrating an increased risk of pulmonary complications amongst patients with obesity and controlled asthma, findings corroborated by additional studies.[3,19] However, obesity remains a risk factor for development of venous thromboembolic complications including pulmonary embolism and thus remains an important patient factor clinicians should consider when assessing risk.

While the ACP guideline provides a necessary evidence-based framework for assessing risk for PPC, the data have not been systematically updated for nearly 10 years. More recent data indicate that additional risk factors include respiratory infections within 1 month prior to surgery, diabetes, preoperative hypoxemia, and preoperative anemia.[3,4,20–22] Currently, prospective studies are under way to improve PPC definition and identify more accurate risk factors.[23]

Given that most patients requiring urologic intervention are over the age of 50, and many patients undergoing procedures associated with urothelial carcinoma have a history of significant tobacco use, the risk of PPC in this patient cohort warrants special attention. Prior exposure to bleomycin presents an additional urologic-specific patient risk factor that will be discussed in more detail later in the chapter.

CIGARETTE SMOKING

When considering patient-related factors, cigarette smoking warrants additional commentary. Smoking tobacco is a well-known risk factor for cardiac, vascular, infectious, and respiratory complications.[24–27] Many PPCs occur at a higher rate in smokers, conferring an increased risk with an odds ratio of 1.4 to 4.3.[6,28] Tobacco smoking confers additional risk for urologic malignancies, increasing the risk of renal cell carcinoma and urothelial carcinoma, and continued tobacco use is associated with disease progression and recurrence.[29–34]

In their prospective study of 200 patients undergoing coronary artery bypass surgery, Warner and associates demonstrated that patients who had stopped smoking ≥8 weeks preoperatively had a significantly lower risk of pulmonary complications than did patients who were active smokers (14.5% vs 33%).[35] Moreover, patients who had stopped smoking for >6 months had pulmonary complication rates similar to those patients who had never smoked (11.1% vs 11.9%). Surprisingly, patients who had quit smoking <8 weeks preoperatively experienced more untoward pulmonary events than did active smokers (57% vs 33%). This appears to be due to increased airway reactivity and sputum production that occur in the initial period after smoking cessation.[9] Recent studies have corroborated these findings, indicating that smoking cessation ultimately benefits patients but requires 4 to 6 weeks to provide benefit.[36–38]

Given the important role cigarette smoking plays both in the pathophysiology of urologic malignancies and on the risk of PPC as well as other major postoperative complications, the urologist is in a unique position to intervene and provide guidance on smoking cessation. In addition, observational studies indicate that patients may be more receptive to quitting during the perioperative period.[39,40] A recent prospective study within a urology clinic demonstrated the efficacy of this approach.[41]

Patients who smoke should be identified preoperatively. The duration and amount of smoking should be quantified. These patients should be informed that smoking increases the perioperative risk and also that quitting can decrease this risk. Preoperative abstinence should be recommended for a minimum period of 4–6 weeks prior to surgery.

PROCEDURE FACTORS

Procedure-related factors associated with risks for PPC are displayed in Table 2.2 and are adapted from the ACP guideline.[1,2] In terms of risk assessment, procedure relegated factors represent the most significant predictors for development of PPC.

Table 2.2 Primary Procedure Related Risk Factors (American College of Physicians Guideline)

Risk Factor	Odds Ratio for PPC	95% Confidence Interval
Surgical site		
Aortic	6.90	2.74–17.3
Thoracic	4.24	2.89–6.23
Any abdominal	3.01	2.43–3.72
Upper abdominal	2.91	2.35–3.60
Emergency surgery	2.21	1.57–3.11
Surgery >3 hours	2.26	1.47–3.47

PPC, postoperative pulmonary complication

(Adapted from Smetana GW, Lawrence VA, Cornell JE, American College of Physicians. Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. Ann Intern Med. 2006;144(8):581-95.)

Surgical Approach and Anatomic Considerations

Special consideration should be paid toward surgical anatomic location. While certain surgical sites (aortic, thoracic, neurologic) are not common in urologic procedures, it is important to note that abdominal surgical location confers an odds ratio of 3.01 and thus is a critical factor to be considered as risk factor for PPC. Regardless of proximity to the diaphragm, surgical trauma to the abdomen impacts respiratory function. Abdominal incisions decrease lung volumes in a restrictive pattern, resulting in reduction in vital capacity of 50–60% and functional residual capacity by approximately 30%.[42,43] Incisional pain limits respiratory motion, often subconsciously, promoting development of atelectasis and pleural effusions. Manipulation of the intestines during transperitoneal dissection can result in phrenic nerve stimulation, which further impairs normal respiratory function.[9,44]

Urologic procedures that involve, or are in close proximity to, the diaphragm represent a high-risk group. This category includes surgical approaches utilizing flank, subcostal, and thoraco-abdominal incisions. Additionally, minimally invasive renal surgery, upper retroperitoneal dissection, and percutaneous renal access present anatomic risk to the pulmonary space. Proximity to the diaphragm and pleural cavity may result in both obvious and discrete injury to the diaphragm and entry into pleural space. While these injuries are often recognized and managed intraoperatively, opportunity for occult injury should still be considered and postoperative assessment should address this risk.

Additional procedure-related factors include prolonged surgical time (>3 hours) and emergent procedures.[1,2]

Anesthesia

Evidence also supports that general anesthesia represents an independent risk factor for PPC. Induction of general anesthesia results in an immediate development of atelectasis due to collapse of lung tissue, ventilation-perfusion mismatch, creation of respiratory dead-space, hypoxemia, and decreased surfactant function.[9] Narcotics, sedatives, and other centrally acting drugs may depress respiratory drive and further increase risk for PPC.

PREOPERATIVE PATIENT EVALUATION

The evaluation of preoperative pulmonary risk begins with a detailed history and physical examination. Patients with a smoking history should be identified and efforts included to quantify duration and amount of smoking. A history of chronic lung disease should also be identified. A recent history of an upper respiratory infection, including influenza, should also be identified. A thorough pulmonary evaluation such as exercise intolerance, chronic cough, sputum production, previous pulmonary surgery, previous chemotherapy (see later), dyspnea at rest or on exertion, wheezing, rales, cyanosis, or weakness or debilitation assist in identifying patients at risk. Evaluation for signs of sleep apnea may also help with risk stratification.[17]

A chest radiograph should be obtained in all patients with any of the above risk factors or over the age of 50. However, clear evidence supporting a benefit from obtaining routine chest radiographs does not currently exist.

Evidence indicates that preoperative pulmonary function testing (PFT) does not accurately predict PPC, and consequently these tests are overused.[45,46] Debate continues regarding the value of PFT. The ACP systematic review concluded that PFT is reasonable for patients with COPD or asthma in whom clinical evaluation is unclear and in patients with unexplained dyspnea or exercise intolerance.[2,47]

As preoperative hypoalbuminemia predicts PPC, recognition of patients with severe malnourishment should be identified, and it is reasonable to recommend preoperative nutritional supplementation.[48,49]

Several risk calculators have been developed to provide quantitative assessment for pulmonary risk and are available online.[50–53] One example is the ARISCAT risk score, which was developed and validated from European surgical databases and uses five risk factors to predict risk of PPC.[3,54] This is shown in Table 2.1. Implementation of these risk calculators offers numerical risk evaluations that may assist with clinical decision making.

Preoperative Strategies

Once a patient is identified as having a higher risk for PPC, several strategies may be employed to decrease that risk. As stated above, urologists should instruct and guide current smokers to stop smoking and provide tools and referrals for patients that are willing to attempt cessation.

Patients with chronic lung disease who do not appear to be in optimal status should have surgery deferred until their pulmonary status has been medically optimized. Patients with a history of asthma should also be assessed for how well their asthma is controlled. At the time of surgery these patients should be free of wheezing and should use beta-agonist inhalers prior to intubation and during the perioperative period. Any signs of a recent upper respiratory infection, even for patients without a history of lung impairment, should prompt further workup and delay in surgical timing until recovery from respiratory infection is complete.

Preoperative pulmonary physical therapy has been shown to reduce postoperative atelectasis and hospital stay in patients undergoing cardiac surgery.[55–57] Preoperative teaching with incentive spirometry for all patients undergoing elective abdominal surgery results in improved understanding of the techniques and perioperative use.[58] It is our practice to provide this teaching and to reinforce incentive spirometry in the perioperative period.

Postoperative Strategies

The strategy for postoperative pulmonary rehabilitation differs from preoperative approaches in that postoperative tactics apply to every patient, regardless of preoperative risk level. These strategies focus on maneuvers aimed to counteract the surgical and anesthesia impairments on respiratory function. Adequate pain control and lung expansion through incentive spirometry (IS), coughing, and early ambulation comprise the cornerstone of such strategies.

INCENTIVE SPIROMETRY AND DEEP BREATHING EXERCISES

While a few small trials demonstrate that utilizing postoperative IS and deep breathing exercises (DBE) reduces PPC and length of stay,[58–60] a recent Cochrane review found no evidence that IS prevented PPC.[61] The authors concluded that the evidence for this conclusion is of low quality and that higher quality trials are needed to evaluate the efficacy of IS.

It remains our practice to employ incentive spirometry in both the immediate postoperative and perioperative period.

EARLY MOBILIZATION

Facilitating early ambulation improves fluid mobilization and increases respiratory utilization. While very few studies provide guidance on the impact of mobilization, one study demonstrated an increase in PPC for each additional day of delay in mobilization following abdominal surgery.[62] It remains our practice to encourage ambulation and routinely employ the assistance of physical therapists for patients that require assistance.

PAIN CONTROL

Adequate pain control ameliorates the pulmonary functional depression induced by surgical incisions and assists with early ambulation. However, use of opioid efficacy is limited by subsequent depression of the respiratory drive. Epidural anesthesia has been associated with fewer PPCs, including in patients with COPD undergoing abdominal surgery.[63,64] Intercostal nerve blockade may also help reduce PPC in patients undergoing subcostal or upper abdominal incisions.[65] For patients with risk factors for PPC, consideration of epidural anesthesia or intercostal nerve block should be considered when applicable.

NASOGASTRIC TUBE

Routine use of postoperative nasogastric tube (NGT) increases PPC including pneumonia and atelectasis, although on Cochrane review this trend was insignificant ($p = 0.07$).[66,67] We do not recommend routine postoperative NGT and employ this only in situations when worsening postoperative nausea, distention, or bilious vomiting develop due to postoperative ileus.

Bronchospasm

Bronchospasm in the postoperative setting may occur due to histamine release from perioperative medications, aspiration, and exacerbation of underlying pulmonary disease such as asthma or COPD. Management rests upon the identification of an underlying cause and treating it appropriately. Removal of offending drugs or allergens and addressing asthma or COPD with inhaled bronchodilators represent first-line strategies.[68] For refractory cases, consider employing systemic steroids.

Atelectasis

Atelectasis commonly occurs following abdominal surgery, affecting patients in all ranges.[69,70] A consequence of surgical and anesthetic effects, atelectasis develops when dependent airways collapse, ultimately leading to changes in the compliance of lung tissue, decreased ventilation in these regions, and reduced movement of airway secretions.[71,72–74] These changes result in decreased oxygen exchange and increase respiratory effort and hypoxemia. In younger healthy patients, these changes may result in little to no clinical effect but may significantly alter the clinical course for patients with impaired lung function or comorbidities such as CHF.

Atelectasis develops in the early postoperative course with symptoms of hypoxemia typically becoming apparent approximately 48–60 hours following surgery and continuing for several days.[75,76] Hypoxemia that develops earlier than this time course (i.e., in the postanesthesia recovery unit) should prompt urgent investigation into alternative PPC including pneumothorax, upper airway edema, and anesthesia effects. Atelectasis in the postoperative setting is most commonly nonobstructive in nature and typically results from loss of contact between the parietal and visceral pleurae or compression of the lower and middle lobes, often due to pleural effusion.[77]

Within the appropriate clinical time course, atelectasis should be suspected when findings such as tachypnea, dyspnea, and hypoxemia are noted. Patients should be assessed for abundant sputum production and secretions, evident as rhonchi on auscultation or frequent productive cough. Obtaining a chest radiograph or chest computed tomography (CT) often confirms the diagnosis.[78]

Management of atelectasis through methods to reverse the process of lung volume collapse can prevent further PPC.[79] Strategies for managing atelectasis differ depending upon the level of airway secretions. For those without abundant secretions, implementation of supplemental oxygen, DBE, and IS remains the mainstay of initial management. Patients without secretions that remain hypoxic and with continued increased respiratory effort may benefit from positive airway pressure (CPAP). Squadrone et al. reported results of a randomized multicenter controlled trial of CPAP versus supplemental oxygen alone and demonstrated a decrease in intubation, pneumonia, and sepsis for patients receiving CPAP.[80] For patients with significant secretions, frequent chest physiotherapy and suctioning (nasogastric if the patient is not able to expectorate) should be implemented. In patients who fail to improve with these methods, consider pulmonary consultation for consideration of possible bronchoscopy and other techniques aimed to improve secretion clearance.[81,82]

Pleural Effusion

Limited data suggest that pleural effusion detected on chest radiograph occurs in up to 49% of patients' surgery when evaluated between 48 to 72 hours following abdominal surgery.[83] Pleural effusion appears to occur more commonly following upper abdominal surgery. Management of effusion is primarily conservative, as most of effusions resolve spontaneously. Pulmonary toilet with DBE and IS as well as early and frequent ambulation may serve to hasten resolution.

In the setting of persistent effusion or clinical concern, diagnostic evaluation of an effusion requires thoracentesis.[84] Thoracentesis can be performed at the bedside, and analysis can help establish diagnoses including malignancy, empyema, chylothorax, urinothorax, hemothorax, and infection.[85]

Postoperative Pneumonia

The incidence of postoperative pneumonia ranges from 1.5% to 50%, depending on a wide array of conditions including type of anesthesia, surgical details, and patient risk factors.[86,87] Postoperative pneumonia can present similarly to other conditions, such as atelectasis, pulmonary edema, and pulmonary embolism.[88] However, postoperative pneumonia requires accurate diagnosis as it is associated with mortality rates from 1.5% to 10%.[87,89]

Postoperative pneumonia should be considered in patients with fevers, increased white blood cell count, and chest imaging demonstrating new onset pulmonary infiltrates. Clinical symptoms of fever, tachypnea, dyspnea, and hypoxemia typically develop within the first five postoperative days.[90,91] Risk factors such as COPD, altered lung defenses, and active smoking should increase clinical suspicion.

Successful management of postoperative pneumonia requires accurate pathogen identification and appropriate antibiotics.[92] These infections, primarily nosocomial in origin, frequently result from resistant organisms, although multiple organisms may be identified. One study demonstrated that the most common pathogens were gram-negative bacteria (e.g., *Pseudomonas aeruginosa, Klebsiella pneumoniae, Acinetobacter* species), *Staphylococcus aureus,* and *Streptococcus pneumoniae.*[90] Prolonged intubation and exposure to antibiotics as well as COPD may increase risk for pneumonia due to organisms such as *Pseudomonas aeruginosa, Acinetobacter,* or methicillin-resistant *S. aureus* (MRSA).[93–96] Postoperative treatment requires accurate collection of respiratory culture and prompt empiric antibiotics.

Aspiration pneumonia during the postoperative period may result in patients with risk factors for decreased protection of their airways. These include neurologic disorders, impaired cough reflex, impaired mental status following surgery leading to dysfunctional swallowing, and postoperative vomiting. Introduction of gastric fluids or particulate matter into the lower airways may stimulate an inflammatory response and subsequent chemical pneumonitis, bacterial infection, or mechanical obstruction.[97–99]

Chemical pneumonitis occurs quickly after aspiration of gastric contents and may lead to dyspnea and respiratory distress.[100] Clinical symptoms include abrupt dyspnea, cyanosis, severe hypoxemia, and infiltrates on chest imaging. The inflammatory response may rapidly progress to acute respiratory distress syndrome (ARDS) and lead to respiratory failure.[101] As ARDS develops, inflammation of the lung parenchyma results in systemic release of inflammatory mediators and frequently results in multisystem organ failure.[102] Treatment of ARDS involves mechanical ventilation, treatment of underlying causes, supportive care, and antibiotic coverage if indicated.

Gupta and colleagues have developed and validated a risk calculator for predicting postoperative pneumonia. This tool is available for free download at surgicalriskcalculator.com.[53]

Postoperative Management for Patients With Obstructive Sleep Apnea

More than half of patients with clinically important obstructive sleep apnea (OSA) present for surgery without formal diagnosis of OSA.[103,104] As the prevalence of obesity continues to rise, the rates of undiagnosed OSA in surgical patients will likely continue to increase.[105] As a result of the large number of patients that are undiagnosed, heightened vigilance for postoperative complications in patients with risk factors for OSA, such as obesity, should be considered.

Hypoventilation, periods of central apnea and upper airway obstruction combine to lead to rapid hypoxemia for patients with OSA. Consequently, careful monitoring of blood oxygen saturation both in the postanesthesia recovery unit, and also during postoperative hospital stays, is prudent. Patients who use CPAP at home should resume as soon as possible following surgery.[106,107] The evidence does not demonstrate a clear benefit for initiating CPAP in patients with OSA who have not used this preoperatively unless these patients are at high risk for PPC.[80] For patients demonstrating hypoxemia or hypoventilation following surgery, implementation of CPAP should be considered on an individual basis.

Complications Involving the Pleural Space

Injury involving the pleural cavity can occur during abdominal surgery, although risk is highest during surgery in the upper abdomen or upper retroperitoneum. These injuries occur both during open and laparoscopic procedures. Oftentimes violation of the pleural space is noted intraoperatively and should be suspected during laparoscopic surgery when excessive diaphragm motion, or billowing, is noted.[108] During open procedures, bubbling noted in the operative field may indicate an occult violation of the diaphragm and pleural cavity. Occult injuries should be considered when hypoxemia or dyspnea is discovered in the postanesthesia recovery unit. However, clinical manifestation of pleural space injury is variable, ranging from an asymptomatic small apical pneumothorax to a large tension pneumothorax that may lead to hemodynamic instability.

PNEUMOTHORAX

Gas may enter the pleural cavity through several routes. These range from patent congenital diaphragm defects to direct injuries to the diaphragm, chest wall, or via musculofascial planes of the diaphragm and mediastinum during insufflation with carbon dioxide (CO_2).[109,110] The volume of gas that enters the pleural cavity determines the degree of pneumothorax (PTX). Small amounts of gas often do not increase the pressure in the pleural cavity above atmospheric pressure, and thus clinical symptoms may not develop. Often these PTX are detected on chest radiograph and do not demonstrate mediastinal shift or represent clinically significant entities.[111] As the amount of gas increases, the pressure in the affected pleural space increases above atmospheric pressure, eventually compressing the ipsilateral lung. Mediastinal shift to the contralateral hemithorax and flattening of the ipsilateral hemidiaphragm are classically noted on radiograph.

Reported rates of PTX following urologic surgeries range from 0.6% to 25%.[112–116] The rates for PTX following laparoscopic surgeries appear lower (0.6% to 8.5%).[112,114–116]

Management of PTX begins with clinical suspicion. When noted intraoperatively, the anesthesiologist should be informed to confirm hemodynamic and respiratory stability. Once established, repair of diaphragm injury should be performed using running, absorbable suture. Prior to closure of this suture, a red rubber catheter can be introduced into the pleural space with its distal end submerged under water or saline. The anesthesiologist then ventilates the patient in order to evacuate gas from the pleural space via the catheter until no further bubbles are noted from the red rubber catheter. The catheter is removed when the diaphragm suture is tied down.[117] This approach can be performed with equal effectiveness for both open and laparoscopic procedures and is necessary when repairing diaphragmatic incisions used during thoraco-abdominal approaches. In the case of a diaphragmatic excision due to malignancy, successful mesh replacement of the diaphragmatic defect secured with nonabsorbable sutures has been reported.[117]

Despite the low rates of these injuries, clinical suspicion for occult injury should remain. Postoperative hypoxemia or dyspnea should be investigated with chest radiography. If central venous catheterization was performed for intraoperative fluid management, postoperative chest radiography should be performed. Routine use of postoperative chest imaging may identify small PTX without clinical sequelae.[111] These can be safely managed conservatively and followed with serial chest radiograph. In the event that a large PTX is noted, management with a tube thoracostomy is recommended.

CHYLOTHORAX

Chyle accumulation within the pleural space is a rare complication. Case reports describe chylothorax following retroperitoneal surgeries; however, these occur in conjunction with chylous ascites and subsequent communication into the pleural space.[116,118] Management includes diagnosis through thoracentesis and treatment of the underlying lymph leakage. Successful pleural space drainage and sclerotherapy have been reported.[118]

HEMOTHORAX

Accumulation of blood within the pleural space following urologic surgery is also a rare complication, with only a few case reports available. Abreu et al. report one case (0.08%) in their review of 1129 patients undergoing laparoscopic urologic surgery.[116] In this case, an intercostal artery was injured, and repair required emergent open thoracotomy. Additional case reports describe hemothorax following laparoscopic pyeloplasty, laparoscopic cryoablation of renal masses, and percutaneous nephrolithotomy.[119–121] Management requires identification of the bleeding source and possible tube thoracostomy.

HYDROTHORAX

Iatrogenic instillation of irrigation fluid into the pleural space may result in hydrothorax. Hydrothorax has been reported as a complication following percutaneous nephrolithotomy.[122,123] Supracostal percutaneous nephrolithotomy may result in higher risk than standard approaches.[122] Management may require tube thoracostomy. For additional discussion of hydrothorax, please refer to the section below regarding percutaneous nephrolithotomy.

Pulmonary Embolism

Pulmonary embolism (PE) results from venous thromboembolism (VTE) and is a potentially fatal pulmonary complication. Prior to use of VTE prophylaxis, studies estimated the incidence of deep vein thrombosis (DVT) in hospitalized patients to range between 10% and 80%, with fatal PE ranging from 0.1% to 7% in patients undergoing elective surgery.[124] While DVT prophylaxis has decreased these rates, VTE and PE remain serious postoperative complications.

Risk for postoperative patients varies depending upon multiple factors.[124–127] Significant risk factors include age, prior VTE, history of malignancy, obesity, and hypercoagulable state (e.g., Factor V Leiden) as well as medical comorbidities. The American College of Chest Physicians provides guidelines to risk stratify surgical patients.[128] Many urologic procedures fall into the moderate risk or higher categories.

Strategies for prevention of VTE aim to decrease risk of subsequent PE. Nonpharmacologic approaches employ frequent, early ambulation, compression stocking, and pneumatic compression devices and may be sufficient for patients who are very low or low risk. In patients who are moderate or higher risk, including pharmacologic prophylaxis is preferred. Pharmacologic prevention must be balanced with potential for bleeding complications in the perioperative period. Options for pharmacologic prevention include unfractionated heparin, low-molecular-weight heparin, fondaparinux, and novel oral antithrombotic agents (e.g., rivaroxaban, dabigatran, apixaban). Regional and spinal anesthesia should be considered for higher risk patients undergoing lower abdominal or pelvic urologic procedures as these have been associated with a decreased risk of PE when compared with general anesthesia.[129]

The clinical presentation of PE is varied and nonspecific. Patients may be asymptomatic but may also experience sudden shock and death.[130] The most common symptoms of PE, according to two large prospective studies, are dyspnea (73%), pleuritic chest pain (44%), cough (37%), orthopnea (28%), calf/thigh pain/swelling (44%), wheezing (21%), and hemoptysis (13%).[130] Despite these clinical indicators, patients with large PE may report mild or no symptoms.[130–132] The most common clinical signs include tachypnea (54%), calf/thigh swelling/edema/erythema (47%), tachycardia (24%), and rarely fever (3%). Unfortunately, given the nonspecific nature of this clinical presentation, the sensitivity and specificity for PE on clinical evaluation are 85% and 51%, respectively.[133]

Clinical suspicion for PE in the postoperative phase prompts urgent investigation. Multiple clinical calculators may assist with stratifying patients to empiric therapy.[134–136] For the majority of patients with suspicion for PE, the preferred diagnostic study is a computed tomographic pulmonary angiogram (CTPA).[130,137] For patients unable to undergo CTPA, ventilation/perfusion (V/Q) scan remains a sensitive but poorly specific test that can be obtained. In addition, lower extremity duplex ultrasound should be obtained to assess for presence of DVT, and, if found, anticoagulation should be initiated.

Once clinical evaluation confirms suspicion for DVT or PE, empiric anticoagulation should be promptly initiated. In postoperative patients anticoagulation may result in bleeding. Absolute contraindications to heparin therapy are active bleeding, severe bleeding diathesis, a platelet count ≤20,000/mm^3, neurosurgical or ocular surgical procedures performed within the past 10 days, or intracranial bleeding within the past 10 days.[138] In essence these contraindications to anticoagulation serve as indications for placement of an IVC filter. Consideration may also be given to placement of an IVC filter in the setting of recurrent PE while therapeutic on anticoagulation or when bleeding risk outweighs risk of DVT or PE such as poor cardiopulmonary reserve.

If PE embolism results in hemodynamic instability, more aggressive treatment with thrombolytic therapy is indicated. Thrombolytic therapy should also be considered when patients are at high risk of bleeding from systemic anticoagulation or if systemic anticoagulation fails. Finally, for hemodynamically unstable patients who are not candidates for thrombolytic therapy, embolectomy via either catheter-based approaches or open surgical approaches may be warranted.

It is our practice to administer unfractionated heparin preoperatively (5000 units subcutaneously administered prior to surgery) and throughout the perioperative period for all patients undergoing pelvic surgery for urologic malignancy. This practice is also applied to patients undergoing retroperitoneal oncologic surgery when bleeding risk is deemed acceptable.

Air Embolism

Introduction of insoluble gas into the vasculature system is a rare but dangerous event. Amongst urologic procedures, laparoscopic procedures carry the highest risk for this complication.[139–142] Gas may gain entry into open vasculature via any vascular invasion, surgical or traumatic, within the pleural or peritoneal cavity as well as through endoscopic approaches. Venous air emboli have been reported following retrograde pyelogram, transurethral resection of the prostate, percutaneous nephrostomy tube placement, and percutaneous nephrolithotomy as well as open radical prostatectomy and laparoscopic prostatectomy.[143–150] Additionally, case reports describe gas embolism following sexual intercourse, autoerotic exercises, and following urethral insufflation.[151–155]

Air or gas embolism occurs when insoluble gas enters an open venous or arterial channel with sufficient pressure differential to allow for gas entry into the vascular system. Gas emboli should be considered in appropriate clinical situations (trauma, laparoscopic surgery, neurosurgery, central venous catheterization) when patients experience sudden onset respiratory distress or neurologic decompensation. However, there are no clear clinical indicators to differentiate gas emboli from other potential complications including pulmonary emboli, myocardial infarction, stroke, and other acute adverse events.

When gas emboli are suspected, the optimal diagnostic test is transthoracic echocardiography (TTE) followed by CTPA.[156,157] When the diagnosis is suspected, patients should be immediately positioned into left lateral decubitus position (Durant's maneuver) or Trendelenburg for suspected venous emboli or supine for suspected arterial emboli. These positioning maneuvers aim to contain the gas emboli.[158] Once the diagnosis has been confirmed, management options range from hyperbaric oxygen, manual aspiration of air from the right ventricle via central venous catheter, or, as a last resort, chest compressions.[159–162]

Pulmonary Complications of Open and Laparoscopic Urologic Surgery

Pleural space violation during surgical procedures in the flank and upper abdomen may occur due to the fact that the pleura extends down to the 11th rib in the posterior axillary line and to just below the 12th rib in the area of the vertebral column.[163] At times, pleural space entry is planned for flank or thoraco-abdominal incision. In addition, upper pole renal tumors have been reported involving or invading the diaphragm.[117,164] As noted above, upper abdominal and flank surgical procedures are associated with a risk of pleural injury and pneumothorax.[165,166] Modifications of the traditional flank incision, such as the supra-11th mini-flank incision, help to prevent pleural injury during open surgical procedures of the kidney.[167]

Pleurotomies recognized intraoperatively during open and laparoscopic surgical procedures can usually be repaired without difficulty or sequelae.[113] A 12Fr rubber catheter is initially placed through the defect and into the pleural cavity, and the pleura is closed with absorbable suture in a running pattern. The lung is then expanded with positive pressure ventilation by the anesthesiologist, removing the remaining gas within the pleural cavity through the end of the catheter. Once all gas has been expelled, the catheter is removed, and the running suture is tied while the lung remains expanded.

Suspected diaphragmatic tears during laparoscopy should be repaired primarily, if possible. Depending on the severity of the injury and the clinical status of the patient, these repairs can be performed either immediately or when the primary operation has been completed. Delaying repair until after specimen extraction may provide better visualization of the injury; however, unstable patients require immediate attention to this complication. If repair can be delayed, then it is advised to decrease the pneumoperitoneum to 10 mm Hg to limit the extent of any present pneumothorax, to facilitate patient ventilation, and to allow for tension-free anastomosis.[168] A thoracostomy tube is indicated for pleural hemorrhage, lung injury, or inability to primarily close the diaphragm.[115]

It is our practice to obtain routine postoperative chest radiograph, although this is up to surgeon discretion. Several studies recommend that postoperative chest radiographs are obtained following cases involving central line placement, intraoperative diaphragm injury, postoperative respiratory distress, or abnormal postoperative clinical findings.[169,170]

Pulmonary Complications of Percutaneous Nephrolithotomy

Pulmonary injuries following percutaneous nephrolithotomy (PCNL) occur during the percutaneous access. Pleural complication rates have been reported to range from 2.3% to 23%, depending on the definition of injury.[171–175]

Rates of PPC are higher with the supracostal PCNL, with a reported 16-fold greater risk of pleural injury when access is above the 12th rib.[176,177] While an infracostal approach is associated with fewer PPC, a supracostal puncture improves stone access and clearance for certain situations (e.g., scoliosis, upper calyx stone).[178] Supracostal punctures should be performed using C-arm fluoroscopic guidance and following maximal exhalation.[179,180] In addition, utilization of an adequately sized working sheath and maintaining low pressure saline irrigation minimize entry of gas and fluid into the pleural space.[176]

The incidence of pulmonary complications that necessitate surgical intervention after a supra-12th rib approach ranges from 3% to 23%.[179,181] Intraoperative placement of an 8 to 10Fr loop nephrostomy tube into the pleural space under fluoroscopic guidance for significant hydropneumothorax following supracostal PCNL may obviate the need for a traditional thoracostomy tube insertion.[182] Traditional thoracostomy tube placement is recommended for when parenchymal injury is suspected or significant drainage of fluid or blood is required.

Nephropleural fistula is a rare pulmonary complication of PCNL and may present as urinothorax. Rates of nephropleural fistula range from 2.3% for supra-12th rib approaches to 6.3% for supra-11th rib access.[183] Management involves collecting system decompression with ureteral stent and concomitant thoracostomy tube. Refractory cases may require decortication with pleural sclerosis.

Patients With Prior Bleomycin Chemotherapy

Men with a history of testicular cancer may have been exposed to chemotherapy regimens including bleomycin, an antibiotic with antineoplastic activity. Often, men with poor-risk or intermediate-risk testicular cancer are subjected to bleomycin.

Patients exposed to bleomycin are at risk for developing bleomycin-related toxicities, which include interstitial pneumonitis (bleomycin-induced pneumonitis [BIP]). The pathophysiology of this toxicity is multifactorial and is related to cumulative bleomycin exposure, age, thoracic radiation, renal insufficiency, tobacco use, and exposure to high oxygen concentration.[184] Pulmonary fibrosis can develop in 2–40% of those who develop BIP.[185] Symptoms of BIP include cough, dyspnea on exertion, and fever. These symptoms often begin gradually but may manifest as late as 6 months following discontinuation of therapy. In some cases, symptoms progress to worsening respiratory failure.[186]

Patients with a history of bleomycin exposure should undergo pulmonary function testing prior to elective surgical procedures. Management of these patients requires careful perioperative fluid managements and minimization of exposure to high concentration oxygen.[187,188]

Conclusion

Postoperative pulmonary complications occur at a similar frequency and result in similar rates of morbidity and mortality as cardiovascular complications. The most important patient-related factor that should be considered by the urologist when assessing preoperative risk is patient age, although COPD, CHF, and cigarette smoking also increase risk. Many urologic procedures involve risk for PPC, and thus elevated vigilance in the perioperative period is recommended. Venous thromboembolism prophylaxis should be utilized whenever possible.

References

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