Peritonitis continues to be a serious complication for patients on peritoneal dialysis (PD). Peritonitis is one of the major causes of hospitalization, accounting for 23% of admissions in the CANUSA study (1). Peritonitis is the leading cause of technique failure and catheter loss (2,3). Patients with frequent peritonitis are at increased risk of dying, independent of other factors (4). Although the rates of peritonitis have decreased dramatically from the inception of CAPD, rates above 0.5 episodes per year still commonly occur. The success of this dialysis technique is closely tied to the ability of the dialysis team to reduce the risk of peritonitis, and when it occurs, manage the patient appropriately.

Clinical Presentation

The usual presentation of peritonitis is abdominal pain, cloudy effluent or, most often, both. The pain can range from extremely severe to nonexistent. In the inexperienced patient, the absence of pain may lead him/her to ignore the cloudy effluent initially, leading to a delay in presentation and subsequent treatment. All patients must be instructed to call immediately if the effluent is even slightly cloudy. Peritonitis is present if the white blood cell (WBC) count in the effluent is 100/μL or greater, with at least 50% polymorphonuclear cells. If the specimen is collected from a short cycle, an aspirate from a drained abdomen, or obtained from a patient already on antibiotics, the percentage of polymorphonuclear cells (i.e., more than 50%) is a more reliable marker for peritonitis than the absolute number of WBC.

Occasionally, blood-tinged effluent will be confused with cloudiness, but trained personnel can readily detect the difference. Other causes of cloudy effluent include chylous ascites, which have a milky appearance, intra-abdominal malignancy, diagnosed by cell cytology, and pancreatitis, which can be differentiated by an effluent amylase level of >50 U/L. Also included in the differential diagnosis is eosinophilic peritonitis, which is rarely associated with unusual fungi, but more often is idiopathic; recent reports suggest that treatment with steroids is effective in reducing the cellularity.

In up to 6% of the episodes of peritonitis, the patient presents with abdominal pain but has clear effluent (5). Koopmans et al. reported 60 such episodes of peritonitis, all with positive cultures, that had initial effluent WBC count less than 100/μL. In 40 episodes (67%) the inflammatory response was delayed, in 16 episodes (27%) the effluent cell count eventually reached 30 to 100 WBC/μL, and in four episodes (7%) the cell count never exceeded 30 WBC/μL. The Gram stain was positive in 70% of these cases. The organism was more likely to be Staphylococcus aureus compared to episodes of peritonitis with a normal inflammatory response. Half of the patients with the impaired cell reaction to peritonitis had more than one such episode, and in the absence of peritonitis had lower peritoneal macrophage cell count than a group of control patients. In view of this study, all patients on PD presenting with abdominal pain should be considered to have peritonitis until proven otherwise.

Etiologies of Peritonitis

The most common microorganisms responsible for peritonitis are listed in Table 1 (6,7). Many studies on peritonitis will list the different organisms as a percentage of the total. This makes it difficult to compare the results of one study to another, if the overall rates are dissimilar. For example, in the two studies shown in the table, the percentages of peritonitis episodes due to Staphylococcus aureus are 23 and 12%, respectively, yet the actual difference in rates is only 0.04 episodes per year. It is striking that the rates of coagulase-negative Staphylococcus are almost identical in the two studies. This remains the most frequent microorganism responsible for peritonitis in many centers.

The leading cause of peritonitis continues to be contamination at the time of the PD exchange. Peritonitis due to skin organisms such as coagulase-negative Staphylococcus, Corynebacterium, Bacillus species, and Branhamella catarrhalis are generally believed to be due to contamination. However, PD patients may also have on their (unwashed) fingers Streptococcus viridans, Staphylococcus aureus, Micrococcus, Proteus species, Klebsiella pneumoniae, Enterobacter species, Escherichia coli, and Acinetobacter species (8). If the patient is questioned closely about contamination, the source may become obvious. For example, a patient from our center had frequent bouts of peritonitis related to contamination; an episode due to Streptococcus viridans occurred after performance of an exchange while his granddaughter was in the room without a mask. A second episode of peritonitis followed an exchange in which the patient overheated his bag, which was then cooled by running under cold water. Miller and Findon performed an elegant study examining the level of bacterial contamination associated with touching the connector during an exchange (8). Touching the connection after hand washing...
Table 1. Common microorganisms causing peritonitis

<table>
<thead>
<tr>
<th>Microorganism</th>
<th>Holley et al. (6)</th>
<th>Van Biesen et al. (7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coagulase-negative <em>Staphylococcus</em></td>
<td>0.17</td>
<td>0.18</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>0.13</td>
<td>0.09</td>
</tr>
<tr>
<td><em>Streptococcus</em></td>
<td>0.04</td>
<td>0.06</td>
</tr>
<tr>
<td>Other Gram-positive</td>
<td>&lt;0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Gram-negative</td>
<td>0.09</td>
<td>0.16&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Polymicrobial</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Fungal</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Culture-negative</td>
<td>0.11</td>
<td>0.20</td>
</tr>
<tr>
<td>Total rate</td>
<td>0.56&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.73&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup> Rates provided as episodes per year at risk.
<sup>b</sup> *Pseudomonas* accounted for 0.06.
<sup>c</sup> Rates do not add to total due to rounding.

using chlorhexidine followed by thorough drying reduced the numbers of bacteria reaching the peritoneal cavity to <5. In contrast, absence of hand washing resulted in considerably higher numbers of organisms on the connection, and wet hands resulted in the transport of up to 4500 organisms. Proper training of the patient in aseptic technique is obviously critical in reducing peritonitis from contamination.

Fifteen to 20% of peritonitis episodes are due to catheter infections, with *Staphylococcus aureus* and *Pseudomonas aeruginosa* accounting for the great majority (9). Patients at greatest risk for *Staphylococcus aureus* catheter-related peritonitis are those who are *Staphylococcus aureus* carriers (10,11). Approximately one-half of PD patients are *Staphylococcus aureus* nasal carriers (11,12). *Staphylococcus aureus* in the nose is transmitted to the hands; Boelaert et al. found that 75% of dialysis patients with *Staphylococcus aureus* in their nares had *Staphylococcus aureus* on their hands, in contrast to 10% of dialysis patients without nasal carriage (13). From the hands, the *Staphylococcus aureus* is quickly transmitted to the catheter exit site and/or to the connection port at the time of an exchange. This risk factor is important to recognize, because the risk of *Staphylococcus aureus* peritonitis can be considerably reduced by prophylaxis for *Staphylococcus aureus* carriage (14,15) (Figure 1). Several different approaches have been identified (15–18). Either at the time the catheter is placed or, ideally, before placement, the nose should be cultured, and, if positive, the patient should be treated with intranasal mupirocin. Once training begins, mupirocin can be used at the exit site as part of routine care, or the intranasal course can be repeated monthly. Patients who have three negative nose cultures are at very low risk for infection and do not require prophylaxis, unless immunosuppressed (12,19).

Peritonitis due to Gram-negative bacilli may be the result of contamination, catheter infection, or may come from the bowel. Treatment of constipation with enemas, diarrhea, and gastric acid inhibitors may predispose to enteric peritonitis (20–22). Although frank bowel perforation is distinctly uncommon (23), Harwell et al. reported that enteric peritonitis was associated with underlying intra-abdominal pathology such as cholecystitis, ischemic colitis, and appendicitis in one-third of patients (24). Because few of these episodes resolved with antibiotic therapy alone (Figure 2), early recognition followed by laparotomy is critical to decrease the risk of death.

Iatrogenic peritonitis could be classified under bacteremia, enteric, or gynecologic categories, but is listed separately to emphasize this unusual but important (potentially preventable) cause of peritonitis. It is well recognized that peritonitis may follow colonoscopy with polypectomy, endoscopy with sclerotherapy, and dental procedures (24–26). Vaginal leak of dialysate, the use of intrauterine devices, endometrial biopsy, and hysteroscopy with polypectomy have all been associated

- Reference 15: daily exit site mupirocin
- Reference 16: rifampin 600 mg qd for 5 days every 12 weeks
- Reference 17: intranasal mupirocin bid for 5 days every month in nasal carriers
- Reference 18: intranasal mupirocin tid for 7 days for each positive nose culture

![Figure 1](https://example.com/figure1.png)

*Figure 1. Protocols to prevent *Staphylococcus aureus* peritonitis.*
with peritonitis (26–28). Antibiotic prophylaxis prior to any procedure associated with the risk of peritonitis is warranted.

**Risk Factors for Peritonitis**

Modifiable risk factors for peritonitis are listed in Table 2. In addition, certain patient populations are at higher risk for coagulase-negative peritonitis, including African-American and native American patients (3,29,30). This risk can be minimized by using a disconnect system. Also at increased risk for peritonitis are immunosuppressed patients (19). Andrews et al. reported that those receiving immunosuppressive therapy in the past 12 months or with a disease predisposing to infection, such as HIV, had a peritonitis rate of 1.8 episodes per patient year compared with a rate of 0.68 episodes per patient year in other patients (19). The risk of *Staphylococcus aureus* and fungal peritonitis was especially high.

**Evaluation**

Upon presentation, a rapid assessment of the patient should include questions on breaks in technique, recent procedures that may have led to peritonitis, change in bowel habits, prior peritonitis, and catheter infection history. The exit site and tunnel should be closely examined for evidence of infection. In addition, the patient’s abdomen should be drained, and the effluent sent for cell count with differential, Gram stain, and culture. The cell count with differential will confirm the presence of peritonitis. Centrifuging 10 ml of effluent results in a positive Gram stain of the infected sediment in 93% of episodes (31). The culture should be obtained by placing 5 ml in each of two tryptic soy broth blood culture bottles (aerobic and anaerobic); Lyé et al. found that the rate of culture-negative peritonitis was reduced from 42 to 25% by this technique (32). In addition to inadequate culture techniques, culture-negative peritonitis may also be due to the presence of antibiotics, so the patient should be questioned closely about recent antibiotic use (33). In one-third of culture-negative peritonitis episodes, reculturing will result in identification of an organism (34).

A decision must be made about whether to hospitalize the patient. This will depend on the severity of the peritonitis, and the need for intravenous analgesia and fluids. Because the disconnect systems have primarily diminished peritonitis due to less virulent organisms such as coagulase-negative *Staphylococcus*, as opposed to Gram-negative and *Staphylococcus aureus*, a high proportion of the patients will require admission (23).

**Initial Treatment**

Often, the clinician does not know the causative organism when antibiotic therapy is ordered. Therefore, the initial therapy should be active against the most commonly offending organisms, including *Staphylococcus* (both coagulase-negative

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**Table 2. Factors associated with decreased risk of peritonitis**

<table>
<thead>
<tr>
<th>Prophylactic antibiotics at time of catheter placement</th>
<th>2-cuffed catheter (versus one-cuff catheter)</th>
<th>Downward pointing tunnel</th>
<th>Twin bag connection system</th>
<th>Treatment of <em>Staphylococcus aureus</em> carriage</th>
</tr>
</thead>
</table>
and -positive), Streptococcus, and Gram-negative bacilli. In the absence of clinical data suggestive of bowel perforation, anaerobic coverage is generally not given initially. Coverage for fungus should be implemented immediately only if the Gram stain is positive for yeast.

The Ad Hoc treatment guidelines of 1996 recommended using a first-generation cephalosporin, in combination with gentamicin for the initial therapy (35) (Table 3). This change from the prior recommendation for vancomycin in conjunction with Gram-negative coverage was due to the increasing incidence of vancomycin-resistant enterococcus and the fear that this resistance will be transferred to staphylococci, leaving us without drugs to treat these infections. Indeed, peritonitis due to vancomycin-resistant Staphylococcus has been reported (36).

Lai et al. reported on the results of once daily intraperitoneal cefazolin and gentamicin, with subsequent modification of therapy as needed, for treatment of peritonitis (37). It is important to note that episodes of peritonitis related to catheter infection were excluded from this study. All 19 episodes of Gram-positive peritonitis resolved. Three episodes of Staphylococcus epidermidis were resistant in vitro to both gentamicin and cephalosporin, yet responded in vivo to these antibiotics, presumably due to the high local concentration in one exchange each day. These data, therefore, support the Ad Hoc Committee's recommendations.

Unfortunately, data from other dialysis programs suggest that use of a first-generation cephalosporin for initial therapy may result in a considerable number of untreated or inadequately treated patients. Vas et al. reported that cefazolin (1.5 g intraperitoneally once daily for 3 wk) resulted in resolution of only 45% of episodes of peritonitis due to methicillin-resistant coagulase-negative Staphylococci (38). This was in contrast to a 73% response during the historical period for which vancomycin (2 g intraperitoneally once weekly for three doses) was used. The proportion of Staphylococci with methicillin resistance is reported to range from 33 to 67% (7,39). We have found an increase in methicillin-resistant coagulase-negative Staphylococci in our program, as shown in Figure 3. Using the Ad Hoc 1996 recommendations for initial therapy, Van Biesen noted that only 76.5% patients with Gram-positive infections and 81% of patients with Gram-negative infections would have been effectively treated, based on sensitivities (7). These authors propose an alternative approach, shown in Table 4, which would provide 100% coverage of Gram-positive organisms and 87.5% coverage of Gram-negative organisms.

Within 2 to 3 d, the organism is usually identified and sensitivities are available. Subsequent therapy is chosen to provide narrow coverage with the least toxicity. Dosing schedules for some commonly used antibiotics are given in Table 5. Guidelines by organism are provided below. If the culture is negative, generally the aminoglycoside is stopped and a single drug such as a first-generation cephalosporin or vancomycin is continued alone.

### Coagulase-Negative Staphylococcus Peritonitis

These patients often do not require hospitalization, because the pain is less severe than that due to other organisms (23). Cefazolin or cephalothin should be changed to vancomycin if the organism is methicillin-resistant (38). Although vancomycin is often given weekly, this may lead to underdosing in many patients, especially those with residual renal function. Low trough levels in the dialysate increases the risk of relapsing peritonitis (40). We prefer the use of 30 mg/kg vancomycin intraperitoneally (maximum of 2 g) in the initial exchange, which is allowed to dwell for a minimum of 6 h, with one-half of this dose repeated in 4 to 5 d. Before the second dose, a blood level is measured, which in outpatients is not back before redosing, but the value guides the timing of the third dose, usually about 5 d later. These three doses then provide antibiotic coverage for a minimum of 2 wk.

### Staphylococcus Aureus Peritonitis

Patients with Staphylococcus aureus peritonitis often have severe abdominal pain and generally require hospitalization. If Staphylococcus aureus exit site or tunnel infection is present, the catheter should be removed without delay (9). If there is no clinically obvious tunnel infection, an ultrasound of the tunnel to confirm the absence of involvement is helpful, as catheter infections may be occult (41). Peritonitis associated with a catheter infection will prove to be either refractory or relapsing (9,42). The course can be deceptive because the episode may appear to resolve with clearing of the effluent, yet the effluent culture remains positive with recrudescence of full-blown peri-

<table>
<thead>
<tr>
<th>Table 3. Ad Hoc 1996 guidelines for initial therapy (35)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment Protocol</strong></td>
</tr>
<tr>
<td>In the first exchange</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Subsequent therapy</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

* Increase dosing frequency based on serum and/or dialysate levels.
Coagulase negative staphylococcus peritonitis, episodes / year

![Graph showing rates of coagulase-negative Staphylococcus peritonitis over time.](image)

Figure 3. Rates of coagulase-negative *Staphylococcus* peritonitis over time.

**Table 4. Empiric treatment of peritonitis, Van Biesen approach (7)*

<table>
<thead>
<tr>
<th>Initial therapy</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>vancomycin</td>
<td>15 mg/kg single dose IP</td>
</tr>
<tr>
<td>gentamicin</td>
<td>1.5 mg/kg for urine output &gt;500 ml/d, 0.5 mg/kg for urine output &lt;500 ml/d</td>
</tr>
<tr>
<td></td>
<td><strong>After 24 hours</strong></td>
</tr>
<tr>
<td>outpatient</td>
<td>ciprofloxacin 500 mg twice a day</td>
</tr>
<tr>
<td>inpatient</td>
<td>cefazolin 250 mg IP/exchange, and ciprofloxacin 50 mg/IP, intraperitoneally.</td>
</tr>
</tbody>
</table>

**Table 5. Intraperitoneal antibiotic dosages for adults (35)**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Intraperitoneal Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>125 mg/L continuously</td>
</tr>
<tr>
<td>Ampicillin/sulbactam</td>
<td>1 g/L load, then 100 mg/L continuously</td>
</tr>
<tr>
<td>Cefazolin and cephalothin</td>
<td>500 mg/L load, then 125 mg/L continuously</td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>1000 mg/exchange, once daily</td>
</tr>
<tr>
<td>Gentamicin and tobramycin</td>
<td>0.6 mg/kg, once daily</td>
</tr>
<tr>
<td>Impipenem/cilistat</td>
<td>500 mg/L load, then 200 mg/L continuously</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>15 to 30 mg/kg every 5 to 7 d</td>
</tr>
</tbody>
</table>

Streptococcus Peritonitis

The source for peritonitis due to *Streptococcus* species (non-enterococcus) may be the respiratory tract (either transient bacteremia or absence of a mask during an exchange), the skin (touch contamination), or the bowel (43). Consistent with this, the incidence of *Streptococcus viridans* has decreased with use of the disconnect systems. *Streptococcus* (especially group A and group B) causes severe peritonitis and may quickly result in death. Therefore, treatment must be implemented rapidly. Ampicillin appears to be more effective than vancomycin (43).

Enterococcal peritonitis is severe, responds slowly to antibiotics, and carries an increased risk of death (23,43). In the Network 9 Peritonitis Study, peritonitis due to enterococcus resulted in death in 7.4% of the episodes (23). Peritonitis due to enterococcus, compared with *Streptococcus viridans* peritonitis, occurs in older patients and is likely spread from the bowel. The incidence has not fallen with the use of disconnect systems. Fortunately, vancomycin-resistant enterococcal (VRE) peritonitis, with a reported mortality rate of 55%, is still rare (44). VRE peritonitis is associated with prior use of both cephalosporins and vancomycin, as well as hospitalization. Treatment is problematic. Troidle *et al.* used chloramphenicol, mostly unsuccessfully (44). We have had one case of VRE peritonitis at the University of Pittsburgh, in a patient with a failed renal transplant given three weekly doses of vancomycin.

...
for culture-negative peritonitis; she did well with prompt catheter removal and institution of quinupristin/dalfopristin, an experimental drug. Stool carriage of VRE is still uncommon in PD patients, perhaps because PD is a home dialysis modality (39).

**Gram-Negative Peritonitis**

Once the organism is identified and sensitivities are known, the antibiotic therapy is adjusted to minimize use of aminoglycosides as much as possible, in view of the risk of vestibular toxicity. Alternatives to aminoglycosides, such as ceftazidime and quinolones, should be used whenever possible. An alternative approach has been to use a low dose of aminoglycoside in one exchange per day, which provides high local levels during the period of the dwell, yet very low systemic levels. Lai et al. treated 14 Gram-negative peritonitis episodes with gentamicin, 20 mg/L in one exchange per day intraperitoneally, combined with 500 mg/L in the same exchange of cefazolin (37). For the eight non-*Pseudomonas* Gram-negative organisms, only two infections due to *Acinetobacter* species required alteration in therapy, and all resolved. Bailie et al. used 0.6 mg/kg in one exchange of gentamicin (in combination with an initial dose of vancomycin) with resolution of two-thirds of the episodes of non-*Pseudomonas* Gram-negative peritonitis (45). The nonresponders included *Acinetobacter* and *Alcaligenes* species. *Acinetobacter* peritonitis is difficult to treat, and two antibiotics should be used to treat this organism.

*Pseudomonas aeruginosa* peritonitis is frequently associated with a tunnel infection, which may be occult. Should the effluent culture reveal a *Pseudomonas* infection, the subcutaneous tunnel should be examined carefully, and if it appears normal, ultrasonography should be performed to further assess possible tunnel involvement. If the tunnel is involved, the catheter should be promptly removed, as resolution with antibiotic therapy is highly unlikely (9). If there is no tunnel infection, *Pseudomonas aeruginosa* will usually resolve with aminoglycoside therapy (generally given as 5 to 8 mg/L in each exchange) and the addition of a second agent active against *Pseudomonas*. Of note, in a recent article from Belgium, 7% of patients with peritonitis died (3 of 42) from sepsis; two of these patients had *Pseudomonas* peritonitis (7).

In view of the results of a recent article by Harwell et al., in every case of peritonitis due to enteric organisms, intra-abdominal pathology underlying the infection should be considered (24). This can be evaluated with a computed tomography scan of the abdomen. Early surgical consultation should be considered, as prompt laparotomy may decrease the risk of death.

**Polymicrobial Peritonitis and Intra-Abdominal Abscesses**

Approximately 6% of all episodes of peritonitis will have multiple organisms (46). If only Gram-positive organisms are present, the patient generally does well, with resolution of the infection with antibiotic therapy in the absence of catheter infection. However, if multiple enteric organisms grow from the culture, intra-abdominal pathology must be considered, which requires surgical exploration. This is especially true if an anaerobe grows in culture. Metronidazole, 500 mg intravenously every 8 h, should be added to the other antibiotics. Intra-abdominal abscesses occur in <1% of peritonitis episodes. These are more common with *Pseudomonas aeruginosa*, *Candida albicans*, and polymicrobial peritonitis, and require drainage. Computed tomography scan of the abdomen is useful to evaluate the patient.

**Fungal Peritonitis**

Fungal peritonitis accounts for 3% of all episodes (47). Usually the patient has severe abdominal pain, and the effluent WBC count is high. Gram stain is often helpful in establishing the diagnosis early. *Candida* is by far the most common organism. Risk factors include frequent peritonitis, immunosuppression, and antibiotic therapy. At our institution, the catheter is removed as soon as the diagnosis is established. In an uncontrolled trial, Goldie et al. found that 15% of patients in whom the catheter was removed within 1 wk of diagnosis died, in contrast to 50% when the catheter was left in place (47). Therapy with fluconazole (200 mg orally each day), flucytosine (1 g orally each day), and, if necessary, amphotericin, should be continued after catheter removal for at least an additional 10 d. The catheter can be reinserted, but a waiting period of 1 to 2 mo is advisable. Approximately 10% of patients will have peritoneal fibrosis making PD no longer an option.

**Peritonitis due to Mycobacterium**

Tuberculous peritonitis occurs more frequently in Asia than in Western countries, but may become more common in view of the current epidemic of mycobacteria infections. As with other microorganisms, the effluent WBC are predominately polymorphonuclear cells. Because the effluent acid-fast bacillus stain is generally negative and there is usually no tuberculous disease elsewhere, the diagnosis may be difficult (48). Ultrafiltration failure may occur but is not inevitable. Therapy should consist of three drugs (isoniazid, rifampicin, and pyrazinamide) for 9 to 12 mo. Reportedly, the catheter does not always require removal (48).

**Refractory and Relapsing Peritonitis**

Refractory peritonitis is defined as an episode in which there is no improvement 5 d after appropriate antibiotic therapy is initiated. There may be apparent resolution of the episode with antibiotic therapy, but a cell count will often show persistence of an abnormal inflammatory response. Recurrent or relapsing peritonitis is defined as a second episode of peritonitis with the same organism as the first within 2 wk of stopping antibiotics. Refractory and relapsing peritonitis may be due to a catheter infection, which may not be clinically apparent. In every case of relapsing and refractory peritonitis, the catheter subcutaneous tunnel should be examined clinically, and, if it appears normal, an ultrasonographic examination should be performed.

Recurrent peritonitis, in the absence of a tunnel infection, may be due to sequestration of bacteria (most often coagulase-negative *Staphylococcus*) in the biofilm surrounding the intra-
abdominal portion of the catheter. Inadequate treatment of peritonitis predisposes to this complication. There are two options: fibrinolytic therapy or catheter replacement. Urokinase, 5000 U in 5 ml of normal saline injected into the catheter with the abdomen drained and allowed to dwell for 2 h, is successful in 29 to 67% of patients with recurrent peritonitis (49,50). This procedure has been primarily used on patients with recurrent coagulase-negative Staphylococcus peritonitis, and should be reserved for those recurrent episodes of peritonitis for which tunnel infection has been excluded as a cause by careful examination using ultrasonography. Alternatively, if the peritoneal cell count can be suppressed to less than 100 WBC/μl, then the catheter can be safely replaced at one setting, allowing the avoidance of hemodialysis in many patients (50,51). One of the most common problems in managing peritonitis is delay in removing the catheter in episodes that are not responding or that are likely to result in relapsing peritonitis. Table 6 lists examples in which catheter removal is often necessary.

Prevention of Peritonitis

Prevention of peritonitis is a key component of a successful PD program, and is based on collaboration between patient, the nurses, and the physician. Decreasing risk from contamination is highly dependent on both patient selection and training techniques. The nurses should reinforce the concepts of sterility and compulsiveness in performing the exchange, even after the initial training is completed. The best connection technology, the twin bag system, should be chosen for continuous ambulatory PD patients, and for cycler patients who must do multiple spikes, use of the Compact Assist Device (Baxter Healthcare Corp., Deerfield, IL) is helpful in reducing the risk of infection. Each program must monitor and periodically review cases of peritonitis to identify problem areas. Patients with high peritonitis rates should be encouraged to transfer permanently to hemodialysis.

The risk of Staphylococcus aureus peritonitis and catheter infections can be reduced by monitoring and treating for nasal carriage (Figure 1). We found that prophylaxis with mupirocin at the exit site resulted in a reduction in Staphylococcus aureus exit site infections and related peritonitis episodes (16). These results have been recently confirmed by Thodis et al. (52). This protocol is well accepted by the patients, who use a Q-tip to place a thin smear of mupirocin around the catheter exit site after bathing. One tube lasts for approximately 2 mo. This method should not be used with polyurethane catheters, which may be damaged. Alternative approaches are intranasal mupirocin for Staphylococcus aureus nasal carriage. In the multicenter European trial, this reduced Staphylococcus aureus catheter infections but not peritonitis (17). Perez-Fontan et al. found a reduction in both Staphylococcus aureus catheter infections and peritonitis with a course of intranasal mupirocin for each positive nose culture (18). Rifampin has also been shown to reduce both Staphylococcus aureus catheter infections and peritonitis rates, but is associated with side effects in 12% of patients (14–16).

Prophylaxis with nystatin, given to the patient taking antibiotics, successfully reduces the risk of Candida peritonitis (53). Additional studies will be needed to identify those patients who would benefit most from such prophylaxis. This may prove to be patients at high risk for fungal peritonitis, including those with frequent bacterial peritonitis, on prolonged courses of antibiotics or with impaired immune systems.

Procedure-related causes of peritonitis may be decreased by asking the patient to inform the dialysis center before extensive dental work, colonoscopy, and endometrial biopsies. We believe that antibiotic prophylaxis should be given for all such procedures. In addition, the abdomen should be drained prior to pelvic and colonic procedures. Aggressive treatment of constipation may result in enteric peritonitis; therefore, every effort should be made to prevent constipation in the PD patient.

In conclusion, peritonitis remains one of the most serious problems facing the PD patient and PD health care worker. Reducing rates of peritonitis can be achieved by careful patient selection and training, use of the best connection technology, and screening for and treating nasal carriage. Once peritonitis occurs, the treatment should be prompt. There should be no hesitation to remove the catheter if this appears to be appropriate.

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