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Management of Peritonitis in the Critically Ill Patient

Carlos A. Ordoñez, MD^a and Juan Carlos Puyana, MD^{b,*}

^aUniversidad del Valle, Fundación Clínica Valle del Lili, Autopista Simón Bolívar, Carrera 98 No. 18-49, Cali, Colombia

^bDivision of Trauma and General Surgery, University of Pittsburgh Medical Center Presbyterian, Suite F-1265, 200 Lothrop Street, Pittsburgh, P A 15213, USA

The terms peritonitis, intra-abdominal infection, and abdominal sepsis are not synonymous, yet sometimes they are used indistinctly to define similar clinical states. Peritonitis is defined as an inflammatory process of the peritoneum caused by any irritant/agent such as bacteria, fungi, virus, talc, drugs, granulomas, and foreign bodies. Intra-abdominal infection is defined as the local manifestations that occur as a consequence of peritonitis. Intra-abdominal sepsis entails a systemic manifestation of a severe peritoneal inflammation.

The clinical spectrum of peritonitis may also be classified according to the pathogenesis as primary, secondary, or tertiary peritonitis. Alternatively, a more localized phenomenon in peritonitis is the formation of abscesses, a condition characterized by the isolation and walling off of the infectious process from the rest of abdominal cavity [1–3].

The mortality of an intra-peritoneal infection in the early 1900s was close to 90%. This condition was managed nonoperatively until Kishner introduced the basic principles of surgery in intra-abdominal infections: (1) elimination of the septic foci, (2) removal of necrotic tissue, and (3) drainage of purulent material. By the 1930s, mortality had been reduced to 50%. With the introduction of antibiotics, the mortality continued to decrease slowly. The use of cephalosporins by the early 1970s was associated with a reduction of mortality to less than 30% to 40%. Subsequent advances in the understanding of physiology, the monitoring and support of the cardiopulmonary systems, the rational use of new drugs, and ICU care aided in stabilizing mortality at around 30% [1,4].

There is no controversy regarding the standard treatment that includes control of the source and intra-abdominal lavage (washing); however, in patients who have advanced peritonitis, the source of the infection may not be completely eradicated with a single operation. Thus controversy arises, specifically regarding issues such as time and frequency of repetitive laparotomies, and management of the open wound/abdomen. Furthermore, the aggressive resuscitation required in these patients causes gut and abdominal wall edema that may be associated with increased intra-abdominal pressure, worsened by a premature closing of the abdominal wall. To date, it is clear that the reduction of mortality below 20% has been the result of a better understanding of the role of damage control, prevention of intra-abdominal compartment syndrome, and improved antibiotic alternatives with broad-spectrum newer medications [5–15].

Anatomy and physiology of the peritoneum

The peritoneum is a single layer of mesothelial cells resting on a basal membrane, and a bed of conjunctive tissue formed by adipose cells, macrophages, fibroblasts, lymphocytes, and

* Corresponding author. puyanajc@upmc.edu (J.C. Puyana).

some elastic fibers of collagen. The abdominal cavity is covered by the parietal peritoneum and it turns into the visceral peritoneum to cover the abdominal viscera. The total surface of the peritoneum is approximately 1.7 m². In normal conditions it is sterile, and it contains 50 mL of yellow fluid, which contains a few macrophages; mesothelial cells generally and lymphocytes. Most of the peritoneal membrane behaves like a passive barrier, semipermeable to the bidirectional diffusion of water and most solutes. The total surface of interchange of the peritoneal cavity is approximately 1 m². Unlike liquids and most solutes, larger particles are eliminated through the larger orifices that exist between the specialized mesothelial cells that cover the lymphatic conduits on the diaphragmatic surface of the peritoneal cavity. These intracellular orifices correspond to fenestrations of the basal membrane, and together serve as conduits of the peritoneal cavity to the underlying lymphatic drainage system of the diaphragm, called “lakes” or “lagoons.” The reabsorption of particles or bacteria is only possible in the subdiaphragmatic peritoneal surface through numerous stomas or intracellular lagoons, to which a network of lymphatic vessels flows into the diaphragmatic lacunas. These lacunas have a diameter of 8 to 12 microns, subject to variations depending on the diaphragmatic movements and the changes of thoraco-abdominal pressure. Smaller particles, such as bacteria that by general are approximately 2 microns in diameter, are readily absorbed through diaphragmatic lacunes into the thoracic duct. Intra-peritoneal fluid and exudates circulate constantly in the cavity toward the decanting zones via gravity, and toward the subphrenic spaces by the suction caused by diaphragmatic contraction. This works like a suction pump. It accelerates the flow during inspiration, and diminishes or restrains it during expiration, and it is probably the most important mechanism in charge of the “defensive” cleansing of the peritoneum [1,4].

After peritoneal inoculation of bacteria in dogs, the micro-organisms can be identified within 6 minutes in the thoracic duct and within 12 minutes in the bloodstream. This is the first mechanism of peritoneal defense, and after the insaturation of a peritoneal infection, bacteremia can occur. If the host is healthy or bacteremia is not massive, it will be controlled without any other systemic repercussion. If, on the contrary, the host is jeopardized or bacteremia is very great, a systemic inflammatory response with commitment of the patient (sepsis) can be produced [16,17].

Simultaneously to the first physical contact between the bacteria and the peritoneum, there is an associated injury to the mesothelial cells, with subsequent activation of inflammatory mediators, which will activate both cellular and humoral immunological responses.

The initial response of the peritoneum against the bacterial contamination is characterized by hyperemia and increased exudates of fluid with phagocytes into the peritoneal cavity. In this initial stage these are predominately macrophages. Neutrophils arrive within 2 to 4 hours, and become the predominant cells of the peritoneal cavity by the first 48 to 72 hours. These cells release great amount of cytokines such as interleukin (IL)-1, IL-6, and tumor necrosis factor (TNF), leukotriens, platelet activating factor, C3A and C5A, that promotes even more local inflammation. The combined effect of these mediators contributes to observed inflammatory response during peritonitis. As there is destruction of the bacteria, the lipo-polysacharids of gram-negative *Enterobacteria* constitute a powerful stimulus for further generation of inflammatory cytokines [18,19].

As a consequence of this inflammation there is production of fibrinogen in the septic foci, with fast formation of fibrin, creating a mesh of fibrin that temporarily reduces and blocks the reabsorption of fluid from peritoneal cavity and “traps” bacteria. This phenomenon may generate the formation of an abscess. In addition, the omentum migrates toward the inflamed area and aids in delivering mediators and cells to facilitate the abscess formation. The most common location is the subphrenic areas.

This response can be controlled and the peritonitis can be resolved, or it may continue to produce a residual or persistent peritonitis or may form an abscess. Therefore, there are three defense mechanisms in the peritoneum against the bacteria that have contaminated it, but these three mechanisms also have paradoxical effects in the organism. The first one, the early mechanical elimination of the bacteria through the diaphragmatic gaps, may in fact produce bacteremia, that if massive, will generate septic shock and eventually death. The second, the liberation of large exudates, rich in phagocytic cells and opsonins, may also produce a severe displacement of fluids and proteins toward this “third space,” which would cause a state of hypovolemia and shock with loss of albumen toward the peritoneal cavity.

The systemic response of to a severe bacterial peritonitis will then include the liberation of catecholamines, and an increase of the secretion of hormones adrenocorticoids, as well as the secretion of aldosterone and antidiuretic hormone. The hemodynamic alterations that are observed in patients who have peritonitis have several causes. The hypovolemia decrease the extracellular volume by the massive shift of fluids toward the peritoneal cavity, producing a decrease in cardiac index, increase of the resistance vascular peripheral, and oxygen consumption increase in the periphery. IL-2 and IL-8 favor cell recruitment; this stimulus of the cell abduction is so profound that pancytopenia can be observed some 4 to 6 hours after the initial stimulus. It has been difficult, however, to show the direct correlation between the magnitude of the septic answer and the concentration of circulating cytokines [20–23].

Classification of intra-abdominal infection

Primary peritonitis

Primary peritonitis is an inflammation of the peritoneum by an extraperitoneal source, frequently occurring from hematogens dissemination. It occurs in children and adults, and can endanger life, particularly in patients who have cirrhosis or in children who have nephrosis. Primary or spontaneous peritonitis relates to the deterioration of the immune defenses of the guest. It is generally produced by a single micro-organism, and the main pathogens in adults are the coliforms. In fact, 70% of these infections they are caused by *Escherichia coli*, 10% to 20% by gram-positive cocci, and 10% by anaerobes. The management includes antibiotics and fluid resuscitation. Occasionally the patient may have to undergo surgery, usually as a diagnostic laparotomy characterized by finding purulent material with gram-positive cultures without obvious perforation or abscesses in solid organs [1,24].

Secondary peritonitis

This is an infection that results from an inflammation or mechanical break of the integrity of the intestinal or the urogenital tract or solid organs, thus exposing the peritoneal cavity to the resident flora of the gastrointestinal (GI) tract.

Secondary peritonitis is classified as acute peritonitis by perforation, postoperative peritonitis, or post-traumatic peritonitis.

Acute peritonitis by perforation

This is the most common type of acute intra-abdominal. Almost 80% of cases result from necrosis of the digestive conduit [1–4]. The perforation of the small intestine caused by the inflammation and necrosis of the intestine, such as in typhoid fever and mesenteric ischemia secondary to the intestinal obstruction, occurs initially as a paralytic ileus, with subsequent progression to necrosis and perforation. Peritonitis caused by appendicitis in most cases presents as a localized peritonitis that if left long enough, may become a generalized peritonitis. Close to 22% of peritonitis is secondary to inflammation of the colon

(diverticulitis and colitis); less common causes are perforations of the colon by cancer, incarcerated hernia, or intussusceptions. Necrotizing pancreatitis may be associated with subsequent peritonitis if there is contamination or infection of the necrotic pancreas and/or exudates.

Postoperative peritonitis

The incidence of postoperative peritonitis occurs in 1% to 20% of patients undergoing laparotomy, and its occurrence is related to the primary reason for laparotomy and other risk factors that are discussed below. The most common cause of postoperative peritonitis is anastomotic failure/leak. In the majority of the cases there is a delay in diagnosis. As a general rule, symptoms are evident between the fifth and the seventh postoperative days, contributing to a very high mortality. The infected intestinal content and the proteolytic enzymes leak into the peritoneal cavity, inflaming it and producing the local and systemic response described above just as the diagnosis is performed. Morbidity and mortality also depend on the anatomical location and the magnitude of the leak. Thus, for example, it is more difficult to repair a duodenum that is fixed and leaks toward the retro-peritoneum than a leak in the distal small bowel or colon that can be easily externalized. In some instances of postoperative peritonitis, the anastomosis may be intact; however, the patient may remain sick because of residual peritonitis from diverse causes. Among them is the inadequate drainage of the initial septic focus, in which the surgeon failed to drain completely, or more commonly, the peritoneum does not have the sufficient defense capacity to control the problem [1,2,25].

Post-traumatic peritonitis

Peritonitis in trauma patients may occur because of missed injuries, such as mesenteric tear with loss of blood supply and subsequent ischemia and intestinal perforation. This type of intra-abdominal infection is usually serious because of the delay in diagnosis, especially in patients who have multiple injuries and associated traumatic brain injury. Patients who suffer a penetrating trauma by sharp weapon or by firearm, with an acute abdomen, are operated on immediately and the injury is controlled. Generally, the intra-abdominal infection secondary to contamination seen in penetrating traumatism is a function of the time lapsed between the injury and the surgery. Only a third of the patients who have penetrating trauma of the colon have contamination of the cavity peritoneal that requires treatment with antibiotics [26–28].

Tertiary peritonitis

Tertiary peritonitis is defined as a persistent or recurrent intra-abdominal infection, after an apparently adequate treatment of a primary or secondary peritonitis. The standard treatment of secondary peritonitis consists of draining the septic focus to remove the necrotic material and to prevent the reaccumulation of pus with an adequate management of antibiotics from 5 to 7 days. If, having completed adequate surgical and antibiotic treatment, the infection persists or recurs after 48 hours, then it can be considered as a tertiary peritonitis. Furthermore, the term tertiary or recurrent peritonitis may also be used when there is persistent infection after a third reintervention for secondary peritonitis that has been managed with staged or planned relaparotomies. The microbiology of this infection is characterized by low virulence germs or sterile peritonitis. The flora changes with time, and the local mechanisms of defense of the abdominal cavity are overwhelmed. Ongoing organ dysfunction should indicate inadequate drainage or unidentified focus. This presentation is classically seen in immune-compromised patients. The patient is septic, showing a hyperdynamic cardiovascular state, fever, and failure to thrive. The operative findings are characterized by lack of formation of abscess and rather diffuse exudates without fibrin-purulent membranes, and the appearance is of a liquid more or less clear. Cultures of this

fluid reveal coagulase negative *Staphylococcus*, enterococcus, *Pseudomonas*, yeast, and *Enterobacter* among others. The septic foci are rarely amenable to percutaneous drainage, and they are in difficult locations within the abdomen. An inexperienced surgeon may describe a sero-sanguineous fluid, and may conclude that the abdominal cavity is clean and even think that the laparotomy was unnecessary; however, these patients usually experience a severe systemic response immediately after the procedure, with tachycardia, hypotension, and bacteremia. It is therefore necessary to aggressively manage the immediate postoperative resuscitation in the ICU with fluids combined with inotropic and vasopressor support. Progressive multiple organ dysfunction with death may follow if this cycle is not controlled [29–33].

The management of tertiary peritonitis should be done in the ICU, and should be performed by a multidisciplinary team. The patient will require metabolic and nutritional support, as well as hemodynamic and respiratory care; early physical therapy is ideal. An opportune and timely decision to adjust antibiotics according to the most recent culture report of blood and of the abdominal cavity should be made in conjunction with the surgeon and the intensivist. From the surgical viewpoint, the decision for on-demand relaparotomy should be made in advance, and decisions for reoperation should not be delayed. If the decision has been made for management with an open abdomen with planned laparotomy using a mesh, then it should be decided when to suspend the repeated lavages of the abdominal cavity, because these can cause more damage than benefit after the fifth or sixth procedure. Antimicrobial therapy should not exceed 14 days, except in patients who have fungal infections [34–38].

Diagnosis

The diagnosis of peritonitis is a clinical diagnosis, based mostly on history and physical examination. The main symptom in all cases is abdominal pain. The pain can be sharp or insidious; often the pain is constant and intense, and is aggravated with movement. The majority of patients lie still, with their knees bent and the head raised; these maneuvers diminish the tension of the abdominal wall and alleviate the pain. Anorexia, nausea, and vomiting are frequent symptoms. Nevertheless, depending on the etiology of the peritonitis and of their time of evolution, the symptoms can vary. The majority of patients are seen in poor general conditions, showing a sharp and severe illness. The temperature is usually above 38° centigrade, but patients who have septic shock may have hypothermia. Tachycardia and the decrease in the amplitude of the pulse are indicatives of hypovolemia, and they are common in the majority of patients. Patients present with a high cardiac output and decreased systemic vascular resistance. They may have increase pulse pressure. Pain to palpation is the most characteristic sign of peritonitis, to both deep and superficial touch. Initially there is voluntary guarding; subsequently the muscular wall undergoes an involuntary and severe spasm. Bowel sounds may or may not be present, and they may resemble an early ileus. Localized peritonitis generates pain localized toward the inciting organ. Percussion of the abdomen may aid in accurately localizing the place of maximum peritoneal irritation. Rectal examination, although indispensable in the physical examination, rarely orients toward the origin of the peritonitis. In the first hours of peritoneal irritation the pain may be intense, but to the extent that time elapses, the pain becomes more insidious and more difficult to assess. A high index of suspicion may be the difference in making an early rather than a late diagnosis with dire consequences. The patient may have an elevated white cell count greater than 11,000 cells per mL with left shift. Leucopenia suggests generalized sepsis and is associated with a poor prognosis. Blood chemistry may be normal, but in serious cases it may indicate severe dehydration, such as increased blood ureal nitrogen (BUN) and hypernatremia. Metabolic acidosis helps in the confirmation of the diagnosis. A urinalysis is indispensable to rule out urinary tract infection, pyelonephritis, and nephrolithiasis. Plain film of the abdomen is not ordered

routinely. When obtained, however, it could reveal paralytic ileus with bowel distension or air fluid levels. An upright chest radiograph is useful if perforated viscera is suspected. Free air in the abdomen may occur in 80% of cases of duodenal ulcer perforation, but it is observed with less frequency when there is colon, small bowel, or intra-peritoneal rectum perforation [1,39–42].

When the diagnosis is made clinically, an abdominal CT only delays the surgical intervention. Nevertheless, an abdominal CT can be useful in for suspected recurrent or undrained infection in the postoperative period. Velmahos and colleagues [43] recommend obtaining an abdominal CT in critically ill post-trauma patients who have sepsis of unknown origin. The CT usually aids in guiding therapy in two of every three cases. Abdominal ultrasound (US) may also aid in working up patients who have postoperative septic complications. Depending on the operator, fluid collection may be identified; however, this finding by itself may be nonspecific. The greatest advantage of US is that it can be done at the bedside. Bowel loops may be identified by their peristalsis, and bedside percutaneous drainage may be done in some cases, thus facilitating the procurement of samples for cultures. Go and coworkers [44] performed a comparative study to validate the use of US versus CT in patients who had postoperative intra-abdominal sepsis. They showed that CT is the procedure of choice in these patients, and that US may be used in selected cases.

Diagnostic peritoneal lavage

Diagnostic peritoneal lavage (DPL) is a trustworthy and safe method for the diagnosis of generalized peritonitis, specifically in patients who do not have conclusive signs on physical examination, who have poor medical history, or who have sedation or post brain injury, advanced age, or spinal cord injury. Patients on steroids or immune-compromised patients may or may not give conclusive results in the DPL. A positive DPL (greater than 500 leukocytes/mL) suggests peritonitis.

Laparoscopy has been used, and there have been recent reports about its efficacy. It is useful in selected cases that have similar characteristics as those described for DPL. The limiting step is the ability of creating an adequate optical cavity in patients who have abdominal distension, anasarca, or previous surgery/scars. Finally, the gold standard diagnostic intervention continues to be exploratory laparotomy. The risks of an added surgery versus the benefits of obtaining a diagnosis are critical to aid in making an individual decision. In summary, the clinical assessment takes precedence over diagnostic tests that may delay a life-saving intervention. One must avoid unnecessary delays that will ultimately compromise the patient's physiology and will impact the ability to successfully resuscitate the patient after surgery.

Treatment

The management of severe peritonitis is complex and requires a multidisciplinary approach. The surgeons and intensivists must work together with practitioners in nutritional support, personal respiratory therapy, infectious disease, and radiology. The use of standard protocols for resuscitation and hemodynamic/ventilatory support to facilitate overall management should have a positive impact on outcome. The authors have developed a strict surgical management protocol with aggressive interventional approach to eradicate septic foci in the abdomen (AAST).

When the decision for re-exploration is made because of patient deterioration or failure to thrive associated with early organ dysfunction, an aggressive preoperative resuscitation is implemented, including management with mechanical ventilation with low tidal volumes (6–8 mL/kg), placement of a pulmonary artery catheter (PAC), and judicious fluid

resuscitation. Elderly patients require more aggressive cardiac monitoring, and may need perioperative support to maintain an adequate cardiac output. These patients may also require monitoring of intra-abdominal pressure to prevent and identify abdominal compartment syndrome. Once the patient is adequately resuscitated, he is taken to the operating room.

Goals of resuscitation

As a general guideline, the authors use the following parameters to guide resuscitation: (1) central venous pressure (CVP) and pulmonary occlusion pressure (POP) between 8 and 12 mmHg, (2) mean arterial pressure (MAP) greater than 65 mmHg, (3) urine output greater than 0.5 mL/Kg/h, and (4) a mixed venous O₂ saturation greater than 70%.

The authors attempt to correct the patient's compromised hemodynamic and respiratory status, and in most cases achieve these parameters within 6 hours of ICU care. Effective circulatory volume and inotropic/vasopressor support are often needed. We use dobutamine, and we aim for a hemoglobin above 7 gr/dL. Transfusions of packed red cells are given when there is active bleeding or if the mixed oxygen saturation is less than 70% and the hemoglobin is less than 7. We use crystalloids, and a patient may easily require three to six liters in the first hours, depending on the state of the patient and the illness. Blood glucose is also closely monitored, aiming to maintain values around 220 mg/dL. All patients receive prophylactic medications, including ranitidine or omeprazole for stress ulcer prevention and subcutaneous heparin for prevention of thromboembolic disease if there is no evidence of coagulopathy. Enteral nutrition is preferred to intravenous as soon as possible. The use of low-dose steroids for 7 days is indicated if the state of shock persists despite adequate resuscitation, or if there is poor response to vasopressors or adrenal insufficiency [45–57].

Antibiotic therapy should be initiated as soon as possible. The initial therapy is administered on an empirical basis. The selection of antimicrobial agents should be based on the suspicion of the responsible micro-organisms and the capacity of the antibiotics to reach adequate levels in the peritoneal cavity. Generally upper GI tract perforations are associated with gram-positive bacteria, which are sensitive to cephalosporin and penicillins. Perforations of the distal small bowel and colon generally present with polymicrobial aerobic and anaerobic bacteria.

Secondary peritonitis is characterized by positive cultures with combinations of the following flora: *E Coli*, *Streptococcus*, *Enterobacter* spp, *Klebsiella* spp, enterococci, *Pseudomonas aeruginosa*, *Proteus* spp, *Staphylococci aureus*, and epidermides. The are *Bacteroides fragilis*, *Clostridium* spp and *Peptostreptococci*. Antifungal therapy should be used if their presence is documented in the peritoneal cavity [58–60].

In cases of moderate or community-acquired peritonitis, monotherapy may be sufficient. In these cases the authors suggest the use of ampicillin/sulbactam or ertapenem. Combined therapy is best guided by the local ICU/hospital antibiogram. The first line of therapy may differ from one institution to the next, and may include anti-anaerobic medications such as metronidazole or clindamicina, combined with aminoglycosides in selected cases (gentamycin or amikacyn), ciprofloxacin and third- or fourth-generation cephalosporins (ceftriaxone or cefotaxime).

In severe cases and patients who have high risk of nosocomial infections, the authors use initial monotherapy with piperacillin—tazobactam, or carbapenem type (imipenem or meropenem). The alternative is the use of fourth-generation cephalosporin and metronidazole.

Careful attention to dosing must be given and corrective measurements implemented daily according to drug levels, because many of these patients have unsteady volume of distribution caused by marked fluid shifts [61–65]. The duration of therapy should be established and guided by the surgical findings. The absence of fever, elevated white count, or left shift may aid in discontinuing antibiotic therapy because the incidence of recurrence is low when these parameters have been met. If leukocytes and rectal temperature are normal for 48 hours, they can be suspended by postoperative day 4, depending on the pathology that produced the peritonitis [66,67]. The incidence of recurrent intra-abdominal sepsis has been reported as high as 33% to 50% in patients who remain febrile and have persistent leukocytosis. The 1996 European consensus recommends short as possible duration of antibiotic use, depending on the condition that caused the peritonitis, without exceeding 5 days [68].

Surgical management

The goals of surgical treatment are eliminating the cause of the contamination, reducing the bacterial inoculum, and preventing persistent or recurrent sepsis. The surgical approach is best made via a midline incision to ensure adequate and complete exploration of the abdominal cavity. Diligent hemostasis and thorough exploration are of primary importance. Suctioning of all fluid cavities is done and quantified. Samples are taken for Gram's stain fungal studies and culture. In general terms, control is achieved by excluding or resecting the perforated viscera. If a colonic perforation is found, the proximal segment is externalized with a col and a mucous fistula is made on the distal segment. In cases that have small bowel perforation, resection is followed by primary anastomosis whenever possible. Not infrequently, the small bowel may require entry both of the proximal and distal segment, especially if the peritoneal contamination is severe and the viability of the bowel is doubtful. The other fundamental objective in the surgical treatment of peritonitis consists in reducing the size of the bacterial load to prevent sepsis and recurrent reaccumulation of purulent material. A thorough lavage of the abdominal cavity removing all detritus and particles should be done. Special attention should be given to areas where abscess may form such as the pelvis, the gutters, and the subphrenic spaces. These areas should be carefully exposed and debrided, avoiding bleeding by excessive peeling of the fibrin [1–4]. Warm saline solution is used until a clear return is obtained. The authors do not recommend using iodine or other chemical agents to lavage the peritoneal cavity, because they may compromise the local inflammatory response. Excess fluid must be suctioned as well.

In closing the abdominal cavity, ideally the fascia should be reapproximated with nonabsorbable material. The skin should be left open and covered with wet gauze dressings for a period from 48 to 72 hours. The incidence of wound infection is close to 42% in these cases; however, if the wound is clean after 3 to 4 days, a secondary closure of the skin maybe attempted, depending of the underlying cause of peritonitis [8,23,69–71].

Some patients may not improve despite adequate initial operative management. These patients may require several laparotomies to achieve the goals listed above. Therefore the surgeon should decide during the first or “index” laparotomy whether or not the patient may benefit from a reoperation, which could be done either as needed or through a planned/scheduled reintervention [72–76]. Postoperative peritonitis has a very high mortality, ranging from 30% to 50%. High mortality may be caused in part by delayed diagnoses and delayed reintervention of complications with associated organ dysfunction. The mortality and morbidity associated with aggressive re-operation strategy in complicated patients who have peritonitis has been reported in the literature. Because the decision to reoperate may not always be done soon enough, several authors suggest the use of open abdomen or strict daily re-exploration to avoid delays in management. During the 1980s this was a procedure

recommend often. More recent reviews indicate that laparotomy on demand may be as beneficial and may have lower morbidity; however, there is no definitive evidence indicating which strategy is better [77–85].

Re-laparotomy “on demand”

The authors refer to relaparotomy on demand (ROD) when the decision for further intervention is not planned in a fixed or scheduled manner, but is rather based on the clinical progress of the patient during the immediate postoperative course. These patients manifest clinical evidence of an intra-abdominal complication characterized by generalized peritonitis or abscess. These complications can manifest at any time, as early as during the first week postoperative. The most common scenario is a patient who has ongoing septic picture, caused by a persistent peritonitis with reaccumulation of pus. Some suggest that these patients may have ongoing immunosuppression and are more susceptible to endotoxemia or true “translocation.” Most commonly these patients develop new pathogens and manifest a variety of symptoms suggesting ongoing infection. The surgeon must be very attentive and have a high index of suspicion. The clinical presentation may include persisting pain, prolonged ileus, abdominal distension, or intolerance to enteral nutrition. Patients may have persistent fever and leucocytosis, as mentioned earlier. The surgeon should make the diagnosis within the first 48 hours after any of these nonspecific findings occur and avoid waiting until the fifth or seventh postoperative day, even though some authors suggest that anastomotic failure or leaks may take that long to manifest themselves. Koperna and Schulz [86] showed that patients reoperated after 48 hours had mortality significantly higher than those operated earlier (76.5% versus 28%; $P = .001$); however, the timing of the relaparotomy did not have any repercussion on survival in those patients who had an acute physiology and chronic health (APACHE) II score greater than 26. This finding suggested that under these circumstances of severely impaired physiology the early operation had little effect.

Unfortunately, the symptoms described above may be a manifestation of systemic inflammatory response (SIRS). Furthermore, patients may manifest respiratory distress syndrome and renal dysfunction as well. The most challenging dilemma for the surgeon is to decide whether or not these findings are the result of an untreated abdominal source. Therefore the decision to reoperate is extremely difficult. The clinical criteria are subjective. The authors frequently obtain feedback from all those involved in the daily care of the patient, including the intensivist, infectious disease consultant, and the radiologists. Early postoperative changes in the CT may be difficult to interpret. After a laparotomy there is distortion of the planes, and there may be fluid collections that can be blood, serum, saline solution, intestinal liquid or pus that can complicate the diagnosis even further. Yet the radiologist is key to guide the surgeon to perform a limited exploration, or to avoid the surgery if a transcutaneous approach is safe and effective. Occasionally the surgeon is obliged to carry out a complete exploration [6]. The advantage of deciding to do a laparotomy on demand at the time of the index laparotomy is that the patient is not submitted to unnecessary surgery or anesthesia. The most serious disadvantage is when a needed reintervention is done late, when the patient's underlying physiology is compromised and has progressed to deterioration and multiple organ dysfunction. Lamme and colleagues [87] compared these two strategies in a retrospective study of 278 patients. The ROD had a mortality of 21.8% ($n = 197$) compared with 36% in the planned relaparotomy (PR) group ($P = 0.016$). The average numbers of surgeries in the ROD group was 0.9/patient compared with 1.3 in the PR group. Fifty-four percent of the patients assigned to ROD did not need a relaparotomy.

A meta-analysis of all reports on ROD and PR for secondary peritonitis showed that there is not a significant difference in mortality when these two modalities are compared [88]. Hutchins and coworkers [89] found that the combined positive predictive value of the clinical assessment made in concert with the input from the laboratory and radiological data as well as the assessment made by the intensivist and the surgeons is close to 83%. In this study the hospital mortality was 43% and the average time for execution of the relaparotomy was 5 days. Not surprisingly, outcome was highly associated to age and organ dysfunction [89].

Planned re-laparotomy

The decision to re-explore the abdomen is made during the initial surgery. The patient is programmed to undergo a repeat laparotomy every 24 hours until the septic focus is fully controlled. Daily debridement and lavage is performed removing all necrotic tissue via a “laparostomy” or open abdomen. To have easy access to the abdomen, a protective nonadhesive mesh is used to cover the bowel. The indications for PR at the authors' institutions are to cover the bowel. The indications for PR at Universidad del Valle, Fundación Clínica Valle del Lili and the University of Pittsburgh are

- Failure in obtaining an adequate control of the source during the laparotomy index
- Inadequate or poor drainage
- Diffuse fecal peritonitis
- Hemodynamic instability
- Reassessment of the anastomosis (tenuous)
- Intra-abdominal hypertension

The authors use a mesh for staged abdominal repair [7]. We achieve adequate control after two to three reinterventions planned every 24 hours. The advantages of PR in our opinion are the ability to clean and treat the whole abdominal cavity as a giant abscess. By placing a mesh with Velcro (Velcro, Manchester, New Hampshire) we do not add unnecessary straining on the skin or the fascia that otherwise would be necessary in some cases of ROD. The very approach of PR should avoid the occurrence of abdominal compartment syndrome and intra-abdominal hypertension. The technique itself requires that the surgeon be extremely gentle; ideally the same surgeon who performed the index laparotomy should do the subsequent procedures. He or she decides whether to break all the interloop adhesions and possible collections, and should know how accessible some of the areas in the peritoneum may be. The amelioration of the inflammatory response, the presence of clean granulation tissue, and the reduction of the systemic response should all aid in deciding when to discontinue the PR and when to proceed with removing the mesh and closing the abdomen. In the authors' experience, we accomplish this stage with two to three laparotomies. At this point, we remove the Velcro or zipper that we use occasionally, and in most patients the wound is then covered with a Bogota bag, allowing for formation of granulation tissue underneath. In most of our cases, the authors carry on the subsequent laparostomies in the ICU under general anesthesia using intravenous agents [6,8]. Alternatively, the PR strategy can be modified by using a vacuum pack system as another technique that may facilitate reduction of the size of the wound, and in some cases facilitate closure of the fascia. Additional advantages of the vacuum pack include managing the possible intra-abdominal hypertension and better control of drainage and fluid production from the wound. Several reports have been published regarding the use of the vacuum pack system [90–92]. In the authors' institutions we prefer the method described by Barker and coworkers [93]. In the initial step during the index laparotomy when a decision for PR is

made, a large sheet of polyethylene is used that is displayed laterally all the way into the colic gutters bilaterally, thus preventing the bowel from adhering to the abdominal cavity. Then several small cuts are made on the plastic sheet and the plastic sheet is covered with three or four abdominal surgical towels. On top of the surgical towels, two Jackson-Pratt drains are placed and these are then covered with two more abdominal surgical towels. In some patients the authors close the skin partially with 1-0 vicryl and a large adhesive dressing is used to cover the abdominal wall. We place a negative pressure close to 100 to 150 mm Hg.

The disadvantages of PR include higher requirements of anesthesia and surgery; increased manipulation of the viscera that may result in fistula formation, evisceration, increased loss of fluid, electrolyte, and proteins; and potential contamination with exogenous flora. This is a more labor-intensive approach and requires great dedication from the multidisciplinary team. Several studies suggest the use of APACHE II to assess the level of physiologic compromise on a daily bases. There have been reports indicating that PR reduces significantly the mortality in patients who have APACHE II scores ranging between 10 to 25. Patients who have initial APACHE II scores greater than 26 have a high mortality regardless of the strategy used, and patients who have APACHE II scores less than 10 have a good prognosis regardless of the approach used, PR or ROD [7,94–96].

Our multidisciplinary team in Cali, Colombia has been working with a protocolized approach to these patients over the last 10 years, and we have accumulated experience of 267 patients who had severe peritonitis managed with the PR approach. The demographic and clinical characteristics of our population are summarized in Tables 1, 2 and 3. The number of laparotomies used in the PR strategy has diminished in our hands, because we have realized over time that most times an adequate lavage and debridement is completed by the third procedure (see Table 2).

The source of infection in this group of patients was colon pathology in 33% of the cases: 22.5% from the small bowel, 22% from hepatobiliary and pancreas sources, 10.5% from stomach and duodenum, and 5% originated from appendicitis. Cultures obtained during the index laparotomy yielded negative results in 25% of the cases, most likely because of previous use of antibiotics. *E coli* was found in the 23% of the patients, enterococci in the 11.6%, *Pseudomona* in 7.7%, *Klebsiella* in 6%, *Staphylococcus* in 5.1%, and other bacteria in 21%. The authors used combination metronidazol-cefotaxime in 43% of the cases, imipenem in 11.3%, carbapenem alone in 20.6%, and combination carbapenem with fluconazol or vancomiyn in 8.6%. The overall mortality was 19.9%, (95% confidence interval [CI], 15.1–24.7) and multiple regression analyses revealed age older than 50 years, APACHE II greater than 25, and septic as independent predictors of death (see Table 3).

In the authors' hands, PR is a safe and viable technique. We experienced a reduction in mortality from 28.4% seen during the period 1995 to 1999 to a mortality of 14.5% (95% CI, 9.1–19.1) from the year 2000 until now. We were able to diminish the number of relaparotomies to 3.4 ± 2.3 . It is likely, however, that the reason for this lower mortality is not merely the result of diminishing the number of laparotomies, but rather the combined effect of a protocolized management that included aggressive resuscitation and improved metabolic, ventilatory, and hemodynamic support [45,53,54,97].

Intraoperative management and surgical approach to bowel anastomosis: the role of deferred early anastomosis

Control of the source of infection and prevention of further contamination can be achieved quickly by performing an ostomy. This procedure is strongly indicated, especially in the face

of massive contamination, hemodynamic instability, and hypoperfusion. The bowel can be exteriorized at any level, most commonly as a colostomy or an ileostomy, and in some cases as a distal or even proximal jejunostomy. Yet these ostomies could become a source of significant morbidity in those patients who survive the acute phase of peritonitis. Complications range from electrolyte and fluid imbalance to skin and wound complications such as hernias and stenosis, as well as serious psychological problems. Furthermore, there is a significant morbidity associated with the surgical approach to re-establish bowel continuity [98,99]. Primary anastomoses of the small bowel are done routinely in patients who have peritonitis caused by small bowel pathology. In patients who have peritonitis secondary to perforated diverticulitis, the decision to perform a primary anastomosis should be based on an individual basis, according to the patient's stability and the surgeon's judgment [100–103]. More recently, primary colonic anastomosis has been advocated in the setting of acute trauma of the colon [104–111]. In trauma, the risk of intra-abdominal infection is independent of the surgical procedure, as shown by Demetriades and colleagues [112], who recommend primary anastomosis in most cases. These data, however, cannot be extrapolated to cases of advanced peritonitis.

De Graaf and coworkers [113] described the use of primary anastomosis as a feasible technique in the management of severe secondary peritonitis in critically ill patients; its use, however, has been limited because of the high risk of failure and leak, which in these patients may result in worsening sepsis, multiple organ dysfunction syndrome (MODS), and death. The determinants that dictate whether or not a patient should undergo primary repair or undergo exteriorization of the bowel are

- Hemodynamic stability
- Extent of inflammation of the peritoneal cavity
- Viability of the bowel

The bowel should not be anastomosed in the face of severe contamination or diffuse peritonitis; in the setting of a failed anastomosis (postoperative peritonitis); in cases who have severe edema of the bowel, severe malnutrition, chronic use of steroids, mesenteric ischemia; and during the initial phases of damage control surgery.

The concept of performing a deferred primary anastomosis (DPA) in septic patients who have peritonitis stems from the authors' experience with the concept of staged laparotomy and damage control in trauma patients. Despite the use of damage control in trauma patients, the mortality-associated damage control has been reported above 40% in some series [114–120].

The authors implemented a protocol of damage control in septic patients who had peritonitis and followed our patients in a prospective observational and descriptive study at the Fundación Valle de Lili in Cali, Colombia, from November 2000 to May 2004 [121]. Our protocol was designed with the objective of performing definitive management of the bowel whenever possible. Specific criteria were defined and patients were submitted to DPA, thus minimizing the numbers of ostomies. The inclusion criteria were

- Severe peritonitis with hemodynamic instability and sepsis
- Septic shock
- Aged 18 years and older

Patients were excluded if they died in the first 24 hours, or if the bowel anastomosis and definitive closure of the abdomen was accomplished during the first-index laparotomy.

All patients were operated within 6 hours of arrival to the emergency room. They underwent aggressive resuscitation, and were submitted to bowel resection and ligature. The abdominal cavity was lavaged and the pus drained. They were placed in the authors' planned laparotomy schedule, explained above, and managed in the ICU as described earlier. When the peritonitis was controlled, a DPA was performed using either a GIA 80 automatic suture device or a hand-sewn anastomosis with vicryl 3-0 continuous suture in a single plan (Figs. 1–4) [122–125].

The demographic data of 26 patients submitted to DPA are depicted in Table 4. Fifteen patients underwent small bowel resection in the initial surgery (57.7%), and 11 (42.3%) had a colon resection. The abdominal wall was closed with a Velcro mesh as described earlier. The average number of relaparotomies until peritonitis was controlled was four. The relaparotomies were performed every 24 hours. The authors performed 14 anastomoses in the small bowel, five colo-colonic anastomoses, and four ileo-colonic anastomoses. Three patients could not undergo bowel anastomosis because of severe protracted inflammation. Eighteen anastomoses were done with stapler; 5 were hand- sewn. All patients underwent one-more-look relaparotomy to ascertain the integrity of the anastomosis before closing the abdomen. Twenty patients had septic shock, and 4 patients developed acute respiratory distress syndrome (ARDS). The most frequent micro-organisms were *E coli*, followed by *Candida albicans* and *Pseudomona aeruginosa*. Only 3 patients developed fistulas. The first patient had a colonic leak colon that closed in the first 5 days without surgery. Two fistulas of the small bowel were difficult to manage and required prolonged support with total parenteral nutrition (TPN). One patient closed spontaneously in 3 months and the third one was taken to the surgery for resection of the fistula 4 months later. Twenty patients received TPN while their bowel was ligated, and enteral nutrition was given to 24 patients about 30 hours after the anastomosis was accomplished. We closed the fascia in 14 patients, 11 underwent skin closure only, and one died before closing.

The mortality was 11.6% (3 patients). Twenty three patients left the hospital alive. The causes of death were line sepsis in one patient, a cardiac event in the second, and the third patient died of multiple organ dysfunction and gut failure with untreatable fistulas (Table 5).

The results of this study indicate that damage control and DPA in severe peritonitis is a viable technique. The numbers of ostomies was reduced, and this was accomplished with a low mortality.

Closure of the abdominal wall

The technique to close the abdomen is decided on during the last relaparotomy when the Velcro mesh is removed. Ideally by this time there is formation of granulation tissue, and in most patients the bowel is adhered to the abdominal wall.

The authors contemplate the following options:

- Remove the Velcro mesh, bring the fascia together with no tension, and leave the skin open.
- Place a plastic bag (Bogota bag) on the abdominal cavity and suture it to the abdominal wall (skin or fascia); wait a few more days for granulation tissue to form.
- Close the skin only because the fascia is adhered and cannot be closed. An incisional hernia repair is done 6 months later.

- When the granulation has been completed and is not possible to close the fascia or the skin, then a split-thickness skin graft is placed over the laparostomy with subsequent correction of the incisional hernia.
- In some cases is possible to “shrink” the defect by bringing the Velcro closer and closer until the last relaparotomy, at which point the foci can be sutured. The authors achieved closing of the fascia in the 50% of the cases of 267 patients managed with Velcro [8,126].
- Using a vacuum pack system in 33 of our patients, the authors were also able to close the fascia in 50% of them and skin in the other 50%. Clearly the vacuum pack has the added advantages of maintaining sterility of the cavity, facilitating prompt access to the abdomen, preventing loss of domain, preserving the quality of the fascia, facilitating wound care by sucking the drainage around the wound, and diminishing the chance of intra-abdominal hypertension and the syndrome of abdominal compartment (Sistema vacuum pack en peritonitis severa. Carlos A. Ordóñez, MD, personal communication, 2006).

The authors do not advise closing by doing an extensive dissection of the fascia, nor do we use a mesh at this time to prevent colonization. [8,126]. When the patient is totally recovered, in approximately 6 to 8 months, a repair of the incisional hernia is performed.

Summary

Severe secondary peritonitis is an entity that continues to carry a significant mortality, despite advancements in critical care support and antibiotic therapies. Surgical management is pivotal and requires a multidisciplinary approach to guide the timing and the number of interventions that may be necessary to eradicate the septic foci and create the optimal conditions for healing with the fewest possible complications. Further research is needed to determine the best surgical strategy for very severe cases. The use of DPA seems to be a safe option in patients presenting with hemodynamic instability and hypoperfusion. These patients have a high-risk anastomotic failure and fistula formation. Allowing for aggressive resuscitation and judicious assessment of the progression of the local inflammation are safe strategies to achieve the highest success and minimize serious and protracted complications in patients who survive the initial septic insult.

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Fig. 1.
Temporary closure proximal and distal with umbilical tape.

Fig. 2.
Side-to-side anastomosis with GIA 80 4.8.

Fig. 3.
GIA 80 4.8 stapled anastomosis to close bowel ends.

Fig. 4.
Anastomosis completed.

Table 1

Demographic and clinical characteristics of patients with peritonitis 1995–2004

Total number	267
Age (years)	52.2 ± 20
Male gender, %	62.5
APACHE II, %	
0–10	24
11–25	70.4
26–35	5.6
Median APACHE II, %	14
Source of infection, %	
Colon	33.3
Small bowel	22.5
Liver biliary tract	8.6
Stomach—duodenum	10.5
Pancreas	13.9
Others	11.3
Number of re-laparotomies	
Mean ± SD	4 ± 3
Median	3
Length of hospital stay, days	
Mean ± SD	25.7 ± 18
Median	20
Length of ICU stay, days	
Mean ± SD	15.8 ± 13
Median	12
Fistula, %	15.3
Septic shock, %	54.3
ARDS, %	30
Days on ventilator	
Mean ± SD	9.5 ± 11
Median	6
Mortality, % (95% CI)	19.9 (15.1–24.7)

Abbreviations: ARDS, acute respiratory distress syndrome; CI, confidence interval.

Table 2

Comparison between patients from 1995–1999 with patients operated on from 2000–2004

	1995–1999	2000–2004	P
Number	102	165	–
Age	53.9 ± 18	51 ± 20	0.24
Male gender, %	61.8	63	0.83
APACHE II			
0–10	23.5	24.2	0.89
11–25	65.7	71.6	0.3
26–35	7.8	4.2	0.2
Median, APACHE II, %	14	14	–
Source of infection, %			
Colon	39.2	33.3	0.93
Small bowel	16.7	26.1	0.07
Liver biliary tract	7.8	9.1	0.68
Stomach—duodenum	12.7	9.1	0.85
Pancreas	16.7	12.1	0.29
Others	12.8	10.3	0.79
Number of relaparotomies			
Mean ± SD	4.9 ± 3.8	3.4 ± 2.3	0.00007
Median			
Length of hospital stay, days			
Mean ± SD	27.2 ± 19	24.8 ± 18	0.3
Median	22	20	
Length of ICU stay, days			
Mean ± SD	18 ± 14	14.4 ± 11.6	0.08
Median	14	11	
Fistula, %	16.5	14.2	0.19
Septic shock, %	52.9	55	0.72
ARDS, %	49	18.2	<0.0001
Days on ventilator			
Mean ± SD	12 ± 12	8 ± 9	0.04
Median	9.5	5	
Mortality, % (95% CI)	28.4 (19.6–37.1)	14.5 (9.1–19.1)	0.0005

Abbreviation: CI, confidence interval.

Table 3

Predictors of death multivariate analysis

Variable	OR	95% CI	P
Age >50 years	1.02	1.001–1.04	0.037
APACHE II >25	1.06	0.99–1.13	<0.05
Period before year 2000	1.98	0.78–3.41	0.3
Septic shock	17.7	6.2–50.7	0.0001

Abbreviations: CI, confidence interval; OR, odds ratio.

Table 4

Characteristics of patients who underwent deferred primary anastomosis

Number	26
Age, mean, years	54.5
Male, %	61.5
Type of peritonitis, %	
Secondary	30.7
Post-traumatic	6.7
Postsurgery	61.6
Median, APACHE II, %	14.5
ICU, LOS, mean, days	17.9
LOS hospital, mean, days	27

Abbreviation: LOS, length of stay.

Table 5

Overall outcome in 26 patients with deferred primary anastomosis

Outcome	n	%
Primary success	20	77
Fistula	3	11.5
Stoma	3	11.5
Mortality at 28 days	3	11.5