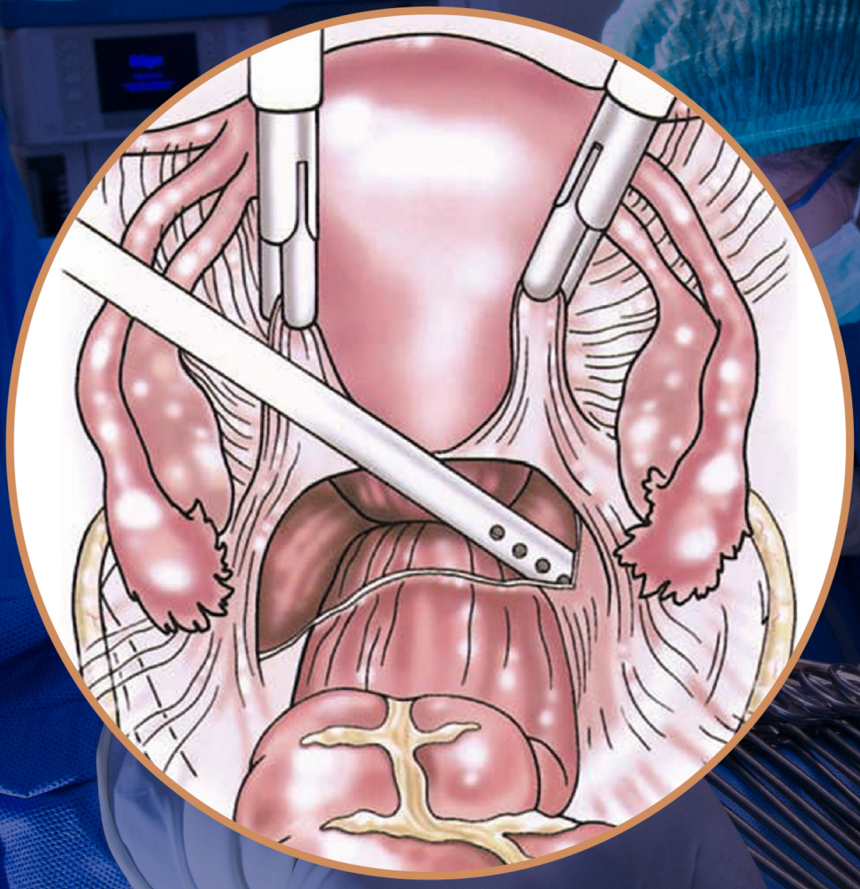
FOURTH EDITION

AN ATLAS OF GYNECOLOGIC ONCOLOGY

Investigation and Surgery



Edited by

J. RICHARD SMITH • GIUSEPPE DEL PRIORE

ROBERT L. COLEMAN • JOHN M. MONAGHAN

CRC Press
Taylor & Francis Group

FOURTH EDITION

AN ATLAS OF GYNECOLOGIC ONCOLOGY

To my four children, Cameron, Victoria, Madeleine and Lara, thank you for being there for Dad.

JRS

To my family—from the smallest latest joyous addition, to the oldest and wisest, some departed, and in the center of them all, my wife, Men-Jean Lee.

GDP

To my extraordinary wife, Fay, for her unwavering support and understanding, mentorship, love and friendship and to our six blessing children, of whom I could not be prouder. And, to my Parents, who through their years of sacrifice and guidance enabled me to pursue my dreams.

RLC

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Investigation and Surgery

Edited by

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CRC Press
Taylor & Francis Group
Boca Raton London New York

CRC Press is an imprint of the
Taylor & Francis Group, an **informa** business

CRC Press
Taylor & Francis Group
6000 Broken Sound Parkway NW, Suite 300
Boca Raton, FL 33487-2742

Printed on acid-free paper

International Standard Book Number-13: 978-1-4987-2906-2 (Hardback)

Library of Congress Cataloging-in-Publication Data

Names: Smith, J. Richard, editor. | Del Priore, Giuseppe, editor. | Coleman, Robert L., editor. | Monaghan, John M., editor.
Title: An atlas of gynecologic oncology: investigation and surgery/edited by J. Richard Smith, Giuseppe Del Priore, Robert L. Coleman and John M. Monaghan.
Description: Fourth edition. | Boca Raton, FL: CRC Press/Taylor & Francis Group, [2018] | Includes bibliographical references and index.
Identifiers: LCCN 2017046627| ISBN 9781498729062 (hardback: alk. paper) | ISBN 9781351141680 (ebook: alk. paper)
Subjects: | MESH: Genital Neoplasms, Female—surgery | Atlases
Classification: LCC RC280.G5 | NLM WP 17 | DDC 616.99/46—dc23
LC record available at https://lccn.loc.gov/2017046627

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http://www.crcpress.com

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Preface

Welcome to the fourth edition of this Atlas. When we looked back to the first edition, instituted 20 years ago now, that text was approximately half the size of this current volume. Much of what is now included would have appeared to be science fiction 20 years ago, but it has become reality. Inevitably, the book gets larger with each expanding edition as the gynecologic oncologist's repertoire of operations gets progressively larger. There are virtually no operations which fail to remain in the skill set, only ever more to know about and ever more equipment and energy sources available. A number of the procedures described are nowadays often performed as laparoscopic or robotically assisted procedures; however, we are also aware that not all surgeons have access to the same equipment, and this is an international book designed for an international audience.

The "cookbook" formula of the previous editions remains; nobody is advising which operation to do, but you do get a "road map" to whichever operation you have decided upon. Many years ago, before satellite navigation systems in cars, if you were going on a long drive you would consult your road atlas the previous night; this book, it is hoped, fulfils a similar role, as well as opening our minds to new things, some of which we may develop, others not.

New chapters have been added and all the text updated by a combination of the chapter authors and the editors. As in previous editions, innovative surgeons have been keen to contribute. The wonderfully clear artwork of Dee McLean and Joanna Cameron continues to enhance this book, allowing easy step-by-step breakdowns of procedures.

Once again it has been a great pleasure and privilege to be the editors and to read so many clear expositions written by experts for experts. We hope you enjoy reading this book as much as we have enjoyed editing it.

J. Richard Smith
Giuseppe Del Priore
Robert L. Coleman
John M. Monaghan

Acknowledgments

J. Richard Smith would like to thank Miss Rodena Kelman and Miss Alison Irvine Smith for their secretarial support and Dr. Charles Lockwood and Professor Philip J. Steer for the academic environment which encouraged the editors in the early days. He would also like to express his gratitude to his research fellow, Mr. Benjamin Jones, for editorial assistance and also to Dr. David Keefe, of NYU Medical Center, for his ongoing support and collaboration. In addition, he thanks all his colleagues at the West London Gynaecological Cancer Centre, particularly Mr. Alan Farthing, Ms. Sadaf Ghaem-Maghami, Ms. Christina Fotopoulou, Ms. Maria Kyrgiou, Mr. Joseph Yazbek, and Professor Hani Gabra, for the supportive environment they engender for this project and many others.

Giuseppe Del Priore wishes to thank his family for indulging his trainees for constant inspiration to learn and teach more. He thanks his colleagues for their continued guidance and constructive feedback.

Robert L. Coleman would like to thank his wife, Fay, and his expanding family for their inspiration, sacrifice, and support to make this all possible; his colleagues and trainees who confidently and consistently ask "why?" and "what if?" as they care for our patients; his trusted friends and brilliant collaborators, Professors Anil Sood, Tom Herzog, and Bradley Monk, who continually challenge him to think bigger and reach deeper; and Ms. Kathleen Collins, Ms. Elizabeth DelBosque, Ms. Ljiljana Milojevic, Ms. Marlana Klinger, and the dedicated team of research nurses, data coordinators, and regulatory staff, whom he's been privileged to work alongside, for their tireless attention to their research program, which strives to better understand the disease process and move the needle on treatment efficacy and safety.

John M. Monaghan would like to thank his fellow editors for keeping him on side and for allowing him to use the experience of time to occasionally add a comment or two. He would particularly like to thank Maggie, his wife of over 50 years, for her continuing patience and encouragement. It has been a privilege to be involved in this book for over 20 years and to see how fully the field of gynecological oncology has progressed to be the major surgical care system for women. When the editors began, laparoscopic surgery was an occasionally used tool; it is now the major route of access for even the most massive of procedures. The resulting shortening of inpatient care and rapid discharge of patients has had many benefits but occasional problems. Careful scientific analysis of systems of care and techniques of management are now standard in gynecological oncology, yet the subject remains innovative and not confined by the stifling atmosphere of safety and nil risk. He sees a strong future of innovation and development for the subject and is happy to have contributed to this important text.

In Memoriam: Andrew D. Lawson

In 2014 Andrew Lawson, pain specialist, anesthetist, and ethicist, succumbed to a pleural mesothelioma after a 7-year battle with his disease. The editors wish to acknowledge his great contribution to the field of chronic pain management and to record his efforts for the first three editions of this book, the latter while very much under the cloud of his diagnosis. He was known personally to many involved with this book and is much missed.

1 Introduction: Preparing a patient for surgery

Srdjan Saso, Benjamin P. Jones, J. Richard Smith, and Giuseppe Del Priore

INTRODUCTION

This chapter reviews three specific areas relevant to virtually all surgical procedures and surgeons: infection prophylaxis; deep venous thrombosis (DVT) prophylaxis; and universal precautions. Universal precautions facilitate the protection of surgeons and their assistants, medical and nursing, and patients. Preoperative and postoperative checklists now form a vital part of risk reduction. James Reason, PhD, formulated the "Swiss cheese theory" of risk. This is based on a piece of Swiss cheese with holes in it. The more slices one puts in the cheese, the less likely it is that an arrow could fly through the holes, and thus the holes are less likely to tally with each other. Therefore the more layers of checking that one puts in pre- and postoperatively, the less likely it is that the antibiotic prophylaxis will be forgotten or the postoperative DVT prophylaxis will not be given. A simple checklist is shown in Figure 1.1. The purpose of any such checklist is to systematically and efficiently ensure that all operative conditions are optimal with respect to patient safety. The hope is that by completing such a checklist, the lives and well-being of surgical and thus gynecological patients will be minimized as errors in patient identity, site, and type of procedure are avoided completely.

More well known is the surgical checklist published by the World Health Organization (WHO) in 2008 in order to increase the safety of patients undergoing surgery. It is officially known as the *WHO Surgical Safety Checklist and Implementation Manual.* It is now used in general surgery, orthopedics, and obstetrics and gynecology. The operation is divided into three distinct phases by the checklist. Each phase corresponds to one of the following periods: (a) before the induction of anesthesia, (b) before the skin incision (known as "time out"), and (c) before the patient leaves the operating facility (known as "sign out"). A "checklist coordinator" must confirm that the surgical team has completed a phase before moving on to the next. Only when all three phases have been completed can the procedure commence.

Phase I: Before Induction of Anesthesia. The following must be confirmed first: patient identity, site of operation, procedure to be carried out, and consent. Type of anesthetic required, allergies (if any), and expected blood loss should be discussed. Phase I is to be completed by the anesthetist.

Phase II: Time Out. This refers to a process before the first incision where all present in the room must introduce themselves by name and role. The patient name and the planned procedure are then confirmed as well as any surgical or anesthetic critical events that may occur. The need for antibiotics, DVT prophylaxis, and imaging is highlighted. Phase II is to be completed by the surgeon and anesthetist.

Phase III: Sign Out. The final phase is performed before the patient leaves the operating room. Swabs, instruments, and needle counts are done, the equipment is checked (including disposables), and the specimens are checked as properly labeled. The postoperative recovery process is discussed. Phase III is to be completed by the surgeon or nursing staff.

Figure 1.1 shows the checklist on admission for surgery.

THROMBOEMBOLIC DISEASE

Venous thromboembolic disease (VTE) is a significant cause of morbidity and mortality in gynecologic oncology patients. If sensitive methods of detection are employed and no preventive measures are taken, at least 20% and as many as 70% of gynecologic cancer patients may have some evidence of thrombosis. In certain situations, such as with a long-term indwelling venous catheter of the upper extremity, nearly all patients will have some degree of VTE, though it may not be clinically significant. On the other hand, lower extremity VTE has a much more certain and clinically significant natural history. Venous thromboses below the knee may spread to the upper leg in approximately 10% to 30% of cases or resolve spontaneously in approximately 30%. Once the disease has reached the proximal leg, the risk of pulmonary embolism (PE) increases from less than 5% for isolated below-the-knee VTE to up to 50% for proximal VTE. The mortality rate for an undiagnosed PE is high. Up to two-thirds of patients who die from PE do so in the first 30 minutes after diagnosis.

Early recognition and effective treatment can reduce this mortality. However, postoperative VTE is still a leading cause of death in gynecologic oncology patients. In the past, it was clear that only one-third of hospitalized high-risk patients received appropriate prophylaxis; this figure has now much improved, particularly with the use of checklists. Risk factors are listed in Table 1.1 (NICE 2015).

PREVENTION AND RISK ASSESSMENT

Patients may be considered for prevention of VTE based on their clinical risk category. Laboratory tests such as euglobulin lysis time do correlate with the risk of VTE but are no more helpful than clinical risk assessment in selecting patients for prophylaxis. Low-risk patients are young (less than 40 years old), undergoing short operative procedures (less than 1 hour), and do not have coexisting morbid conditions such as malignancy or obesity that would elevate the risk of VTE. Moderate-risk patients include those undergoing longer procedures, older or obese patients, and patients having pelvic surgery. High-risk patients include otherwise moderate-risk patients who have cancer and those with a previous history of VTE. Positioning for vaginal surgery lowers the risk of VTE when compared with the abdominal approach.

All patients should be assessed for risk of bleeding before being offered pharmacological VTE prophylaxis. This should not be offered to patients with any of the risk factors for

CHECKLIST FOR SURGERY ON ADMISSION
Date:

NAME: DATE OF BIRTH:

OPERATION PLANNED:

RISKS	
Infection	
Anesthetic	
Hemorrhage	
Deep Venous Thrombosis	
Uterine Perforation	
Other Organ Damage	
Others	

PAST GYNECOLOGICAL HISTORY	
Last Menstrual Period	
Contraception	
x/y	
Pregnancy Test	

PERTINENT MEDICAL/ANESTHETIC PROBLEMS

Signed _____

POST-OPERATIVE CHECKLIST	
Low molecular weight heparin or other measures prescribed	
Antibiotics prescribed	
Photographs taken and collected	

Signed _____

1

Figure 1.1 Checklist for surgery on admission.

Table 1.1 Risk Factors for VTE (NICE 2015)

- Active cancer or cancer treatment
- Age over 60 years
- Critical care admission
- Dehydration
- Known thrombophilias
- Obesity (body mass index [BMI] over 30 kg/m^2)
- One or more significant medical comorbidities (for example, heart disease; metabolic, endocrine, or respiratory pathologies; acute infectious diseases; inflammatory conditions)
- Personal history or first-degree relative with a history of VTE
- Use of hormone replacement therapy
- Use of estrogen-containing contraceptive therapy
- Varicose veins with phlebitis

bleeding shown in Table 1.2, unless the risk of VTE outweighs the risk of bleeding. Patients should be advised to consider stopping estrogen-containing oral contraceptives or hormone replacement therapy 4 weeks before elective surgery. If stopped, advice must be provided on alternative contraceptive methods (NICE 2015). All patients should have some form of VTE prevention. This first begins with risk reduction. Patients should not become dehydrated unless clinically indicated. They must mobilize as soon as possible. Aspirin or other antiplatelet agents should not be considered as adequate prophylaxis for VTE. Finally, temporary inferior vena caval filters should be offered to patients who are at very high risk of VTE (such as patients with a previous VTE event or an active malignancy) and for whom mechanical and pharmacological VTE prophylaxis are contraindicated (NICE 2015).

VTE prophylaxis can be in the form of mechanical or pharmacological prophylaxis. The ultimate decision is based on individual patient factors including clinical condition, surgical procedure, and patient preference. Mechanical prophylaxis can be anti-embolism stockings (thigh or knee length), foot impulse devices, or intermittent pneumatic compression devices (thigh or knee length). Pharmacological prophylaxis is based on local policies and individual patient factors, including clinical condition (such as severe renal impairment or established renal failure) and patient preferences (NICE 2015).

Table 1.2 Risk of Bleeding (NICE 2015)

- Active bleeding
- Acquired bleeding disorders (such as acute liver failure)
- Concurrent use of anticoagulants known to increase the risk of bleeding (such as warfarin with international normalized ratio [INR] higher than 2)
- Lumbar puncture/epidural/spinal anesthesia expected within the next 12 hours
- Lumbar puncture/epidural/spinal anesthesia within the previous 4 hours
- Acute stroke
- Thrombocytopenia (platelets less than 75×10^9/L)
- Uncontrolled systolic hypertension (230/120 mmHg or higher)
- Untreated inherited bleeding disorders (such as hemophilia and von Willebrand disease)

Low-risk patients, with an incidence of approximately 3% for VTE, may be adequately protected with early ambulation, elevation of the foot of the bed, and graduated compression stockings. "Early ambulation" has been defined by some investigators as walking around the nursing station at least three times within the first 24 hours. Graduated compression stockings are readily available; however, ensuring their proper application and size can be difficult. Obese patients may suffer from a "tourniquet" effect if the stocking rolls off the thigh; this may actually increase the risk of VTE, not prevent it.

Moderate-risk patients include the majority of general gynecology patients and have approximately 10% to 40% chance of developing VTE. These patients should receive the same measures as low-risk patients with the addition of low-dose unfractionated low molecular weight heparin (LMWH), 5000 units subcutaneously twice a day. An alternative to the administration of heparin is the application of pneumatic compression devices to the lower extremities. High-risk category patients require even more measures owing to the estimated 40% to 70% risk of VTE.

The vast majority of gynecologic oncology cases will fall into the high-risk category. Standard unfractionated heparin (UH) is ineffective in these cases in low doses; for example, 5000 units twice daily. If given three times daily, UH is effective but no better than pneumatic calf compression. Unfortunately, more frequent dosing is associated with significantly more wound hematoma formation and blood transfusions. It also requires additional nursing and pharmacy personnel time, and is more uncomfortable for the patient. These may be some of the reasons only a minority of surgeons regularly use UH prophylaxis. Unfortunately, although compression devices are effective in gynecologic oncology patients, the devices are somewhat cumbersome, and are disliked by patients and nursing staff. In fact, improper application of the devices occurs in approximately 50% of patients on routine inpatient nursing stations. Compression devices are also contraindicated in patients with significant peripheral vascular disease.

The LMWHs have many potential advantages over the previously cited alternatives. Excellent bioavailability allows for single daily dosing, which in turn reduces nursing effort while improving patient satisfaction. This form of prophylaxis is also associated with less thrombocytopenia and postoperative bleeding. Patients with UH-associated thrombocytopenia will usually tolerate LMWH. In summary, in high-risk patients such as gynecological patients, LMWH may be more efficacious, more cost-effective, and less toxic than the alternatives.

Many other agents have been tried in an attempt to overcome the imperfections of existing options. All have limitations and are not used routinely. However, all are effective to some degree and may be appropriate in highly selected patients. Some of these agents include aspirin, warfarin, and high molecular weight dextran. The most promising are direct thrombin inhibitors and oral factor Xa inhibitors such as Rivaroxaban. In comparison with LMWH, aspirin results in more bleeding complications and is less effective than heparin in preventing VTE. Warfarin has a prophylactic effect similar to aspirin, but again is less effective than heparin and is associated with a higher risk of complications and requires more intensive monitoring. Dextrans are effective but have been associated with rare cases of allergic reactions. Other complications reported include fluid overload and nephrotoxicity. Further research to avoid some of these limitations may improve the therapeutic value of these alternatives.

PREVENTION AND TREATMENT

The duration of prophylaxis has traditionally been limited to the duration of hospital stay. In many older studies, when health care was less cost-conscious, this may have been several days to weeks. Lengths of stay are now much shorter, and as a result so is the duration of VTE-preventive measures. Even before this forced change in clinical practice, it was recognized that a significant minority of VTE either developed or was diagnosed long after discharge from the hospital. The optimal duration of prophylaxis is still not known and depends on the method used. For instance, patients should be instructed to walk every day once discharged from the hospital. Similarly, graduated compression stockings may be worn after surgery until discharge with little risk and possibly some benefit. Some authors also advocate compression stockings to be worn at home following discharge. Conversely, pharmacologic therapies have side effects, may require some training (e.g., self or nurse injections), and are associated with considerable cost. General guidance used to be to use prophylaxis until the patient is fully mobile. However, following the demonstration that administering LMWH for 4 weeks, when compared to a single-week course, reduces VTE risk by 60% in postoperative cancer patients, generally a 4-week course is now prescribed (Bergqvist et al. 2002).

The agents discussed above are all designed to prevent VTE and thereby reduce the risk of developing a clinically significant PE. When these methods are used properly, most patients will not develop VTE and therefore will be at low risk for a PE. However, it is not uncommon for a gynecologic oncology patient to present with VTE as the first manifestation of disease. For instance, it is the presenting symptom in up to 10% of ovarian cancer patients. In these patients, and in those who develop VTE despite appropriate prophylaxis, something must be done to prevent the progression to a potentially fatal PE. This becomes especially difficult if the patient requires surgical treatment for the malignancy.

One common management technique for these difficult situations is mechanical obstruction of the inferior vena cava. This can be accomplished preoperatively via peripheral venous access and interventional radiologic techniques. Care must be taken to delineate the extent of the clot so that no attempt is made to pass the filtering device through an occluded vein. If peripheral caval interruption is not possible, a vena caval clip may be applied intraoperatively. However, large pelvic masses, not uncommon in gynecologic cancer patients, may prevent access. Additional problems with vena caval interruption include migration of the device, complete occlusion of the cava, perforation, and infection. In preoperative cases where the patient cannot have a filter or clip placed, one option is the discontinuation of intravenous UH 1 hour before the perioperative period, with resumption approximately 6 hours after completion of the surgery. Most patients will do well with this technique, but they are still vulnerable to intraoperative PEs. Another pharmacologic option may include the preoperative lysis of the thrombus with thrombolytic agents such as urokinase followed by resumption of standard prophylactic measures. Oral anticoagulation is used after caval interruption, if not contraindicated, to prevent postthrombotic venous stasis of the lower extremity. Therefore, mechanical devices, while reducing perioperative pulmonary emboli, do not obviate the need for long-term anticoagulation. It is vitally important if one is operating on patients who have traveled on long-haul flights that major surgery should be avoided within 48 hours of this flight. NICE guidance on venous thromboembolism in patients undergoing surgery (NICE 2012) states "immobility associated with continuous travel of more than three hours in the four weeks before or after surgery may increase the risk of VTE." For those patients traveling by airplane postoperatively, the relatively new oral preparations of Dabigatran and rivaroxaban, licensed for the prevention of VTE after hip and knee replacement surgery, may be prescribed (Gomez-Outes et al. 2009).

DIAGNOSIS

Given the imperfection of prophylaxis and the high risk of VTE in gynecologic oncology patients, all physicians caring for these women should be familiar with the treatment and diagnosis of VTE including PE. Fewer than one-third of patients with VTE of the lower extremity will present with the classic symptoms of unilateral edema, pain, and venous distension. A positive Homan sign (calf pain with dorsiflexion of the foot) is also unreliable and is seen in less than half of patients with VTE. Calf VTE occurs bilaterally in approximately 40% of cases and is more common on the left (40%) than on the right (20%). Only a high index of suspicion and objective testing can correctly identify patients with VTE.

In high-risk patients with a high baseline prevalence of VTE, sensitive but non-specific tests are useful owing to their high positive predictive value. To exclude disease in these same high-risk patients, repeat testing on subsequent days or more sensitive techniques are needed. Noninvasive diagnostic testing should always be considered before interventional techniques including venography and arteriography. Lower extremity Doppler and real-time two-dimensional ultrasonography scans are fairly sensitive (85%) and specific (>95%) for VTE. If results are positive in high-risk patients, including those with symptoms suggestive of PE, no further testing is indicated and therapy may be initiated. In the past, ventilation–perfusion scans and more recently spiral CT thoracic scanning may be used similarly in patients in whom PE is suspected. If the scan indicates an intermediate or high probability of PE, treatment is usually advisable. In patients at higher risk for hemorrhagic complications, such as during the immediate postoperative period where there is residual tumor, confirmatory tests may be indicated before therapy.

TREATMENT

If there is no contraindication to anticoagulation, therapy should be started as soon as the diagnosis of VTE is made. Outcomes are correlated with the time it takes to achieve therapeutic anticoagulation, so the fastest means available should be employed. LMWH has an advantage over UH in that a single daily dose of approximately 175 units/kg subcutaneously will be therapeutic almost immediately. Unfractionated heparin may require approximately 24 hours and repeated blood testing before becoming therapeutic. Treatment with warfarin can be started once the anticoagulation effect of either heparin is confirmed. With UH, this may be as early as day 1, although 2 to 3 days of therapy may be needed before anticoagulation is achieved. With LMWH, warfarin can be started within a few hours, and definitely on the same day. Either heparin should be continued until the warfarin has achieved an international normalized ratio of 2 to 3. Anticoagulation with warfarin should continue for at least 3 months. Patients with recurrent VTE or persistent precipitating events, e.g., vessel compression by tumor, may need indefinite anticoagulation.

Disseminated cancer and chemotherapy will unavoidably increase the risk of complications from anticoagulation. Cancer patients who have nutritional deficits, organ damage, and unknown metastatic sites are particularly vulnerable. Chemotherapeutic agents alter the metabolism of anticoagulants through their effect on liver and renal function, making dosing more difficult. Chemotherapeutic drugs may also share similar toxicities with anticoagulants and thereby worsen hemorrhagic complications from thrombocytopenia and anemia. For these reasons, treatment of VTE may be neither desired by the patient nor recommended by her physician in all situations.

INFECTION PROPHYLAXIS

Most gynecology units now routinely use antibiotic prophylaxis prior to both minor and major surgery. In the absence of such prophylaxis, abdominal hysterectomy is complicated by infection in up to 14% of patients, and following vaginal hysterectomy, infection rates of up to 38% have been reported (Sweet and Gibbs 1990). This results in much morbidity, increased length of hospital stay, increased prescribing of antibiotics, and a large financial burden. By its very nature, oncological surgery carries greater risks of infection than routine gynecological surgery, owing to the length of the procedures and increased blood loss (Table 1.3).

It is difficult to compare many of the studies on prophylaxis, as diagnosis and antibiotic regimens are not standardized. However, there seems to be general agreement that approximately 50% of infections are prevented in this way and that the potential dangers of increased microbial resistance do not justify withholding prophylaxis. Prophylaxis is thought to work by

Table 1.3 Risk Factors for Postoperative Infection

1	Hospital stay for more than 72 hours before surgery
2	Prior exposure to antimicrobial agents in the immediate preoperative period
3	Morbid obesity
4	Chronic illness, e.g., hypertension, diabetes
5	History of repeated infection
6	Prolonged operative procedure (>3 hr)
7	Blood loss in excess of 1500 mL

reducing, but not eradicating, vaginal flora. The antibiotic used, its dose, and the duration of therapy do not appear to influence results. It is therefore suggested that short courses of antibiotics should be used, involving a maximum of three doses. First-generation cephalosporins, broad-spectrum penicillins, and/or metronidazole are all reasonable choices on grounds of efficacy and cost. Antibiotic prophylaxis should not detract from good surgical technique, with an emphasis on strict asepsis, limitation of trauma, and good hemostasis.

INFECTION CONTROL

There is increasing awareness of the risks of transmission of blood-borne pathogens from surgeon to patient and vice-versa during surgical practice. These risks have been highlighted by the publicity surrounding human immunodeficiency virus (HIV), but are generally greater from other pathogens including hepatitis B virus (HBV). Infection with hepatitis C virus (HCV) also poses a risk of transmission from patient to surgeon. The prevalence of these viral infections varies widely with different populations, and this exerts an influence on the surgeon's risk, as does the number of needlestick (or sharps) injuries sustained and the surgeon's immune status. The risks of transmission of these viruses and their subsequent pathogenicity are discussed below. The necessity for universal precautions in surgical practice need not affect overmuch operator acceptability or cost.

Antenatal anonymous surveys have shown a seroprevalence of HIV in metropolitan areas of the United Kingdom to be as high as 0.26% (Evans et al. 2009). HIV prevalence has increased in the United Kingdom over the last decade, with an estimated 110,000 individuals living with HIV by 2013.

The risk of acquiring HIV from a single-needlestick injury from an infected patient is in the region of 0.10% to 0.36% (Cardo et al. 1997a,b). Pooled data from several prospective studies of healthcare personnel suggest that the average risk of HIV transmission is approximately 0.3% (95% confidence interval, 0.2–0.5) after a percutaneous exposure to HIV-infected blood and approximately 0.09% (95% confidence interval, 0.006–0.5) after a mucous-membrane exposure (Gerberding 2003). However, using mathematical models to predict lifetime risks of acquiring the infection in a population with a low HIV seroprevalence (0.35%), it has been suggested that 0.26% of surgeons would seroconvert during their working lives (Howard 1990). Needlestick injuries pose a significant occupational risk for surgical trainees. A study by Makary et al. (2007) in *The New England Journal of Medicine* found that virtually all surgical residents (99%) had had a needlestick injury by their final year of training, and concluded that needlestick injuries are common among surgeons in training and are often not reported. Improved prevention and reporting strategies are needed to increase occupational safety for surgical providers (Makary et al. 2007).

In December 2001, 57 healthcare workers in the United States had seroconverted to HIV as a result of occupational exposure. Of the adults reported with acquired immune deficiency syndrome (AIDS) in the United States through December 31, 2002, 24,844 had a history of employment in healthcare. These cases represented 5.1% of the 486,826 AIDS cases reported to the Centers for Disease Control and Prevention (CDC) for whom occupational information was known (www.cdc.gov). This website is a valuable resource, particularly with respect to new and ever-changing drug regimens currently in use in the management of blood-borne pathogens. Intact skin and mucous membranes are thought to be effective barriers against HIV. Only a very few cases of transmission via skin contamination are known to have occurred, and these healthcare workers had severe dermatitis and did not observe barrier precautions when exposed to HIV-infected blood (CDC 1987). Aerosol transmission of HIV is not known to occur, and the principal risks are related to injuries sustained from hollow-bore needles, suture needles, and lacerations from other sharp instruments. Infectivity is determined by the volume of the inoculum and the viral load within it: thus, a hollow-bore needlestick injury carries greater risk than injury from a suture needle. Prior to highly active antiretroviral therapy, infection with HIV results in AIDS in 50% of patients over a 12-year period and had a long-term mortality approaching 100%. The situation is now radically different. For HIV seropositive surgeons, further operative practice involving insertion of the fingers into the body cavity is precluded owing to the potential risk of doctor-to-patient transmission: for gynecologic surgeons, this encompasses virtually their entire surgical practice, with the exception of laparoscopic and hysteroscopic procedures.

There is a whole classification related to exposure-prone procedures (EPP) which is categorized into nonexposure-prone (category 0) and exposure (1–3). Category 3 encompasses all open procedures. This classification is available from the UK Department of Health website related to UKAP (United Kingdom Advisory Panel for Health Care Workers infected with blood-borne pathogens). At present there is no vaccine available to prevent infection with HIV. Should needlestick injury occur, the injured area should be squeezed in an attempt to expel any inoculum, and the hands should be thoroughly washed. There is good evidence that after exposure prophylactic zidovudine (azidothymidine [AZT]) reduces transmission by 79%. Most occupational health departments now advise their healthcare workers to commence treatment within 1 hour of injury with multiple therapy which depending on the risk of HIV exposure should either be a two-drug regimen for 4 weeks, or for those at higher risk a three-drug regimen. These used to commonly include zidovudine (AZT), as it is the only drug which has proven to reduce HIV risk following occupational exposure. However, as AZT is often poorly tolerated, newer medications such as tenofovir and emtricitabine are being increasingly utilized instead, mostly in combination with a protease inhibitor.

This type of regimen may well reduce the risks of seroconversion further. In some countries, surgeons with a persistently undetectable viral load (less than 50 copies) may be allowed to return to performing EPPs under occupational health supervision.

Intraoperative transmission of HBV occurs more readily than with HIV, and exposure of skin or mucous membrane to blood from a hepatitis B e antigen (HBeAg) carrier involves a highly significant risk of transmission for those who are not immune (West 1984). The risk of seroconversion following an accidental inoculation with blood from an HBeAg carrier, in the absence of immunity, is up to 30% for susceptible healthcare workers without post-exposure prophylaxis (PEP) or sufficient hepatitis B vaccination (Wicker et al. 2008). Hepatitis B surface antigen (HBsAg) is found in 0.5% to 1% of patients in inner cities and in 0.1% of patients in rural areas and blood donors. Given a needlestick rate of 5% per operation, the risk of acquiring the virus in a surgical lifetime is potentially high. Prior to the introduction of HBV vaccination an estimated 40% of American surgeons became infected at some point in their careers, with 4% becoming carriers. Acute infection with HBV is associated with the development of fulminant hepatitis in approximately 1% of individuals. Carriers may go on to develop chronic liver damage, cirrhosis, or hepatocellular carcinoma, carrying an overall mortality of approximately 40%.

Transmission of HBV from infected healthcare workers to patients is rare but well documented. Welch et al. (1989) reported a case of an infected gynecologist who transmitted HBV to 20 of his patients; the operations carrying greatest risk of infection were hysterectomy (10/42) and caesarean section (10/51). In view of this risk, government guidelines in most countries stipulate that surgeons should be immune to HBV, either through natural immunity or vaccination, the exceptions being staff who fail to respond to the vaccine (5%–10%) and those who are found to be HBsAg positive in the absence of "e" antigenemia (United Kingdom Advisory Group on Hepatitis 2003). In the United Kingdom, the United States, and other countries this is a statutory obligation. Those who fail to respond to vaccination should receive hepatitis B immunoglobulin following needlestick injury where the patient is HBV positive.

HCV, the commonest cause of non-A non-B hepatitis in the developed world, is also known to be spread by blood contamination. Routine screening for antibodies among blood donors in the United Kingdom has shown that 0.05% were seropositive in 2001; many of these were seemingly healthy asymptomatic carriers. However, as many as 85% of injecting drug users may be seropositive. In the United Kingdom, infection with HCV is second only to alcohol as a cause of cirrhosis, chronic liver disease, and hepatocellular carcinoma, although the clinical course in seemingly healthy individuals is unclear.

A recent anonymous seroprevalence study of staff at an inner London teaching hospital reported that infection with HCV was no higher than that previously seen in blood donors. The seroprevalence was no different for workers involved with direct clinical exposure (medical and nursing staff) compared with those at risk of indirect clinical exposure (laboratory and ancillary staff) (Zuckerman et al. 1994). However, these findings should not lead to complacency. From epidemiological data, it would appear that HCV infection is less contagious than HBV, but more so than HIV. The risk of a HCV infection is estimated at between 3 and 10%; it increased tenfold if the source patient has high levels of virus load (Wicker et al. 2008). It would, however, appear that transmission is very rare with solid-bore needles, i.e., almost exclusively follows inoculation with hollow bore needles. Transmission has rarely followed mucous membrane exposure and never via non-intact or intact skin. The possibility of HCV infection should be considered in the event of needlestick injury. Immunization and PEP are not available for those exposed to HCV. Recently, in the United Kingdom the same restrictions have been introduced to healthcare workers infected with HIV and hepatitis C i.e., preclusion from performing exposure-prone procedures. This is not the case in any other country.

PREVENTION OF BLOOD-BORNE INFECTION

Some surgeons have advocated preoperative screening of patients for HIV infection. They argue that patients shown to be infected should be treated as high-risk, while the remaining patients would be labeled as low-risk, with the consequent development of a two-tier infection control policy. However, such an approach is fraught with political, ethical, logistical, and financial implications, and furthermore, wrongly assumes that infected patients can always be identified by serological testing. The universal precautions suggested below are practicable, and effectively minimize the intraoperative infection risk of both surgeon and patient. These precautions are based on the procedure rather than the perceived risk status of the patient. As discussed above, the greatest risk of contracting a blood-borne pathogen is from needlestick injury. Vaginal hysterectomy has been shown to have the highest rate (10%) of needlestick injury of any surgical procedure (Tokars et al. 1991). Glove puncture has been used as a measure of skin contamination and a reflection of needlestick injury; the highest rate of glove puncture reported in any surgical procedure was 55% at caesarean section. Double gloving has shown a sixfold diminution in inner glove puncture rate, and anecdotally appears to result in a reduction in needlestick injury, but it is uncomfortable, particularly during protracted procedures, making it unsuitable for many gynecologic

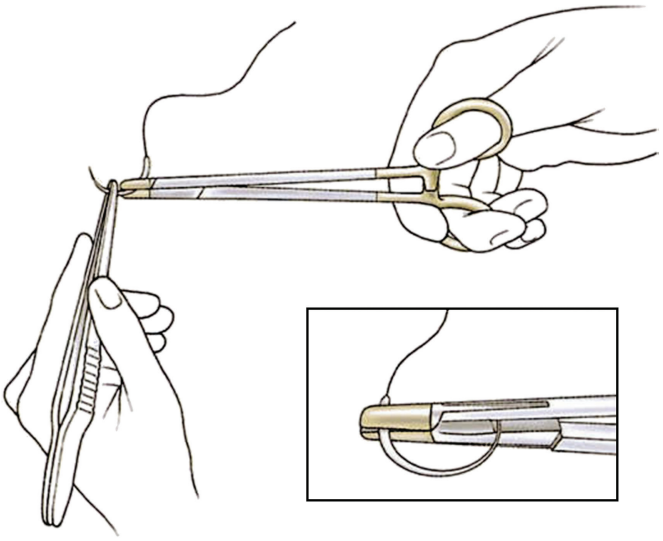

Figure 1.2 Safety needle holder.

Table 1.4 Risk Factors for Transmission of Blood-Borne Pathogens during Surgical Practice

1	Prolonged surgical procedure
2	Heavy blood loss
3	Operating within a confined space, e.g., pelvis or vagina
4	Poor lighting
5	Guiding the needle by feel

Table 1.5 Simple Precautions Available to Reduce Needlestick Injury

1	Blunt-tipped needles: available from Davis & Geck (Protec Point) and Ethicon (Ethiguard needle)
2	Staple guns for skin closure: available from Autosuture and Ethicon Endosurgery
3	Staples for bowel anastomosis: available from Autosuture and Ethicon Endosurgery
4	Spectacles/protective eyewear: blood-borne pathogens have, however, only been shown to be transmitted very rarely and usually only in the presence of gross ocular contamination
5	Magnet for picking up sharps
6	Hands-free disposable sharp boxes for needles and blades
7	Blunt towel clips
8	Self-adhesive drapes

oncological operations. Blunt-tipped needles, such as the Protec Point (Davis & Geck, Gosport, United Kingdom) and Ethiguard (Ethicon, Edinburgh, United Kingdom), appear to reduce the rate of glove puncture, and one of the authors (JRS) has never sustained a needlestick injury in 8 years of continuously using these needles. The newer needles are capable of penetrating the majority of tissues including uterine muscle, vaginal vault, cervix, peritoneum, and rectus sheath. They are unsuitable for bowel and bladder surgery and do not penetrate skin, but they have been used subcutaneously for abdominal wound closure. Abdominal skin closure can also be safely undertaken with the use of staples. This is particularly important since it has been shown that 5% of glove punctures occurred during this stage of the procedure. Just under half of punctures occur in the right hand—a surprising finding considering that most surgeons are right-handed and therefore grasp the needle holder with the dominant hand. Injury appears to occur during knot tying, and a safety needle holder with provision for guarding the needle tip at this stage and when returning the needle to the scrub nurse is now available (Thomas et al. 1995) (Figure 1.2). The use of a kidney dish for passing scalpels between staff should also be encouraged, as should safe needle and blade disposal in hands-free surgical sharps boxes. Blades or needles that have fallen on the floor should be retrieved with a magnet prior to disposal. Blunt towel clips are also available to prevent injury while draping. Reusable self-adhesive drapes are available, as are disposable self-adhesive drapes with a surrounding bag to prevent gross contamination.

Skin and mucous membrane contamination should be avoided by the use of masks and waterproof gowns. Glasses or other protective eyewear should be worn to prevent contamination by facial splashes of blood and other body fluids.

The risks and safety measures discussed above are summarized in Tables 1.4 and 1.5. Table 1.4 demonstrates that oncological surgery carries the greatest risk. However, the simple and relatively cheap procedures and precautions suggested in Table 1.5 can reduce the risk for both surgeon and patient to extremely low levels.

REFERENCES

Advisory Group on Hepatitis. 1993. *Protecting Health Care Workers and Patients from Hepatitis B*. London: HMSO.

Bergqvist D, Agnelli G, Cohen AT, et al. 2002. Duration of prophylaxis against venous thromboembolism with enoxaparin after surgery for cancer. *N Engl J Med* **346**(13):975-80.

Cardo DM, Culver DH, Ciesielski CA, et al. 1997a. A case-control study of HIV seroconversion in health care workers after percutaneous exposure. *N Engl J Med* **337**:1542–3.

Cardo DM, Culver DH, Ciesielski CA, et al. 1997b. A case control study of HIV seroconversion in health care workers after percutaneous exposure. *N Engl J Med* **337**:1485–90.

Centers for Disease Control (CDC). 1987. Update: Human immunodeficiency virus infection in health care workers exposed to blood of infected patients. *MMWR* **36**:285–9.

Evans HE, Mercer CH, Rait G, et al. 2009. Trends in HIV testing and recording of HIV status in the UK primary care setting: A retrospective cohort study 1995–2005. *Sex Transm Infect* **85**:520–6.

Gerberding JL. 2003. Occupational exposure to HIV in health care settings. *N Engl J Med* **348**:826–33.

Gomez-Outes A, Lecumberri R, Pozo C, Rocha E. 2009. New anticoagulants: Focus on venous thromboembolism. *Curr Vasc Pharmacol* **7**:309–29.

Howard RJ. 1990. Human immunodeficiency virus testing and the risk to the surgeon of acquiring HIV. *Surg Gynaecol Obstet* **171**:22–6.

Makary MA, Al-Attar A, Holzmueller CG, et al. 2007. Needlestick injuries among surgeons in training. *N Engl J Med* **356**:2693–9.

NICE. 2015. Guidance on venous thromboembolism in patients undergoing surgery.

Reason J. 1990. *Human Error*. Cambridge: University Press, Cambridge.

Sweet RL, Gibbs RS. 1990. *Infectious Diseases of the Female Genital Tract*. 2nd ed. Baltimore: Williams & Wilkins.

Thomas PB, Falder S, Jolly M, et al. 1995. The role of blunt-tipped needles and a new needle-holder in reducing needlestick injury. *J Obstet Gynaecol* **15**:336–8.

Tokars J, Bell D, Marcus R, et al. 1991. Percutaneous injuries during surgical procedures (Abstract). VII International Conference on AIDS, Florence, Italy.

Wicker S, Cinatl J, Berger A, et al. 2008. Determination of risk of infection with blood-borne pathogens following a needlestick injury in hospital workers. *Ann Occup Hyg* **52**:615–22.

Welch J, Webster M, Tilzey AJ, et al. 1989. Hepatitis B infections after gynaecological surgery. *Lancet* 1989; **1**:205–7.

West DJ. 1984. The risk of hepatitis B infection among health professionals in the United States: A review. *Am J Med Sci* **287**:26–33.

WHO. *Surgical Safety Checklist and Implementation Manual*. Who.int. 2010-12-08.

Zuckerman J, Clewley G, Griffiths P, Cockroft A. 1994. Prevalence of hepatitis C antibodies in clinical health-care workers. *Lancet* **343**:1618–20.

2 Preoperative workup

Jessica Thomes-Pepin and Chris Stephenson

INTRODUCTION

Surgical planning for the patient with a gynecologic malignancy begins with a detailed assessment of perioperative risk determined by pre-existing medical comorbidities. Reducing perioperative-associated complications and improving outcomes remains a prudent goal in procedural preparation.

Additional considerations in surgical planning include discussions with the patient regarding postoperative expectations, the need for blood products, the need for subsequent additional therapy including surgical procedures or chemotherapy, the possibility for ostomy or placement of other tubes and/or catheters, potential changes in sexual function, and the effect of the procedure on quality of life.

The more informed a patient can be regarding expectations surrounding surgical management, the more likely they are to make sound judgment regarding therapy and to be satisfied with their overall care. The following recommendations help assess surgical "fitness" by organ system. Highlights are indicated in a box, as above.

ASSESSMENT OF PERIOPERATIVE CARDIAC RISK

The most treatable cause of morbidity and mortality associated with noncardiac surgery remains perioperative cardiovascular complications. Nearly one-third of all patients undergoing major elective surgery have at least one major cardiac risk factor, with the greatest risk of perioperative death stemming from associated cardiac stress (Mangano 1990).

Acute myocardial infarction (MI) comprises 50% of all perioperative cardiovascular complications, which most commonly occur within the first three days after surgery (Ashton et al. 1993).

A postoperative MI carries a 28-fold increase in risk of cardiovascular complications within the first 6 months following surgery; including a 40% to 70% increased risk of death (Shah et al. 1990). The 2014 ACC/AHA perioperative cardiac risk guidelines determine preoperative risk according to patient and procedural factors applied within an evidence-based algorithm (Fleisher et al. 2008). Preoperative cardiac assessment allows the determination of fitness for surgery, minimizes major adverse cardiac events (MACE) in the postoperative period, and identifies those at risk for long-term adverse outcomes.

2014 ACC/AHA PERIOPERATIVE CARDIAC RISK GUIDELINES

Step 1: Determine the Urgent or Emergent Nature of the Procedure

Emergent (<6 hours) or urgent (6–24 hours) procedures allow for limited to no clinical evaluation. The risk of cardiovascular complications in these procedures is increased two- to fivefold in comparison to elective procedures (Goldman et al. 1977). This prompts the operative team to employ more aggressive perioperative surveillance and management. A procedure performed on an elective basis allows further evaluation and assessment, and possibly treatment of active cardiac conditions, lowering the overall risk of MACE. The majority of oncologic procedures fall into the time-sensitive category (<1–6 weeks), allowing for evaluation and further assessment without significant time for intervention.

Step 2: Determine the Presence of Active Cardiac Disease or Active Clinical Risk Factors of Cardiac Disease

There remains a persistent underestimation of cardiac disease in women evaluated preoperatively. In patients with established cardiovascular disease, preoperative assessment must include eliciting any recent change in symptoms including shortness of breath, palpitations, fatigue, or chest pain. Unstable angina, MI, significant arrhythmias, cardiomyopathy, or severe cardiac valvular disease all increase the risk of MACE. MACE after noncardiac surgery is often associated with prior coronary artery disease (CAD), and the timing of a recent MI impacts perioperative morbidity and mortality (Fleisher et al. 2008).

In a retrospective chart analysis, the incidence of postoperative MI decreased as the length of time from MI to procedure increased (0–30 days, 32.8%; 31–60 days, 18.7%; 61–90 days, 8.4%; 91–180 days, 5.9%) (Livhits et al. 2011).

A recent MI occurring within 6 months of noncardiac surgery has been found to be an independent risk factor for perioperative stroke and associated with an eightfold increase in perioperative mortality (Mashour et al. 2011). Obviously, most cancer patients cannot wait 6 months for surgery, however a delay to beyond 30 days may be acceptable in certain circumstances. Age, smoking, hyperlipidemia, and diabetes mellitus are important historical factors that portend further investigation.

Patients with clinical heart failure (HF) or history of HF are at significant risk for perioperative complications (Detsky et al. 1986). Risk-adjusted 30-day mortality and readmission rates in patients undergoing major noncardiac surgery was 50% to 100% higher in patients with HF than in elderly controls without a history of CAD or HF (Hammill et al. 2008).

Decompensated HF confers the highest perioperative risk, while severely decreased (<30%) left ventricular ejection fraction (LVEF) independently contributes to perioperative morbidity and mortality (Healy et al. 2010).

It is recommended that patients with clinically suspected moderate or greater degrees of valvular stenosis or regurgitation undergo preoperative echocardiography if not performed within the last year or if there has been a change in clinical status or the physical examination since the last evaluation (Douglas et al. 2011).

Preoperative recommendations regarding non-ischemic cardiomyopathies must be made in conjunction with the patient's cardiologist or a gynecological oncologist with a thorough understanding of the pathophysiology of the cardiomyopathy who can integrate assessment and management of the underlying process and associated HF (Fleisher et al. 2015). For those patients requiring intervention, perioperative risk may be lowered if performed prior to elective noncardiac surgery (Nishimura et al. 2014). There are a paucity of data regarding cardiac arrhythmias and conduction disorders regarding true contribution to perioperative risk; however, their presence within the preoperative setting warrants further investigation. Patients with an implantable electronic device (IED) should be managed in conjunction with the clinician following the patient regarding the device and underlying cardiac disease. If feasible, a patient with pulmonary hypertension should undergo evaluation by a specialist prior to proceeding with surgery, and continue all chronic pulmonary vascular targeted therapy unless contraindicated (Fleisher et al. 2015).

Step 3: Calculation of Risk to Determine Perioperative Morbidity

In the 2007 ACC/AHA guidelines, the committee separated clinical risk factors into major, intermediate, and minor categories (Eagle et al. 2002) (Table 2.1). The presence of one or more active cardiac conditions with major clinical risk warrants further investigation prior to proceeding with surgery. In conjunction with estimation of procedural risk, the specific combined incidence of cardiac death and nonfatal MI helps determine whether further preoperative cardiac testing is indicated (Tables 2.2A and 2.2B). The 2014 ACC/AHA clinical practice guidelines (CPG) recommend use of a validated risk-prediction tool to predict the risk of perioperative MACE in patients undergoing noncardiac surgery. Different calculators include the Revised Cardiac Risk Index (RCRI), the American College of Surgeons National Surgical Quality Improvement Program (NSQIP), Myocardial Infarction and Cardiac Arrest (MICA), and the American College of Surgeons NSQIP Surgical Risk Calculator (Cohen et al. 2013, Gupta et al. 2011, Lee et al. 1999) For patients with a low risk of perioperative MACE, further testing is not recommended prior to proceeding with the planned procedure.(Schein et al. 2000)

Step 4: Determine the Patient's Functional Capacity, or their Ability to Perform Common Daily Tasks

Functional capacity is measured in METS (metabolic equivalents), and correlates with oxygen demands in stress testing (Hlatky et al. 1989) (Table 2.3).

Functional status is a reliable predictor of perioperative and long-term cardiac events (Fleisher et al. 2015). A high functional status usually requires no further testing.

Table 2.1 The Presence of One or More Active Cardiac Conditions with Major Clinical Risk Warrants Further Investigation Prior to Proceeding with Surgery

Major cardiac risk factors	Unstable coronary artery syndromes
	Unstable or severe angina
	Recent myocardial ischemia
	Uncertain timing of historic MI-Q waves on EKG
	Acute MI: acute event 7 days or prior
	Recent MI: >7 days or ≤1 month prior
	Decompensated heart failure
	Significant arrhythmias
	Severe valvular disease
Intermediate cardiac risk factors	History of heart failure
	History of compensated heart disease or prior heart failure
	History of cerebrovascular disease
	Diabetes mellitus
	Renal insufficiency
Minor cardiac risk factors	Abnormal EKG: LBBB, LVH, ST abnormality
	Rhythm other than sinus
	Uncontrolled systemic hypertension

Abbreviations: MI, myocardial infarction; EKG, electrocardiogram; LBBB, left bundle branch block; LVH, left ventricular hypertrophy.
Sources: Adapted from Freeman WK, Gibbons RJ, *Mayo Clin Proc* **84**(1):79–90, 2009; Flleisher et al. (2007), Eagle KA, Berger PB, Calkins H, et al., *Anesth Analg* **94**:1052–64, 2002.

In patients without a recent exercise test, functional status can be estimated from the ability to perform activities of daily living (Reilly et al. 1999). Functional capacity is classified as excellent (10), good (7−10), moderate (4–6), poor (4 or less), or unknown. Perioperative cardiac and long-term risks are increased in patients unable to perform 4 METs of work during daily activities (Fleisher et al. 2015). In patients with poor or unknown functional capacity, the number of active clinical risk factors should guide the need for further testing. The 2014 ACC/AHA CPG provides a preoperative algorithm for guidance on perioperative management to minimize associated risk on the basis of available evidence and expert opinion (Figure 2.1).

Step 5: Supplemental Preoperative Evaluation

Supplemental testing allows the clinician to obtain prognostic information, further guiding therapy and perioperative management. A preoperative electrocardiogram (ECG) within 30 days of surgery is useful in patients with established coronary heart disease, providing a useful baseline standard to measure changes postoperatively (Beattie et al. 2006).

An ECG is not useful in asymptomatic patients undergoing low-risk surgical procedures (Liu et al. 2002, Turnbull and Buck, 1987).

Left ventricular (LV) function should be preoperatively evaluated in patients with dyspnea of unknown origin, patients with HF with worsening dyspnea, other changes in clinical status, or stable patients with a history of LV dysfunction and no assessment within a year (Healy et al. 2010).

Table 2.2 A) Procedural-Based Risk

High risk (5%)	Advanced upper abdominal extensive debulking
	Segmental liver resection
	HIPEC
Intermediate risk (1%–5%)	Intraperitoneal and intrathoracic surgery
	Head and neck surgery
	Orthopedic surgery
	Simple and radical hysterectomy
	Robotic hysterectomy
	Extensive MIS debulking
Low risk (<1%)	Simple endoscopic procedures (IP port)
	Superficial procedures
	Wide local excision ambulatory surgery
	Vascular port placements

Source: Adapted from Fleisher LA, Beckman JA, Brown KA, et al., *Anesth Analg* **106**:685–712, 2008.

Table 2.2 B) Preoperative Cardiac Evaluation Algorithm

Without known cardiac issue	Low-risk procedure	Proceed with planned procedure
	Intermediate- or high-risk procedure	Determine functional capacity, proceed with planned procedure if ≥4 METs without symptoms
Low or unknown functional	Intermediate- or high-risk procedure	Consider cardiac testing if it will change capacity management Proceed with heart rate control or consider noninvasive testing if it will change management

Source: Adapted from Fleisher LA et al., *J Am Coll Cardiol* **50**:1707–32, 2007.

Table 2.3 Metabolic Equivalents

I.	Eat, dress, use the toilet without assistance
II.	Walk indoors and around the house without assistance
III.	Walk a block or two on level ground at 2–3 mph without assistance
IV.	Perform light work around the house including dusting or washing dishes
V.	Climb a flight of stairs or walk up a hill
VI.	Walk on level ground at 4 mph
VII–X.	Run a short distance
	Perform heavy housework including scrubbing floor or lifting or moving heavy furniture
	Participate in moderate recreational activity including golf, bowling, dancing, tennis
	Perform strenuous sport activity like running, swimming
	Participate in singles tennis, football, basketball, and skiing

Sources: Adapted from Hlatky MA, Boineau RE, Higginbotham MB, et al., *Am J Cardiol* **64**:651–4, 1989; Fletcher GF, Balady G, Froelicher VF, et al., *Circulation* **91**:580–615, 1995; Fleisher LA, Beckman JA, Brown KA, et al., *Anesth Analg* **106**:685–712, 2008.

For patients with elevated risk who have an excellent functional capacity (>10 METs), and possibly moderate to good (>4 to 10 METs) functional capacity, it is reasonable to forego further testing and proceed with surgery (Carliner et al. 1985). For patients with elevated risk and poor (<4 METs) or unknown functional capacity, it may be reasonable to perform exercise testing with cardiac imaging or noninvasive pharmacologic stress testing to assess for myocardial ischemia if it will change management (Das et al. 2000). Perioperative cardiac risk is directly linked to the extent of jeopardized viable myocardium identified by stress cardiac imaging (Beattie et al. 2006). Cardiopulmonary exercise testing may be considered for patients undergoing elevated risk procedures where functional capacity is unknown (Snowden et al. 2013).

PERIOPERATIVE THERAPY

In patients where preoperative risk stratification recommends revascularization prior to surgery, proceeding with therapy should be dictated according to existing clinical practice guidelines (Hillis et al. 2012, Levine et al. 2011). There are no randomized controlled trials to support routine coronary revascularization prior to noncardiac surgery exclusively to reduce perioperative cardiac events (McFalls et al. 2004).

In patients who have had prior percutaneous coronary intervention (PCI), elective noncardiac surgery should be delayed 14 days after balloon angioplasty, 30 days following bare metal stent (BMS) placement, and 365 days following drug-eluding stent (DES) placement (Berger et al. 2010, Nuttall et al. 2008, van Kuijk et al. 2009).

In situations where noncardiac surgery is necessary, a consensus decision regarding the relative risks of surgery and antiplatelet therapy can be helpful.

Patients with implantable cardioverter defibrillators (ICDs) who have inactivated programming preoperatively should have continuous cardiac monitoring utilized intraoperatively with external defibrillation equipment readily available (Fleisher et al. 2015).

PERIOPERATIVE MEDICAL MANAGEMENT

Several randomized control trials have suggested perioperative beta-blockade reduces cardiac events in high-risk patients during noncardiac procedures. However, other studies, including a

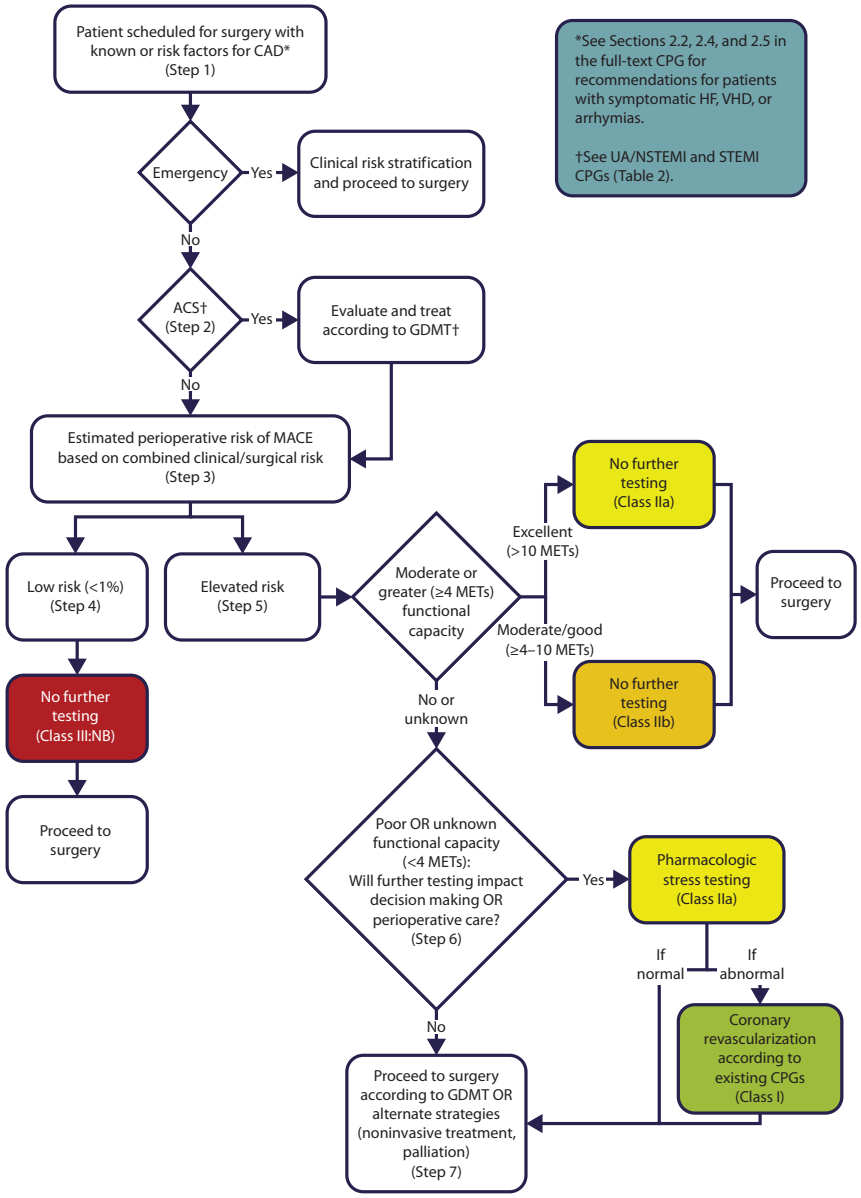

Figure 2.1 Stepwise approach to perioperative cardiac assessment for CAD. Colors correspond to the Classes of Recommendations in Table 2.1. *Step 1:* In patients scheduled for surgery with risk factors for or known CAD, determine the urgency of surgery. If an emergency, then determine the clinical risk factors that may influence perioperative management and proceed to surgery with appropriate monitoring and management strategies based on the clinical assessment (see Section 2.1 in Fleisher et al. 2014 for more information on CAD). (For patients with symptomatic HF, VHD, or arrhythmias, see Sections 2.2, 2.4, and 2.5 in Fleisher et al. 2014 for information on evaluation and management.) *Step 2:* If the surgery is urgent or elective, determine if the patient has an ACS. If yes, then refer patient for cardiology evaluation and management according to GDMT according to the UA/NSTEMI and STEMI CPGs (18,20). *Step 3:* If the patient has risk factors for stable CAD, then estimate the perioperative risk of MACE on the basis of the combined clinical/surgical risk. This estimate can use the American College of Surgeons NSQIP risk calculator (http://www.surgicalriskcalculator.com) or incorporate the RCRI (131) with an estimation of surgical risk. For example, a patient undergoing very low-risk surgery (e.g., ophthalmologic surgery), even with multiple risk factors, would have a low risk of MACE, whereas a patient undergoing major vascular surgery with few risk factors would have an elevated risk of MACE (Section 3, Fleisher et al. 2014). *Step 4:* If the patient has a low risk of MACE (<1%), then no further testing is needed, and the patient may proceed to surgery (Section 3, Fleisher et al. 2014). *Step 5:* If the patient is at elevated risk of MACE, then determine functional capacity with an objective measure or scale such as the DASI (133). If the patient has moderate, good, or excellent functional capacity (≥4 METs), then proceed to surgery without further evaluation (Section 4.1, Fleisher et al. 2014). *Step 6:* If the patient has poor (<4 METs) or unknown functional capacity, then the clinician should consult with the patient and perioperative team to determine whether further testing will impact patient decision making (e.g., decision to perform original surgery or willingness to undergo CABG or PCI, depending on the results of the test) or perioperative care. If yes, then pharmacological stress testing is appropriate. In those patients with unknown functional capacity, exercise stress testing may be reasonable to perform. If the stress test is abnormal, consider coronary angiography and revascularization depending on the extent of the abnormal test. The patient can then proceed to surgery with GDMT or consider alternative strategies, such as noninvasive treatment of the indication for surgery (e.g., radiation therapy for cancer) or palliation. If the test is normal, proceed to surgery according to GDMT (Section 5.3, Fleisher et al. 2014). *Step 7:* If testing will not impact decision making or care, then proceed to surgery according to GDMT or consider alternative strategies, such as noninvasive treatment of the indication for surgery (e.g., radiation therapy for cancer) or palliation. ACS indicates acute coronary syndrome; CABG, coronary artery bypass graft; CAD, coronary artery disease; CPG, clinical practice guideline; DASI, Duke Activity Status Index; GDMT, guideline-directed medical therapy; HF, heart failure; MACE, major adverse cardiac event; MET, metabolic equivalent; NB, No Benefit; NSQIP, National Surgical Quality Improvement Program; PCI, percutaneous coronary intervention; RCRI, Revised Cardiac Risk Index; STEMI, ST-elevation myocardial infarction; UA/NSTEMI, unstable angina/non–ST-elevation myocardial infarction; and VHD, valvular heart disease. * and †: see further Fleischer et al. 2014. (Reproduced with permission from Fleisher LA, Fleischmann KE, Auerbach AD et al. 2015. *J Am Coll Cardiol* 2014;64(22):e77-e137.)

systematic review, have suggested no benefit, with instead an increase in risk of bradycardia and stroke (Juul et al. 2006, Shammash et al. 2001, Yang et al. 2006). According to the 2014 ACC/AHA CPG, beta blockers should be continued in those chronically dependent (Andersson et al. 2014, Lindenauer et al. 2004). Initiating perioperative beta-blockade in patients at intermediate or high risk for myocardial ischemia as determined by preoperative risk stratification may be reasonable (Boersma et al. 2001); however, initiating the day of surgery is not recommended (Devereaux et al. 2006). Similarly, statins should be continued perioperatively (Desai et al. 2010, Kennedy et al. 2005, Lindenauer et al. 2004, Raju et al. 2013). A meta-analysis by Hindler et al. uncovered a 44% reduction in mortality with perioperative statin use, while perioperative statin withdrawal is an independent predictor of myonecrosis (Hindler et al. 2006, Le Manach et al. 2007). The majority of data on perioperative statin use is derived from observational studies demonstrating a protective effect on cardiac complications (Lindenauer et al. 2004, Raju et al. 2013).

Studies have suggested alpha-2 agonists reduce mortality and MI in vascular procedures; however, 2014 ACC/AHA recommendations note these benefits do not transcend to patients undergoing noncardiac surgery (Ellis et al. 1994, Oliver et al. 1999, Stuhmeier et al. 1996, Thomson et al. 1984, Wijeysundera and Beattie 2003). Perioperative calcium channel blockers may also reduce perioperative ischemia and SVT with a trend toward reduced MI and death; however, large-scale trial studies are needed (Wijeysundera and Beattie 2003). Calcium channel blockers with significant negative inotropic effect (i.e., diltiazem and verapamil) have potential to worsen HF (Fleisher et al. 2015). Perioperative angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blocker (ARB) data are limited to observational studies demonstrating an increased risk of hypotension on the day of surgery, without change in cardiovascular outcomes (Rosenman et al. 2008, Turan et al. 2012). The 2014 ACC/AHA CPG states it is reasonable to continue ACE inhibitors and ARBs perioperatively, and if they are held preoperatively, to restart them as soon as clinically feasible postoperatively (Fleisher et al. 2015). In order to lower the risk of renal failure, ACE inhibitors and ARBs should be used judiciously in procedures where large fluid shifts can be anticipated (Bertrand et al. 2001, Comfere et al. 2005, Coriat et al. 1994).

Antiplatelet therapy for the prevention of BMS and DES thrombosis is most protective within the first 4 to 6 weeks after stent implantation (Nuttall et al. 2008, van Kuijk et al. 2009). If urgent surgery is required within this time period, antiplatelet therapy should be continued unless the relative risk of bleeding outweighs the benefit of the prevention of stent thrombosis. If the antiplatelet therapy must be discontinued, it is recommended to continue aspirin and to restart antiplatelet therapy as soon as possible after surgery. Perioperative planning should be determined in concert with the entire medical team to optimize therapy plans and outcomes. The initiation or continuation of aspirin is not beneficial in patients undergoing elective, noncardiac, noncarotid surgery in those without stents unless the risk of ischemic events outweighs the risk of surgical bleeding (Fleisher et al. 2015).

In patients with atrial fibrillation (AF) and prosthetic valves, vitamin K antagonists are prescribed for stroke and thromboembolic prevention. Factor Xa inhibitors are additionally used in patients for stroke prevention with AF but are associated with an increased risk of thrombotic events compared to warfarin (Fleisher et al. 2015). In patients requiring surgery, the risks of bleeding must be weighed against the benefit of remaining on anticoagulants. In procedures with minimal risk of bleeding, it may be reasonable to continue the anticoagulants perioperatively. Patients on vitamin K antagonists with prosthetic valves may require bridging therapy with unfractionated heparin (UFH) or low-molecular weight heparin (LMWH), depending on the associated risks. Reversal agents include vitamin K and fresh frozen plasma. Of note, vitamin K response is delayed, including a delayed return to therapeutic level of anticoagulation, when the antagonists are restarted. Vitamin K antagonists should be discontinued 5 days preoperatively, with bridging therapy at the same time, if utilized. The antagonists may be discontinued earlier, if the international normalized ratio (INR) is higher. The INR should be checked the day prior to surgery. If elevated (1.5 or higher) the day prior, 1–2 mg of oral vitamin K should be administered with a repeat INR the morning of surgery (Douketis et al. 2012). Vitamin K antagonist therapy may then be reinitiated 12–24 hours postoperatively following confirmation of adequate hemostasis. LMWH may be initiated according to risk 48–72 hours postoperatively and continued until the INR reaches the therapeutic range of 2–3. For procedures with an elevated risk of surgical bleeding, it is recommended to discontinue the inhibitors 48 hours or more prior to the procedure (Fleisher et al. 2015). Retrievable inferior vena cava (IVC) filters may be utilized in patients where the risk of bleeding with anticoagulation outweighs the utilization of antithrombotic agents. Temporary IVC filters have been found to effectively capture thrombi and protect against thromboembolic complications (Linsenmaier et al. 1998).

ASSESSMENT OF HEMATOLOGIC RISK

Thromboembolic Disease

Forty percent of postoperative gynecologic deaths and the most preventable cause of hospital deaths are directly related to pulmonary emboli.

Thromboembolic disease is the most frequent cause of postoperative death in patients with uterine or cervical carcinoma (Martino et al. 2006).

Without thromboprophylaxis, the postoperative gynecologic cancer patient has between a 17% and 40% estimated risk of developing a venous thromboembolism (Clarke-Pearson et al. 1984b). Risk stratification, dependent upon patient-specific and procedural-specific risk factors, may be implemented within models to determine the need for therapy, balanced with the risk of bleeding (Geerts et al. 2008). One such model, the Caprini score, estimates risk according to a point system (Caprini 2005). An adaptation provided in the American College of Chest Physicians consensus statement published in 2012 categorizes risk as very low (0–1 point), low (2 points), moderate (3–4 points), or high (≥ 5 points) (Gould et al. 2012). However, gynecologic patients have not been validated individually with the

Caprini score, and are instead stratified according to abdominal or pelvic surgeries. An alternative risk classification system is provided within the ACOG Practice Bulletin 84, modified from the 2004 Chest guidelines (Geerts et al. 2008) (see Figure 2.2). Commonly placed within the highest risk categories, the cancer patient is often subject to additional risks including chemotherapy, radiation therapy, and hormonal treatment, further necessitating the need for long-term thromboprophylactic therapy.

Options for perioperative thromboembolic prophylaxis include pharmacologic and mechanical methods. The risk of venous thromboembolism incidence is decreased to 2% to 6% with standard preventive measures, including intermittent pneumatic compression (IPC), UFH, and LMWH. (Prevention of deep vein thrombosis and pulmonary embolism (2007)) According to the ENOXACAN II study, 4 weeks of postoperative anticoagulation decreases the incidence of VTE from 12% to 4.8% in cancer patients undergoing abdominal, gynecological, or urological surgery (Bergqvist et al. 2002).

IPC devices reduce venous stasis and promote endogenous fibrinolysis. A threefold reduction of venous thromboembolism was found in gynecologic cancer patients undergoing surgery when IPCs are used intraoperatively and continued for 5 days postoperatively (Clarke-Pearson et al. 1984c).

Comparisons of LMWH to UFH have shown overall superiority to LMWH. However, UFH in individualized PTT directed treatment may be therapeutically equivalent. Paradoxically, the overall cost of LMWH is less than UFH when all the nursing and lab costs are included. UFH may be associated with low pharmacy cost.

Concerns with UFH include an increased risk of postoperative bleeding and heparin-induced thrombocytopenia (Clarke-Pearson et al. 1984a). LMWH is associated with decreased risk of bleeding complications, has increased bioavailability and greater ease of use with once-daily dosing. When compared to UFH, LMWH is rarely associated with Heparin-Induced Thrombocytopenia (HIT). Dual prophylaxis with pharmacologic and mechanical methods may benefit the high-risk oncology patient, and is possibly cost-effective (Agnelli et al. 1998; Clarke-Pearson et al. 2003).

Lowest preoperative risk patients do not require prophylaxis, but should begin early ambulation (Figure 2.2). Moderate-risk patients should have at least one type of preventative measure (mechanical or pharmacologic). High-risk patients should receive both mechanical and pharmacologic prevention with IPCs and LMWH (Douketis et al. 2012). IPCs should be initiated preoperatively and continued until ambulation. While all methods are cost-effective, patients in the high-risk group benefit most from the use of IPC with LMWH (Dainty et al. 2004). High-risk patients subjected to a major cancer procedure or with multiple risk factors should receive thromboprophylaxis after hospital discharge for up to 28 days postoperatively. Extended prophylaxis for this is supported by the American College of Chest Physicians and American College of Gynecologists (Geerts et al. 2008).

Inherited risk factors for VTE typically do not result in VTE until an additional precipitating event induces formation (Middeldorp et al. 1998). Factor V Leiden mutation and prothrombin gene mutation G20210A are the most common mutations uncovered with VTE occurrence. Factor V Leiden is carried by 5% of Caucasians and in up to 20% of patients with VTE (Dahlback et al. 1993). Prothrombin G20210A mutations are less common, almost exclusively found in Caucasians, and found in 6% of patients with VTE (Poort et al. 1996). Antithrombin III, protein C, and protein S are additional inherited deficiencies that also result in an increased risk of VTE. Although rare, patients with a strong family history of clots who are negative for Factor V Leiden or prothrombin mutation should consider additional testing (Rosendaal 2005). Antiphospholipid syndrome is an acquired thrombophilia associated with arterial and venous thrombosis. Testing for antiphospholipid syndrome includes serum analysis for lupus anticoagulant and anticardiolipin antibodies (de Groot and Derksen, 2005).

Duplex ultrasonography is ordered with suspicion for the presence of deep venous thromboembolism (DVT).

The sonogram duplex may need to be repeated as the risk of DVT continues throughout the postop period and the sensitivity of the test is only approximately 80% and highly variable.

Treatment is with heparinization to 1.5 times control prothrombin time or with therapeutic doses of LMWH. Increasing sensitivity of dynamic contrast-enhanced computerized tomography has confirmed the replacement of the prior gold standard of pulmonary arteriogram in the diagnosis of pulmonary embolism. Upon diagnosis of a pulmonary embolism, the patient is anticoagulated with UFH or LMWH. LMWH and direct thrombin inhibitors are generally preferred. Long-term anticoagulation should last for 3 months in the case of DVT and 6 months in the case of pulmonary embolism. Some patients on thrombogenic chemotherapy regimens may benefit from lifelong anticoagulation.

ASSESSMENT OF PULMONARY RISK

Pulmonologic-associated procedural-based risk may be specific to the patient, the procedure, or both. Approximately 25% of morbidity in the early postoperative period is pulmonary related, including atelectasis, pneumonia, respiratory failure, and exacerbation of underlying chronic lung disease (Fisher et al. 2002). Major abdominal surgery induces a 20% to 30% overall risk of pulmonary complications (Ferguson 1999). Vital capacity is reduced by 45% and functional residual capacity is reduced by 20% with laparotomy (Qaseem et al. 2006). The supine position results in a reduction of functional residual capacity below alveolar closing volume, significantly increasing the postoperative risk of atelectasis. Several additional intraoperative factors increase the risk of perioperative pulmonary complications (Table 2.4). Procedural-based pulmonary risk factors include duration of surgery, choice of anesthetic, the emergent nature of the procedure, and incision location. Risk factors specific to the patient include increasing age, chronic lung disease, cigarette use, functional status, obesity, congestive heart failure, asthma, obstructive sleep apnea, poor mental status, alcohol use, and neurologic impairment (Doyle 1999, Smetana et al. 2006).

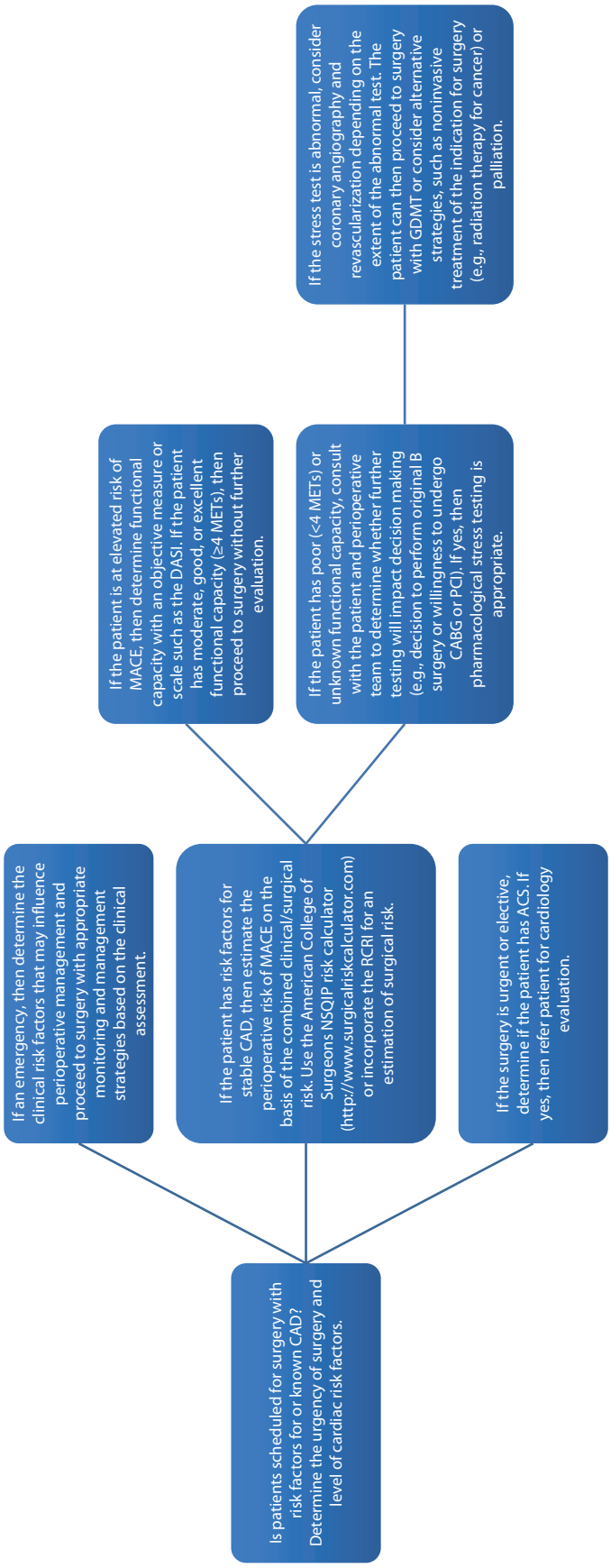

Figure 2.2 Risk classification system. ACS indicates acute coronary syndrome; CABG, coronary artery bypass graft; CAD, coronary artery disease; CPG, clinical practice guideline; DASI, Duke Activity Status Index; GDMT, guideline-directed medical therapy; MACE, major adverse cardiac event; MET, metabolic equivalent; NSQIP, National Surgical Quality Improvement Program; PCI, percutaneous coronary intervention; RCRI, Revised Cardiac Risk Index.

Table 2.4 Intraoperative Factors that Increase the Risk of Perioperative Pulmonary Complications

- Length of surgery
- Amount of IV crystalloids
- Amount of colloid
- Hyperthermia
- Advanced age
- Low preoperative oxygen saturation
- Respiratory infection within the past month
- Preoperative anemia
- Upper abdominal or thoracic surgery
- Emergency surgery

Congestive obstructive pulmonary disease (COPD) remains the most common risk factor for postoperative pulmonary complications. Patients with COPD retain carbon dioxide, have poor gas exchange, and have an increased residual volume. Smoking increases the risk of postoperative complications even in the absence of chronic lung disease. Perioperative pulmonary risk is particularly increased in those who have been smoking more than 20 years, and is highest in patients still smoking within 2 months of surgery (Moller et al. 2002).

Obstructive sleep apnea increases risk for airway management difficulties in the immediate perioperative period; however, with the epidemic of obesity, almost all patients are at risk for some complication.

Patients with a history of asthma or other restrictive lung diseases are at a minimal risk for postoperative complications (Smetana et al. 2006).

There is no predictive value in obtaining a chest x-ray in a well, normal adult and it should not be included in the preoperative evaluation. Alternatively, patients at increased risk for perioperative pulmonary complications, including those older than 50 years of age and those with diagnosed lung disease, may benefit from a baseline chest x-ray. Pulmonary function testing (PFT) may be used to assess the extent of disease and predict the risk of postoperative complications. However, few clinical trials actually support PFT, with the exception of restrictive lung disease (Qaseem et al. 2006). Patients with longstanding restrictive lung disease are at a significantly elevated risk for pulmonary hypertension. Preoperative functional status and coordination with the patient's pulmonologist can be helpful for perioperative pulmonology care. Spirometry may be helpful in diagnosing obstructive lung disease; however, it has not been proven to be predictive of postoperative pulmonary complications. In the setting of unacceptably poor preoperative PFTs, the urgent nature of a procedure should be considered. Management may be determined in concert with the anesthesiologist, and may require cancellation or pulmonary rehabilitation. Preoperative arterial blood gases are not considered an acceptable routine test; however, when indicated, an elevated $PaCO_2$ above 45 mmHg has been proven to increase perioperative complications, while surgery is contraindicated in patients with hypoxemia (PO2 < 50 mmHg). A low serum albumin (<35g/L) is an additional marker for increased risk of postoperative pulmonary complications, particularly in patients with more than one risk factor (Gibbs et al. 1999).

Risk reduction strategies in the postoperative period include pulmonary expansion by means of incentive spirometry, chest wall expansion, deep breathing, and cough, none of which has been proven to be superior to the others. Increased use of bronchodilators and steroids, exacerbations, and smoking are risk factors for perioperative bronchospasm. Prophylaxis in reactive airway disease is with perioperative inhaled beta agonists by inhaler or nebulizer therapy. Steroid therapy should be reserved for those patients already using them as a part of their current regimen, which may decrease inflammation preoperatively and minimize bronchospasm postoperatively. Prophylactic antibiotics have no place in perioperative therapy to prevent pulmonary complications. Patients on oral steroids for prolonged periods of time should receive preoperative stress dose steroids (see below; "Adrenal Suppression"). Preoperative consultation with an anesthesiologist may be helpful in this patient population for planning medication use, optimization of therapy, and communication.

ASSESSMENT OF ENDOCRINOLOGIC RISK

Diabetes Mellitus

Perioperative hyperglycemia has been found to increase the risk of adverse events in patients undergoing elective noncardiac surgery (Frisch et al. 2010).

Postoperative glucose levels greater than 200 mg/dL are associated with prolonged hospital stays and increased risk of postoperative complications including wound infections and cardiac arrhythmias (Ramos et al. 2008).

Postoperative infections make up two-thirds of postoperative complications in diabetics with vascular disease, which additionally increase the risk of postoperative MI and acute renal failure (Dronge et al. 2006).

Diabetes-associated perioperative risk can be determined by evaluating the extent of the disease. Microvascular changes induce long-term complications and end-organ damage, including retinopathy, neuropathy, nephropathy, and cardiovascular disease (Meneghini 2009). The presence of disease for 10 years or more even further increases the risk of microvascular complications (Schiff and Welsh 2003). Preoperative assessment includes a thorough history and physical, an ECG, and serologies evaluating renal function with a glycosylated hemoglobin (HBA1C) level. Understanding the extent of neuropathy prior to the administration of chemotherapy provides a baseline for post-therapy assessments. Preoperative medical management in a diabetic may include holding medications the night prior to surgery (such as metformin and thiazolinediones for risk of lactic acidosis and postoperative fluid retention) or the morning of surgery (most oral antihyperglycemics). Insulin-requiring diabetics should continue regular short-acting insulin the night prior and halve the a.m. dose. Long-acting basal insulin should be continued at full dose unless additionally taking oral antihyperglycemics, when the basal insulin dose should be cut in half (Meneghini 2009). A diabetic controlled by diet alone does not require additional antihyperglycemic therapy preoperatively or intraoperatively.

Operative physiologic stress induces a hyperglycemic state in the diabetic patient. This is caused by an adrenal stress response releasing epinephrine, norepinephrine, cortisol, and growth hormone, all of which suppress insulin function. Gluconeogenesis and lipolysis support the stress response by mobilizing glucose precursors, inducing a net protein catabolism. Intraoperative glucose assessment in procedures lasting longer than 2 hours monitors for signs of ketosis or acidosis resulting from this hyperglycemic stress response (Hoogwerf 2006).

Diabetic patients have an increased risk for postoperative cardiac complications including ischemia and infarction and acute renal failure. Large fluid shifts, peritoneal evaporative loss, anesthetic agents, and gastrointestinal and respiratory losses result in decreased intravascular volume, which may impact postoperative renal function, particularly in the diabetic. Large amounts of crystalloids should be avoided in all perioperative patients but especially in diabetics with reduced renal function. Wound complications and postoperative infections are driven by the hyperglycemic impairment of phagocytes, granulocytes, and collagen synthesis at glucose levels >200 mg/dL. This impairment and microvasculopathy place the uncontrolled diabetic patient at a significantly elevated risk for wound and fascial dehiscence. The microvascular changes of diabetes impair oxygen delivery to tissues, compounding the already poor ability to ward off infection within the wound. Several retrospective studies have found that tighter glycemic control lowers incidence of postoperative wound complications, including reduced infectious morbidity (Marks 2003).

Postoperative glycemic control has undergone recent modification. A large randomized study by Van den Berghe et al. (2001) found that aggressive insulin therapy (glucose levels between 80 and 110 mg/dL) reduced episodes of septicemia and in-hospital mortality over standard insulin therapy (glucose levels between 180 and 200 mg/dL). In contrast, the NICE-SUGAR trial (Normoglycemia in Intensive Care Evaluation and Survival Using Glucose Algorithm Regulation) found an increased risk of mortality (27.5% vs. 24.9%) with intensive control, with the majority due to an increased risk of hypoglycemia (Finfer et al. 2009). Because of these results, management of glycemic control will depend upon the postoperative status, the type and management of the patient's diabetes, and oral intake status to maintain blood glucose levels below 180 mg/dL in the critically ill and 140 mg/dL in the non-critically ill (Moghissi et al. 2009). Converting total insulin requirements to long-acting insulin (50%–80% of total requirements) will more frequently achieve the glycemic goal of <140 mg/dL with lower risk of postoperative infections (Umpierrez et al. 2011).

Thyroid Dysfunction

Thyroid dysfunction increases the risk of perioperative complications associated with cardiac, vascular, metabolic, and central nervous systems. Thyroid-stimulating hormone and thyroxine (T4) levels should be obtained preoperatively in patients with a diagnosis of thyroid dysfunction or those with a history of fatigue and new-onset depression. Avoidance of rare but serious complications (myxedema coma and thyroid storm) can be accomplished by appropriate preoperative assessment.

Retrospective studies have demonstrated that euthyroid to mild or even moderate hypothyroidism may safely undergo surgery (Weinberg et al. 1983).

Perioperative risks associated with hypothyroidism include intraoperative hypotension, gastrointestinal complications including ileus, postoperative neuropsychiatric complications, and inability to mount fever. Patients with severe hypothyroidism (myxedema coma, decreased mentation, pericardial effusions, heart failure, or very low levels of T4) who are in need of an urgent/emergent procedure should receive intravenous T4 and stress dose glucocorticoids (Ladenson et al. 1984). Signs of myxedema coma, a medical emergency, include seizures, coma, unexplained heart failure, hypothermia, prolonged ileus, or postoperative delirium (Stathatos and Wartofsky 2003).

Hyperthyroidism poses perioperative cardiac risk due to the ability of both T4 and triiodothyronine (T3) to impose inotropic and chronotropic effects on cardiac function. The most common cause of hyperthyroidism is Graves' disease, an autoimmune disorder resulting in increased thyroid hormone production. Hyperthyroidism is characterized by tachycardia, atrial fibrillation, fever, tremor, goiter, and ophthalmopathy. The greatest perioperative risk to an untreated hyperthyroid patient is the development of thyroid storm and should be considered in any patient suffering postoperative fever, tachycardia, hyperpyrexia, nausea and vomiting, or delirium. Treatment includes beta-blockade, thionamides, iodine, and corticosteroids in addition to admittance to an intensive care unit for appropriate monitoring. Until control is achieved, moderate to severe hyperthyroidism necessitates surgery cancellation.

Patients with mild disease may proceed with surgery with the support of perioperative beta-blockade.

Moderate to severe (thyrotoxic) patients should have surgery delayed unless the procedure is emergent or urgent. Premedication for these patients includes antithyroid agents, beta-blockade, and corticosteroids.

Adrenal Suppression

Exogenous corticosteroid use over a prolonged period of time poses a potential risk for hypothalamic pituitary axis (HPA) suppression. In preoperative evaluation, the surgeon must determine the type of steroid used, the duration of treatment, and whether a taper was used if the medication was discontinued. Doses seldom resulting in HPA suppression and not requiring stress-dose corticosteroids include steroid equivalents to 5 mg of prednisone as a single daily dose, alternate-day steroids given as a morning dose, and any steroid used for less than 3 weeks. Alternatively, patients taking 20-mg equivalents of prednisone daily for more than 3 weeks require stress-dose steroids in the perioperative period (Salem et al. 1994). Theoretically, the steroid doses typically used every 3 weeks for prevention of hypersensitivity reactions could be associated with adrenal insufficiency (Del Priore et al. 1995). Preoperative stress-dose steroids are used for the prevention of HPA suppression and its life-threatening sequelae. Administering stress-dose glucocorticosteroids must be weighed against the potential side effects of

the medication including poor wound healing, fluid retention, and increased risk for infection.

ASSESSMENT OF RENAL RISK

The prevalence of renal disease in surgical patients continues to rise alongside the incidence of diabetes and hypertension. Advancements in dialysis are allowing many patients to live with end stage renal disease (ESRD). These patients are subject to increased risks of perioperative morbidity and mortality. Patients with ESRD commonly suffer from coronary artery disease and peripheral vascular disease. Half of patients with ESRD die of cardiovascular disease (Go et al. 2004). Contributing factors include microalbuminuria/proteinuria, hypertension, diabetes, dyslipidemia, and smoking (Weir 2011). Preoperative evaluation of ESRD patients includes a cardiac evaluation, electrolyte and fluid management, assessing for anemia or bleeding diatheses, and optimizing glycemic control. Postoperatively, these patients tend to have difficulty with fluid balance, anemia, electrolyte, acid-base abnormalities, and postoperative wound complications secondary to an immunocompromised state. Engaged surgeons can help ensure euvolemia, periprocedural electrolyte replacement, and postoperative fluid shift management. Goal-directed fluids, with as little crystalloid as needed, will help in maintaining euvolemia.

Serum creatinine levels are a poor indicator of renal function.

Day-to-day variations in creatinine more likely reflect acute changes in volume of distribution. All patients have age-related reduction in renal function. All chemotherapy patients have some degree of renal impairment despite normal creatinine.

Erythropoetin is commonly administered to ESRD patients to maintain hemoglobin levels chronically (Eschbach et al. 1989). Within the immediate perioperative period, transfusion may be required to achieve acceptable preoperative hemoglobin levels. ESRD patients frequently suffer increased risk of bleeding secondary to platelet dysfunction due to uremic inhibition, abnormal von Willebrand factor binding, abnormal platelet arachidonic acid metabolism, excess vascular prostacyclin, and nitric acid production. 1-deamino-8-D-arginine vasopressin (dDAVP) intravenously can be used to treat uremic platelet dysfunction or may be administered intranasally with cryoprecipitate to prevent intraoperative bleeding (Rabelink et al. 1994).

ASSESSMENT OF HEPATIC RISK

The most common cause of chronic liver disease in the United States is nonalcoholic fatty liver disease. Routine testing of liver function rarely yields an abnormality or changes perioperative management in the routine surgical patient. However, liver disorders can impact perioperative risk enough to significantly confer unnecessary morbidity and mortality. Decompensated liver disease increases the perioperative risk of acute hepatic failure, sepsis, bleeding, and renal dysfunction. A patient presenting with a history of jaundice, blood transfusions, alcohol or recreational drug use, acute hepatitis, or physical findings of icterus, hepatosplenomegaly, palmar erythema, or spider nevi should be tested to rule out occult or active liver disease (Hoetzel et al. 2012).

The extent of liver dysfunction and type of surgery play key roles in determining perioperative risk. The Child-Turcott-Pugh (CTP) classification and the model of end-stage liver disease (MELD) assist in determining overall surgical risk by assessing the severity of underlying liver disease. The CTP score was found to correlate with overall mortality depending on the procedure (Hoetzel et al. 2012). Liver disease easily affects many other organ systems in the body including the cardiorespiratory and circulatory systems, the brain, kidneys, and the immune system. Patients with chronic hepatitis without cirrhosis have very minimal perioperative morbidity; however, those patients with acute hepatitis have an associated mortality rate of up to 50% and should not undergo non-emergent or urgent procedures until resolution of the acute phase. Cirrhotic disease significantly increases perioperative surgical risk. Cirrhotic patients additionally suffer coagulopathies and frequently require administration of vitamin K, fresh frozen plasma, or cryoprecipitate prior to surgery.

REFERENCES

Agnelli G, Piovella F, Buoncristiani P, et al. 1998. Enoxaparin plus compression stockings compared with compression stockings alone in the prevention of venous thromboembolism after elective neurosurgery. *N Engl J Med* **339**:80–5.

Andersson C, Merie C, Jorgensen M, et al. 2014. Association of beta-blocker therapy with risks of adverse cardiovascular events and deaths in patients with ischemic heart disease undergoing noncardiac surgery: A Danish nationwide cohort study. *JAMA Intern Med* **174**:336–44.

Ashton CM, Petersen NJ, Wray NP, et al. 1993. The incidence of perioperative myocardial infarction in men undergoing noncardiac surgery. *Ann Intern Med* **118**:504–10.

Beattie WS, Abdelnaem E, Wijeysundera DN, et al. 2006. A meta-analytic comparison of preoperative stress echocardiography and nuclear scintigraphy imaging. *Anesth Analg* **102**:8–16.

Berger PB, Kleiman NS, Pencina MJ, et al. 2010. Frequency of major noncardiac surgery and subsequent adverse events in the year after drug-eluting stent placement results from the EVENT (Evaluation of Drug-Eluting Stents and Ischemic Events) Registry. *JACC Cardiovasc Interv* **3**:920–7.

Bergqvist D, Agnelli G, Cohen AT, et al. 2002. Duration of prophylaxis against venous thromboembolism with enoxaparin after surgery for cancer. *N Engl J Med* **346**:975–80.

Bertrand M, Godet G, Meersschaert K, et al. 2001. Should the angiotensin II antagonists be discontinued before surgery? *Anesth Analg* **92**:26–30.

Boersma E, Poldermans D, Bax JJ, et al. 2001. Predictors of cardiac events after major vascular surgery: Role of clinical characteristics, dobutamine echocardiography, and beta-blocker therapy. *JAMA* **285**:1865–73.

Caprini JA. 2005. Thrombosis risk assessment as a guide to quality patient care. *Dis Mon* **51**:70–8.

Carliner NH, Fisher ML, Plotnick GD, et al. 1985. Routine preoperative exercise testing in patients undergoing major noncardiac surgery. *Am J Cardiol* **56**:51–8.

Clarke-Pearson DL, Delong ER, Synan, IS, et al. 1984a. Complications of low-dose heparin prophylaxis in gynecologic oncology surgery. *Obstet Gynecol* **64**:689–94.

Clarke-Pearson DL, Dodge RK, Synan I, et al. 2003. Venous thromboembolism prophylaxis: Patients at high risk to fail intermittent pneumatic compression. *Obstet Gynecol* **101**:157–63.

Clarke-Pearson DL, Synan IS, Colemen, RE, et al. 1984b. The natural history of postoperative venous thromboemboli in gynecologic oncology: Aprospective study of 382 patients. *Am J Obstet Gynecol* **148**:1051–4.

Clarke-Pearson DL, Synan IS, Hinshaw, WM, et al. 1984c. Prevention of postoperative venous thromboembolism by external pneumatic calf compression in patients with gynecologic malignancy. *Obstet Gynecol* **63**:92–8.

Cohen ME, Ko CY, Bilimoria KY, et al. 2013. Optimizing ACS NSQIP modeling for evaluation of surgical quality and risk: Patient risk adjustment, procedure mix adjustment, shrinkage adjustment, and surgical focus. *J Am Coll Surg* **217**:336–46.e1.

Comfere T, Sprung J, Kumar MM, et al. 2005. Angiotensin system inhibitors in a general surgical population. *Anesth Analg* **100**:636–44

Coriat P, Richer C, Douraki T, et al. 1994. Influence of chronic angiotensin-converting enzyme inhibition on anesthetic induction. *Anesthesiology* **81**:299–307.

Dahlback B, Carlsson M, Svensson PJ. 1993. Familial thrombophilia due to a previously unrecognized mechanism characterized by poor anticoagulant response to activated protein C: Prediction of a cofactor to activated protein C. *Proc Natl Acad Sci U S A* **90**:1004–8.

Dainty L, Maxwell GL, Clarke-Pearson DL, et al. 2004. Cost-effectiveness of combination thromboembolism prophylaxis in gynecologic oncology surgery. *Gynecol Oncol* **93**:366–73.

Das MK, Pellikka PA, Mahoney DW, et al. 2000. Assessment of cardiac risk before nonvascular surgery: Dobutamine stress echocardiography in 530 patients. *J Am Coll Cardiol* **35**:1647–53.

De Groot PG, Derksen RH. 2005. The antiphospholipid syndrome: Clinical characteristics, laboratory features and pathogenesis. *Curr Opin Infect Dis* **18**:205–10.

Del Priore G, Gurski KJ, Warshal DP, et al. 1995. Adrenal function following high-dose steroids in ovarian cancer patients. *Gynecol Oncol* **59**(1):102–4.

Desai H, Aronow WS, Ahn C, et al. 2010. Incidence of perioperative myocardial infarction and of 2-year mortality in 577 elderly patients undergoing noncardiac vascular surgery treated with and without statins. *Arch Gerontol Geriatr* **51**:149–51.

Detsky AS, Abrams HB, Mclaughlin JR, et al. 1986. Predicting cardiac complications in patients undergoing noncardiac surgery. *J Gen Intern Med* **1**:211–9.

Devereaux PJ, Yang H, Guyatt GH, et al. 2006. Rationale, design, and organization of the PeriOperative ISchemic Evaluation (POISE) trial: A randomized controlled trial of metoprolol versus placebo in patients undergoing noncardiac surgery. *Am Heart J* **152**:223–30.

Douglas PS, Garcia MJ, Haines DE, et al. 2011. ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR 2011 Appropriate Use Criteria for Echocardiography. *J Am Coll Cardiol* **57**:1126–66.

Douketis JD, Spyropoulos AC, Spencer FA, et al. 2012. Perioperative management of antithrombotic therapy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest* **141**:e326S–50S.

Doyle RL. 1999. Assessing and modifying the risk of postoperative pulmonary complications. *Chest* **115**:77s–81s.

Dronge AS, Perkal MF, Kancir S, et al. 2006. Long-term glycemic control and postoperative infectious complications. *Arch Surg* **141**:375–80; discussion 380.

Eagle KA, Berger PB, Calkins H, et al. 2002. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *Anesth Analg* **94**:1052–64.

Ellis JE, Drijver G, Pedlow S, et al. 1994. Premedication with oral and transdermal clonidine provides safe and efficacious postoperative sympatholysis. *Anesth Analg* **79**:1133–40.

Eschbach JW, Kelly MR, Haley NR, et al. 1989. Treatment of the anemia of progressive renal failure with recombinant human erythropoietin. *N Engl J Med* **321**:158–63.

Ferguson MK. 1999. Preoperative assessment of pulmonary risk. *Chest* **115**:58s–63s.

Finfer S, Chittock DR, Su SY, et al. 2009. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* **360**:1283–97.

Fisher BW, Majumdar SR, McAlister FA, et al. 2002. Predicting pulmonary complications after nonthoracic surgery: A systematic review of blinded studies. *Am J Med* **112**:219–25.

Fleisher LA, Beckman JA, Brown KA, et al. 2008. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: Executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *Anesth Analg* **106**:685–712.

Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2015. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: Executive summary: A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *J Nucl Cardiol* **22**:162–215.

Fletcher GF, Balady G, Froelicher VF, et al. 1995. Exercise standards: A statement for healthcare professionals from the American Heart Association. *Circulation* **91**:580–615.

Freeman WK, Gibbons RJ. 2009. Perioperative cardiovascular assessment of patients undergoing noncardiac surgery. *Mayo Clin Proc* **84**(1):79–90.

Frisch A, Chandra P, Smiley D, et al. 2010. Prevalence and clinical outcome of hyperglycemia in the perioperative period in noncardiac surgery. *Diabetes Care* **33**:1783–8.

Geerts WH, Bergqvist D, Pineo GF, et al. 2008. Prevention of venous thromboembolism: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* **133**:381s–453s.

Gibbs J, Cull W, Henderson W, et al. 1999. Preoperative serum albumin level as a predictor of operative mortality and morbidity: Results from the National VA Surgical Risk Study. *Arch Surg* **134**:36–42.

Go AS, Chertow GM, Fan D, et al. 2004. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med* **351**:1296–305.

Goldman L, Caldera DL, Nussbaum SR, et al. 1977. Multifactorial index of cardiac risk in noncardiac surgical procedures. *N Engl J Med* **297**:845–50.

Gould MK, Garcia DA, Wren SM, et al. 2012. Prevention of VTE in nonorthopedic surgical patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest* **141**:e227S–77S.

Gupta PK, Gupta H, Sundaram A, et al. 2011. Development and validation of a risk calculator for prediction of cardiac risk after surgery. *Circulation* **124**:381–7.

Hammill BG, Curtis LH, Bennett-Guerrero E, et al. 2008. Impact of heart failure on patients undergoing major noncardiac surgery. *Anesthesiology* **108**:559–67.

Healy KO, Waksmonski CA, Altman RK, et al. 2010. Perioperative outcome and long-term mortality for heart failure patients undergoing intermediate- and high-risk noncardiac surgery: Impact of left ventricular ejection fraction. *Congest Heart Fail* **16**:45–9.

Hillis LD, Smith PK, Anderson JL, et al. 2012. 2011 ACCF/AHA guideline for coronary artery bypass graft surgery: Executive summary: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Thorac Cardiovasc Surg* **143**:4–34.

Hindler K, Shaw AD, Samuels J, et al. 2006. Improved postoperative outcomes associated with preoperative statin therapy. *Anesthesiology* 105:1260–72; quiz 1289–90.

Hlatky MA, Boineau RE, Higginbotham MB, et al. 1989. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index). *Am J Cardiol* **64**:651–4.

Hoetzel A, Ryan H, Schmidt R. 2012. Anesthetic considerations for the patient with liver disease. *Curr Opin Anaesthesiol* **25**:340–7.

Hoogwerf BJ. 2006. Perioperative management of diabetes mellitus: How should we act on the limited evidence? *Cleve Clin J Med* **73**:Suppl 1, S95–9.

Juul AB, Wetterslev J, Gluud C, et al. 2006. Effect of perioperative beta blockade in patients with diabetes undergoing major noncardiac surgery: Randomised placebo controlled, blinded multicentre trial. *BMJ* **332**:1482.

Kennedy J, Quan H, Buchan AM, et al. 2005. Statins are associated with better outcomes after carotid endarterectomy in symptomatic patients. *Stroke* **36**:2072–6.

Ladenson PW, Levin AA, Ridgway EC, et al. 1984. Complications of surgery in hypothyroid patients. *Am J Med* **77**:261–6.

Le Manach Y, Godet G, Coriat P, et al. 2007. The impact of postoperative discontinuation or continuation of chronic statin therapy on cardiac outcome after major vascular surgery. *Anesth Analg* **104**:1326–33.

Lee TH, Marcantonio ER, Mangione CM, et al. 1999. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation* **100**:1043–9.

Levine GN, Bates ER, Blankenship JC, et al. 2011. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. *J Am Coll Cardiol* **58**:e44–122.

Lindenauer PK, Pekow P, Wang K, et al. 2004. Lipid-lowering therapy and in-hospital mortality following major noncardiac surgery. *JAMA* **291**:2092–9.

Linsenmaier U, Rieger J, Schenk F, et al. 1998. Indications, management, and complications of temporary inferior vena cava filters. *Cardiovasc Intervent Radiol* **21**:464–9.

Liu LL, Dzankic S, Leung JM. 2002. Preoperative electrocardiogram abnormalities do not predict postoperative cardiac complications in geriatric surgical patients. *J Am Geriatr Soc* **50**:1186–91.

Livhits M, Ko CY, Leonardi MJ, et al. 2011. Risk of surgery following recent myocardial infarction. *Ann Surg* **253**:857–64.

Mangano DT. 1990. Perioperative cardiac morbidity. *Anesthesiology* **72**:153–84.

Marks JB. 2003. Perioperative management of diabetes. *Am Fam Physician* **67**:93–100.

Martino MA, Borges E, Williamson E, et al. 2006. Pulmonary embolism after major abdominal surgery in gynecologic oncology. *Obstet Gynecol* **107**:666–71.

Mashour GA, Shanks AM, Kheterpal S. 2011. Perioperative stroke and associated mortality after noncardiac, nonneurologic surgery. *Anesthesiology* **114**:1289–96.

McFalls EO, Ward HB, Moritz TE, et al. 2004. Coronary-artery revascularization before elective major vascular surgery. *N Engl J Med* **351**:2795–804.

Meneghini LF. 2009. Perioperative management of diabetes: Translating evidence into practice. *Cleve Clin J Med* **76** :Suppl 4, S53–9.

Middeldorp S, Henkens CM, Koopman MM, et al. 1998. The incidence of venous thromboembolism in family members of patients with factor V Leiden mutation and venous thrombosis. *Ann Intern Med* **128**:15–20.

Moghissi ES, Korytkowski MT, Dinardo M, et al. 2009. American Association of Clinical Endocrinologists and American Diabetes Association consensus statement on inpatient glycemic control. *Endocr Pract* **15**:353–69.

Moller AM, Villebro N, Pedersen T, et al. 2002. Effect of preoperative smoking intervention on postoperative complications: A randomised clinical trial. *Lancet* **359**:114–7.

Nishimura RA, Otto CM, Bonow RO, et al. 2014. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: Executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* **63**:2438–88.

Nuttall GA, Brown MJ, Stombaugh JW, et al. 2008. Time and cardiac risk of surgery after bare-metal stent percutaneous coronary intervention. *Anesthesiology* **109**:588–95.

[No authors listed]. Practice Bulletin No. 84: Prevention of deep vein thrombosis and pulmonary embolism: Correction. *Obstet Gynecol.* 2016 Jan;127(1):166. Erratum for ACOG Practice Bulletin No. 84: Prevention of deep vein thrombosis and pulmonary embolism. [*Obstet Gynecol.* 2007]

Oliver MF, Goldman L, Julian DG, et al. 1999. Effect of mivazerol on perioperative cardiac complications during noncardiac surgery in patients with coronary heart disease: The European Mivazerol Trial (EMIT). *Anesthesiology* **91**:951–61.

Poort SR, Rosendaal FR, Reitsma PH, et al. 1996. A common genetic variation in the 3'-untranslated region of the prothrombin gene is associated with elevated plasma prothrombin levels and an increase in venous thrombosis. *Blood* **88**:3698–703.

Qaseem A, Snow V, Fitterman N, et al. 2006. Risk assessment for and strategies to reduce perioperative pulmonary complications for patients undergoing noncardiothoracic surgery: A guideline from the American College of Physicians. *Ann Intern Med* **144**:575–80.

Rabelink TJ, Zwaginga JJ, Koomans HA, et al. 1994. Thrombosis and hemostasis in renal disease. *Kidney Int* **46**:287–96.

Raju MG, Pachika A, Punnam SR, et al. 2013. Statin therapy in the reduction of cardiovascular events in patients undergoing intermediate-risk noncardiac, nonvascular surgery. *Clin Cardiol* **36**:456–61.

Ramos M, Khalpey Z, Lipsitz S, et al. 2008. Relationship of perioperative hyperglycemia and postoperative infections in patients who undergo general and vascular surgery. *Ann Surg* **248**:585–91.

Reilly DF, Mcneely MJ, Doerner D, et al. 1999. Self-reported exercise tolerance and the risk of serious perioperative complications. *Arch Intern Med* **159**:2185–92.

Rosendaal FR. 2005. Venous thrombosis: The role of genes, environment, and behavior. *Hematology Am Soc Hematol Educ Program* 1–12.

Rosenman DJ, Mcdonald F S, Ebbert JO, et al. 2008. Clinical consequences of withholding versus administering renin-angiotensin-aldosterone system antagonists in the preoperative period. *J Hosp Med* **3**:319–25.

Salem M, Tainsh RE, Jr, Bromberg J, et al. 1994. Perioperative glucocorticoid coverage. A reassessment 42 years after emergence of a problem. *Ann Surg* **219**:416–25.

Schein OD, Katz J, Bass EB, et al. 2000. The value of routine preoperative medical testing before cataract surgery. Study of Medical Testing for Cataract Surgery. *N Engl J Med* **342**:168–75.

Schiff RL, Welsh GA. 2003. Perioperative evaluation and management of the patient with endocrine dysfunction. *Med Clin North Am* **87**:175–92.

Shah KB, Kleinman BS, Sami H, et al. 1990. Reevaluation of perioperative myocardial infarction in patients with prior myocardial infarction undergoing noncardiac operations. *Anesth Analg* **71**:231–5.

Shammash JB, Trost JC, Gold JM, et al. 2001. Perioperative beta-blocker withdrawal and mortality in vascular surgical patients. *Am Heart J* **141**:148–53.

Smetana GW, Lawrence VA, Cornell JE. 2006. Preoperative pulmonary risk stratification for noncardiothoracic surgery: Systematic review for the American College of Physicians. *Ann Intern Med* **144**:581–95.

Snowden CP, Prentis J, Jacques B, et al. 2013. Cardiorespiratory fitness predicts mortality and hospital length of stay after major elective surgery in older people. *Ann Surg* **257**:999–1004.

Stathatos N, Wartofsky L. 2003. Perioperative management of patients with hypothyroidism. *Endocrinol Metab Clin North Am* **32**:503–18.

Stuhmeier KD, Mainzer B, Cierpka J, et al. 1996. Small, oral dose of clonidine reduces the incidence of intraoperative myocardial ischemia in patients having vascular surgery. *Anesthesiology* **85**:706–12.

Thomson IR, Mutch WA, Culligan JD. 1984. Failure of intravenous nitroglycerin to prevent intraoperative myocardial ischemia during fentanyl-pancuronium anesthesia. *Anesthesiology* **61**:385–93.

Turan A, You J, Shiba A, et al. 2012. Angiotensin converting enzyme inhibitors are not associated with respiratory complications or mortality after noncardiac surgery. *Anesth Analg* **114**:552–60.

Turnbull JM, Buck C. 1987. The value of preoperative screening investigations in otherwise healthy individuals. *Arch Intern Med* 147:1101–5.

Umpierrez GE, Smiley D, Jacobs S, et al. 2011. Randomized study of basal-bolus insulin therapy in the inpatient management of patients with type 2 diabetes undergoing general surgery (RABBIT 2 surgery). *Diabetes Care* **34**:256–61.

Van den Berghe G, Wouters P, Weekers F, et al. 2001. Intensive insulin therapy in critically ill patients. *N Engl J Med* **345**:1359–67.

Van Kuijk JP, Flu WJ, Schouten O, et al. 2009. Timing of noncardiac surgery after coronary artery stenting with bare metal or drug-eluting stents. *Am J Cardiol* **104**:1229–34.

Weinberg AD, Brennan MD, Gorman CA, et al. 1983. Outcome of anesthesia and surgery in hypothyroid patients. *Arch Intern Med* **143**:893–7.

Weir MR. 2011. Recognizing the link between chronic kidney disease and cardiovascular disease. *Am J Manag Care* **17**:Suppl 15, S396–402.

Wijeysundera DN, Beattie WS. 2003. Calcium channel blockers for reducing cardiac morbidity after noncardiac surgery: A meta-analysis. *Anesth Analg* **97**:634–41.

Yang H, Raymer K, Butler R, et al. 2006. The effects of perioperative beta-blockade: Results of the Metoprolol after Vascular Surgery (MaVS) study, a randomized controlled trial. *Am Heart J* **152**:983–90.

3 Complications

David Warshal and James Aikins

INTRODUCTION

Complications are a frequent consequence of surgery. A clear understanding of surgical principles and meticulous technique are essential but are not always sufficient to prevent complications, particularly when normal anatomical relationships have been altered by the presence of a malignancy. Furthermore, some complications are beyond the control of the surgeon. The judicious surgeon must always be cognizant of the potential complications associated with each step of a particular surgical procedure and actively work to minimize these risks. The prompt detection and management of perioperative complications is of paramount importance in order to minimize adverse sequelae.

For this chapter, we have chosen to address what we believe are the most relevant issues in regard to complications associated with gynecologic surgery. Urinary tract complications have not been included in this section since they are discussed in Chapter 30.

BOWEL COMPLICATIONS

Preoperative bowel preparation was considered an essential component in preventing complications associated with colorectal surgery for over a century. However, over the past several years, a series of studies have challenged this belief. A 2011 Cochrane review examining this issue concluded, based on over 5800 subjects participating in 18 trials, that there was no benefit conferred by preoperative bowel preparation. Mechanical bowel preparation versus rectal enema was also examined with no differences detected. In fact, a trend toward increased postoperative infectious complications with bowel preparation was discovered. It has been suggested that this association may be due to leakage of liquid stool from inadequately prepped bowel or from local structural and inflammatory changes of the bowel wall that can result from a mechanical bowel prep. If bulky stool is encountered intraoperatively, it should be gently milked away from the area of resection or washed out from the anus to facilitate reanastomosis. Intravenous antibiotics with both aerobic and anaerobic coverage, such as a second-generation cephalosporin with metronidazole or amoxicillin/clavulanic acid, should be administered preoperatively. Ciprofloxicin or clindamycin may be substituted for the cephalosporin in penicillin-allergic cases. Preoperative use of oral antibiotics has been suggested by multiple studies to reduce the risk of surgical site infection following colectomy.

Historically, injury to the colon, particularly with gross contamination of the peritoneal cavity, was managed by colostomy formation. Recent prospective randomized studies examining the management of traumatic colon injuries have demonstrated either equal or improved outcomes with primary repair rather than colostomy. Though the risk for intra-abdominal sepsis is increased with multiple associated abdominal injuries, massive blood transfusion, and severe peritoneal contamination, the method of management of the colon injury does not affect the incidence of sepsis. In addition, the repair technique, handsewn versus stapled, also does not influence the complication rate. In the face of a colon injury with peritoneal contamination, broad-spectrum antibiotic prophylaxis should be continued for 24 hours.

Intraoperative bowel injuries are most likely to occur during entry into the abdominal cavity and during lysis of adhesions. If entering the abdomen through an old scar, the risk of injury is reduced if entry is gained just beyond the limit of the old scar. Sharp entry is preferred over use of an electrocoagulation device due to the clean, defined nature of a sharp injury. Thermal injuries are more difficult to detect and evaluate due to the potential for delayed tissue necrosis up to a few centimeters beyond the point of visible damage. When a significant thermal injury to the bowel occurs, a wide resection up to 3 to 5 cm from the edges of the injury with primary reanastomosis is recommended. Thin filmy intra-abdominal adhesions can be safely lysed using blunt dissection and the electrocautery devise. Thicker, less yielding adhesions require sharp dissection to avoid injury to the bowel.

Following difficult bowel dissections, direct visual inspection of all bowel surfaces is important. Of note, the risk of compromise of the distal sigmoid colon is increased in cases of ovarian cancer with extensive pelvic disease and with endometriosis where the cul-de-sac may be obliterated. Injury in this area may be particularly difficult to visualize. When concern is raised, a large-gauge foley catheter should be inserted into the rectum and the balloon inflated. With the pelvis filled with saline and the proximal sigmoid occluded with gentle pressure, air is injected into the foley to inflate the bowel. Air will bubble to the surface if a laceration is present.

Small bowel lacerations involving less than half of the circumference of the bowel are repaired without resection. A single layer of full thickness delayed absorbable 3-0 sutures are placed 3 mm apart. The closure is oriented perpendicular to the path of the bowel to limit narrowing of the lumen. A second seromuscular layer imbricating the first layer is sometimes placed, provided that it does not compromise the bowel lumen. The closure should be watertight and is tested by gently milking bowel contents and intraluminal gas past the repair site. Pinching the bowel lumen at the anastomotic site should confirm a luminal diameter of at least 1 cm. If a larger laceration occurs, the edges are devascularized, or multiple small enterotomies involve a short segment of bowel, resection of the injured area with primary reanastomosis is warranted.

Repair of large bowel lacerations is similar to that for the small bowel with a few exceptions. Lacerations of up to 30% of the circumference of the bowel are closed primarily with

larger injuries requiring bowel resection. Two-layer closures as described above are the standard. There is generally no concern regarding narrowing of the large bowel lumen by repair.

Routine use of a nasogastric tube following extensive gynecologic procedures or bowel resection has recently been reexamined. Nasogastric tube suctioning does not reduce the duration of ileus and may actually delay return of normal bowel function. Following bowel resection, the presence of a tube did not affect the incidence of anastomotic leakage or incisional hernia development. In addition to the substantial discomfort associated with nasogastric tubes, they are a major risk factor for postoperative pulmonary complications. Two recent meta-analyses suggested that only up to 10% of patients undergoing bowel resection and managed without nasogastric decompression would warrant insertion later in their postoperative course.

Several studies have recently evaluated the feasibility of early feeding of patients who have undergone bowel resection and other types of intraabdominal surgery. Early feeding was found to be safe and not associated with the development of a prolonged ileus or anastomotic leakage. A reduced length of stay and a reduction in the postoperative infection rate have also been reported. Conversely, many of these studies have also shown that those fed early have an increased risk for nausea, vomiting, and abdominal distention.

Following laparotomy, a *postoperative* ileus occurs routinely. Small bowel motility and absorption generally returns within a few hours of surgery followed by stomach emptying which begins after 24 hours. The colon remains inactive for approximately 48 to 72 hours. This process is controlled by the autonomic nervous system. Occasionally a paralytic ileus may develop that can last from days to weeks. A *paralytic* ileus is associated with bowel mucosal injury secondary to bowel manipulation, hypoxia, endotoxins, and/or hypoperfusion, and all bowel segments are affected. Pain and opioid use can prolong both postoperative and paralytic ileuses. Techniques to reduce the risk of ileus include gentle handling of tissue, appropriate intraoperative fluid management, minimizing opioid use, epidural infusion of local anesthetics for pain management, and use of peripherally acting gastrointestinal opioid receptor antagonist. Alvimopan, approved by the FDA for postoperative use, has been found to shorten the time to return of bowel function without compromising pain control in patients undergoing bowel resection and radical hysterectomy. Concern has been raised regarding a potential association between use of COX 2 inhibitors and impaired intestinal healing following bowel resection. Patients with a paralytic ileus develop abdominal bloating, anorexia, nausea, and vomiting if early feeding is initiated. Abdominal cramping and pain in excess of that anticipated by the patient's postoperative state are usually absent. Physical exam reveals a distended, tympanitic abdomen without bowel sounds. Obstruction series imaging will show a nonspecific bowel gas pattern with dilated loops of small and often large bowel. It can often be difficult radiographically to distinguish an ileus from an early obstruction. It is important to rule out infectious and metabolic causes such as peritonitis, abscess, and electrolyte abnormalities such as hypokalemia and hypomagnesemia. Patients are kept nil per orum (NPO) and observed with supportive measures instituted. For patients with persistent nausea and vomiting, a nasogastric tube should be inserted. However, nasogastric tubes have not been shown to shorten the duration of an ileus. If possible, narcotic use should be minimized. No currently available medications have been demonstrated to relieve a postoperative ileus once it is established. Watchful waiting with periodic obstruction series imaging to exclude an obstruction and blood work to exclude infection and metabolic derangement is recommended. For a prolonged ileus lasting more than 1 week, hyperalimentation should be considered. We have anecdotally found that hunger develops shortly before flatus and that diarrhea is common during the first 24 hours following the onset of bowel movements.

Bowel obstructions, characterized as partial or complete, prevent passage of bowel contents through the intestines. Obstruction most commonly involves the small bowel, with adhesions followed by hernias accounting for the majority of postoperative causes. Symptoms associated with bowel obstructions include colicky abdominal pain that comes in waves, bloating, and rapid onset of nausea, often with forceful emesis that temporarily relieves these symptoms. Auscultation reveals high-pitched, rushing bowel sounds and borborygmi. An obstruction series imaging will show distended loops of bowel with air-fluid levels arranged in a stepladder fashion. Conservative management with placement of a nasogastric tube is appropriate if evidence of bowel strangulation, such as fever, tachycardia, abdominal guarding, rebound tenderness, and leukocytosis are absent. A spontaneous resolution rate of approximately 80% is seen, with partial obstructions responding better than complete blockages. If improvement is not evident within the first 1 to 2 days of conservative management, or if signs and symptoms of bowel compromise develop, surgical exploration should be performed.

Patients who have undergone extensive enterolysis or bowel resection either due to injury or to disease are at risk for perforation or leakage at the anastomotic site with the subsequent development of peritonitis, an abscess, or an enterocutaneous fistula. Leakage from small bowel anastomoses occurs in up to 3% of cases whereas the risk rises to up to 20% for colorectal anastomoses. Patients with perforation or free anastomotic leaks allowing soiling throughout the peritoneum present with fever, tachycardia, increasing abdominal pain, and acute abdominal signs such as guarding and rebound tenderness. In the immediate postoperative period, intra-abdominal free air detected by x-ray will not be diagnostic. Septic shock with hypotension and end-organ dysfunction can rapidly ensue. A high level of suspicion must be maintained when evaluating such patients since the use of postoperative narcotics can minimize these signs and symptoms. If significant concern for peritonitis is present, medical stabilization should be promptly initiated, including the use of broad-spectrum antibiotics, and the patient returned to the operating room for re-exploration. Intraoperative management must be individualized based on the condition of the patient and the complexity of the complication. Often a simple perforation or a small bowel anastomotic leak can be repaired primarily. Distal colonic and rectal anastomotic leaks will usually necessitate colostomy formation. Postoperative abscess formation following contamination of the peritoneal cavity during gynecologic surgery has become much less frequent due to the use of preoperative prophylactic antibiotics. Simple vaginal cuff abscesses can often be opened and allowed to drain

through the vagina. Deeper pelvic or abdominal abscesses can occur spontaneously or in association with a contained leakage from the bowel. Intravenous antibiotics and drainage are usually required. Percutaneous placement of a drainage catheter is favored as a safe approach.

The second group of agents are biologically active. Topical thrombin can be sprayed on an area of light bleeding or can be used in conjunction with a collagen or gelatin matrix. FloSeal® (Baxter, Deerfield, IL) and SURGIFLO combine topical thrombin with a gelatin matrix to provide a framework for clot initiation and limit the spread of thrombin. As noted above, a similar strategy is to saturate Gelfoam® with thrombin. These are applied to areas with light active bleeding and rely on the conversion of the patient's fibrinogen to fibrin to complete hemostasis. Products using bovine thrombin carry a black box warning from the FDA regarding the potential development of antibodies to bovine thrombin and/or factor V that can cross-react against human factor V, causing a factor V deficiency. This can lead to hematologic abnormalities that affect the prothrombin (PT) and the partial thromboplastin (PTT) times and can cause severe bleeding or thrombosis. For more brisk venous or arterial bleeding, fibrin sealants are indicated. These include Tisseal® (Baxter Dearfield, IL), Evicel® (Ethicon Somerville, NJ), and Vitagel® (Orthovita Malvern, PA), and contain thrombin and fibrinogen. Vitagel is unique in that it uses plasma obtained from the patient to supply concentrated autologous fibrinogen, platelets, and other coagulation factors. However, the thrombin in this product is bovine-derived. Fibrin glue can also be made by filling separate syringes with thrombin and cryoprecipitate. The contents of the syringes are applied simultaneously to the area of bleeding. An additional alternative has been developed that impregnates thrombin and fibrinogen onto an oxidized regenerated cellulose patch (EVARREST) to create a seal at the point of bleeding.

There are few studies directly comparing these agents. A rat neurosurgical model was recently used to compare the safety and efficacy of Surgicel®, FloSeal, Arista®, and Avitene® against a negative control. A standardized defect was made in the rats' brain and the agents were then applied to the area. Time to hemostasis was recorded. The rats were sacrificed according to a predetermined schedule and their brains were examined for inflammation and residual hemostatic agent. In this relatively small study, all the hemostatic agents performed better than the negative control, with hemostasis at 1 minute achieved in approximately 65% to 95% of active cases. Avitene and FloSeal showed a propensity to promote granuloma formation and residual material remained for all of the agents but Arista. Clearly these latter two attributes are less critical in abdominal/pelvic surgery.

If the above steps are unsuccessful, suturing of a venous defect in a large vessel such as the vena cava is performed using a 5-0 monofilament suture. Proximal and distal occlusion of the vessel around the site of injury using sponge sticks will facilitate ease of repair. Alternatively, a finger may be placed over the vascular defect and slowly moved down the length of the vessel as successive stitches are placed. For bleeding deep in the pelvis, a bilateral hypogastric artery ligation will reduce the pulse pressure in the more distal vessels and control bleeding in up to 50% of cases.

Recent reports including a meta-analysis have shown that intravenous infusion of recombinant activated factor VIIa has an approximately 75% likelihood of reducing or stopping major abdominal bleeding. Thromboembolic complications occurred in 16% of cases. If all else fails, a base of hemostatic agents are applied to the area of bleeding and packing is placed in an effort to apply pressure to this area when the abdominal wall is closed. A variety of techniques have been described including a "parachute" packing that comes out through the vagina and is placed on traction to apply pressure to the deep pelvis. The patient remains intubated and sedated while medical stabilization is achieved. Prophylactic antibiotics are given and the patient is returned to the operating room for pack removal in 24 to 72 hours.

Where deep pelvic sidewall bleeding is experienced and does not respond to internal iliac ligation but can be controlled by application of clamps deep in the pelvis, it is valuable to be able to leave the clamps in situ and remove them 48 hours postoperatively in the theatre under light anesthesia. Close monitoring thereafter usually reveals no evidence of bleeding.

In situations of excessive hemorrhage, the surgeon must remain aware of the extent of blood loss. If this loss is rapid or extreme, it may be necessary to stop active efforts to identify and repair bleeding sites, which often allows ongoing loss of blood, in favor of controlling the bleeding with pressure and allowing the anesthesiologist to stabilize the patient with crystalloid and blood products. Additional assistants and specialists should be summoned as needed. As blood loss mounts, monitoring of the patient's coagulation profile with replacement using fresh frozen plasma, platelets, and cryoprecipitate as indicated becomes essential. Blood calcium levels can become deranged and be a cause of continuous hemorrhage.

WOUND COMPLICATIONS

The incidence of postoperative wound complications is associated with patient-related factors such as obesity, older age, poor nutritional status, and intercurrent medical conditions such as diabetes and pulmonary disease. Intraoperative factors adversely affecting wound healing include extended duration of surgery, inadequate wound hemostasis, and poor surgical technique. Wound infections occur in up to 12% of cases, while fascial dehiscences are discovered in up to 3% of wounds. Superficial wound separations affect up to 20% of cases. The choice of abdominal incisions is dependent primarily on issues related to access to the pelvis and upper abdomen. Transverse incisions provide excellent exposure to the pelvis while minimizing the cosmetic side effects of pelvic surgery when laparoscopy is not feasible. In addition, many studies, including a recent Cochrane review, have found that when compared to vertical incisions, transverse incisions are associated with less pain, less compromise of pulmonary function, and lower rates of dehiscence and hernia formation. Despite the increased operative time, greater blood loss, and increased risk for nerve damage with transverse incisions, they are the default surgical route when access to the upper abdomen is not needed or large masses do not require intact removal. Entrapment of the ilioinguinal or iliohypogastric nerve within the fascial closure of a transverse incision can occur when the fascial incisions have extended beyond the lateral border of the rectus muscles. Patients present with sharp,

moderate to severe pain localized to the lower quadrant. Relief of the pain following injection of local anesthetic helps to establish the diagnosis. Under extreme circumstances, the fascial stitch may need to be modified.

When pelvic exposure is limited with a Pfannenstiel incision, we recommend conversion to a Cherney incision in which the tendinous insertions of the rectus muscles onto the symphysis pubis are divided. A portion of the tendon is left on both the muscle and the insertion site to facilitate reapproximation with permanent suture at the completion of the procedure. The inferior epigastric vessels are isolated along the lateral edge of each muscle and divided. Partially or fully cutting the rectus muscles under these circumstances is discouraged since the attachment of the muscles to the fascia was taken down as part of the Pfannenstiel incision and closure of the fascia at the completion of the procedure will not reapproximate the cut portion of muscle.

The obese patient presents a special challenge in regard to incision location. The inclination to make a suprapubic incision below the pannus must be resisted due to the high rate of wound breakdown and infection associated with this location. The lone exception to this rule is when a panniculectomy is performed which facilitates the intra-abdominal portion of the procedure and reduced postoperative complications. Gallup has described a technique in which the pannus is retracted caudally and a vertical incision is made either periumbilically or, for those with a very large pannus, entirely supraumbilically. Care must be taken to not extend the incision on to the pannus and inadvertently go through it and on to the mons. The fascial incision is taken down to the symphysis pubis. Issues in regard to closure are discussed below.

Epithelialization begins within hours following wound closure with a watertight seal established within 48 hours. Wounds should be covered with a clean, dry dressing for 24 to 48 hours. The wound's tensile strength increases rapidly during the initial 6 weeks following surgery. Staples may be removed from low-tension, transverse incisions in 7 days. For vertical incisions that are under increased tension, particularly in the obese, staples should remain in place for up to 14 days despite the increased scarring that can develop at the staple sites when they remain in place beyond 7 to 10 days. Tapes such as Steri-Strips are placed across the wound following removal of staples to reduce tension on the skin edges. Alternatives to standard staples for large wounds or those under mild tension are subcuticular stitches or use of copolymer subcutaneous staples that are absorbed over several months and therefore do not need to be removed. For smaller incisions, dermal glues provide fast closure with good cosmesis.

The role of surgical preparation and technique in the development of wound complications has been extensively studied. There is no clear evidence that bathing preoperatively with chlorhexidine reduces the risk for skin infections. Furthermore, scrubbing and painting the abdomen holds no advantage over an iodine-based paint-only skin prep, and using a second scalpel after opening the skin also does not reduce the incidence of wound infections. Clipping rather than shaving pubic hair that might interfere with skin closure has been shown to be beneficial. Incising the subcutaneous fat with either a scalpel or with electrocautery using cutting current also does not appear to affect wound outcome. Coagulation current should not be used for general opening of the subcutaneous tissue or fascia due to the wider path of thermal injury caused by this mode.

Closure of the peritoneum is associated with adhesion formation, infection, and delayed return of bowel function. A running mass closure of the abdominal wall using either delayed-absorbable or permanent monofilament suture with stitches placed 1.5 to 2 cm from the fascial edge and 1 cm apart has a dehiscence rate of less than 0.5%. It is important when closing the fascia to reapproximate the tissue but to not strangulate it by pulling too tightly on the sutures, which can predispose to dehiscence.

Management of the subcutaneous tissue in overweight and obese women remains controversial. A meta-analysis from 2004 examined suture closure of subcutaneous fat greater than 2 cm in thickness during caesarean section. Though only one of the studies independently showed benefit, the analysis concluded that closure decreased the risk of wound disruption by 34%. However, a prospective, randomized study involving 222 evaluable subjects compared a control group to subcutaneous closure or closed suction drainage of the subcutaneous space in gynecologic patients with vertical incisions and 3 cm or more of subcutaneous fat. The overall wound complication rates and wound disruption rates were similar for all groups. Of additional interest is an obstetrical study that showed no difference between suture closure with or without closed suction drainage.

Superficial wound separations occur when excessive tension is placed on the skin edges. Often the subcutaneous tissue has not reapproximated and an infection, seroma, or hematoma may be present. Loculated subcutaneous fluid will usually begin to seep through the wound within 3 to 7 days following surgery, heralding an impending wound separation. If the drainage is copious and persistent, fascial dehiscence must be considered and gentle probing of the fascia with a long Q-tip or a gloved finger should be performed. Purulent drainage due to infection needs to be cultured and drained by opening the incision. Debridement of the wound as described below is usually sufficient. If cellulitis of the skin is present, characterized by erythema, warmth, tenderness, and swelling, antibiotic therapy using a first-generation cephalosporin or a quinolone is prescribed for 10 days.

When a superficial wound separation is apparent, the extent of the defect in the subcutaneous tissue is assessed. If a significant portion of the defect tunnels under an intact area of the wound, particularly if access for debridement and packing is limited, the overlying skin is opened. In the occasional case where the wound surfaces are clean, immediate closure with permanent monofilament suture is performed. Mattress stitches are placed approximately 2 cm apart and tied tight enough to reapproximate but not necrose the tissue. Steri-Strips can be placed between sutures to further approximate the wound edges. It is important to close the deep subcutaneous space to avoid seroma development. We have successfully utilized a modification of the figure-of-eight closure described by Dodson et al. (1992) for patients with particularly deep wounds. Sutures are removed in 10 to 14 days. Antibiotics are used only when infection is present.

If necrotic or infected tissue is present, debridement is performed. Studies evaluating various means of wound debridement including sharp dissection, mechanical debridement using wet-to-dry normal saline dressing changes, and enzymatic

or autolytic agents have failed to identify significant outcome differences between these methods. Once the wound is free of necrotic or infected debris and granulation tissue is present, the wound may be closed using the techniques noted above. Secondary closure significantly reduces recovery time versus healing by secondary intention and is successful in approximately 90% of cases. An additional option to speed healing is a vacuum-assisted closure (VAC) device which cyclically applies negative pressure to the wound bed, facilitating the removal of interstitial fluid and formation of granulation tissue and reducing bacterial colonization. A 2004 study from M.D. Anderson showed that this devise could be used for a variety of complex gynecologic oncology wounds.

Fascial dehiscence (separation of the fascial closure) and evisceration (dehiscence with protrusion of the bowel through the wound) are surgical emergencies that historically have been associated with a mortality rate of up to 35%. Recent series have demonstrated much lower mortality rates, possibly due to earlier recognition and better supportive care. Fascial dehiscence usually occurs 1 to 2 weeks following surgery. When suspected, the incision must be thoroughly inspected, preferably using a gloved finger on the fascia. When a dehiscence is discovered, broad-spectrum antibiotics are started, and the patient is immediately moved to the operating room. Under most circumstances, the point of failure will be the fascia rather than breakage or untying of the suture. The wound should be opened entirely and cleaned of any necrotic or infected tissue. The bowel should be inspected for injury, and copious irrigation of the abdominal cavity performed. A nasogastric tube is placed to help decompress the bowel. A continuous mass closure technique as described above is used to close the abdominal wall. In addition, many surgeons continue to place retention sutures using large permanent sutures placed through the entire thickness of the abdominal wall, spaced approximately 3 cm apart, and secured using skin bridges that allow for adjustment of the tension of the suture. The skin is usually closed secondarily.

BIBLIOGRAPHY

Brown SR, Tiernan J. 2005. Transverse verses midline incisions for abdominal surgery. *Cochrane Database Syst Rev* (4). DOI:10.1002/14651858.CD005199.pub2.

Chelmow D, Rodriguez EJ, Sabatini MM. 2004. Suture closure of subcutaneous fat and wound disruption after cesarean delivery: A meta-analysis. *Obstet Gynecol* **103**:974–80.

Dodson MK, Magann EF, Meeks GR. 1992. A randomized comparison of secondary closure and secondary intention in patients with superficial wound dehiscence. *Obstet Gynecol* **80**(3):321–4.

Guenaga KKFG, Matos D, Wille-Jorgensen P. 2009. Mechanical bowel preparation for elective colorectal surgery. *Cochrane Database Syst Rev* (1).

Güenaga KF, Matos D, Wille-Jørgensen P. 2011. Mechanical bowel preparation for elective colorectal surgery. *Cochrane Database Syst Rev* (9). DOI: 10.1002/14651858.CD001544.pub4.

Pursifull NF, Morey AF. 2007. Tissue glues and nonsuturing techniques. *Curr Opin Urol* **17**:396–401.

Schimp VL, Worley C, Brunello S, Levenback CC, Wolf JK, Sun CC, Bodurka DC, Ramirez PT. 2004. Vacuum-assisted closure in the treatment of gynecologic oncology wound failures. *Gynecol Oncol* **92**(2):586–91.

Stany MP, Farley JH. 2008. Complications of gynecologic surgery. *Surg Clin N Am* **88**:343–59.

Sweeney KJ, Joyce M, Geraghty JG. 2002. Management of intraoperative bowel injuries. *CME J Gynecol Oncol* **7**:178–82.

Tanguy M, Seguin P, Malledant Y. 2007. Bench-to-bedside review: Routine postoperative use of the nasogastric tube—Utility of futility? *Crit Care* **11**:201–7.

Von Heymann C, Jonas S, Spies C, et al. 2008. Recombinant activated factor VIIa for the treatment of bleeding in major vascular and urological surgery: A review and meta-analysis of published data. *Crit Care* **12**:R14.

Wydra D, Emerich J, Ciach K, et al. 2004. Surgical pelvic packing as a means of controlling massive intraoperative bleeding during pelvic posterior exenteration—A case report and review of the literature. *Int J Gynecol Cancer* **14**:1050–4.

4 Anatomy

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INTRODUCTION

Surgical anatomy is the synthesis of topographic, functional, and clinical anatomy and surgical techniques applied to diagnosis and treatment. It presents more than a systematic description of anatomic structures; with particular emphasis on anatomical relationships. Cancer biology and tumor spread are also considered with different surgical techniques. Thus, to achieve the primary goal of cancer treatment, and to completely extirpate tumor masses and preserve important anatomic structures—a detailed knowledge of the anatomy of the pelvis and abdomen is essential. This skill directly influences complication rate (morbidity) and optimal debulking rate (survival) of patients with gynecologic tumors. Further, anatomical knowledge lends insight into pathogenesis, influences treatment decisions, and is critical for effective communication between surgeons and pathologists. Studies have shown that the strongest clinician-driven predictor of survival is the optimal surgical outcome (Barlin et al. 2009, Bristow et al. 2002, Jemal et al. 2008, Lichtenegger et al. 1998). A survey on patients with ovarian carcinoma from 904 American hospitals demonstrated that gynecologic oncologists performed more hysterectomies, oophorectomies, omentectomies, and lymph node, and peritoneal biopsies, and yielded higher debulking rates than other specialists (Nguyen et al. 1993). With the exception of patients with Stage I disease, patients treated by general surgeons had significantly reduced survival compared with those treated by gynecologic oncologists ($p < 0.004$). To optimize clinical management and to eliminate unnecessary steps and improve safety and efficacy, systematic and continual teaching in anatomy is required for all physicians who are involved in the surgical treatment of patients with gynecologic malignancies.

PELVIC FASCIA AND PELVIC SPACES

The pelvic fascia occupies space between the membranous peritoneum and the muscular pelvic walls and floor not occupied by viscera (Figure 4.1). It can be further characterized as membranous pelvic fascia (parietal and visceral) and endopelvic (subperitoneal) fascia. The latter includes numerous synonyms such as intrapelvic fascia, connective tissue body, neurovascular plate, corpus intrapelvicum, paratissue (Stoeckel) parametrium, parangium hypogastricum (Pernkopf), transverse ligament of the collum (Mackenrodt), cardinal ligament (Kocks), web (Meigs), broad ligament, and hypogastric sheets. Some fascia line muscles and viscera, or provide scaffolding, and in doing so form reflections and actual and potential spaces. Their nature and association with pelvic structures explains their inclusion in the critical knowledge base.

Parietal pelvic fascia lines the muscles that form the pelvic walls and floor, and is continuous with transversalis and iliopsoas fascias. Visceral pelvic fascia encloses pelvic organs and forms their adventitial layers. Both parietal and visceral fascias are continuous where viscera penetrate the pelvic floor. Here, parietal fascia thickens, forming the bilateral tendinous arch (arcus) that courses from pubis to sacrum (Figures 4.2 and 4.3), adjacent to the viscera. In females, the arcus is divided into the anterior pubovesicular ligament and the posterior sacrogenital ligaments. This lateral attachment of visceral fascia of the vagina with the arcus is called the paracolpium. The paracolpium supports the vagina and assists in the weight bearing of the urinary fundus. Because of its anatomical course and thickness, the arcus can be used to anchor sutures during reconstructive procedures.

The remaining fascia is endopelvic fascia; it varies in density and content, and forms the matrix surrounding pelvic viscera. Using blunt dissection, surgeons can easily create potential spaces within this loose tissue: the prevesicular (retropubic), the paravesicular (posterolateral), the pararectal and the presacral (retrorectal) spaces (Moore et al. 2014). More fibrous areas of endopelvic fascia form condensations known as pelvic ligaments. One of these, the hypogastric sheath, serves as a conduit for passage of all neurovascular structures passing from the lateral pelvic wall to the viscera, but also separates the retropubic and presacral spaces. Medially, this sheath divides into three pillars (laminae or ligaments) that pass between pelvic organs and convey neurovascular and structural support: the bladder pillar; the lateral rectal pillar; and the uterovaginal pillar (cardinal, or transverse cervical ligament).

The cardinal ligament (see Figure 4.3) is the strongest thickening of pelvic fascia, providing the majority of support for the uterus. It can be used clinically to anchor wide loops of suture during surgical repair. Further, it emits the rectal and bladder pillars. The paracolpium (part of the uterovaginal pillar below the level of the ureter) reaches the vagina and cervix at the level of the vaginal fornix. Additional loose connective tissue lies between the uterus and the ureter (*mesoureter*) containing the blood supply for the ureter.

The bladder pillar courses from the body of the corpus intrapelvicum to the bladder, conveying superior vesicular arteries and veins. Viewed from the vagina, the distal pillar lies in the sagittal plane and rises to the bladder forming a *vesicouterine ligament*, part of which, covering the ureter (ureteral roof), forms the upper limit of the paracystium.

The rectal pillar extends from the cardinal ligament to the sacrum, and conveys the middle rectal arteries and veins, and rectal nerve plexuses. The upper portion deviates laterally to accommodate the pouch of Douglas (rectouterine; cul-de-sac; Moore et al. 2014); bringing it close to the pelvic wall. The rectouterine ligament splits into an anterior leaf that emits rectal fascia, and a posterior leaf, which reaches the sacrum at the level

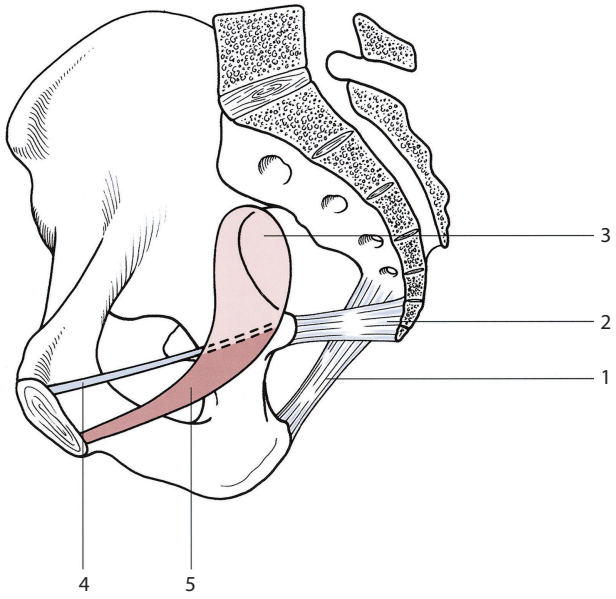

Figure 4.1 1: Ischiosacral ligament; 2: sacrospinous ligament; 3: origin of corpus intrapelvicum at lateral pelvic sidewall; 4: arcus tendineus levatoris ani; 5: arcus tendineus fascia pelvis.

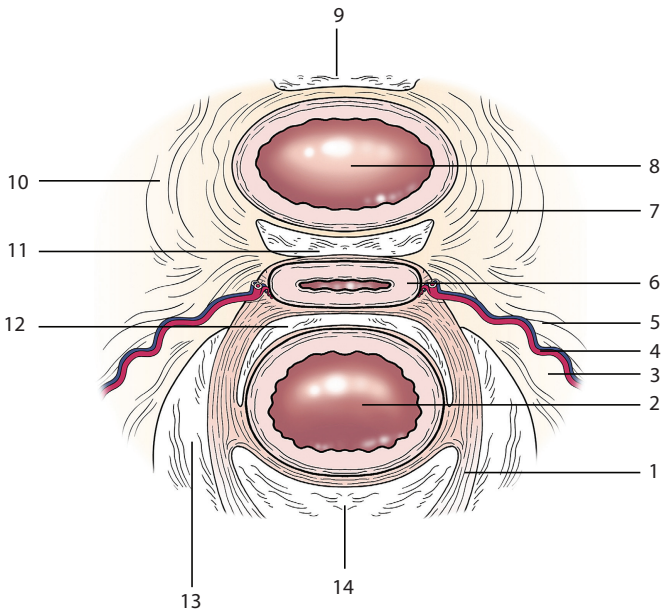

Figure 4.3 1: Uterosacral ligament; 2: rectum; 3: adventitia; 4: vessels; 5: cardinal ligament; 6: vagina; 7: bladder pillar; 8: urinary bladder; 9: prevesical space; 10: paravesical space; 11: vesicovaginal space; 12: rectovaginal space; 13: pararectal space; 14: retrorectal space.

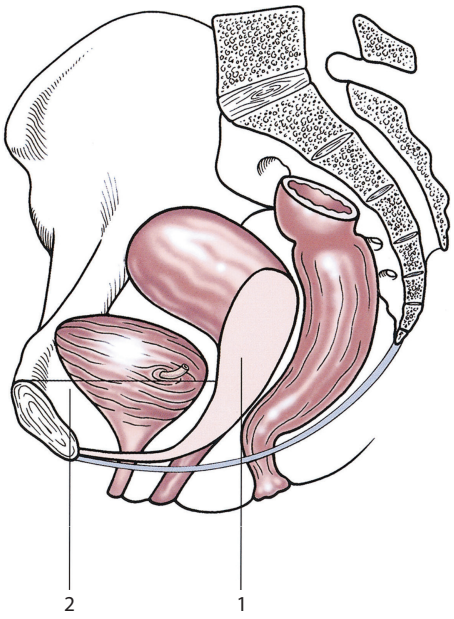

Figure 4.2 1: Corpus intrapelvicum; 2: arcus tendinous levatoris ani.

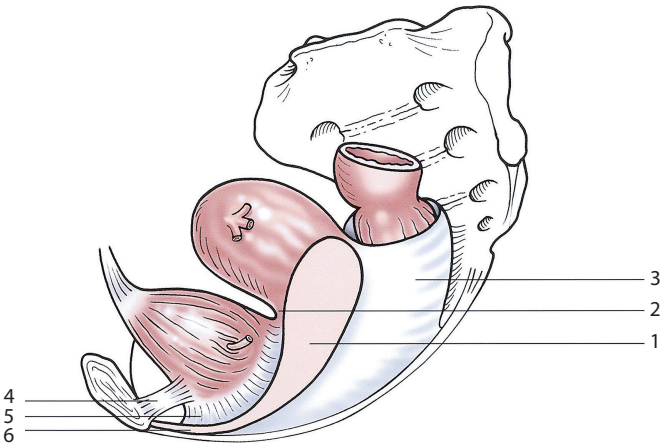

Figure 4.4 1: Origin of connective tissue, detached at lateral pelvic wall; 2: cardinal ligament; 3: uterosacral ligament; 4: pubovesical ligament; 5: laterovesical ligament; 6: arcus tendineus fascia pelvis.

of the anterior sacral foramina II to IV, but can extend upward beyond the sacral promontory (Figures 4.3 and 4.4). As such, this fascia creates a surgically important pelvirectal space superior to the pelvic diaphragm, in contrast to the ischio-anal fossa in the perineum. It is divided into the rectouterine and rectorectal (presacral) spaces by the lateral rectal ligaments. The retrorectal space is limited by rectal fascia and the parietal pelvic fascia, and is separated from the pararectal spaces by the rectal pillar.

The rectouterine pouch opens laterally into the pararectal space (Moore et al. 2014). After being opened from the abdomen, the pararectal space is narrow, because the rectal pillar lies close to the pelvic wall. Surgeons can gain access to this space pulling the uterus anteriorly so that the rectal pillar is lifted off the pelvic wall.

The paravesical space is limited medially by the obliterated umbilical artery (umbilical ligament), vesical fascia, and the ligament of the bladder. At its lateral margin, it merges into the retropubic space. The body of the corpus intrapelvinum and the cardinal ligament form the posterior boundary. The roof of the paravesical and prevesical space is formed by the vesico-umbilical fascia.

The pararectal space is limited medially by the ureter, rectal fascia, and the rectal pillar, and laterally by the parietal pelvic fascia and internal iliac vessels. Its anterior border is the cardinal ligament. After being opened from the abdomen, the pararectal space is narrow, because the rectal pillar lies close to the pelvic wall. The space is best demonstrated by pulling the uterus anteriorly so that the rectal pillar is lifted off the pelvic wall.

The retrorectal/presacral space lies behind the rectum and is limited by rectal fascia and the parietal pelvic fascia. The retrorectal space is separated from the pararectal spaces by the part of the rectal pillar that joins the pelvic sacral foramina II to IV.

Between the vaginal and rectal fascia lies the rectovaginal space extending caudally to the centrum tendinum. Superiorly, it is limited by the peritoneum of the cul-de-sac, and bilaterally by the rectal pillars. The vesicocervical and vaginal spaces are limited by vesical fascia and the cervix, reach the peritoneum, and are separated by the supravaginal septum. The vesicovaginal space reaches caudally to the origin of the urethra and between the bladder pillars.

The rectouterine folds contain a considerable amount of fibrous tissue and muscular fibers which are attached to the front of the sacrum and constitute the uterosacral ligaments (rectouterine ligaments). These ligaments are major ligaments of the uterus (uterosacral, cardinal, and pubocervical ligaments) and course from the uterus near the cervix to the anterior aspect of the sacrum. Pelvic splanchnic nerves run on top of the uterosacral ligaments, and the ligaments are palpable during rectal examination.

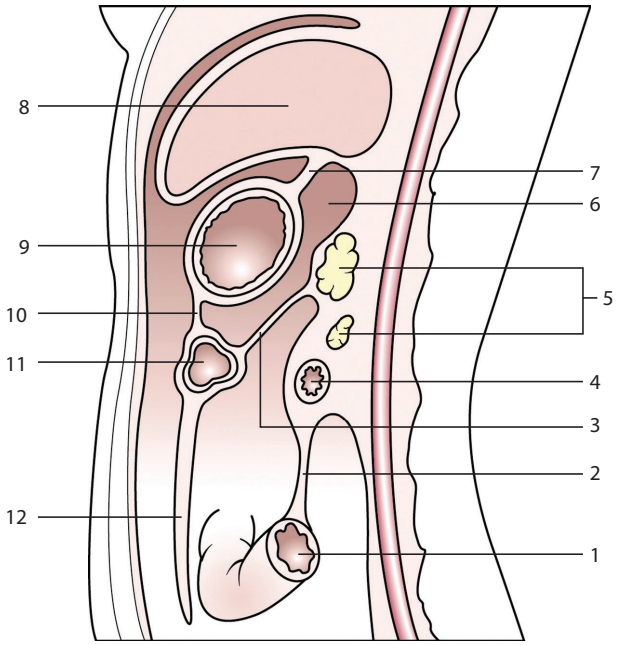

Figure 4.5 1: Small intestine; 2: mesentery; 3: transverse mesocolon; 4: duodenum; 5: pancreas; 6: omental bursa; 7: lesser omentum; 8: liver; 9: stomach; 10: gastrocolic ligament; 11: transverse colon; 12: greater omentum.

UPPER PART OF THE ABDOMEN

In most primary gynecologic cancers, the highest tumor mass is concentrated in the pelvis, whereas the upper abdominal quadrants are predominantly involved by metastases in patients with recurrence. Even so, given the physical proximity of the gastrointestinal (GI) tract to the reproductive tract, as well as the fact that signs and symptoms of GI pathology can mimic those of gynecologic cancers, the abdominal viscera and relationships to peritoneum and peritoneal reflections are important (Le 2013). Gynecologic oncology surgeons are uniquely qualified to balance the surgical effort with the potential therapeutic gain by virtue of their expertise in the relevant cancers, and by virtue of this chapter and similar materials, the relevant surgical anatomy.

The abdominal peritoneal cavity continues inferiorly into the pelvic cavity, and is a potential space devoid of organs but containing a thin film of peritoneal fluid. The intraembryonic coelom (embryonic body cavity) serves as the primordial peritoneum. During development, the primordial abdominal cavity is lined with peritoneum derived from this mesoderm that forms a closed sac; the lumen of this peritoneal sac is the peritoneal cavity. As viscera migrate into this sac, their vessels and nerves remain connected to their extraperitoneal sources or destinations; between fused layers of peritoneum (mesenteries).

Various terms are used to describe parts of the peritoneum/mesentery (Figure 4.5). The small intestine mesentery is referred to as "the mesentery," but other mesenteries of specific parts of the GI tract are named accordingly: mesoesophagus, mesogastrium, transverse and sigmoid mesocolons, and mesoappendix. Omentum describes a double-layered extension of peritoneum passing from the stomach and proximal duodenum to adjacent organs. The greater omentum descends from the greater curvature of the stomach and then ascends to the anterior transverse colon and mesocolon. Similarly, the lesser omentum extends from the lesser curvature of the stomach and duodenum to the liver. Peritoneal ligaments are named based on which organs or parts of the abdominal wall they connect: falciform ligament, hepatogastric, hepatoduodenal ligament (thickened free edge of the lesser omentum conducting the portal triad), gastrophrenic ligament, gastrosplenic ligament, and gastrocolic ligament.

The omental bursa is a sac-like cavity posterior to the stomach, lesser omentum, and gastrocolic ligament that communicates with the greater sac via the epiploic (omental) foramen (of Winslow). Surgeons can explore the omental bursa by preparing the space between the gastrocolic ligament and transverse colon or via the foramen of Winslow by palpation. During primary surgery of ovarian cancer, the greater omentum is often resected incompletely by design. As a consequence, residuals of omentum are frequently detected during surgery in relapse. In the case of an acute pancreatitis, necrosis or effusion can also affect this pouch. In cases of diffuse peritoneal carcinomatosis, peritonectomy is often applied to achieve debulking. This can also be performed in the case of involvement of the right diaphragm. Thus, the falciform ligament of the liver is cut to completely inspect the diaphragm.

Peritoneal recesses/gutters refer mainly to four spaces in the abdomen: left and right paracolic gutters, and left and right paramesenteric gutters. Other smaller recesses include those around the duodenojejunal flexure, cecum, and sigmoid colon. These gutters are clinically important because they allow a passage for infectious fluids from different abdominal compartments. Along the lateral edge of the paracolic gutters (Moore et al. 2014), the White line of Toldt is formed. Surgeons can perform the Cattell maneuver by dividing along the White line of Toldt lateral to the cecum and ascending colon exposing the inferior vena cava (IVC), right renal vessels, fourth part of the duodenum, aorta, and uncinate process of the pancreas.

The duodenum is about 25 cm long, C-shaped, and aside from its ampulla, it is entirely retroperitoneal. The duodenum has four parts: superior, descending, horizontal, and ascending. The ligament of Treitz is a musculofibrous band that extends from the upper aspect of the ascending part of the duodenum to

the right diaphragmatic crus and tissue around the celiac trunk (CT). Always remember that the head of the pancreas lies in the "C" of the duodenum.

VASCULAR SUPPLY

Most vessels encountered during oncologic procedures can be interrupted without ill effect secondary to rich collateral circulation (Figures 4.6 through 4.8). These anastomoses prevent ischemia unless more than one major vessel is occluded. However, patchy ischemia, induced by atherosclerosis, fibrosis, or irradiation, can occur since small vessels entering the gut wall are essentially terminal arteries. Obstruction of these vessels results in segmental ischemia. Whenever possible, vessels should be spared to promote healing and to optimize chemo- and radiotherapy. Certain vessels, such as the superior mesenteric artery (SMA), can never be interrupted without reanastomosis. In advanced cancer, regions of the GI tract are frequently involved; therefore, knowledge of the blood supply of mesenteric and pelvic arteries is required to determine areas of intestinal resection and to obtain maximal debulking.

Blood vessels are not entirely consistent in their course and origin. Guidelines for locating vessels include bony landmarks and cutaneous and muscle relationships. The descending aorta pierces the diaphragm at vertebra level T12 and usually bifurcates at L4. Renal arteries originate near L2. The ovarian arteries arise directly from the aorta at L3. The three major arteries originating from the aorta are the CT (T12–L1); the SMA (L1–L2); and the inferior mesenteric artery (IMA) (L3; 2–3 cm inferior to the SMA) (Figures 4.6 and 4.7). Lastly, external iliac vessels are landmarks of the pelvis that are easily palpated during surgery (Figures 4.7 and 4.8).

The CT supplies mostly structures of the embryonic foregut. From the CT, the common hepatic becomes the proper hepatic; then left and right hepatic (usually giving rise to the cystic artery) arteries. The right gastric and left gastric arteries anastomose, forming the lesser epiploic artery. The common hepatic artery divides into the supraduodenal artery and the gastroduodenal artery, which divides into the right gastro-omental (gastroepiploic) and the superior pancreaticoduodenal. The splenic artery traverses the splenorenal ligament and gives rise to the left gastro-omental artery that anatastomoses with the right gastro-omental artery.

The SMA supplies viscera derived from the embryonic midgut. The SMA runs in the root of the mesentery to the ileocecal junction, gives rise to jejunal and ileal branches, the inferior pancreaticoduodenal artery, the middle and right colic arteries, and terminates as the ileocolic artery. The ileocolic artery divides into ileal and colic branches, and an appendicular artery. The inferior pancreaticoduodenal artery anastomoses with the superior pancreaticoduodenal artery.

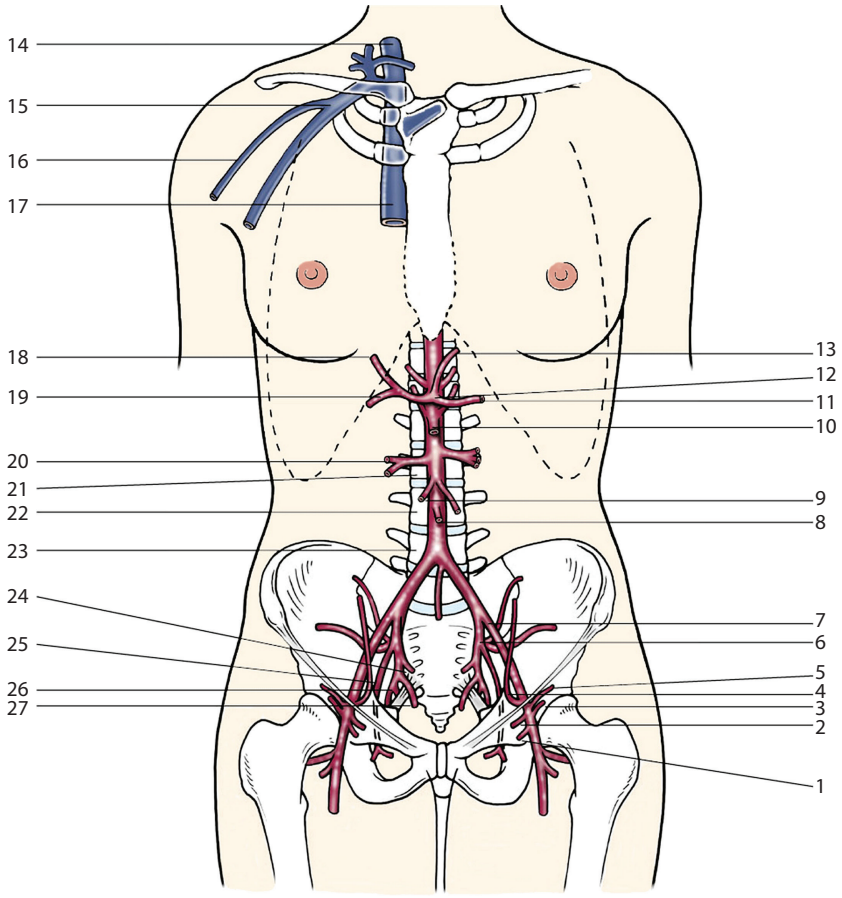

Figure 4.6 1: External pudendal artery; 2: superficial epigastric artery; 3: superficial circumflex iliac artery; 4: inferior epigastric artery; 5: deep circumflex iliac artery; 6: internal iliac artery; 7: external iliac artery; 8: inferior mesenteric artery; 9: gonadal artery; 10: superior mesenteric artery; 11: splenic artery; 12: CT; 13: left gastric artery; 14: internal jugular vein; 15: subclavian vein; 16: cephalic vein; 17: superior vena cava (SVC); 18: hepatic artery; 19: gastroduodenal artery; 20: renal artery; 21: L2; 22: L3; 23: L4; 24: inferior rectal artery; 25: obturator artery; 26: internal pudendal artery; 27: uterine artery.

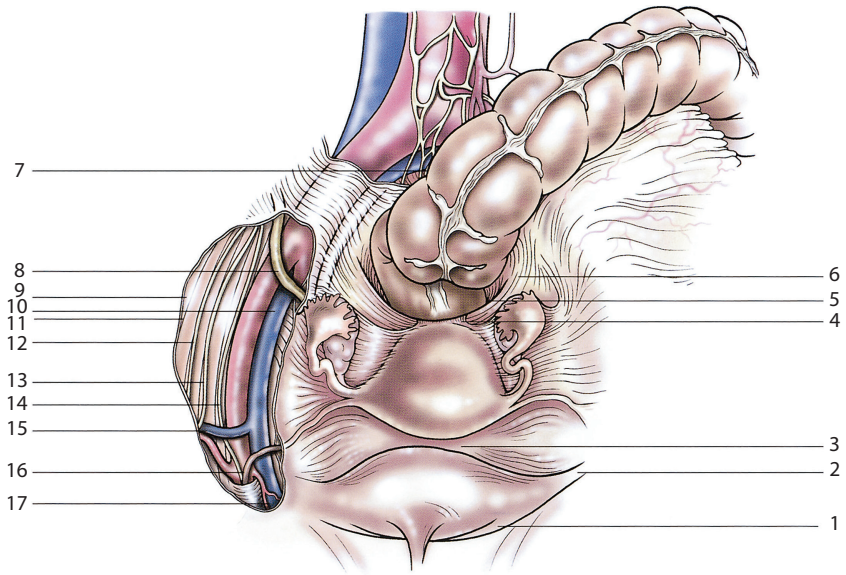

Figure 4.7 1: Paravesical fossa; 2: transverse vesical fold; 3: uterovesical pouch; 4: rectovesical pouch; 5: sacrogenital fold of uterosacral ligament; 6: pararectal fossa; 7: superior hypogastric plexus; 8: ureter; 9: psoas muscle; 10: internal iliac artery; 11: iliohypogastric nerve; 12: ilioinguinal nerve; 13: lateral femoral cutaneous nerve; 14: genitofemoral nerve; 15: circumflex iliac artery; 16: round ligament inserting into internal inguinal ring; 17: inferior epigastric artery.

The IMA descends retroperitoneally supplying the GI tract derived from the embryonic hindgut. It gives rise to the left colic supplying the descending colon, and the sigmoid artery that supplies the distal descending and sigmoid colons. The IMA terminates as the superior rectal artery.

Middle suprarenal arteries arise on the lateral aorta near the SMA. With the superior and inferior suprarenal arteries, they form approximately 60 branches that penetrate the capsule. Usually, four pairs of lumbar arteries arise from the posterior aorta. A fifth pair can originate from the median sacral artery. Ovarian arteries descend in the suspensory ligament of the ovary, and supply the ureter, ovary, and tubular ampulla. The marginal artery of the colon (of Drummond) runs in the mesentery along the border of the bowel as a part of the vascular arcade that connects the SMA and IMA. This is in contrast to Riolan's arch that when present is found near the mesenteric root and parallel to the middle colic artery. Riolan's arch connects the proximal middle colic artery with the left colic artery, and can be identified near the left colic flexure. The marginal artery and Riolan's arch may be enlarged providing significant blood flow to ischemic colonic segments. Critically, when Riolan's arch is not developed, or is narrowed, then ligation of the IMA can induce necrosis of the descending colon because arterial perfusion by the middle colic artery is interrupted.

Venous drainage from the abdominal esophagus, stomach, upper duodenum, jejunum, ileum, cecum, ascending colon, transverse colon, pancreas, and spleen is into the portal vein and the superior mesenteric vein. The inferior mesenteric vein joins the splenic vein before entering the portal vein, and receives blood from the descending and sigmoid colons, and rectal vein plexus. The middle rectal vein drains into the internal iliac vein, and the inferior rectal vein into the internal pudendal vein. The ovarian venous drainage is asymmetric. The right ovarian vein joins the IVC, whereas the left joins the left renal vein. The renal veins are direct tributaries to the IVC.

The internal iliac artery divides into anterior and posterior divisions (Figure 4.8). The branches that arise from the posterior division are the iliolumbar, sacral arteries, and the superior gluteal artery. The first branch to arise from the anterior division may be the iliolumbar artery. This aside, the umbilical artery (obliterated hypogastric vessel) is the first major branch, and it runs along the lateral pelvic wall then ascends toward the umbilicus giving rise to superior vesicular arteries and terminating as the medial umbilical ligament. This ligament raises a fold of peritoneum (medial umbilical fold), and identification

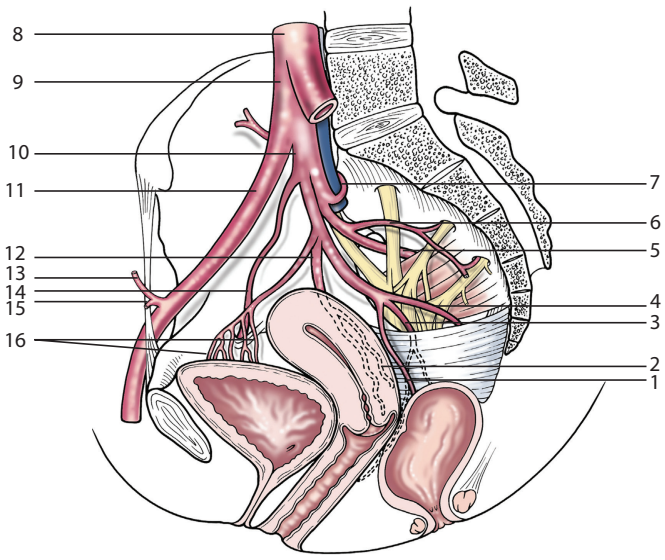

Figure 4.8 1: Middle rectal artery; 2: descending cervical of uterine artery; 3: internal pudendal artery; 4: inferior gluteal artery; 5: superior gluteal artery; 6: lateral sacral artery; 7: iliolumbar artery; 8: aorta; 9: common iliac artery; 10: internal iliac artery; 11: external iliac artery; 12: uterine artery; 13: circumflex iliac artery; 14: obturator artery; 15: inferior epigastric artery; 16: superior vesical arteries.

of the umbilical ligament is very helpful in the preparation of the parametrium during radical hysterectomy.

Near to where the umbilical artery is crossed by the ureter, the obturator artery courses along the obturator fascia. Before exiting the obturator foramen, it will give off a pubic branch that will anastomose with a "superior" pubic branch from the external iliac artery. The obturator artery may arise from the inferior epigastric artery.

The inferior vesicular artery is replaced by the vaginal artery, and arises from the uterine artery. The uterine artery may arise from the internal iliac artery, the anterior division, or from the umbilical artery, and its branches course in the broad and cardinal ligaments. Near to the cervix at the superior vagina, the ureter passes inferior to the uterine artery. The artery divides into an ascending uterine branch and a descending vaginal branch. The ascending branch will anastomose with the ovarian artery, and the vaginal artery with the vaginal branch of the uterine artery and superior vesicular arteries.

The internal pudendal artery will course inferolaterally and exit along the inferior border of the piriformis muscle in the greater sciatic foramen. Then, it will pass around the ischial spine (or sacrospinous ligament), re-entering the pelvis through the lesser sciatic foramen. It exits near the pudendal canal giving rise to the perineal artery and the dorsal artery of the clitoris.

Pelvic venous plexuses and tributaries to the internal iliac vein are important in the presence of metastases and are variable, but generally accompany the arteries that supply the same territory and viscera. One major difference is that there are no veins accompanying the umbilical arteries. Second, iliolumbar veins generally bypass the internal iliac vein, draining directly into the common iliac veins. Third, outside of pregnancy or pelvic congestion syndrome where the uterine veins enlarge, the superior gluteal vein is the largest tributary to the internal iliac vein. Finally, the lateral sacral veins provide a collateral route to the IVC or superior vena cava (SVC) via anastomotic connections with the internal vertebral venous plexus. This collateral pathway may allow ovarian cancer to spread to spinal or cranial sites. The pelvic vessels continue below the inguinal ligament into the femoral triangle (Figures 4.8 and 4.9).

Generally, lymphatic drainage parallels the course of venous blood supply. However, lymph node metastases can obstruct flow and lead to retrograde metastases, which appear to skip regional chains. For example, some endometrial and ovarian cancers can have isolated para-aortic lymph node spread through the lymph vessels of the infundibulopelvic ligament and show a retrograde lymphatic spread (Burghardt et al. 1991).

The groups of regional lymph nodes responsible for drainage of female pelvic viscera are shown in Figure 4.10. Lymph drainage from the rectum is via three pathways: from the *superior rectum* to pararectal and/or sacral nodes to the inferior mesenteric nodes, from the *middle rectum* to the internal iliac nodes, and from the *inferior rectum* directly into the sacral nodes. The inferior mesenteric nodes drain into the lumbar (caval or aortic) lymph nodes, and also collect lymph drainage from the sigmoid and descending colons. Critically, lymphatic drainage from the vagina is from four zones: the *upper vagina* drains into external and internal iliac nodes, to common iliac nodes and then lumbar

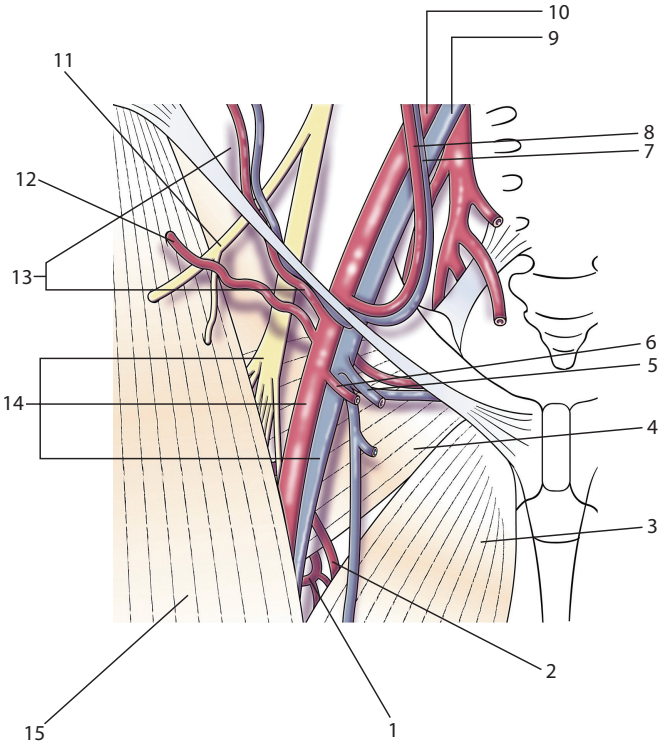

Figure 4.9 1: First perforating artery; 2: femoral pudendis artery; 3: adductor longus muscle; 4: pectineus muscle; 5: external pudendal vein; 6: external pudendal artery; 7: inferior epigastric vein; 8: inferior epigastric artery; 9: external iliac vein; 10: external iliac artery; 11: lateral femoral cutaneous nerve; 12: superficial circumflex iliac artery; 13: deep circumflex iliac artery + vein; 14: femoral nerve, artery + vein; 15: sartorius muscle.

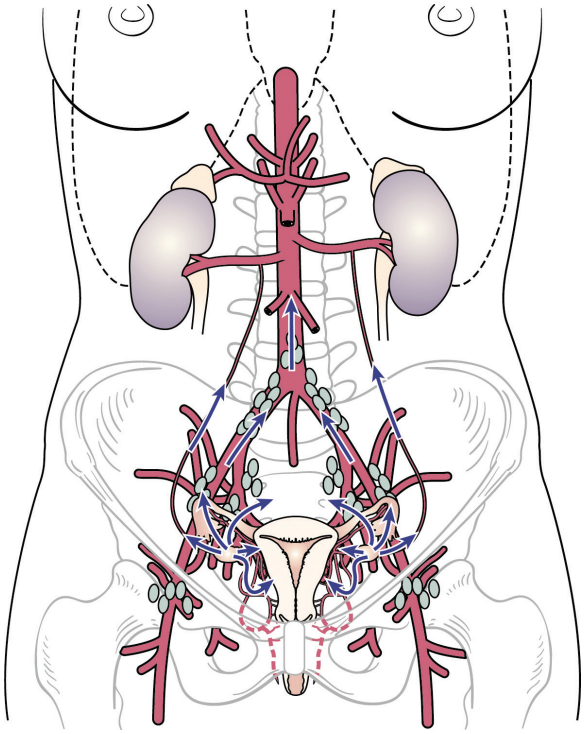

Figure 4.10 Lymphatic spread in ovarian cancer.

nodes; the *middle vagina* drains into internal iliac nodes; the *inferior vagina* drains into sacral and common iliac nodes; and the *vaginal introitus* drains into superficial inguinal nodes, that also receive drainage from the perineal integument, valve, prepuce of the clitoris, perianal integument, and anal canal. The deep inguinal nodes receive lymph flow from the glans of the clitoris.

NERVES

Few procedures require a complete dissection of nerves in gynecologic oncology, although this is advocated by some investigators. Larger nerves are sometimes used as landmarks during surgical dissections; for instance, the obturator nerve may serve as the near-to-inferior border of pelvic lymphadenectomy (obturator fossa) and the phrenic nerve as the posterior border of the scalene node dissection. Smaller nerves, such as the genital femoral nerve, may be transected during the removal of suspicious lymph nodes. To avoid injury, at the very beginning of a surgical procedure, the anatomy of the nervous system should be kept in mind when positioning the patient. For example, because laparoscopy requires the surgeon to be further cephalad than during the same procedure done by laparotomy, both arms should be tucked at the patient's side to avoid excessive superior traction on the brachial plexus. During vaginal procedures, resting an arm on the medial anterior thigh may compress the femoral nerve. This nerve may also be injured by an abdominal retractor placed too deeply over the psoas muscle. Further, the sympathetic trunk and hypogastric nerves are responsible for sympathetic innervation of the pelvis. Injury to the sympathetic trunk can cause ipsilateral vasodilatation postoperatively (hyperthermia in the lower extremity). The splanchnic nerves carry parasympathetic innervation of the pelvis and control micturition and defecation. The nerves of the pelvis and abdomen show a wide spectrum of variation in topographic anatomy. Nevertheless, the general course and function of many nerves must be known in order to avoid their injury and minimize surgical complications (Figures 4.8 and 4.9).

Components of both the somatic and autonomic (visceral) nervous systems are associated with the posterior abdominal wall. These include the subcostal nerves (anterior rami of T_{12}), the lumbar spinal nerves (L_1–L_5), and the lumbar plexus of nerves (anterior rami of L_1–L_4).

The anterior and posterior rami of the lumbar spinal nerves contain sensory and motor fibers. L_1 and L_2 (occasionally L_3) give rise to white rami communicantes conveying presynaptic sympathetic fibers to the lumbar sympathetic trunks. Postsynaptic fibers leave the trunks within gray rami communicantes and enter spinal nerves. The medial aspect of the lumbar sympathetic trunk also gives rise to lumbar splanchnic nerves carrying presynaptic fibers responsible for sympathetic innervation of the pelvic viscera.

The three largest branches of the lumbar plexus are consistent and can be used as surgical landmarks: the femoral nerve (L_2–L_4), the obturator nerve (L_2–L_4), and the lumbosacral trunk (L_4–L_5). The femoral nerve originates at the lateral border of psoas major, and courses deep to the inguinal ligament into the anterior compartment of the thigh. The obturator nerve originates at the medial border of the psoas major and enters the lesser pelvis, then passes through the obturator foramen and into the adductor. Finally, the lumbosacral trunk passes over the ala of the sacrum and into the pelvis, participating in the formation of the lumbosacral plexus with the anterior rami of the S_1–S_4.

Lesser nerves of the lumbar plexus are the ilioinguinal and iliohypogastric nerves (L_1), the genitofemoral nerve (L_1–L_2), the lateral femoral cutaneous nerve (L_2–L_3), and the accessory obturator nerve (L_3–L_4; present about 10% of the time).

The sacral plexus (S_1–S_5) also gives rise to nerves coursing through the pelvis that can be affected by cancers and surgical procedures. The main nerves are the sciatic (L_4–S_3), pudendal (S_3–S_4), and the superior (L_4–S_1) and inferior (L_5–S_2) gluteal nerves. Lesser nerves are the nerves to quadratus femoris (L_4–S_1), pirformis (S_1–S_2) and levator ani and coccygeus (S_3–S_4); a posterior cutaneous nerve to the buttocks and superior posteromedial thigh (S_2–S_3), and the nerve to obturator internus (L_5–S_2). Pelvic splanchnic (S_2–S_4) nerves supply pelvic viscera via the inferior hypogastric and pelvic plexuses. The sciatic nerve is located laterally to the internal iliac artery where anterior rami converge on the surface of the piriformis. Usually, the sciatic nerve leaves the pelvis along the inferior border of piriformis. However, branches may pass above and/or below (or through) the piriformis, and then merge to form the sciatic nerve. The sciatic nerve can be compromised by inadequate positioning during surgery and by lateral pelvic wall metastases.

The pudendal nerve is the main nerve of the perineum and the main sensory nerve of the external genitalia. Throughout its course it is accompanied by the pudendal artery. It exits the pelvis through the greater sciatic foramen between piriformis and coccygeus, then hooks around the ischial spine and the sacrospinous ligament, re-entering the perineum through the lesser sciatic foramen.

The inferior one-quarter of the vagina has somatic innervation from the deep perineal nerve that conveys sympathetic and visceral fibers. In contrast, the superior three-quarters of the vagina are visceral with respect to innervation, and derived from the uterovaginal plexus, which comprises sympathetic, parasympathetic, and visceral afferent fibers. Surgically important, the uterine plexus courses a route paired with the uterine artery along the lateral wall of the uterus within the broad ligament and at the junction of the base of the broad ligament and the superior part of the transverse cervical ligament. Between the layers of the broad ligament, it communicates with the ovarian plexus.

The ovaries and fallopian tubes are innervated in part from the ovarian plexus and the uterine plexus. The ovarian plexus arises from the renal plexus and descends through the suspensory ligament of the ovary. Because the ovaries and tubes are intraperitoneal and superior to the pelvic pain line, afferent pain fibers ascend to cell bodies located in the T_{11}–L_2 spinal ganglia. Afferent reflex fibers course in a retrograde fashion along parasympathetic fibers through the uterine portion of the uterovaginal plexus, and pelvic splanchnic nerves to cell bodies in the $S_{2–4}$ spinal ganglia. Thus, pain secondary to cancer or postoperatively can be controlled in the pelvis by regional anesthetic blockade of the dorsal nerve roots of $T_{10–12}$ to the uterus tubes and ovary, and $S_{2–4}$ to the remaining genital structures.

MUSCLES

The muscles of the abdominal cavity are sometimes involved in either the disease process or surgical procedures in gynecologic oncology. Many of the cutaneous landmarks used in planning gynecologic surgery comprise borders of superficial muscles (Figures 4.11 and 4.12). Muscles are the primary focus of reconstructive procedures (discussed in this book), and can be used as flaps to cover gaping defects created at the time of radical or ultraradical surgery (Possover et al. 1998). One favorite technique involves using the gracilis muscle to close a pelvic defect and create an adequate vagina. These techniques include grafting procedures, realignment of standard incisions, the use of vascular pedicle flaps, and organ substitution. However, most often muscles are structures to be retracted or transected. Nevertheless, they are helpful in identifying related anatomical structures, and therefore surgeons should be familiar with them.

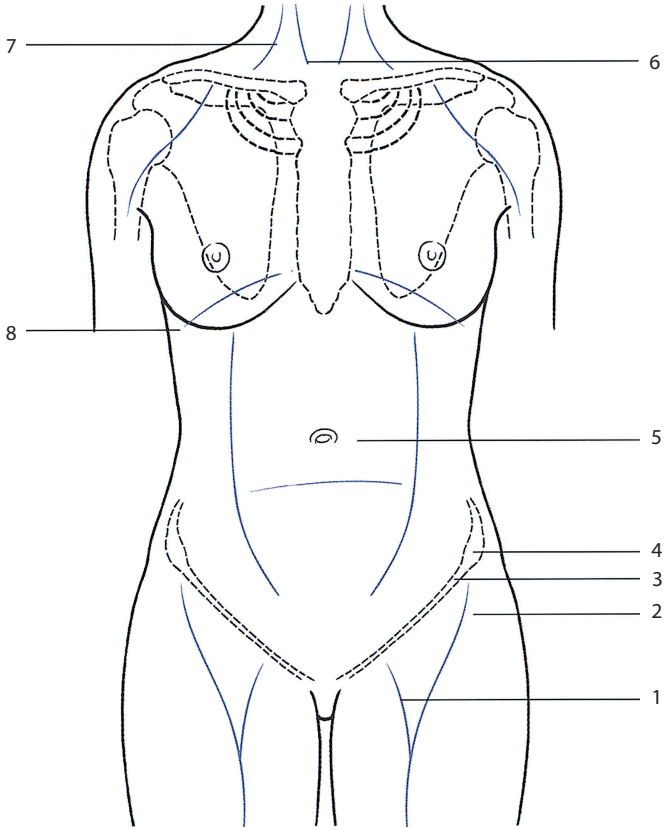

Figure 4.11 1: Adductor longus groove; 2: sartorius muscle groove; 3: inguinal ligament; 4: anterior superior iliac spine; 5: level of L4/L5 vertebral bodies; 6: sternal head; 7: clavicular head and sternocleidomastoid muscle (6 and 7); 8: seventh rib.

One useful relationship is that between the rectus abdominis muscle and the epigastric vessels. When performing laparoscopy (Soper et al. 1989), it is best to place the lateral trocars completely laterally to these muscles to prevent injury of epigastric vessels. This procedure facilitates surgery also by keeping the surgical instruments as far apart as possible. It is this relationship with the epigastrics that makes the rectus abdominis muscle an ideal vascular pedicle flap for reconstructive procedures. The gracilis muscle is also a suitable pedicle flap, but because it is more variable, the rectus is preferred for perineal reconstruction.

Muscles also serve as borders for lymph node dissections. For example, the middle of the psoas muscle marks the lateral extent of the pelvic lymphadenectomy, and the obturator internus muscle does the same for the obturator space lymphadenectomy. The muscles of the proximal lower extremity are similarly used as landmarks in inguinofemoral dissection. During a scalene node biopsy, dissection is carried to the surface of the anterior scalene muscle between the sternocleidomastoid and the trapezius muscles (Figures 4.11 and 4.12).

BONES AND CUTANEOUS LANDMARKS

Experienced surgical oncologists recognize the value of boney and cutaneous landmarks in planning successful gynecologic oncology procedures (Figures 4.11 and 4.12). For example, gaining central venous access always begins with determination of the location of the distal third of the clavicle or the heads of the sternocleidomastoid muscle. Vascular access may also be achieved through a cephalic vein cut-down. This vein is identified by the cutaneous border of the deltoid and pectoralis major muscles (deltoid-pectoral triangle).

These same landmarks are also useful in initiating a scalene node biopsy. An inguinal node dissection may be performed through different incisions provided that the operator recognizes the relationship of the nodes to the inguinal ligament. Tube thoracotomy and thoracocentesis require recognition of the location of the inferior scapula at the seventh and eighth rib. Finally, although the patient's soft tissue dimensions are important, the truly limiting factor for most is the bony confine of the operative field. For instance, a large patient may have a wide and shallow pelvis, making the patient an acceptable candidate for a radical hysterectomy. This may be determined before the incision by noting the distance between the anterior iliac crests in relation to the distance from the crest to the ischial tubercle. Similarly, for vaginal procedures, emphasis should be placed on the distance between the ischial tubercles and the angle of the pubic arch. The best way to assess the patient preoperatively is by recognizing the significance of the bony and cutaneous landmarks of the operative field.

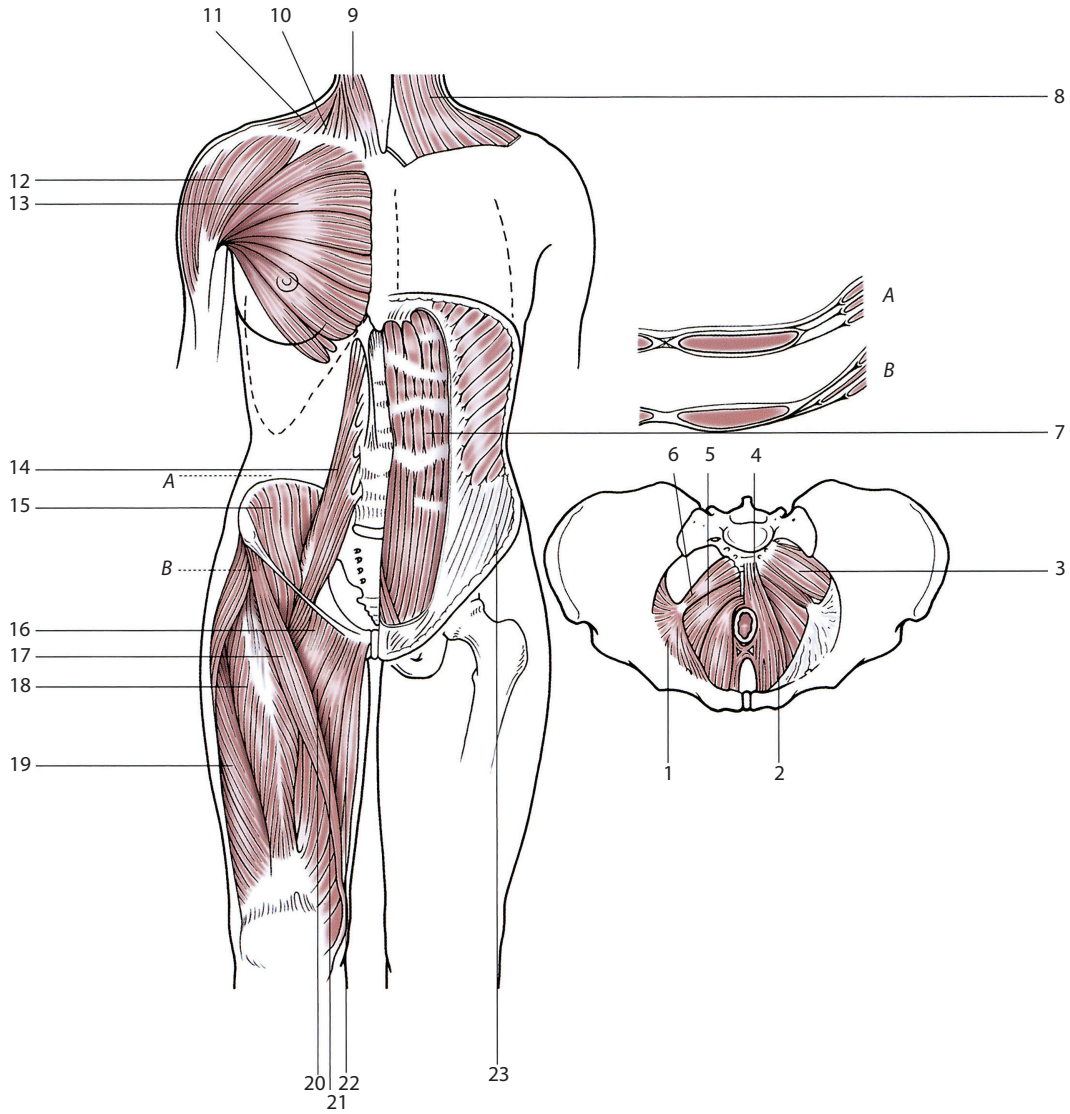

Figure 4.12 A: Transverse level, umbilicus; B: transverse level, arcuate line; 1: obturator internus; 2: puborectalis; 3: piriformis; 4: pubococcygeal muscle; 5: iliococcygeal muscle; 6: coccygeus; 7: rectus abdominis; 8: platysmus; 9: sternocleidomastoid muscle; 10: anterior scalene muscle; 11: trapezius; 12: deltoid; 13: pectoralis major; 14: psoas; 15: iliacus; 16: pectineus; 17: sartorius; 18: rectus femoris; 19: adductor brevis; 20: adductor longus; 21: vastus lateralis; 22: gracilis; 23: anterior superior iliac spine.

REFERENCES

Barlin J, Ueda S, Bristow RE. 2009. Cytoreductive surgery for advanced and recurrent endometrial cancer: A review of the literature. *Women's Health (Lond)* **5**(4):403–11.

Bristow RE, Tomacruz RS, Armstrong DK, et al. 2002. Survival effect of maximal cytoreductive surgery for advanced ovarian carcinoma during the platinum era: A meta-analysis. *J Clin Oncol* **20**(5):1248–59.

Burghardt E, Girardi F, Lahoussen M, et al. 1991. Patterns of pelvic and paraaortic lymph node involvement in ovarian cancer. *Gynecol Oncol* **40**(2):103–6.

Jemal A, Siegel R, Ward E, et al. 2008. Cancer Statistics, 2008. *CA Cancer J Clin* **58**(2):71–86.

Le O. 2013. Patterns of peritoneal spread of tumor in the abdomen and pelvis. *World J Radiol* **28**(3):106–12.

Lichtenegger W, Sehouli J, Buchmann E, et al. 1998. Operative results after primary and secondary debulking operations in advanced ovarian cancer (AOC). *J Obstet Gynaecol Res* **24**(6):447–51.

Moore KL, Dalley AF, Agur AMR. 2014. *Clinically Oriented Anatomy*. 7th ed. Lippincott Williams & Wilkins.

Nguyen HN, Averette HE, Hoskins W, et al. 1993. National Survey of Ovarian Carcinoma: V. The impact of physician's specialty on patient's survival. *Cancer* **73**:3663–70.

Possover M, Krause N, Drahonovsky J. 1998. Left-sided suprarenal retrocrural para-aortic lymphadenectomy in advanced cervical cancer by laparoscopy. *Gynecol Oncol* **71**(2):219–22.

Soper JT, Larson D, Hunter VJ. 1989. Short gracilis myocutaneous flaps for vulvovaginal reconstruction after radical pelvic surgery. *Obstet Gynecol* **74**:823.

5 Cross-sectional and molecular imaging

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INTRODUCTION

Pelvic imaging has seen a revolution in the recent times with increasing availability and utilization at almost all levels from initial assessment of tumors to its role in management and disease response evaluation. Various classification systems are in use for staging gynecological malignancies but the International Federation of Gynecology and Obstetrics (FIGO) is currently widely used in this regard. Although FIGO does not take into account the role of cross-sectional imaging for staging of gynecological malignancies, computed tomography (CT) and magnetic resonance imaging (MRI) have become the mainstay for assessment and staging in developed countries; however FIGO manual and surgicopathological staging is vital for full international comparison of incidence and results of treatment. The role of imaging is valuable in the initial assessment of indeterminate adnexal masses and endometrial assessment but not in diagnosing cervical and endometrial cancers. These tumors are diagnosed by clinical examination supplemented by examination under anesthesia (EUA), biopsy, and hysteroscopy. The gold standard for staging of endometrial cancer remains histopathological. CT and MRI also have a vital role in radiotherapy treatment planning. Following treatment, cross-sectional imaging plays an important role in assessing response to treatment and also for evaluating for any recurrence. 2-(F-18) Fluor-2-deoxy-D-glucose positron emission tomography (18 FGD PET) is also now part of the mainstay of imaging when it comes to staging locally advanced cervical cancer, disease recurrence, and prior to pelvic exenteration. It also has an increasing role in radiotherapy planning and response assessment.

CARCINOMA OF THE ENDOMETRIUM

Endometrial cancer is one of the commonest cancers of the female genital tract with estimates of almost 54,870 new cases in 2015 in the United States and 10,170 deaths, as per the figures released by the American Cancer Society (Anon 2015b). This is a disease of elderly females, with 95% of endometrial cancers occurring in women aged 40 and above. The cancer is also related to unopposed estrogen exposure such as in early menarche, late menopause, infertility, exposure to tamoxifen (an anti-breast cancer drug), and hormone replacement therapy. Obesity markedly increases the risk of developing endometrial cancer. The disease predominantly affects postmenopausal women who present with vaginal bleeding. Because of this, most of the women are diagnosed early, with 75% presenting with disease confined to the endometrium. The majority of endometrial tumors arise from the glandular epithelium and hence are adenocarcinomas of the endometrioid type (75%). The other less common type of tumors are serous papillary, adenosquamous, and the clear cell type, all of which have a worse prognosis. Sarcomas, including the mixed malignant Mullerian tumors (MMMT) and leiomyosarcomas, are also relatively rare. The incidence of uterine body sarcomas is 8%.

The diagnosis of the endometrial cancers is by hysteroscopy and curettage with histological confirmation. Carcinoma of the endometrium is staged using either the tumor, node, metastasis (TNM) classification or the FIGO staging system, which is surgicopathological (Table 5.1).

It is important to add here that the FIGO staging system has been revised, and the new staging system has been changed for Stage I disease to Stage IA where the endometrial tumor either does not invade or invades less than half of the depth of the myometrium (Pecorelli 2009). Previously, Stage IA was disease confined to the endometrium with no myometrial invasion. Stage IB now replaces the previous IC disease in which the tumor invades equal to or more than half of the depth of the myometrium. Similarly, cervical glandular epithelium involvement now is staged as Stage I instead of Stage IIA as per the old staging system.

Role of Imaging

Imaging has a role in early detection of abnormality of the endometrium, which may warrant further assessment by hysteroscopy and dilatation and curettage. This may be in the form of abnormal thickening of the endometrial lining on ultrasound, which would then be assessed by a hysteroscopy, and once the diagnosis of endometrial cancer is made on biopsy, imaging evaluation by MRI would be required. Imaging would also be able to assess for advanced disease, which might affect the choice of treatment or the surgical approach. If it is demonstrated on imaging that the disease is advanced, with peritoneal and nodal disease, then adjuvant therapy may be indicated. Some patients may require lymph node sampling or lymphadenectomy, depending on the protocol. These are patients with high-grade tumor, lymphovascular spread, cervical and deep myometrial involvement, and adenosquamous histology. Of these factors, imaging is able to assess the depth of myometrial invasion, nodal enlargement, and also invasion of the cervix.

Ultrasound

Ultrasound is usually the first imaging modality of choice for assessment of the endometrium in patients who present with vaginal bleeding. Transvaginal ultrasound is more accurate and allows precise measurement of the endometrial thickening compared to the transabdominal ultrasound. In addition, in obese patients and in patients with retroverted uterus, it may be difficult to assess the endometrial lining on a transabdominal ultrasound.

Normal endometrial thickness and appearances vary not only with the age of the patient but also with the phase of the

Table 5.1 Endometrial (Corpus Uteri) Cancer (FIGO Staging)

Stage	Description
I	**Confined to corpus uteri**
Ia	Confined to endometrium or invasion < half of the myometrium
Ib	Invasion ≥ half of the myometrium
II	**Tumor invades cervical stroma, but does not extend beyond uterus**
III	**Local or regional spread**
IIIa	Involvement of serosa of uterus or adnexae
IIIb	Vaginal and/or parametrial spread
IIIc	Metastases to pelvic and/or para-aortic lymph nodes
	IIIc1 – Positive pelvic lymph nodes
	IIIc2 – Positive para-aortic lymph nodes
IV	**Bladder, bowel, distant**
IVa	Invasion of bladder or bowel mucosa
IVb	Distant metastases, including intra-abdominal or inguinal lymph nodes

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menstrual cycle. In the early part of the menstrual cycle, the endometrium is visualized as a thin reflective line (Figure 5.1). During the proliferative phase, the endometrium is thickened and is seen as a triple line (Figure 5.2), and lastly, during the secretory phase, the endometrial lining is at its maximum thickness, with homogenously increased reflectivity and through transmission (Figure 5.3).

In postmenopausal women, the endometrium lining is thin and generally measures less than 4 mm unless the patient is on HRT or tamoxifen for breast cancer. If the patient is on sequential HRT and is asymptomatic, then the endometrial thickness can be up to 8 mm. Endometrial thickness between 5 and 8 mm will require a biopsy if the patient is symptomatic and presents with vaginal bleeding. Thickness greater than 8 mm would require a follow-up ultrasound in asymptomatic patients and a biopsy in symptomatic ones (Levine et al. 1995), although some authors would advise a biopsy in all women with a thickness greater than 5 mm and in all woman with persistent vaginal bleeding regardless of the thickness. Not to biopsy would be indefensible in a postmenopausal woman.

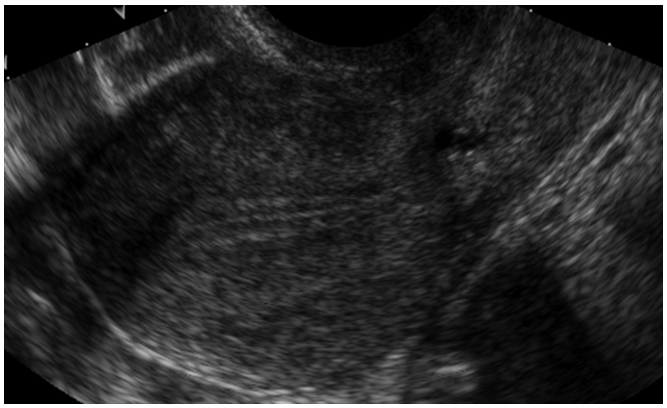

Figure 5.1 Transvaginal ultrasound during the early phase of the menstrual cycle shows a thin reflective endometrial lining.

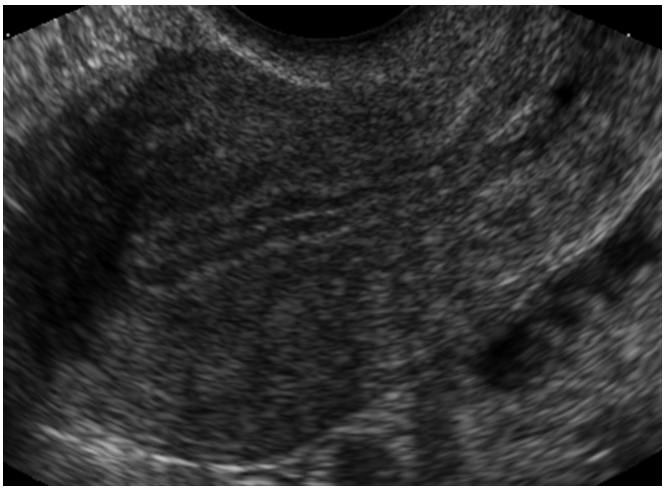

Figure 5.2 Transvaginal ultrasound during the proliferative phase of the menstrual cycle shows a triple line appearance of the endometrial echoes.

Figure 5.3 Transvaginal ultrasound during the secretory phase of the menstrual cycle shows thickened hyperreflective endometrial echoes.

Endometrial cancer is characterized by increased endometrial thickness often associated with heterogeneous reflectivity and irregular and ill-defined margins (Figure 5.4). However, there remains an overlap between endometrial cancer, polyps, and hyperplasia. Transvaginal ultrasound appears to have a sensitivity of about 94.3% for detecting endometrial cancer but has a low specificity of 52.4%. A recent meta-analysis has suggested sensitivity of 68% to 100% and specificity of 71% to 90% for assessing the depth of myometrial invasion (Epstein and Blomqvist 2014).

Computed Tomography

CT has a major role in assessing for distant spread when it comes to staging endometrial carcinoma. MRI, however, best stages the tumor locally. The staging investigation generally includes a contrast-enhanced scan of the chest, abdomen, and pelvis. Although contrast-enhanced CT can detect endometrial thickness and the primary tumor as a hypodense lesion, it is difficult to assess for the depth of myometrial invasion on