pain. There may or may not be pain localized to the infected or diseased organ from which secondary peritonitis has arisen. Patients with secondary peritonitis generally have abnormal findings on abdominal examination, with marked voluntary and involuntary guarding of the anterior abdominal musculature. Later findings include tenderness, especially rebound tenderness. In addition, there may be localized findings in the area of the inciting event. In general, patients are febrile, with marked leukocytosis and a left shift of the WBCs to band forms.

While recovery of organisms from peritoneal fluid is easier in secondary than in primary peritonitis, a tap of the abdomen is rarely the procedure of choice in secondary peritonitis. An exception is in cases involving trauma, where the possibility of a hemoperitoneum may need to be excluded early. Emergent studies (such as abdominal CT) to find the source of peritoneal contamination should be undertaken if the patient is hemodynamically stable; unstable patients may require surgical intervention without prior imaging.

## TREATMENT SECONDARY PERITONITIS

Treatment for secondary peritonitis includes early administration of antibiotics aimed particularly at aerobic gram-negative bacilli and anaerobes (see below). Mild to moderate disease can be treated with many drugs covering these organisms, including broad-spectrum penicillin/β-lactamase inhibitor combinations (e.g., ticarcillin/clavulanate, 3.1 g q4–6h IV), cefoxitin (2 g q4–6h IV), or a combination of either a fluoroquinolone (e.g., levofloxacin, 750 mg q24h IV) or a third-generation cephalosporin (e.g., ceftriaxone, 2 g q24h IV) plus metronidazole (500 mg q8h IV). Patients in intensive care units should receive imipenem (500 mg q6h IV), meropenem (1 g q8h IV), or combinations of drugs, such as ampicillin plus metronidazole plus ciprofloxacin. The role of enterococci and *Candida* species in mixed infections is controversial. Secondary peritonitis usually requires both surgical intervention to address the inciting process and antibiotics to treat early bacteremia, to decrease the incidence of abscess formation and wound infection, and to prevent distant spread of infection. Although surgery is rarely indicated in PBP in adults, it may be life-saving in secondary peritonitis. Recombinant human activated protein C (APC) was considered at one time for treatment of severe sepsis from causes including secondary peritonitis but was withdrawn from the market in 2011 after it was determined that the drug was associated with an increased risk of bleeding and that evidence for its beneficial effects was inadequate. Thus APC should not be used for sepsis or septic shock outside randomized clinical trials.

Peritonitis may develop as a complication of abdominal surgeries. These infections may be accompanied by localizing pain and/or nonlocalizing signs or symptoms such as fever, malaise, anorexia, and toxicity. As a nosocomial infection, postoperative peritonitis may be associated with organisms such as staphylococci, components of the gram-negative hospital microflora, and the microbes that cause PBP and secondary peritonitis, as described above.

### PERITONITIS IN PATIENTS UNDERGOING CONTINUOUS AMBULATORY PERITONEAL DIALYSIS

A third type of peritonitis arises in patients who are undergoing continuous ambulatory peritoneal dialysis (CAPD). Unlike PBP and secondary peritonitis, which are caused by endogenous bacteria, CAPD-associated peritonitis usually involves skin organisms. The pathogenesis of infection is similar to that of intravascular device–related infection, in which skin organisms migrate along the catheter, which both serves as an entry point and exerts the effects of a foreign body. Exit-site or tunnel infection may or may not accompany CAPD-associated peritonitis. Like PBP, CAPD-associated peritonitis is usually caused by a single organism. Peritonitis is, in fact, the most common reason for discontinuation of CAPD. Improvements in equipment design, especially the Y-set connector, have resulted in a decrease from one case of peritonitis per 9 months of CAPD to one case per 24 months.

The clinical presentation of CAPD peritonitis resembles that of secondary peritonitis in that diffuse pain and peritoneal signs are common. The dialysate is usually cloudy and contains >100 WBCs/μL, >50% of which are neutrophils. However, the number of cells depends in part on dwell time. According to a guideline from the International Society for Peritoneal Dialysis (2010), for patients undergoing automated peritoneal dialysis who present during their nighttime treatment and whose dwell time is much shorter than with CAPD, the clinician should use the percentage of PMNs rather than the absolute number of WBCs to diagnose peritonitis. As the normal peritoneum has very few PMNs, a proportion above 50% is strong evidence of peritonitis even if the absolute WBC count does not reach 100/μL. Meanwhile, patients undergoing automated peritoneal dialysis without a daytime exchange who present with abdominal pain may have no fluid to withdraw, in which case 1 L of dialysate should be infused and permitted to dwell a minimum of 1–2 h, then drained, examined for turbidity, and sent for cell count with differential and culture. The differential (with a shortened dwell time) may be more useful than the absolute WBC count. In equivocal cases or in patients with systemic or abdominal symptoms in whom the effluent appears clear, a second exchange is performed, with a dwell time of at least 2 h. Clinical judgment should guide initiation of therapy.

The most common organisms are *Staphylococcus* species, which accounted for ~45% of cases in one series. Historically, coagulase-negative staphylococcal species were identified most commonly in these infections, but these isolates have more recently been decreasing in frequency. *Staphylococcus aureus* is more often involved among patients who are nasal carriers of the organism than among those who are not, and this organism is the most common pathogen in overt exit-site infections. Gram-negative bacilli and fungi such as *Candida* species are also found. Vancomycin-resistant enterococci and vancomycin-intermediate *S. aureus* have been reported to produce peritonitis in CAPD patients. The finding of more than one organism in dialysate culture should prompt evaluation for secondary peritonitis. As with PBP, culture of dialysate fluid in blood culture bottles improves the yield. To facilitate diagnosis, several hundred milliliters of removed dialysis fluid should be concentrated by centrifugation before culture.

## TREATMENT CAPD PERITONITIS

Empirical therapy for CAPD peritonitis should be directed at *S. aureus*, coagulase-negative *Staphylococcus*, and gram-negative bacilli until the results of cultures become available. Guidelines suggest that agents should be chosen on the basis of local experience with resistant organisms. In some centers, a first-generation cephalosporin such as cefazolin (for gram-positive bacteria) and a fluoroquinolone or a third-generation cephalosporin such as ceftazidime (for gram-negative bacteria) may be reasonable; in areas with high rates of infection with methicillin-resistant *S. aureus*, vancomycin should be used instead of cefazolin, and gram-negative coverage may need to be broadened—e.g., with an aminoglycoside, ceftazidime, cefepime, or carbapenem. Broad coverage including vancomycin should be particularly considered for toxic patients and for those with exit-site infections. Vancomycin should also be included in the regimen if the patient has a history of colonization or infection with methicillin-resistant *S. aureus* or has a history of severe allergy to penicillins and cephalosporins. Loading doses are administered intraperitoneally; doses depend on the dialysis method and the patient's renal function. Antibiotics are given either continuously (i.e., with each exchange) or intermittently (i.e., once daily, with the dose allowed to remain in the peritoneal cavity for at least 6 h). If the patient is severely ill, IV antibiotics should be added at doses appropriate for the patient's degree of renal failure. The clinical response to an empirical treatment regimen should be rapid; if the patient has not responded after 48–96 h of treatment, new samples should be collected for cell counts and cultures, and catheter removal should be considered. For patients who lack exit-site or tunnel infection, the typical duration of antibiotic treatment is 14 days. For patients with exit-site or tunnel infection, catheter removal should be considered,