Chapter 16 Complications of Minimally Invasive Gynecologic Surgery

Paula C. Brady and Sarah L. Cohen

Definitions

Laparoscopy

Laparoscopy is a surgical approach in which the abdomen is insufflated with CO_2 and the procedure is completed using instruments inserted through small incisions in the abdomen (Fig. 16.1). Ports, usually 5–12 mm in diameter, are placed into the abdominal incisions to allow for insertion and removal of instruments, and the CO_2 gas source is connected to one of these ports to maintain abdominal insufflation. The surgery is visualized using a laparoscope, which is a telescopic lens 5–10 mm in diameter. At the conclusion of the surgery, the patient will have just a few small incisions on the abdomen. A patient who undergoes a total laparoscopic hysterectomy (meaning the cervix was removed with the uterus) will have an incision at the top of the

P.C. Brady, MD (⊠) Department of Obstetrics, Gynecology and Reproductive Biology, Brigham and Women's Hospital, Boston, MA, USA e-mail: Pbrady2@partners.org

S.L. Cohen, MD, MPH

Division of Minimally Invasive Gynecology, Department of Obstetrics, Gynecology and Reproductive Biology, Brigham and Women's Hospital, Boston, MA, USA e-mail: Scohen20@partners.org

© Springer International Publishing Switzerland 2016 P.C. Brady, *Handbook of Consult and Inpatient Gynecology*, DOI 10.1007/978-3-319-27724-0_16 419

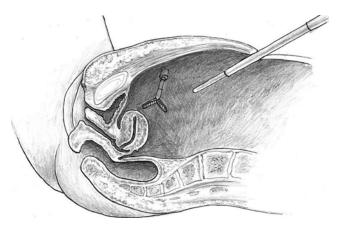


FIG. 16.1 Pelvic laparoscopy

vagina, called the **vaginal cuff**. If a patient has a supracervical hysterectomy (meaning the cervix is left in situ), a myomectomy (removal of fibroids), or removal of a large adnexal mass, one of the abdominal port sites may be enlarged (usually up to 3–5 cm) to accommodate the transabdominal removal of this tissue.

Robot-Assisted Laparoscopy

Robot-assisted laparoscopy involves similar core principles as conventional laparoscopy, with the assistance of a computerized system for enhanced visualization and manipulation of specialized laparoscopic instruments. The advantages of robotic surgery include three-dimensional visualization, articulated robotic arms with additional degrees of freedom, and improved ergonomics for the surgeon [1].

Hysteroscopy

Hysteroscopy is visualization of the endometrial cavity through distention of the uterus with fluid. Using a hysteroscope, fibroids, polyps, intrauterine septa, or adhesions can be removed. Distention media include electrolyte-poor fluids such as glycine 1.5 %, sorbitol 3 %, and mannitol 5 %—with risk of electrolyte abnormalities in the setting of excessive fluid absorption—and

isotonic fluids such as normal saline or lactated Ringer's solution [2]. Due to concerns of excessive intravascular absorption of the distention medium, a procedure should be stopped in the setting of fluid deficits of 1000–1500 mL of an electrolyte-poor solution or 2500 mL of an isotonic solution [2].

Differential Diagnosis by Primary Complaint

Fever

Superficial surgical site infection Vaginal cuff cellulitis Pelvic hematoma or abscess Endomyometritis Cystitis **Pvelonephritis** Clostridium difficile colitis Toxic shock syndrome Necrotizing fasciitis Septic pelvic thrombophlebitis Ovarian vein thrombosis Deep vein thrombosis (DVT) Pulmonary embolism (PE) Pneumonia Medication effect (drug fever) Urinary tract or bowel injury Retained foreign body Alcohol withdrawal Transfusion reaction

The differential diagnosis for fever varies with the interval from surgery. Fevers in the first 24 h after surgery are commonly noninfectious, due to inflammation or medication reactions. Infectious complications of surgery usually present 48 h or more after surgery. Pneumonia (particularly aspiration) and urinary tract infections may present as early as 2–3 days postoperatively, while the presentation of surgical site infections, vaginal cuff complications and pelvic abscesses commonly may be delayed by 5 or more days postoperatively. Bowel and urinary tract injuries typically present in the days following surgery, but may be delayed by 1–2 weeks. Thromboembolism may occur at any point.

Pain

Inadequate analgesic medication Superficial surgical site infection Endomyometritis Vaginal cuff cellulitis Pelvic hematoma or abscess Necrotizing fasciitis Cystitis **Pyelonephritis** Ovarian vein thrombosis Vaginal cuff dehiscence Uterine perforation Bowel injury Bowel obstruction Urinary tract injury Urinary retention Port site hernia or hematoma Persistent pneumoperitoneum Nerve entrapment Musculoskeletal pain

Nausea/Vomiting

Bowel obstruction or injury Ileus Port site herniation Urinary tract infection Urinary ascites (urinary tract injury) Medication effects (including anesthetics and narcotics) Nonsurgery related (e.g., viral gastroenteritis) *When You Get the Call* Ask for a full set of the most recent vital signs. If the patient's complaint is pain, consider requesting that pain medications are temporarily withheld, if possible, to allow for an accurate physical examination.

When You Arrive Review the patient's vital signs to assess for hypotension, tachycardia, or hypoxia. Clarify the date of the patient's surgery, as certain complications may be expected at specific intervals from surgery. Review the operative report, including the extent of dissection, complications, administration of perioperative antibiotics, and use of mechanical or chemical thromboprophylaxis. Also review for any implants-such as clips and mesh-or hemostatic agents, which may affect interpretation of imaging. Hemostatic agents, such as oxidized regenerated cellulose (Surgicel®, Ethicon, Somerville, NJ) and gelatin bioabsorbable sponges (Gelfoam®, Pfizer, New York, NY, and Surgifoam[®], Ethicon, Somerville, NJ), may appear as an abscess on imaging, in the absence of infection [3-5]. If hysteroscopy was performed, note the distention medium and fluid deficit.

History

Review with the patient when her primary symptoms began and any associated symptoms, including but not limited to fever, abdominal distention, nausea, vomiting, or diarrhea. If the patient is presenting with vaginal bleeding, discharge or abdominal pain, review her activities at the time of symptom onset, including heavy lifting or intercourse. Sudden onset of pain after bearing down or intercourse may raise concern for vaginal cuff dehiscence in patients who have had a total hysterectomy.

Review her full medical history, including any chronic diseases such as diabetes, a history of venous thromboembolism, whether she smokes, and menopausal status. Make note of any current medications, including anticoagulant therapy, and whether this medication was withheld surrounding surgery. Review her prior surgical history, as prior surgeries may increase the risk of adhesions and intraoperative injury to other organs.

Physical Examination

In the setting of fever, the patient should have a head-to-toe assessment, including examination of the respiratory tract for signs of pneumonia, the abdomen for signs of peritonitis and port site hernia or infection, and the lower extremities for evidence of thrombus.

For patients with pain and/or fever after a total hysterectomy, a bimanual exam should be performed to confirm that the vaginal cuff is intact and to assess for tenderness or a fullness of the vaginal cuff concerning for abscess or hematoma, as well as pelvic tenderness or pain. On sterile speculum exam, note any sources of bleeding and whether the vaginal cuff is intact; assess for erythema, induration, or vaginal discharge, and culture any purulent vaginal discharge.

Diagnosis

Fever

Postoperatively, a temperature of 100.4 °F ($38 \circ C$) on two occasions more than 4 h apart or a single temperature of 101 °F ($38.5 \circ C$) constitutes a fever [6]. Patients with significant complications, such as pelvic abscess or bowel injury, may present with septic physiology, which must be identified and addressed quickly [7]. The diagnostic criteria of sepsis are shown in Table 16.1; for further management of sepsis, please see Chap. 1., Acute Pelvic Pain [8, 9].

Sepsis	Severe sepsis
 Sepsis Suspected source plus 2 or more: 1. Temperature >38.3 °C (101 °F) or <36 °C (96.8 °F) 2. Heart rate >90 beats per minute 3. Tachypnea (>20 breaths/min) 4. WBC >12,000 µ/L or <4000 µ/L or normal with >10 % immature (band) forms 	 Sepsis plus one or more: Systolic blood pressure <90 mmHg or decrease from baseline by 40 mm Hg Elevated lactate (>1 mmol/L; >4 particularly concerning, sign of organ hypoperfusion) Acute lung injury: PaO₂/FIO₂ <250 (in the absence of pneumonia) or <200 (with pneumonia) Acute oliguria: <0.5 mL/kg/h despite fluid resuscitation Creatinine >2 mg/dL INR >1.5
	 Platelets <100,000/uL Bilirubin >2 mg/dL

TABLE 16.1 Clinical criteria of sepsis and severe sepsis

Criteria from Fischerova [8]; Dellinger et al. [9]

Risk factors for postoperative infection include advanced age, immunosuppression, diabetes, smoking, obesity, operative time greater than 3 h, use of a razor for hair removal at the surgical site, and lack of prophylactic antibiotics [10]. The wound classification also affects risk of infection, with the risk increases progressively along the classes up to 27 % or more with dirty wounds (Table 16.2) [11–14].

Laboratory testing should include a complete blood count with a differential and urinalysis. Patients with possible sepsis (including high fever and hemodynamic changes) should also have electrolytes, creatinine, liver function tests, coagulation studies (prothrombin time and activated partial thromboplastin time) and a lactate checked. In patients with a fever of

426 P.C. Brady and S.L. Cohen

TABLE 16.2 Surgical wound classification

Class I/clean: An uninfected operative wound in which no inflammation is encountered and the respiratory, alimentary, genital, or urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow nonpenetrating (blunt) trauma should be included in this category if they meet the criteria

Class II/clean-contaminated: An operative wound in which the respiratory, alimentary, genital, or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered

Class III/contaminated: Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (e.g., open cardiac massage) or gross spillage from the gastrointestinal tract and incisions in which acute, nonpurulent inflammation is encountered are included in this category

Class IV/dirty-infected: Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation

Adapted from Mangram et al. [12], with permission from Elsevier and the Association for Professionals in Infection Control and Epidemiology, Inc.

38.5 °C (101 °F) or more, obtain blood cultures in addition to a urine culture, cultures of any purulent wound exudate, or a vaginal culture. Obtain imaging as indicated by the history and physical exam, including chest radiograph, pelvic ultrasound, or abdominal CT scan. Abdominal CT scans are particularly helpful in identifying pelvic hematomas or abscesses but can also identify other sources of fever, including bowel injuries and urinary tract injuries. Oral and intravenous CT contrast should be given whenever possible.

Pain

Patients presenting with significant pain postoperatively should have a complete blood count to assess for leukocytosis and anemia. A basic metabolic panel may be obtained to assess for electrolyte derangements and creatinine elevation, particularly in the setting of nausea/vomiting. A urinalysis may reveal urinary tract infection. Abdominal imaging should be obtained, targeted to the suspected source of pain. If the differential diagnosis remains broad, an abdominal CT scan with oral and IV contrast can be helpful, revealing urinary or gastrointestinal tract injuries or obstructions, pelvic fluid collections (hematoma, abscess, or urinary ascites), or thromboses in pelvic vessels.

Management

The most common complications of laparoscopy are shown in Table 16.3, presented in the order they are discussed in the text.

Infection

Superficial Surgical Site Infection

Laparoscopy is associated with a reduced risk of surgical site infection as compared to laparotomy (0–2 % versus 6.5 %, respectively) [15]. Worsening pain at an incision, accompanied by warmth, erythema, or induration, is suggestive of a surgical site infection [7, 11]. If examination or imaging suggests the presence of a fluid collection under the skin—seroma, hematoma, or abscess—or purulent fluid is expressed from the incision, a wound should be opened, irrigated, and managed with wet-to-dry dressings [7, 16]. The fascia should be probed with a sterile cotton swab to ensure no dehiscence has occurred.

Complication	Incidence
Surgical site infection	1 %
Vaginal cuff cellulitis	1.5 %
Pelvic abscess	0.8 %
Urinary tract infection	13 %
Pneumonia	1.6 %
Venous thromboembolism	1 %
Ureteral injury	0.8–1.7 %
Bladder injury	1.6–2.9 %
Urinary retention	7–15 %
Bowel injury	0.03-0.39 %
Small bowel obstruction	0.53 %
Ileus	0.2 %
Vaginal cuff dehiscence	0.31 %
Port site herniation	3.1 % (12 mm ports), 0.57 % (10 mm ports)
Port site hematoma	0.5 %
Nerve injury	2 %
Subcutaneous emphysema	2.3 %

TABLE 16.3 Complications of gynecologic laparoscopic surgery

References for these values are provided in the text. The high end of each range reflects complication rates of laparoscopic hysterectomy, while the low end represents other gynecologic laparoscopy

Opening an infected wound can be sufficient management; however, if surrounding erythema is observed, or fever and/or leukocytosis are documented, antibiotics should be given. For mild symptoms, cephalexin (500 milligrams (mg) PO every 6 h) or trimethoprim-sulfamethoxazole double strength (160– 800 mg PO every 12 h) can be used [17]. The latter provides coverage for methicillin-resistant *Staphylococcus aureus*, as does clindamycin (300–450 mg PO every 6 h) [18].

Vaginal Cuff Cellulitis

Vaginal cuff cellulitis occurs following 1.5 % of hysterectomies [19]. Patients with untreated preoperative vaginal infections, including bacterial vaginosis and *Trichomonas vaginalis*, are at increased risk of developing postoperative cuff cellulitis [20]. Patients usually present 5–10 days after surgery with fever, pelvic or low back pain, the feeling of pelvic fullness, and/or vaginal discharge [6]. Patients will have tenderness localized to the vaginal cuff, which may be erythematous and indurated or have purulent discharge [21]. Any discharge should be cultured in order to direct antibiotic therapy [22].

Hemodynamically stable patients with mild symptoms and no evidence of cuff abscess may be treated with oral antibiotics, including amoxicillin–clavulanate (875–125 mg PO every 12 h) alone, or metronidazole (500 mg PO every 8 h) with either a fluoroquinolone such as ciprofloxacin (500 mg PO every 12 h) or trimethoprim-sulfamethoxazole double strength (160–800 mg PO every 12 h) [21–23]. If a patient's symptoms do not improve in 24–48 h, assessment should be initiated for other sources of infection, including imaging to assess for pelvic abscesses.

In patients with any hemodynamic derangements, severe pain, inability to tolerate oral antibiotics, or evidence of pelvic abscess, intravenous antibiotics should be initiated. If an abscess is present, consideration of surgical drainage should be undertaken per discussion below. Options for parenteral antibiotics include penicillins with beta-lactamase inhibitors. such as ampicillin-sulbactam (3 g IV every 6 h) or piperacillintazobactam (3375 g IV every 6 h), or a later-generation cephalosporin such as cefotetan (2 g IV every 12 h) [23]. If a patient does not improve, or is acutely ill, broader-spectrum antibiotics should be started, such as clindamycin (900 mg IV every 8 h) plus ampicillin (2 g IV every 6 h) plus gentamicin (2 mg per kilogram (kg) IV, then 1.5 mg/kg every 8 h or 5 mg/ kg ideal body weight every 24 h). Metronidazole (500 mg IV every 8 h) is an alternative to clindamycin in this regimen. An alternative regimen is levofloxacin (500 mg IV every 24 h) and metronidazole (500 mg IV every 8 h).

Pelvic Abscess

Pelvic abscesses, including tubo-ovarian abscesses, occur in 0.8 % of patients who have undergone gynecologic surgery [24]. Patients present 10–14 days after surgery with fever, pelvic pain or fullness [6, 16]. Infections are typically polymicrobial and often include anaerobes [6]. Pelvic fluid collections may be imaged using ultrasonography, CT, or MRI. The presence of internal gas bubbles and a capsule or ringenhancing lesion are suggestive of abscess [25]. Of note, some degree of fluid or even hematoma in the pelvis after hysterectomy is commonly detected on imaging; this alone does not indicate that a complication has occurred [7]. Additionally, if a hemostatic agent was utilized during the initial surgical procedure-such as oxidized regenerated cellulose (Surgicel®, Ethicon, Somerville, NJ) and gelatin bioabsorbable sponges (Gelfoam®, Pfizer, New York, NY, and Surgifoam®, Ethicon, Somerville, NJ)-it may appear on postoperative imaging as a complex fluid collection located in the hemostatic bed.

Patients with fever and a pelvic abscess by imaging should be treated with IV broad-spectrum antibiotics until afebrile for 24-48 h. Extrapolating from studies of tubo-ovarian abscesses, 8 cm is the upper limit of abscess size that may be treated with IV antibiotics without drainage, although many clinicians would pursue drainage at a smaller abscess size or when initial antibiotic therapy fails to produce clinical improvement [26]. Similar to severe vaginal cuff cellulitis, presumptive regimens include clindamycin (900 mg IV every 8 h) or metronidazole (500 mg IV every 8 h) plus gentamicin (2 mg/kg IV, then 1.5 mg/kg every 8 h or 5 mg/kg ideal body weight every 24 h), with or without ampicillin (2 g IV every 6 h) [21, 23]. Other broad-spectrum regimens include (1) piperacillin-tazobactam (3375 g IV every 6 h), (2) ceftriaxone (2 g IV every 24 h) plus metronidazole (500 mg IV every 8 h) or clindamycin (900 mg IV every 8 h), or (3) a carbapenem, such as meropenem (1 g IV every 8 h) [21, 27].

If patients do not initially improve, abscess drainage should be pursued. Persistent fevers and leukocytosis, worsening pain, and increase in abscess size by imaging constitute failure of IV antibiotics alone, often due to poor perfusion of the abscess, preventing adequate antibiotic penetration [21]. Development of septic physiology is also an indication for intervention, either surgical or by interventional radiology. Patients with ruptured abscesses may develop septic shock and require emergent surgical intervention. Drainage of pelvic abscesses in sufficiently stable patients is most commonly performed percutaneously (transabdominally or transgluteally) by interventional radiology with CT guidance. Abscesses in the posterior cul-de-sac could potentially be accessed transvaginally. Alternatively, surgical drainage and washout of the abscess can be pursued; surgical treatment of a pelvic abscess can be a complex and morbid procedure, potentially requiring removal of involved gynecologic organs [23].

After the patient is afebrile for 24–48 h, she should be transitioned to oral antibiotics, for 7–10 days as dictated by wound culture results. Oral options include metronidazole (500 mg every 8 h) and trimethoprim-sulfamethoxazole (160–800 mg every 12 h), or amoxicillin–clavulanate (875–125 mg every 12 h alone) [21].

Endomyometritis

Endomyometritis is a polymicrobial infection of the endometrial lining or uterine muscle that can occur after any uterine instrumentation, including hysteroscopy or curettage. Risk of endomyometritis is less than 1 % following hysteroscopy [28, 29]. Patients present with vague abdominal pain and irregular spotting or bleeding. On physical examination, patients have uterine tenderness and may have cervical motion tenderness. Options for treatment include (1) amoxicillin–clavulanate (875–125 mg PO every 12 h), or (2) amoxicillin (500 mg PO every 8 h) and metronidazole (500 mg PO every 8 h) [30]. Please see Chap. 12, Obstetrics in the Emergency Room, for discussion of group A streptococcus endomyometritis, a severe, potentially lethal infection.

Toxic Shock Syndrome (TSS)

A syndrome initially recognized most commonly in menstruating women with *Staphylococcus aureus* infections and associated with tampon use, TSS is now diagnosed most often in nonmenstruating women and can also be caused by *Streptococcus pyogenes* (group A streptococcus, or GAS) or *Clostridium sordellii* [31–33]. TSS can occur postoperatively, usually developing 2 days after surgery; it has been reported following hysterectomy, as well as after cone biopsy, IUD insertion, endometrial biopsy, and medical abortion [31, 34– 37]. TSS also occurs in postpartum women.

General diagnostic criteria of toxic shock syndrome include fever of at least 38.9 °C (102 °F), hypotension (defined as a systolic blood pressure of 90 mm Hg or less in patients over 16 years of age), erythroderma (a diffuse blanching rash resembling a sunburn), desquamation occurring 1-2 weeks after the initial rash, and dysfunction of three or more organ systems, including vomiting or diarrhea, severe myalgias or elevated creatine phosphokinase, elevated creatinine or pyuria without urinary tract infection, liver function testing twice the upper limit of normal, platelets of 100,000/µL or less, mucous membrane hyperemia, and mental status changes [38]. Cultures should be negative for Rocky Mountain spotted fever, leptospirosis, or measles, which can have overlapping features. Criteria specific to GAS TSS include cultures (blood, cerebrospinal fluid, tissue or surgical wound) positive for GAS and hypotension, with evidence of dysfunction in two organ systems, including creatinine greater than 2 mg per deciliter (dL) (or twice the baseline value), platelets of $100,000/\mu$ L or abnormal coagulation factors or fibrinogen, liver function testing twice the baseline or upper limit of normal, acute respiratory distress syndrome, erythroderma, or soft tissue necrosis [39]. Patients with toxic shock syndrome commonly also have 15,000 or more white blood cells per µL with 10 % or more bands present on differential.

Treatment is supportive, involving aggressive fluid resuscitations to address hypotension and correction of coagulopathy; patients usually require admission to an intensive care unit and coordination with infectious disease specialists. Any necrotizing wounds should be debrided, which may include hysterectomy if the uterus is the suspected source of infection [40].

Management also includes antibiotics; clindamycin (900 mg IV every 8 h) is indicated for all TSS as it inhibits toxin production [35]. Patients with GAS or clostridial TSS should also receive penicillin G (4 million units IV every 4 h). Treatment for TSS associated with methicillin-sensitive *Staphylococcus aureus* requires the addition of oxacillin (2 g IV every 4 h), while methicillin-resistant *Staphylococcus aureus* requires vancomycin (15–20 mg/kg IV every 8–12 h) or linezolid (600 mg IV every 12 h) [41, 42]. Intravenous immunoglobulin may be considered as well.

Necrotizing Fasciitis

This rare wound complication is often caused by either Streptococcus pyogenes (group A streptococcus) or infection with one or more anaerobic species. Risk factors include chronic disease such as diabetes or renal insufficiency, immunosuppression, malnutrition, obesity, and age over 60 years [43]. Initial physical findings may resemble cellulitis, but bullae and skin necrosis may develop, with rapid progression of erythema and edema. Patients may develop creptius (subcutaneous gas) [44]. On physical exam, patients will often have fever and exquisite pain out of proportion to their exam findings. A white blood cell count above 25,000/µL is suggestive of necrotizing fasciitis [43]. Patients may also have elevated glucose (above 180 mg/dL), creatinine (above 1.6 mg/ dL), and C-reaction protein (above 4 mg/L), as well as elevated lactate and creatine kinase (600 U/L or greater) [43, 44]. Imaging can be helpful in diagnosing necrotizing fasciitis; depending on the type of surgery performed and the patient's symptoms, ultrasound, CT, or MRI may reveal infectious collections, thickened fascia or subcutaneous air [45]. Definitive diagnosis can only be made following surgery by histopathologic analysis.

Once the diagnosis is suspected, general surgery and infectious disease consultations should be obtained. Expeditious surgical debridement of infected tissue is vital in the management of necrotizing fasciitis. Affected tissues may appear gray and necrotic and can be bluntly dissected with ease; purulence resembling "dishwater" may also be present [44]. Cultures should be obtained to tailor antibiotic treatment.

Broad-spectrum antibiotics should also be initiated as soon as necrotizing fasciitis is suspected; possible regimens include vancomycin (15-20 mg/kg IV every 8-12 h) or linezolid (600 mg IV every 12 h) plus a carbapenem (such as meropenem 1 g IV every 8 h) or piperacillin-tazobactam (3375 g IV every 6 h) [17, 42]. Once confirmed by wound or blood cultures, streptococcal or clostridial necrotizing fasciitis should be treated with penicillin (2-4 million units IV every 4-6 h) and clindamycin (900 mg IV every 8 h). Mixed bacterial infections can be managed with (1) cefotaxime (2 g IV every 6 h) plus metronidazole or clindamycin, (2) piperacillin-tazobactam plus vancomycin, or (3) monotherapy with a carbapenem in the doses described above [17]. During the patient's hospitalization, frequent physical exams and serial white blood cell counts should be performed to assess clinical progress. Serial debridements are often necessary.

Urinary Tract Infections

Cystitis, commonly called a urinary tract infection (UTI), occurs in up to 13 % of patients following gynecologic surgery [46]. Signs and symptoms include low-grade fever, frequency, urgency, and dysuria. On exam, patients may have suprapubic pain or tenderness of the anterior abdominal wall [7]. Urinalysis may show leukocyte esterase (a sign that leukocytes are present), nitrites (a sign that bacteria are present), and, on microscopic analysis, pyuria (10 leukocytes/mL) and/or bacteriuria [47]. Urine cultures should be obtained before starting any antibiotics if possible.

Antibiotic options include trimethoprim-sulfamethoxazole (160–800 mg PO every 12 h for 3 days), ciprofloxacin (250 mg PO every 12 h for 3 days), and nitrofurantoin monohydrate (100 mg PO every 12 h for 7 days); nitrofurantoin is bacterio-static and may be less effective against some infections [47]. Alternatives with less efficacy that are also acceptable include fosfomycin trometamol (3 g PO one dose), amoxicil-lin–clavulanate, cefaclor, or cefpodoxime proxetil for 3–7 days. Antibiotic selection should be adjusted according to urine culture results.

Patients with infection of the upper urologic tract, also called pyelonephritis, will present with fevers and flank pain in addition to the symptoms of a UTI. In patients with severe flank pain, renal calculus should be ruled out with imaging such as CT scan or renal ultrasound. Hemodynamically stable, nongravid patients with mild symptoms may be treated as outpatients with fluoroquinolones, either levofloxacin (750 mg PO once per day for 5 days) or ciprofloxacin (500 mg PO twice daily, or extended release 1000 mg PO once daily, for 7 days) [48, 49]. Alternatively, trimethoprim-sulfamethoxazole (160-800 mg PO every 12 h for 14 days) may be used. In patients with septic physiology and inability to tolerate oral antibiotics or in cases in which resistance to fluoroquinolones is suspected, inpatient management with parenteral antibiotics may be indicated. Options include ceftriaxone (1-2 g IV daily), or, for severe infection, piperacillin-tazobactam (3375 g IV every 6 h), or meropenem (500 mg IV every 8 h) [42, 49, 50]. Severely ill patients, or those whose symptoms have not improved in 48 h, may require CT scan to assess for abscess or other complicating factors.

Pneumonia

Pneumonia occurs postoperatively in approximately 1.6 % of patients following gynecologic surgery [51]. Patients may present with fever, dyspnea, and cough productive of sputum. Patients will have decreased breath sounds on physical exam

and may have hypoxia. Diagnosis is made by chest radiograph; sputum cultures can be sent to direct antibiotic selection.

Risk factors for multidrug-resistant pathogens include antibiotic exposure within the last 90 days, current hospitalization of 5 days or more, hospitalization of 2 days or more in the past 90 days, immunosuppression, home wound care, and residence in a nursing home or extended care facility [52]. Patients who do not meet these criteria can be treated with ceftriaxone (2 g IV daily), fluoroquinolones such as levofloxacin (750 mg IV or PO daily), ampicillin-sulbactam (3 g IV every 6 h), or ertapenem (1 g IV daily). Patients with risk factors for multidrug resistance require broad-spectrum antibiotics: an antipseudomonal cephalosporin (cefepime 1-2 g IV every 8–12 h or ceftazidime 2 g IV every 8 h), an antipseudomonal carbapenem (imipenem 500 mg IV every 6 h or meropenem 1 g every 8 h), or piperacillin-tazobactam (4.5 g every 6 h). In patients with suspected or confirmed methicillin-resistant Staphylococcus aureus, risk factors for MRSA or in areas with high incidence, add linezolid (600 mg IV every 12 h), or vancomycin (12 mg/kg IV every 12 h) [52]. The original American Thoracic Society (ATS) and Infectious Diseases Society of America (IDSA) guidelines propose double coverage with an aminoglycoside such as gentamicin or a fluoroquinolone such as levofloxacin or ciprofloxacin, which may add to the toxicity of the regimen but can be considered in patients with cultures showing gram-negative bacilli [53]. Consider discussion with the hospital's infectious disease staff, as local pathogen and resistance profiles vary. Antibiotics should be adjusted according to sputum and blood culture results.

Thrombotic

Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE)

The incidence of venous thromboembolism after laparoscopic hysterectomy is 1 % [54]. Various risk assessment scores exist to quantify a patient's risk of perioperative venous thromboembolism (VTE), such as the Caprini Risk Assessment Model [55, 56]. Low risk factors (conferring one point each in this scoring system) include age 41-60 years, minor surgery, body mass index greater than 25 kg/m², swollen legs, varicose veins, pregnancy or postpartum state, history of recurrent abortion, oral contraceptives or hormone replacement, sepsis within the last month, significant pulmonary disease (such as pneumonia in the last month), acute myocardial infarction, congestive heart failure, inflammatory bowel disease, and immobilization. Moderate risk factors (conferring two points each) include age 61-74 years, major open surgery or laparoscopic surgery (longer than 45 min), malignancy, and central venous access. Factors associated with highest risk, each conferring 3 points, include age greater than 75 years, a family history of VTE, thrombophilias (particularly factor V Leiden, prothrombin 20210A gene mutation, and antiphospholipid antibodies), and heparin-induced thrombocytopenia. Stroke and spinal cord injury in the past month each confer 5 points. Patients with no risk factors have a VTE risk of 0.5 %, while those with 1–2 points have a risk of 1.5 %; patients with 3-4 points have a VTE risk of 3 %, and those with 5 points or more have a risk of 6 % [57].

Patients with DVT may present with unilateral lower extremity pain, erythema, and edema. Tenderness in the distribution of deep veins of the leg, or a calf swollen to 3 cm greater in diameter than the contralateral side, 10 cm below the tibial tuberosity, is particularly concerning for DVT [58]. Duplex Doppler venous ultrasonography is the definitive method of diagnosis [7].

PE is a highly morbid surgical complication; in a review of venous thromboembolism after hysterectomy, the mortality rate of postoperative PE was 0.91 % [59]. Increased risk of death from PE is associated with age over 80 years, chronic cardiopulmonary disease, and arterial oxygen saturation less than 90 % [60]. Patients with pulmonary embolism may complain of dyspnea, chest pain, cough, or hemoptysis. Patients with PE may present with hypoxia, tachypnea, and tachycardia. D-dimer is artificially elevated in the postoperative setting and may be less useful as a triage test [61]. Patients' risk

TABLE 16.4 Wells criteria for prediction of pulmonary embolism (PE)

Clinical data	Points
Clinical symptoms concerning for deep vein thrombosis (DVT), particularly lower extremity swelling or pain	3
Clinical suspicion of PE as the leading diagnosis	3
Heart rate of greater than 100 beats per minute	1.5
Immobilization or surgery within the past month	1.5
History of a prior DVT or PE	1.5
Hemoptysis	1
Malignancy (treated currently or within the past 6 months, or palliative)	1
Wells et al. [62]	

for pulmonary embolism may be stratified according to Wells Criteria, shown in Table 16.4 [62].

A total of less than 2 points is low risk; in this category, 2 % of patients are diagnosed with PE. Moderate risk is defined as a score of 2–6 points, with a rate of PE of 18.8 %, while a score of greater than 6 points is considered high risk, with a rate of PE of 50 %. Patients with moderate to high risk of PE by Wells Criteria, or otherwise high risk or clinically concerning for PE, should undergo a chest CT with IV contrast, with a protocol specific to the detection of PE [7].

Patients with deep vein thrombosis or pulmonary embolism require anticoagulation. In patients with hemodynamic instability, shock, or severe hypoxemia attributed to pulmonary embolism, an unfractionated heparin IV infusion should be started, in a weight-based algorithm guided by activated partial thromboplastin time. An intensivist and/or vascular medicine specialist should be consulted immediately, for possible thrombolytic therapy and/or surgical or catheter embolectomy [63].

In hemodynamically stable patients, PE and DVT may be treated with unfractionated heparin IV, in a weight-based algorithm guided by activated partial thromboplastin time, or low molecular weight heparin, such as enoxaparin (1 mg/kg subcutaneously twice daily or 1.5 mg/kg subcutaneously once daily), which are equally effective [64]. Patients with renal dysfunction (creatinine clearance less than 30 ml/min) should be treated with unfractionated heparin. Hemodynamically stable patients without significant symptoms, chronic medical illness, or high risk for bleeding, and who are reliable for follow-up, can be treated as outpatients with subcutaneous low molecular weight heparin and eventually transitioned to warfarin if appropriate [65]. These patients should be managed along with a hematologist and anticoagulation clinic.

Septic Pelvic Thrombophlebitis (SPT)

Inflammation of the pelvic vessels may occur in the postpartum or postoperative setting, with thrombus and bacterial infection [66]. SPT is often a diagnosis of exclusion, in patients who are persistently febrile for 3 days despite broad-spectrum antibiotics and negative diagnostic workup for pelvic abscess [27, 67]. The pelvic vasculature can be imaged by CT or MRI; however, absence of a thrombus by imaging does not rule out the condition.

Patients should be treated with antibiotics; the duration of treatment is not standardized, but many are treated until 48 h afebrile. Treatment with ertapenem, a beta-lactam/beta-lactamase inhibitor such as piperacillin-tazobactam, or a combination of ampicillin, gentamicin, and clindamycin, has been described [67]. Though somewhat controversial, antico-agulation is commonly used in the treatment of SPT, with either therapeutic intravenous heparin or enoxaparin (1 mg/kg subcutaneously every 12 h) [67–69]. The duration of anti-coagulation is not clearly defined; in patients without documented thrombosis, anticoagulation may be stopped after 48 h if symptoms have improved [70].

Ovarian Vein Thrombosis

Ovarian vein thrombosis is similar to SPT in risk factors and presenting symptoms; the two can coexist, though ovarian vein thrombus may also be detected in isolation. A thrombus in the ovarian vein is a rare cause of pelvic pain and fever in women postoperatively, occurring most commonly in the right ovarian vein [71]. Patients commonly present with unilateral pain and may have fever; as with SPT, pain and fever refractory to optimal antibiotics should raise concern for this diagnosis. Ovarian vein thrombi are detected most commonly in postpartum women, with an estimated incidence of 0.15–0.18 %, but can also be found in women with recent pelvic surgery, malignancy, pelvic infection, or other hypercoagulable state [71].

CT scan is currently considered the diagnostic modality of choice, though the diagnosis can also be made by MRI or pelvic ultrasound with Doppler [72]. If untreated, ovarian vein thrombosis can result in pulmonary embolism or sepsis. No clear guidelines for management of ovarian vein thrombosis exist; 3–6 months of anticoagulation is often suggested in reviews, and use of antibiotics is controversial [72, 73].

Urinary Tract Injuries

Risk factors for injuries to the urinary system at the time of laparoscopy include malignancy, adhesions from endometriosis, pelvic inflammatory disease or prior surgery, presence of pelvic masses distorting the anatomy, prior pelvic radiation, or congenital anomalies of the urinary tract [74, 75]. Injury to the urinary system during hysteroscopy is very rare (0.1 %) but could occur at the time of uterine perforation, particularly with electrosurgical devices [2, 29].

Reported rates of urologic injuries during gynecologic surgery are highest during laparoscopic hysterectomies, as compared to vaginal or abdominal hysterectomies. Pelvic reconstructive surgeries are associated with higher risk, though injuries have also been reported during oophorectomy, lymphadenectomy, laparoscopic sterilization, and surgeries for endometriosis [46, 76–78]. Bladder injuries occur more commonly than ureteral injuries; in a large metaanalysis, bladder and ureteral injuries occurred in 1.6–2.9 % and 0.8–1.7 % of cases, respectively [79]. In this series, laparoscopic hysterectomies (with or without bilateral salpingooophorectomy) accounted for the upper end of each range, as compared to other gynecologic procedures. Intraoperative cystoscopy has been shown to aid in identification of these injuries at the time of surgery, but despite cystoscopy, 12–15 % of urinary tract injuries may be identified only postoperatively [80].

Ureteral Injury

During surgery, ureteral injuries occur most often at the pelvic brim or near the cardinal ligament [81]. Preoperative stent placement may be used to aid in intraoperative ureteral identification in cases of distorted anatomy [82]. The ureter may be impacted by crush injuries, ligation, transection, perforation, or thermal injury; these injuries can result in strictures, urinomas, or fistulas to the vagina or skin [81, 83]. Particularly in the case of thermal injury, ureteral damage may not be recognized at the time of initial surgery and may lead to delayed necrosis or progressive obstruction [74]. Ureteral injuries are most commonly recognized within 30 days of surgery [83].

Patients with ureteral injury will present with flank pain, fever, hematuria, abdominal distention, or ileus, particularly if urinary ascites is present [74, 81]. If only one ureter is injured, creatinine may only be transiently and mildly elevated. Women with unilateral ureteral obstruction have been shown to have a mean serum creatinine elevation of 0.8 mg/dL post-operatively [84]. Patients also often have a leukocytosis and elevated C-reactive protein, a marker of inflammation [77].

A renal ultrasound is a low-cost imaging modality without radiation exposure that will reveal hydronephrosis, absent ureteric jets, and peritoneal fluid in the presence of a ureteral injury [83]. It can be considered first-line imaging in patients with poor renal function, contrast allergy, or pregnancy. An abdominopelvic CT with IV contrast or a CT urogram may show a contrast medium leak, noncontiguous ureter, hydroureter, and abnormal ureteric enhancement [83]. The most invasive test is cystoscopy with a retrograde intravenous pyelogram, with the advantage that a ureteral injury can be stented concomitantly [74, 83].

If ureteral injury is suspected, urology should be consulted. Patients may require proximal diversion with percutaneous nephrostomy tubes, placement of a ureteral stent, or either immediate or interval operative repair [85, 86].

Bladder Injury

The bladder may be injured on entry to the abdomen in laparoscopic surgery with the Veress needle or trocars; more commonly, the bladder is injured by thermal damage, incision, or trauma during surgery [75]. The dome of the bladder is most commonly injured, particularly in patients who have a history of prior cesarean section; the incidence of bladder injury during laparoscopic hysterectomy rises to 21 % in patients with three prior cesarean sections [75, 87]. The presenting symptoms of bladder injury include abdominal pain, oliguria, hematuria, elevated creatinine, and urinary ascites causing ileus and leakage of urine from incisions, namely, the vaginal cuff in patients who underwent hysterectomy [75, 88, 89]. Fistula formation involving the bladder is a delayed complication of unrecognized bladder injury.

Bladder injuries are optimally diagnosed with cystoscopy to directly visualize any transmural sutures or disruptions. Retrograde or CT cystography is an alternative, involving retrograde instillation of contrast into the bladder followed by radiograph or CT, which may miss small defects [25]. An abdominopelvic CT with IV contrast may also show contrast extravasation [16].

A small (<2 cm) or puncture lesion of the extraperitoneal bladder may be managed with bladder rest and prolonged catheter drainage [89]. Larger full-thickness extraperitoneal bladder wall defects, and any intraperitoneal bladder wall injuries, are best repaired surgically. Full-thickness injury to the bladder

dome is generally repaired in two layers, with polydioxanone or Vicryl sutures, and a catheter should be left in place for at least 7 days [85]. Injury to the bladder trigone requires more extensive assessment and surgical expertise in order to assess injury to the ureters, and ureteral stents may be required [7].

Urinary Retention

Urinary retention is relatively common after gynecologic surgery and occurs following 7–15 % of hysterectomies and 4 % of general surgical procedures [90, 91]. Risk factors for urinary retention also include age over 50 years and urine volume of greater than 270 mL upon arrival to the postoperative recovery unit [92]. Postoperative urinary retention is associated with a higher risk of urinary tract infection; pain and medication effects can also contribute [93, 94]. Patients may report pain and may have tachycardia and suprapubic fullness on examination. For diagnosis, a bedside bladder scan may be performed, or the bladder may be catheterized. A bladder capacity of 600 mL without the urge to void and a postvoid residual of greater than 100–150 mL are diagnostic of postoperative voiding dysfunction [93, 94]. The patient may also report an urge without the ability to void.

For patients with urinary retention, Foley catheter replacement for 24–72 h or intermittent catheterization, ideally 4–5 times per day, are equally effective [91, 95]. Patients can be taught to self-catheterize as needed. Prophylactic antibiotics are not necessary for either indwelling catheter or intermittent self-catheterization.

Gastrointestinal Tract Injury

Bowel Injury

The incidence of bowel injuries during gynecologic laparoscopy ranges from 0.03 % for minor laparoscopies to 0.39 % for laparoscopic hysterectomy, with injury occurring most commonly to the small bowel, followed by the large bowel and stomach [46, 96–98]. Bowel injuries may occur on initial entry into the abdomen, with the Veress needle or trocars, or during adhesiolysis [7, 16]. Alternatively, thermal damage may be inflicted with energy devices or, less commonly, intestinal vascular supply may be compromised during dissection, leading to organ necrosis. Up to 41 % of injuries are not recognized at the time of surgery and patients often become symptomatic within 2 weeks of surgery [98]. Bowel injuries are exceedingly rare at the time of hysteroscopy but could occur with a uterine perforation, particularly if the uterus was perforated with a sharp instrument or with an activated energy device [29].

Patients with bowel injuries will commonly present with fever, nausea, vomiting, and abdominal distention. On examination, patients may be febrile, with hemodynamic changes suggestive of sepsis or septic shock (Table 16.1). Due to spillage of bowel contents, patients may develop peritoneal signs. A complete blood count and complete metabolic panel should be obtained to assess for leukocytosis and electrolyte or metabolic derangements. In acutely ill patients, a serum lactate should also be sent, which has high sensitivity for bowel ischemia; lactate may also be elevated in patients with bowel spillage or bowel obstruction [99]. An abdominal radiograph is not particularly helpful in this setting, as some degree of intra-abdominal free air is expected in the acute postoperative phase. Diagnosis is optimally made using abdominal and pelvic CT with oral contrast, which can reveal contrast extravasation [7, 25].

Patients with iatrogenic bowel injuries should be started on broad-spectrum antibiotics, primarily targeting anaerobes and gram-negative aerobes; one possible regimen is piperacillin-tazobactam (4.5 g IV every 8 h) [100]. Expeditious surgical exploration is most often performed by laparotomy, though laparoscopic exploration and repair have also been reported. Small injuries may be oversewn, though bowel resection may be required for areas of necrosis or thermal injury; bowel diversion, by ileostomy or colostomy, is performed in approximately 11 % of cases [101]. Bowel injury is associated with the highest mortality rate of all complications of gynecologic laparoscopy; mortality is reported in 1 in 31 patients with delayed diagnosis of bowel injury [98, 102].

Ileus and Bowel Obstruction

Diminished bowel function postoperatively can be attributed to ileus—a decrease in intestinal motor activity—or mechanical bowel obstruction. With both conditions, patients may present with nausea, vomiting, or abdominal pain or distention; patients often report an absence of flatus or bowel movements [103].

A complete blood count should be obtained, as leukocytosis may suggest the presence of a complication such as bowel injury or obstruction. In patients presenting with nausea and vomiting, a complete metabolic panel should be obtained to assess for electrolyte or metabolic derangements. Abdominal radiographs and abdominal CT scans with oral contrast can be used to clarify the diagnosis; abdominal CTs have superior sensitivity and specificity for the diagnoses of ileus or bowel obstruction and may reveal bowel or urinary tract injuries contributing to the patient's presentation [104, 105, 110].

In patients with **postoperative ileus** imaged by abdominal CT, oral contrast will pass through the entire digestive tract, and the colon will contain air and fluid [104]. Postoperative ileus is usually a self-limited condition, occurring after 0.2 % of laparoscopic hysterectomies, which resolves in 3-5 days with bowel rest, and decompression with nasogastric tubes is not recommended [106–108]. A postoperative ileus that is persistent despite bowel rest, intravenous hydration, and electrolyte repletion is concerning for bowel obstruction.

Small bowel obstruction occurs after 0.53 % of benign gynecologic surgeries [109]. Patients present with abdominal distention, nausea, and vomiting 2–8 days after surgery [110]. Risk factors for bowel obstruction postoperatively include intraoperative lysis of adhesions and/or concomitant bowel surgery, blood transfusion, and cystotomy [111]. On abdominal CT scan, a transition point may be identifiable, with proximally dilated small bowel and distally collapsed bowel, with no oral contrast beyond this point [104]. A demonstrative CT scan is shown in Chap. 18, Gynecologic Oncology. Initial management with nasogastric tube, antiemetics, and intravenous fluids is advised, though 13–50 % of patients with bowel obstructions after surgery for benign gynecologic disease fail medical management and require a second surgery for bowel obstruction due to adhesions, herniation, or injury [104, 110]. A "closed-loop" obstruction, or obstruction of a segment of bowel at both ends (sometimes by bowel torsion or incarceration), may appear as a "C" on CT scan with mesenteric vessels converging. This can quickly progress to ischemia and perforation, and usually requires urgent surgical management. For diagnosis and management of large bowel obstruction, please see Chap. 18: Gynecologic Oncology.

Vaginal Cuff Dehiscence

Vaginal cuff dehiscence occurs in 0.31 % of hysterectomies and is more common following laparoscopic hysterectomy (0.64 %) as compared to vaginal (0.13 %) or abdominal hysterectomy (0.2 %) [112]. Evisceration occurs in 35–67 % of cases [113, 114]. Risk factors for vaginal cuff dehiscence include postoperative intercourse, obesity, smoking, malnutrition, immunosuppression, prior pelvic radiation, diabetes, corticosteroid use, and menopausal status [115]. Patients can present with cuff dehiscence at any point after hysterectomy, but dehiscence is most common in the first weeks to months [114, 116]. Patients present with vaginal bleeding or discharge, sometimes occurring after intercourse or Valsalva; patients may also report a vaginal bulge due to bowel evisceration [7].

Vaginal cuff dehiscence, with or without evisceration, is diagnosed clinically. The vaginal cuff disruption is palpable on bimanual exam, and speculum exam reveals the dehiscence and/or evisceration; the cuff should also be carefully inspected for evidence of cellulitis or abscess [117]. If an evisceration is diagnosed, any protruding peritoneal contents should be wrapped with a moist towel while expeditiously proceeding to the operating room. A comprehensive metabolic panel and complete blood count should be obtained in all patients with suspected or confirmed vaginal cuff dehiscence, as leukocytosis may suggest peritonitis or bowel ischemia. A CT scan can be obtained in stable patients without evisceration, and with presentations suspicious for pelvic abscess or bowel injury [7].

Patients with vaginal dehiscence require prophylactic broad-spectrum antibiotics, as the peritoneal cavity is exposed to the vaginal flora, and surgical repair [7]. Vaginal cuff dehiscence without evisceration or evidence of peritonitis can be repaired vaginally; patients with abdominal tenderness, leukocytosis, concerning findings on CT scan, or evisceration are best served with an abdominal approach, by either laparoscopy or laparotomy, to allow for visual inspection for bowel injury or pelvic abscess [117, 118]. Regardless of approach, the vaginal cuff edges should be gently debrided and closed with a delayed absorbable suture such as 0-polydioxanone [117].

Abdominal Wall Complications

For diagnosis and management of fascial dehiscence, which is rare following minimally invasive gynecologic surgery, please see Chap. 18, Gynecologic Oncology.

Port Site Hernia

The risk of port site herniation is related to port diameter; the fascia of ports greater than 8 mm should be closed as risk of herniation is higher with larger ports [119, 120]. The incidence of port site hernia is 3.1 % at 12 mm ports and 0.57 % at 10 mm ports [121]. However, up to 12 % of port site herniations occur through 5 mm ports [122].

Patients present, on average, 9 days postoperatively, though they may present a month or more after surgery [120]. Port site herniations usually contain fat or small bowel, though omentum and large bowel may be involved. Patients may present with abdominal pain and symptoms of bowel

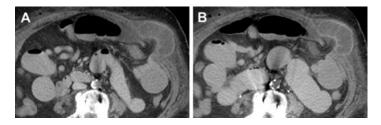


FIG. 16.2 Port site hernia. The herniated loop of bowel is persistently enhanced on the delayed phase image (**b**) as compared to the portal venous phase image (**a**). The patient required resection of a small segment of bowel secondary to ischemia (Reprinted from Santillan [138], with permission from Elsevier)

obstruction, including distention, nausea and vomiting, or port site protrusion [120, 123]. Diagnosis may be made by CT scan or ultrasound of the port site (Fig. 16.2) [120]. Repair may be by laparotomy, by laparoscopy, or through the port site, depending on clinician judgment and the acuity of the patient's presentation [119].

Abdominal Wall Hematoma

The incidence of abdominal wall bleeding is 0.5 %, usually due to injury of the inferior epigastric vessels [124]. Vascular injuries to the abdominal wall are often identified intraoperatively, but hematomas may develop within hours of arriving in the postoperative recovery unit, or after 2–3 days [125]. Signs of an abdominal wall hematoma include pain, ecchymosis, and bleeding from an incision. In patients who are still in the post-anesthesia recovery unit, outline the ecchymosis to allow for ongoing comparison. A rapidly expanding hematoma, acutely declining hemoglobin or hemodynamic instability should prompt aggressive resuscitation and operative management. In patients who are hemodynamically stable with stable hemoglobin, without signs of hematoma expansion or infection, management is supportive while awaiting spontaneous hematoma resolution.

Nerve Injuries

Fewer than 2 % of women develop neuropathies after laparoscopic surgery, and most resolve spontaneously or with addition of physical therapy [126]. Nerve injuries may be due to patient positioning leading to nerve compression or direct nerve injury during dissection. Nerve injuries can occur after hysteroscopy as well, though are less likely as the duration of the positioning is shorter with hysteroscopy than laparoscopy. Postoperative nerve injuries are usually diagnosed clinically, and motor neuropathies require the early involvement of physical therapists to ensure proper resolution [7].

Motor and Sensory Nerves

Femoral nerve injury leads to deficits in hip flexion and adduction and knee extension, with impaired sensation over the anterior and medial thigh and medial calf, and loss of the patellar reflex. This injury occurs in patients positioned with excessive hip flexion or external rotation or excessive stretch placed on the nerve; rarely, this injury may result from retroperitoneal hematoma, or, in open surgery, by deep, lateral retractor placement [7]. Injury to the obturator nerve leads to weakened adduction of the hip and loss of sensation over the medial thigh. Obturator nerve injury occurs most commonly during lymph node dissection or paravaginal defect repair [127]. Obturator nerve transection at the time of surgery should be immediately repaired with microsurgical technique. Injuries to the sciatic nerve, which innervates the posterior thigh and indirectly the lower leg and foot, are rare in gynecologic surgery; sciatic nerve injury may occur due to deep stitches placed in the setting of sudden hemorrhage or due to knee hyperextension with the hip flexed [127]. The peroneal **nerve** is a branch of the sciatic nerve specifically responsible for ankle flexion and sensation over the calf and dorsal foot and can be injured by excessive pressure on the lateral knee against stirrups in the dorsal lithotomy position [7]. Patients with an injury to the peroneal nerve may report foot drop.

Sensory Nerves

Injury of the lateral femoral cutaneous nerve results in numbness or pain over the lateral thigh, following excessive or prolonged hip flexion or external rotation [127]. Genitofemoral nerve injury results in paresthesias or numbness of the ipsilateral mons and inguinal area and is most often caused by direct injury at the time of external iliac lymphadenectomy or, in open surgery, by deep retractor placement over the psoas [7]. Iliohypogastric and ilioinguinal nerve injuries lead to neuropathic pain over the anterior abdominal wall, mons, and/or medial thigh [7]. These nerves are most commonly injured in gynecologic surgery at the lateral edges of Pfannenstiel incisions but can be injured at laparoscopy with placement of lateral ports or incorporation into fascial closures [7, 127]. Local nerve blocks can be both diagnostic and therapeutic for ilioinguinal or iliohypogastric nerve injury [128]. Local injections of anesthetics (2-10 mL of either 1 % lidocaine or 0.5 % bupivacaine), with or without corticosteroids (such as 40 mg of triamcinolone), can greatly improve cutaneous nerve pain in the abdominal wall [129, 130]. Lidoderm patches may also be helpful [128]. Persistent pain attributed to entrapment of these nerves may be relieved with surgical resection of the entrapped nerve [127, 128].

Complications of Pneumoperitoneum

Subcutaneous emphysema, or CO_2 trapped in the subcutaneous tissues, occurs in 2.3 % of patients following laparoscopy [131]. The subcutaneous tissues can be insufflated at the time of Veress needle placement, or via leakage from the intraperitoneal cavity or CO_2 flow through incompletely inserted trocars. Risk factors include age over 65 years, operative time over 200 min, and higher number of ports [131]. Subcutaneous emphysema results in palpable subcutaneous air (crepitus) and generally resolves in 1–2 days as the gas is absorbed and metabolized [132]. Massive subcutaneous

emphysema may result in hypercarbia as the CO₂ is reabsorbed and may rarely result in pneumothorax or pneumomediastinum [133]. Postoperative patients with significant subcutaneous emphysema and hypoxia, tachypnea, or tachycardia should have a chest radiograph to assess for pneumothorax or pneumomediastinum and an arterial blood gas to assess for hypercarbia [133].

Patients commonly report shoulder pain after laparoscopy, attributed to diaphragmatic irrigation by intraperitoneal CO_2 , stretch, or pressure due to the Trendelenburg position, and which resolves with time [119]. Persistent pneumoperitoneum, which may result in abdominal discomfort and distention and shoulder pain, is more common in thinner women and can persist for over 3 weeks [134].

Complications of Hysteroscopy

The most common complications of hysteroscopy are shown in Table 16.5.

Fluid Extravasation

Hysteroscopic fluid, instilled in the uterus to produce distention and allow for visualization, is generally chosen for its electrolyte content; normal saline, which contains electrolytes, is the preferred choice given its safety profile, as compared to an electrolyte-free fluid such as 1.5 % glycine [135].

TABLE 16.5 Complications of hysteroscopy		
Complication	Incidence	
Endomyometritis	0.85 %	
Fluid overload	0.2 %	
Uterine perforation	0.12 % (diagnostic), 0.76 % (operative)	

References for these values are provided in the text

Use of monopolar instruments requires electrolyte-free fluid, while bipolar instruments can be used in a solution with electrolytes [135].

Fluid overload, resulting from absorption of the hysteroscopic distention medium during hysteroscopy, is reported in 0.06–0.2 % of cases [29, 136]. Assessment of the patient's vital signs and respiratory function should be performed once 500 mL of a hypotonic solution has extravasated, and the procedure should be stopped once 1000 mL of fluid has been absorbed [135]. When isotonic solution is used for uterine distention, the procedure should be stopped once 2500 mL of solution has extravasated [135].

Complications of fluid extravasation are more likely at lower absorbed volumes in patients with medical comorbidities, particularly cardiac and pulmonary disease. Patients with fluid overload during hysteroscopy, particularly those with medical comorbidities, may develop complications such as pulmonary edema.

In patients with large absorbed volumes and/or significant comorbidities, a sodium level should be checked intraoperatively. Hyponatremia is more common with the use of electrolyte-poor, hypotonic solutions such as glycine; normal saline is isotonic to serum and less likely to cause hyponatremia [135]. Patients may report mild symptoms such as nausea with serum sodium declines of 5–10 millimole per liter (mmol/L), while levels below 120 mmol/L are severe and potentially life threatening [137]. Patients with symptomatic hyponatremia may present with confusion, nausea, and seizure; severe neurologic complications include cerebral edema and death [2].

Patients with fluid overload or mild hyponatremia can be managed with loop diuretics; hypertonic saline is also used in the treatment of hyponatremia. Patients with symptomatic hyponatremia, particularly those 48 h or more after surgery, may require admission to intensive care for correction, which, if corrected too quickly, risks further neurologic complications [137].

Uterine Perforation

Uterine perforation is estimated to occur in 0.12 % of diagnostic hysteroscopies and 0.76 % of operative hysteroscopies [136]. Adhesiolysis is associated with the highest complication rate, as compared to hysteroscopic myomectomy or polypectomy. Diagnostic laparoscopy for the detection of visceral injuries is required when perforation occurs with sharp or electrosurgical instruments. Hemostatic uterine perforations do not require repair. Uterine perforation occurring with blunt instrument (such as a dilator) is often managed expectantly but may rarely result in significant hemorrhage (0.03 %), requiring laparoscopy or laparotomy and, in extreme cases, hysterectomy [29, 136].

Postoperatively, patients with uterine perforation may report pain, bleeding, fever, or symptoms suggestive of urinary or bowel tract injuries. Diagnostic assessment should focus on the patient's symptoms, and management is dictated by the presence of hemorrhage, infection, and/or visceral injury.

References

- Herron DM, Marohn M, SAGES-MIRA Robotic Surgery Consensus Group. A consensus document on robotic surgery. Surg Endosc. 2008;22:313–25.
- 2. American College of Obstetricians and Gynecologists. Technology assessment No. 7: hysteroscopy. Obstet Gynecol. 2011;117:1486–91.
- 3. Behbehani S, Tulandi T. Oxidized regenerated cellulose imitating pelvic abscess. Obstet Gynecol. 2013;121:447–9.
- 4. Arnold AC, Sodickson A. Postoperative surgical mimicking abscesses following cholecystectomy and liver biopsy. Emerg Radiol. 2008;15:183–5.
- 5. Sandrasegaran K, Lall C, Rajesh A, Maglinte DT. Distinguishing gelatin bioabsorbable sponge and postoperative abdominal abscess on CT. AJR Am J Roentgenol. 2005;184:475–80.
- 6. Faro C, Faro S. Postoperative pelvic infections. Infect Dis Clin North Am. 2008;22:653–63.

- 7. Clarke-Pearson DL, Geller EJ. Complications of hysterectomy. Obstet Gynecol. 2013;121:654–73.
- Fischerova D. Urgent care in gynaecology: resuscitation and management of sepsis and acute blood loss. Best Pract Res Clin Obstet Gynaecol. 2009;23:679–90.
- Dellinger RP, Levy MM, Rhodes A, Annane D, Gerlach H, Opal SM, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2012. Crit Care Med. 2013;41:580–637.
- Beilman GJ, Dunn DL. Surgical infections. In: Brunicardi F, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE, editors. Schwartz's principles of surgery. 10th ed. New York: McGraw-Hill; 2014. http://accessmedicine.mhmedical.com.ezp-prod1.hul.harvard.edu/content.aspx?bookid=980 &Sectionid=59610847. Accessed 11 June 2015.
- Garner JS. CDC guideline for prevention of surgical wound infections, 1985. Supersedes guideline for prevention of surgical wound infections published in 1982. (Originally published in November 1985). Revised. Infect Control. 1986;7:193–200.
- Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for Prevention of Surgical Site Infection, 1999. Centers for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. Am J Infect Control. 1999;27:97–132.
- 13. Reichman DE, Greenberg JA. Reducing surgical site infections: a review. Rev Obstet Gynecol. 2009;2:212–21.
- 14. Ortega G, Rhee DS, Papandria DJ, Yang J, Ibrahim AM, Shore AD, et al. An evaluation of surgical site infections by wound classification system using the ACS-NSQIP. J Surg Res. 2012;174:33–8.
- Colling KP, Glover JK, Statz CA, Geller MA, Beilman GJ. Abdominal hysterectomy: reduced risk of surgical site infection associated with robotic and laparoscopic technique. Surg Infect (Larchmt). 2015;16:498–503.
- Hodges KR, Davis BR, Swaim LS. Prevention and management of hysterectomy complications. Clin Obstet Gynecol. 2014;57: 43–57.
- 17. Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJ, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis. 2014;59:e10–52.

- Pallin DJ, Binder WD, Allen MB, Lederman M, Parmar S, Filbin MR, et al. Clinical trial: comparative effectiveness of cephalexin plus trimethoprim-sulfamethoxazole versus cephalexin alone for treatment of uncomplicated cellulitis: a randomized controlled trial. Clin Infect Dis. 2013;56:1754–62.
- Jennings AJ, Spencer RJ, Medlin E, Rice LW, Uppal S. Predictors of 30 day readmission and impact of same-day discharge in laparoscopic hysterectomy. Am J Obstet Gynecol. 2015;213:344. e1–7.
- Soper DE, Bump RC, Hurt WG. Bacterial vaginosis and trichomoniasis vaginitis are risk factors for cuff cellulitis after abdominal hysterectomy. Am J Obstet Gynecol. 1990;163:1016–21.
- 21. Lachiewicz MP, Moulton LJ, Jaiyeoba O. Pelvic surgical site infections in gynecologic surgery. Infect Dis Obstet Gynecol. 2015;2015:614950.
- 22. Lazenby GB, Soper DE. Prevention, diagnosis, and treatment of gynecologic surgical site infections. Obstet Gynecol Clin North Am. 2010;37:379–86.
- 23. Soper DE. "Chapter 111: Infections of the female pelvis". In: Bennett JE, Dolin R, Blaser MJ, editors. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. Philadelphia: Elsevier Saunders; 2015. https://www-clinicalkeycom.ezp-prod1.hul.harvard.edu/#!/content/book/3-s2.0-B978145574801300326X. Accessed 11 June 2015.
- 24. Mahdi H, Goodrich S, Lockhart D, DeBernardo R, Moslemi-Kebria M. Predictors of surgical site infection in women undergoing hysterectomy for benign gynecologic disease: a multicenter analysis using the national surgical quality improvement program data. J Minim Invasive Gynecol. 2014;21:901–9.
- 25. Gjelsteen AC, Ching BH, Meyermann MW, Prager DA, Murphy TF, Berkey BD, et al. CT, MRI, PET, PET/CT, and ultrasound in the evaluation of obstetric and gynecologic patients. Surg Clin North Am. 2008;88:361–90.
- 26. Dewitt J, Reining A, Allsworth JE, Peipert JF. Tuboovarian abscesses: is size associated with duration of hospitalization & complications? Obstet Gynecol Int. 2010;2010:847041.
- 27. Jaiyeoba O. Postoperative infections in obstetrics and gynecology. Clin Obstet Gynecol. 2012;55:904–13.
- Agostini A, Cravello L, Shojai R, Ronda I, Roger V, Blanc B. Postoperative infection and surgical hysteroscopy. Fertil Steril. 2002;77:766–8.

- Aydeniz B, Gruber IV, Schauf B, Kurek R, Meyer A, Wallwiener D. A multicenter survey of complications associated with 21,676 operative hysteroscopies. Eur J Obstet Gynecol Reprod Biol. 2002;104:160–4.
- Meaney-Delman D, Bartlett LA, Gravett MG, Jamieson DJ. Oral and intramuscular treatment options for early postpartum endometritis in low-resource settings: a systematic review. Obstet Gynecol. 2015;125:789–800.
- 31. Descloux E, Perpoint T, Ferry T, Lina G, Bes M, Vandenesch F, et al. One in five mortality in non-menstrual toxic shock syndrome versus no mortality in menstrual cases in a balanced French series of 55 cases. Eur J Clin Microbiol Infect Dis. 2008;27:37–43.
- 32. Ho CS, Bhatnagar J, Cohen AL, Hacker JK, Zane SB, Reagan S, et al. Undiagnosed cases of fatal Clostridium-associated toxic shock in Californian women of childbearing age. Am J Obstet Gynecol. 2009;201:459 e1–7.
- 33. Nair M, Alabi C, Hirsch PI. Toxic shock syndrome: a silent killer. J Obstet Gynaecol. 2006;26:825.
- 34. Loscar M, Schelling G, Haller M, Polasek J, Stoll C, Kreimeier U, et al. Group A streptococcal toxic shock syndrome with severe necrotizing fasciitis following hysterectomy-a case report. Intensive Care Med. 1998;24:190–3.
- 35. Aldape MJ, Bryant AE, Stevens DL. Clostridium sordellii infection: epidemiology, clinical findings, and current perspectives on diagnosis and treatment. Clin Infect Dis. 2006;43:1436–46.
- Venkataramanasetty R, Aburawi A, Phillip H. Streptococcal toxic shock syndrome following insertion of an intrauterine device-a case report. Eur J Contracept Reprod Health Care. 2009;14:379–82.
- Mourton S, Rich W. Group A streptococcal toxic shock syndrome after an office endometrial biopsy: a case report. J Reprod Med. 2006;51:665–8.
- Toxic shock syndrome (other than Streptococcal) (TSS). Centers for Disease Control and Prevention. http://wwwn.cdc. gov/nndss/conditions/toxic-shock-syndrome-other-than-streptococcal/case-definition/2011/ Accessed 8 June 2015.
- Defining the group A streptococcal toxic shock syndrome. Rationale and consensus definition. The Working Group on Severe Streptococcal Infections. JAMA. 1993;269:390–1.
- 40. Anderson BL. Puerperal group A streptococcal infection: beyond Semmelweis. Obstet Gynecol. 2014;123:874–82.

- Stevens DL, Wallace RJ, Hamilton SM, Bryant AE. Successful treatment of staphylococcal toxic shock syndrome with linezolid: a case report and in vitro evaluation of the production of toxic shock syndrome toxin type I in the presences of antibiotics. Clin Infect Dis. 2006;42:729–30.
- 42. Bartlett J. Soft tissue infections. In: Pham PA, Auwaerter PG, Bartlett JG, Johns Hopkins M, editors. Johns Hopkins ABX guide: diagnosis and treatment of infectious diseases [e-book]. Burlington: Jones and Bartlett Learning; 2012. Available from: eBook Collection (EBSCOhost), Ipswich. Accessed 8 June 2015.
- 43. Rimawi BH, Soper DE, Eschenbach DA. Group A streptococcal infections in obstetrics and gynecology. Clin Obstet Gynecol. 2012;55:864–74.
- 44. Hussein QA, Anaya DA. Necrotizing soft tissue infections. Crit Care Clin. 2013;29:795–806.
- 45. Anaya DA, Dellinger EP. Necrotizing soft-tissue infection: diagnosis and management. Clin Infect Dis. 2007;44:1436–46.
- Nieboer TE, Johnson N, Lethaby A, Tavender E, Curr E, Garry R, et al. Surgical approach to hysterectomy for benign gynaecological disease. Cochrane Database Syst Rev. 2009;8:CD003677.
- 47. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 91: treatment of urinary tract infections in nonpregnant women. Obstet Gynecol. 2008;111:785–94.
- 48. Peterson J, Kaul S, Khashab M, Fisher AC, Kahn JB. A doubleblind, randomized comparison of levofloxacin 750 mg oncedaily for five days with ciprofloxacin 400/500 mg twice-daily for 10 days for the treatment of complicated urinary tract infections and acute pyelonephritis. Urology. 2008;71:17–22.
- 49. Hooton TM. Clinical practice. Uncomplicated urinary tract infection. N Engl J Med. 2012;366:1028–37.
- 50. Soper JD, Kaye D. "Chapter 36: Urinary Tract Infections". In: Bennett JE, Dolin R, Blaser MJ, editors. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. Philadelphia: Elsevier Saunders; 2015. https://www-clinicalkeycom.ezp-prod1.hul.harvard.edu/#!/content/book/3-s2.0-B978145574801300326X. Accessed 11 June 2015.
- Pappachen S, Smith PR, Shah S, Brito V, Bader F, Khoury B. Postoperative pulmonary complications after gynecologic surgery. Int J Gynaecol Obstet. 2006;93:74–6.
- 52. American Thoracic Society; Infectious Diseases Society of America. Guidelines for the management of adults with

hospital-acquired, ventilator-associated, and healthcareassociated pneumonia. Am J Respir Crit Care Med. 2005;171:388–416.

- Ottosen J, Evans H. Pneumonia: challenges in the definition, diagnosis, and management of disease. Surg Clin North Am. 2014;94:1305–17.
- Ritch JM, Kim JH, Lewin SN, Burke WM, Sun X, Herzog TJ, Wright JD. Venous thromboembolism and use of prophylaxis among women undergoing laparoscopic hysterectomy. Obstet Gynecol. 2011;117:1367–74.
- 55. Gould MK, Garcia DA, Wren SM, Karanicolas PJ, Arcelus JI, Heit JA, et al. 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. 2012;141:e227S–77.
- 56. Caprini JA. Thrombosis risk assessment as a guide to quality patient care. Dis Mon. 2005;51:70–8.
- Bahl V, Hu HM, Henke PK, Wakefield TW, Campbell Jr DA, Caprini JA. A validation study of a retrospective venous thromboembolism risk scoring method. Ann Surg. 2010;2512:344–50.
- Wells PS, Hirsh J, Anderson DR, Lensing AW, Foster G, Kearon C, Weitz J, D'Ovidio R, Cogo A, Prandoni P. Accuracy of clinical assessment of deep-vein thrombosis. Lancet. 1995;345:1326–30.
- Swenson CW, Berger MB, Kamdar NS, Campbell Jr DA, Morgan DM. Risk factors for venous thromboembolism after hysterectomy. Obstet Gynecol. 2015;125:1139–44.
- 60. Cushman M, Tsai AW, White RH, Heckbert SR, Rosamond WD, Enright P, Folsom AR. Deep vein thrombosis and pulmonary embolism in two cohorts: the longitudinal investigation of thromboembolism etiology. Am J Med. 2004;117:19–25.
- 61. Hoffman BL, Schorge JO, Schaffer JI, Halvorson LM, Bradshaw KD, Cunningham F, Calver LE. Chapter 39. Perioperative considerations. In: Hoffman BL, Schorge JO, Schaffer JI, Halvorson LM, Bradshaw KD, Cunningham F, Calver LE, editors. Williams gynecology. 2th ed. New York: McGraw-Hill; 2012. http://access-medicine.mhmedical.com.ezp-prod1.hul.harvard.edu/content.aspx?bookid=399&Sectionid=41722331. Accessed 14 June 2015.
- 62. Wells PS, Anderson DR, Rodger M, Ginsberg JS, Kearon C, Gent M, et al. Derivation of a simple clinical model to categorize patients probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer. Thromb Haemost. 2000;83:416–20.

- 63. Cohen AT, Dobromirski M, Gurwith MM. Managing pulmonary embolism from presentation to extended treatment. Thromb Res. 2014;133:139–48.
- 64. Siragusa S, Cosmi B, Piovella F, Hirsh J, Ginsberg JS. Lowmolecular-weight heparins and unfractionated heparin in the treatment of patients with acute venous thromboembolism: results of a meta-analysis. Am J Med. 1996;100:269–77.
- 65. Wells PS, Forgie MA, Rodger MA. Treatment of venous thromboembolism. JAMA. 2014;311:717–28.
- 66. Nezhat C, Farhady P, Lemyre M. Septic pelvic thrombophlebitis following laparoscopic hysterectomy. JSLS. 2009;13:84–6.
- 67. Garcia J, Aboujaoude R, Apuzzio J, Alvarez JR. Septic pelvic thrombophlebitis: diagnosis and management. Infect Dis Obstet Gynecol. 2006;2006:15614.
- 68. Josey WE, Staggers Jr SR. Heparin therapy in septic pelvic thrombophlebitis: a study of 46 cases. Am J Obstet Gynecol. 1974;120:228–33.
- 69. Chirinos JA, Garcia J, Alcaide ML, Toledo G, Baracco GJ, Lichtstein DM. Septic thrombophlebitis: diagnosis and management. Am J Cardiovasc Drugs. 2006;6:9–14.
- Witlin AG, Sibai BM. Postpartum ovarian vein thrombosis after vaginal delivery: a report of 11 cases. Obstet Gynecol. 1995;85:775–80.
- Savader SJ, Otero RR, Savader BL. Puerperal ovarian vein thrombosis: evaluation with CT, US, and MR imaging. Radiology. 1988;167:637–9.
- 72. Dessole S, Capobianco G, Arru A, Demurtas P, Ambrosini G. Postpartum ovarian vein thrombosis: an unpredictable event: two case reports and review of the literature. Arch Gynecol Obstet. 2003;267:242–6.
- Stafford M, Fleming T, Khalil A. Idiopathic ovarian vein thrombosis: a rare cause of pelvic pain - case report and review of literature. Aust N Z J Obstet Gynaecol. 2010;50:299–301.
- 74. Chan JK, Morrow J, Manetta A. Prevention of ureteral injuries in gynecologic surgery. Am J Obstet Gynecol. 2003;188:1273–7.
- 75. Ostrzenski A, Ostrzenska KM. Bladder injury during laparoscopic surgery. Obstet Gynecol Surv. 1998;53:175–80.
- Ostrzenski A, Radolinski B, Ostrzenska KM. A review of laparoscopic ureteral injury in pelvic surgery. Obstet Gynecol Surv. 2003;58:794–9.
- 77. Donnez O, Jadoul P, Squifflet J, Donnez J. A series of 3190 laparoscopic hysterectomies for benign disease from 1990 to 2006:

evaluation of complications compared with vaginal and abdominal procedures. BJOG. 2009;116:492–500.

- Harris RL, Cundiff GW, Theofrastous JP, Yoon H, Bump RC, Addison WA. The value of intraoperative cystoscopy in urogynecologic and reconstructive pelvic surgery. Am J Obstet Gynecol. 1997;177:1367–9.
- 79. Gilmour DT, Das S, Flowerdew G. Rates of urinary tract injury from gynecologic surgery and the role of intraoperative cystos-copy. Obstet Gynecol. 2006;107:1366–72.
- Gilmour DT, Dwyer PL, Carey MP. Lower urinary tract injury during gynecologic surgery and its detection by intraoperative cystoscopy. Obstet Gynecol. 1999;94:883–9.
- Oh BR, Kwon DD, Park KS, Ryu SB, Park YI, Presti Jr JC. Late presentation of ureteral injury after laparoscopic surgery. Obstet Gynecol. 2000;95:337–9.
- 82. Redan JA, McCarus SD. Protect the ureters. JSLS. 2009;13: 139–41.
- 83. Briggs JH, Wing L, Macdonald AC, Tapping CR. Suspected iatrogenic ureteric injury: an approach to diagnostic imaging. Clin Radiol. 2014;69:e454–61.
- Stanhope CR, Wilson TO, Utz WJ, Smith LH, O'Brien PC. Suture entrapment and secondary ureteral obstruction. Am J Obstet Gynecol. 1991;164:1513–7.
- Lam A, Kaufman Y, Khong SY, Liew A, Ford S, Condous G. Dealing with complications in laparoscopy. Best Pract Res Clin Obstet Gynaecol. 2009;23:631–46.
- Kim JS, Lee DH, Suh HJ. Double-J stenting: initial management of injured ureters recognized late after gynecological surgery. Int Urogynecol J. 2010;21:699–703.
- Wang L, Merkur H, Hardas G, Soo S, Lujic S. Laparoscopic hysterectomy in the presence of previous caesarean section: a review of one hundred forty-one cases in the Sydney West Advanced Pelvic Surgery Unit. J Minim Invasive Gynecol. 2010;17:186–91.
- Al-Mandeel H, Qassem A. Urinary ascites secondary to delayed diagnosis of laparoscopic bladder injury. J Minim Access Surg. 2010;6:50–2.
- 89. Delacroix Jr SE, Winters JC. Urinary tract injures: recognition and management. Clin Colon Rectal Surg. 2010;23:104–12.
- Smorgick N, DeLancey J, Patzkowsky K, Advincula A, Song A, As-Sanie S. Risk factors for postoperative urinary retention after laparoscopic and robotic hysterectomy for benign indications. Obstet Gynecol. 2012;120:581–6.

- 91. Lau H, Lam B. Management of postoperative urinary retention: a randomized trial of in-out versus overnight catheterization. ANZ J Surg. 2004;74:658–61.
- 92. Keita H, Diouf E, Tubach F, Brouwer T, Dahmani S, Mantz J, et al. Predictive factors of early postoperative urinary retention in the postanesthesia care unit. Anesth Analg. 2005;101:592–6.
- 93. Ghezzi F, Cromi A, Uccella S, Colombo G, Salvatore S, Tomera S, et al. Immediate Foley removal after laparoscopic and vaginal hysterectomy: determinants of postoperative urinary retention. J Minim Invasive Gynecol. 2007;14:706–11.
- Won HR, Maley P, Chetty N, Chan K, Abbott J. Bladder dysfunction after gynecologic laparoscopic surgery for benign disease. J Minim Invasive Gynecol. 2012;19:76–80.
- 95. Madersbacher H, Cardozo L, Chapple C, Abrams P, Toozs-Hobson P, Young JS, et al. What are the causes and consequences of bladder overdistension? ICI-RS 2011. Neurourol Urodyn. 2012;31:317–21.
- 96. Brosens I, Gordon A, Campo R, Gordts S. Bowel injury in gynecologic laparoscopy. J Am Assoc Gynecol Laparosc. 2003;10:9–13.
- Bishoff JT, Allaf ME, Kirkels W, Moore RG, Kavoussi LR, Schroder F. Laparoscopic bowel injury: incidence and clinical presentation. J Urol. 1999;161:887–90.
- Llarena NC, Shah AB, Milad MP. Bowel injury in gynecologic laparoscopy: a systematic review. Obstet Gynecol. 2015;125: 1407–17.
- Lange H, Jäckel R. Usefulness of plasma lactate concentration in the diagnosis of acute abdominal disease. Eur J Surg. 1994;160:381–4.
- 100. Carpenter CF, Gilpin N. Peritonitis, spontaneous bacterial and secondary. In: Pham PA, Auwaerter PG, Bartlett JG, Johns Hopkins M, editors. Johns Hopkins ABX guide: diagnosis and treatment of infectious diseases [e-book]. Burlington: Jones and Bartlett Learning; 2012. Available from: eBook Collection (EBSCOhost), Ipswich. Accessed 20 Sep 2015.
- 101. van der Voort M, Heijnsdijk EA, Gouma DJ. Bowel injury as a complication of laparoscopy. Br J Surg. 2004;91:1253–8.
- 102. Lawson G. Gynaecological laparoscopy deaths in Australia. Aust N Z J Obstet Gynaecol. 2015;55:477–81.
- 103. Livingston EH, Passaro Jr EP. Postoperative ileus. Dig Dis Sci. 1990;35:121–32.
- 104. Frager DH, Baer JW, Rothpearl A, Bossart PA. Distinction between postoperative ileus and mechanical small-bowel

obstruction: value of CT compared with clinical and other radiographic findings. AJR Am J Roentgenol. 1995;164:891–4.

- 105. Milad MP, Escobar JC, Sanders W. Partial small bowel obstruction and ileus following gynecologic laparoscopy. J Minim Invasive Gynecol. 2007;14:64–7.
- 106. Nelson R, Edwards S, Tse B. Prophylactic nasogastric decompression after abdominal surgery. Cochrane Database Syst Rev. 2005;1:CD004929.
- 107. Prasad M, Matthews JB. Deflating postoperative ileus. Gastroenterology. 1999;117:489–92.
- 108. Mäkinen J, Brummer T, Jalkanen J, Heikkinen AM, Fraser J, Tomás E, Härkki P, Sjöberg J. Ten years of progress–improved hysterectomy outcomes in Finland 1996-2006: a longitudinal observation study. BMJ Open. 2013;3:e003169.
- 109. Muffly TM, Ridgeway B, Abbott S, Chmielewski L, Falcone T. Small bowel obstruction after hysterectomy to treat benign disease. Minim Invasive Gynecol. 2012;19:615–9.
- 110. Allen AM, Antosh DD, Grimes CL, Crisp CC, Smith AL, Friedman S, et al. Management of ileus and small-bowel obstruction following benign gynecologic surgery. Int J Gynaecol Obstet. 2013;121:56–9.
- 111. Antosh DD, Grimes CL, Smith AL, Friedman S, McFadden BL, Crisp CC, et al. A case-control study of risk factors for ileus and bowel obstruction following benign gynecologic surgery. Int J Gynaecol Obstet. 2013;122:108–11.
- 112. Uccella S, Ceccaroni M, Cromi A, Malzoni M, Berretta R, De Iaco P, et al. Vaginal cuff dehiscence in a series of 12,398 hysterectomies: effect of different types of colpotomy and vaginal closure. Obstet Gynecol. 2012;120:516–23.
- 113. Ceccaroni M, Berretta R, Malzoni M, Scioscia M, Roviglione G, Spagnolo E, et al. Vaginal cuff dehiscence after hysterectomy: a multicenter retrospective study. Eur J Obstet Gynecol Reprod Biol. 2011;158:308–13.
- 114. Hur HC, Donnellan N, Mansuria S, Barber RE, Guido R, Lee T. Vaginal cuff dehiscence after different modes of hysterectomy. Obstet Gynecol. 2011;118:794–801.
- 115. Robinson BL, Liao JB, Adams SF, Randall TC. Vaginal cuff dehiscence after robotic total laparoscopic hysterectomy. Obstet Gynecol. 2009;114:369–71.
- 116. Ramirez PT, Klemer DP. Vaginal evisceration after hysterectomy: a literature review. Obstet Gynecol Surv. 2002;57:462–7.

- 117. Matthews CA, Kenton K. Treatment of vaginal cuff evisceration. Obstet Gynecol. 2014;124:705–8.
- 118. Cronin B, Sung VW, Matteson KA. Vaginal cuff dehiscence: risk factors and management. Am J Obstet Gynecol. 2012;206:284–8.
- 119. Magrina JF. Complications of laparoscopic surgery. Clin Obstet Gynecol. 2002;45:469–80.
- 120. Boike GM, Miller CE, Spirtos NM, Mercer LJ, Fowler JM, Summitt R, Orr Jr JW. Incisional bowel herniations after operative laparoscopy: a series of nineteen cases and review of the literature. Am J Obstet Gynecol. 1995;172:1726–31.
- Kadar N, Reich H, Liu CY, et al. Incisional hernias after major laparoscopic gynecologic procedures. Am J Obstet Gynecol. 1993;168:1493–5.
- 122. Montz FJ, Holschneider CH, Munro M. Incisional hernia following laparoscopy: a survey of the American Association of Gynecologic Laparoscopists. J Am Assoc Gynecol Laparosc. 1994;1:S23–4.
- 123. Reardon PR, Preciado A, Scarborough T, Matthews B, Marti JL. Hernia at 5-mm laparoscopic port site presenting as early postoperative small bowel obstruction. J Laparoendosc Adv Surg Tech A. 1999;9:523–5.
- 124. Hashizume M, Sugimachi K. Needle and trocar injury during laparoscopic surgery in Japan. Surg Endosc. 1997;11:1198–201.
- 125. Hurd WW, Pearl ML, DeLancey JO, Quint EH, Garnett B, Bude RO. Laparoscopic injury of abdominal wall blood vessels: a report of three cases. Obstet Gynecol. 1993;82:673–6.
- 126. Cardosi RJ, Cox CS, Hoffman MS. Postoperative neuropathies after major pelvic surgery. Obstet Gynecol. 2002;100:240–4.
- 127. Irvin W, Andersen W, Taylor P, Rice L. Minimizing the risk of neurologic injury in gynecologic surgery. Obstet Gynecol. 2004;103:374–82.
- 128. Shin JH, Howard FM. Abdominal wall nerve injury during laparoscopic gynecologic surgery: incidence, risk factors, and treatment outcomes. J Minim Invasive Gynecol. 2012;19:448–53.
- 129. Boelens OB, Scheltinga MR, Houterman S, Roumen RM. Management of anterior cutaneous nerve entrapment syndrome in a cohort of 139 patients. Ann Surg. 2011;254:1054–8.
- 130. Kanakarajan S, High K, Nagaraja R. Chronic abdominal wall pain and ultrasound-guided abdominal cutaneous nerve infiltration: a case series. Pain Med. 2011;12:382–6.

- Murdock CM, Wolff AJ, Van Geem T. Risk factors for hypercarbia, subcutaneous emphysema, pneumothorax, and pneumomediastinum during laparoscopy. Obstet Gynecol. 2000;95:704–9.
- 132. Celik H, Cremins A, Jones KA, Harmanli O. Massive subcutaneous emphysema in robotic sacrocolpopexy. JSLS. 2013;17:245–8.
- 133. Worrell JB, Cleary DT. Massive subcutaneous emphysema and hypercarbia: complications of carbon dioxide absorption during extraperitoneal and intraperitoneal laparoscopic surgery-case studies. AANA J. 2002;70:456–61.
- 134. Bryant LR, Wiot JF, Kloecker RJ. A study of the factors affecting the incidence and duration of postoperative pneumoperitoneum. Surg Gynecol Obstet. 1963;117:145–50.
- 135. AAGL Advancing Minimally Invasive Gynecology Worldwide, Munro MG, Storz K, Abbott JA, Falcone T, Jacobs VR, Muzii L, et al. AAGL Practice Report: Practice Guidelines for the Management of Hysteroscopic Distending Media: (Replaces Hysteroscopic Fluid Monitoring Guidelines. J Am Assoc Gynecol Laparosc. 2000;7:167–168). J Minim Invasive Gynecol. 2013;20:137–48.
- 136. Jansen FW, Vredevoogd CB, van Ulzen K, Hermans J, Trimbos JB, Trimbos-Kemper TC. Complications of hysteroscopy: a prospective, multicenter study. Obstet Gynecol. 2000;96:266–70.
- 137. Hahn RG. Fluid absorption in endoscopic surgery. Br J Anaesth. 2006;96:8–20.
- 138. Santillan CS. Computed tomography of small bowel obstruction. Radiol Clin North Am. 2013;51:17–21.