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Objective: Intestinal obstruction is a blockage of the intestinal content through bowel. The block must be complete and permanent. Obstruction may be mechanical, simple or strangulated, and paralytic. The purpose of this chapter is to clarify, also evaluating our surgical experience, the steps to diagnose and the ways to treat intestinal obstructions.

Methods: In the period 2011–2015, we have treated 52 patients with the clinical presentation of intestinal obstruction. Acute mechanical small bowel obstructions were the most frequent (71.2%), whereas acute large bowel obstructions were the less frequent (28.8%). Some steps in the physical examination and plain radiography are as follows:

• Preliminary diagnosis: simple versus strangulated obstruction.
• First-step diagnosis: mechanical or paralytic obstruction.
• Second-step diagnosis (define the level of obstruction).
• Third-step diagnosis: kind of obstacle.

Results: In our experience, all complete intestinal obstructions have been treated with urgent surgery. Various types of surgical procedures have been employed based on intraoperative pathological findings.

Conclusion: Surgical emergency is the first choice of treatment in strangulation obstruction and in simple (complete) mechanical obstruction of small and large bowel. Generally, paralytic ileus can be resolved with the treatment that caused it. Intestinal pseudo-obstruction is a syndrome characterized by a complete dilatation generally of large bowel without mechanical obstacle. The chronic pseudo-obstruction can be idiopathic or secondary to systemic disease.

Keywords: intestinal management, obstruction, pathophysiology, strangulation, intestinal pseudo-obstruction
1. Introduction

Intestinal obstruction is a syndrome characterized by a blockage of the intestinal content, gas and liquid, through small or large bowel. The block must be complete and permanent.

There are several data that can diversify the intestinal obstruction syndrome. First, the etiology based on a large number of factors that allow the subdivision into mechanical and functional/paralytic obstruction.

The other feature characterizes the syndrome: the seat of the obstruction along the bowel—upper small gut, distal small gut, and large bowel.

Finally, the cause of obstruction can involve the vascular supply of an intestinal segment, giving rise to strangulation obstruction that should be differentiated from simple obstruction.

The syndrome of intestinal obstruction with these various etiopathological and clinical features develops the same, overlappable, and pathophysiological alterations.

2. Etiology

Bowel obstruction can be caused by several factors.

The causes of mechanical obstruction can be divided into causes within the bowel lumen, causes in the intestinal wall, and extrinsic causes.

The causes within the bowel are infrequent. They can be due to large gallstones passed into the intestinal lumen by spontaneous bilo-digestive fistulas, most frequently cholecystoduodenal fistulas, very rarely phito-thricobezoar, masses of parasites, food bolus, concretions of barium following barium enema X-ray investigation or X-ray studies with opaque medium.

It is useful to point out that the fecaloma, fecal impaction in the rectal ampulla, based on the damage of the autonomic nervous system in the colorectal wall, can cause chronic alteration of intestinal transit with incomplete obstruction without the pathophysiological alterations of acute gut obstruction.

The causes in the gut wall include the neoplasms of small and large bowel, the congenital atresias, the stenosis due to chronic inflammatory disease (Crohn disease, diverticulitis, etc.), and postanastomotic or posttraumatic structures.

The extrinsic causes include a very large range of pathological conditions: compression by external masses, adhesions, bands, strangulated external or internal hernias, volvulus, and intussusception [1]. In the clinical practice, it should be valuable to distinguish between acute and chronic obstructions. Nevertheless to define both clinical pictures with accuracy is very useful. The acute intestinal obstruction, simple or strangulated forms, is characterized by complete and permanent blockage and consequently the acute pathophysiological syndrome of obstruction may develop with all clinical, laboratory, and instrumental features: bowel
dilatation, disturbances of fluids and electrolytes balance, congestion, and ischemic parietal damage, etc.

On the contrary, in the chronic obstruction, the blockage of gut transit is incomplete and the syndrome cannot develop completely and is characterized only by constipation.

3. Classification of intestinal obstruction

Intestinal obstruction may be mechanical or paralytic. Mechanical obstruction can be due to intraluminal, intrinsic to the intestinal wall and extrinsic.

Paralytic ileus due to reduction or the absence of peristalsis can be caused by peritoneal phlogosis, infection, abdominal surgery, pelvic surgery, and some medications such as antidepressant, pain medications, muscle and nerve disorders, and retroperitoneal hemorrhage.

The majority of patients have simple obstruction. On the contrary, there is also strangulation obstruction, usually due to complicated external hernia (abdominal wall) or internal (by congenital defects or postoperative adhesions): in these patients the vascular supply to a strangulated intestinal segment is compromised and consequently intestinal infarction. Strangulation obstruction leads to an increased risk of morbidity and mortality.

In the mechanical occlusion with strangulation, the vascular (arterial and venous) occlusion leads to bowel ischemia and necrosis. The evolution of strangulated bowel is the perforation and peritonitis. The occlusive syndrome becomes worse due to strangulation.

Intestinal pseudo-obstruction is a syndrome characterized by a complete dilatation generally of large bowel without mechanical obstacle. The intestinal pseudo-obstruction can affect small or large bowel and it may be possible to differentiate the syndromes with acute or chronic onset and evolution.

The chronic pseudo-obstruction can be idiopathic or secondary to systemic disease.

4. Epidemiology

The examination of homogeneous clinical cases of a single center allows us to clarify the epidemiological features.

In the period 2011–2015, 52 patients have been admitted in our service with the clinical presentation of intestinal obstruction.

Demographic data are as follows: 52 patients, 26 males, 26 females, and mean age 67 years (range 27–86 years).

Acute mechanical small bowel obstruction was the most frequent (71.2%) with various pathologies: adhesion-relate obstructions, small bowel volvulus, gallstones ileus, malignan-
cies, abdominal wall hernias, internal hernias, carcinomatosis, and ileoceocolic intussusception (Figure 1).

Figure 1. CT scan: ileoceocolic intussusceptions.

The less frequent in our experience were acute large bowel obstructions (28.8%). The more common pathologies were colon and rectal cancer, sigmoid volvulus (Figure 2). We have observed and treated only one patient with acute colonic pseudo-obstruction (Ogilvie’s syndrome) (Table 1).

Figure 2. CT scan: sigmoid volvulus.
<table>
<thead>
<tr>
<th>Pathology</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adhesions</td>
<td>22</td>
<td>42.3</td>
</tr>
<tr>
<td>Small bowel volvulus</td>
<td>4</td>
<td>7.7</td>
</tr>
<tr>
<td>Sigmoid volvulus</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Right colon cancer</td>
<td>3</td>
<td>5.8</td>
</tr>
<tr>
<td>Left colon cancer</td>
<td>5</td>
<td>9.6</td>
</tr>
<tr>
<td>Rectal cancer</td>
<td>5</td>
<td>9.6</td>
</tr>
<tr>
<td>Ogilvie’s syndrome</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Gallstones ileus</td>
<td>2</td>
<td>3.8</td>
</tr>
<tr>
<td>Carcinomatosis</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Strangulated incisional hernia</td>
<td>3</td>
<td>5.8</td>
</tr>
<tr>
<td>Strangulated groin hernia</td>
<td>2</td>
<td>3.8</td>
</tr>
<tr>
<td>Strangulated umbilical hernia</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Internal hernia</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Ileocecocolic intussusception</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td></td>
</tr>
</tbody>
</table>

**Table 1.** Fifty-two acute intestinal obstructions: pathologies.

<table>
<thead>
<tr>
<th>Intervention</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adhesiolysis</td>
<td>18</td>
<td>34.6%</td>
</tr>
<tr>
<td>Small bowel resection</td>
<td>6</td>
<td>11.5</td>
</tr>
<tr>
<td>Right hemicolectomy</td>
<td>4</td>
<td>7.7</td>
</tr>
<tr>
<td>Left hemicolectomy</td>
<td>5</td>
<td>9.6</td>
</tr>
<tr>
<td>Intestinal derotation</td>
<td>4</td>
<td>7.7</td>
</tr>
<tr>
<td>Cecostomy</td>
<td>2</td>
<td>3.8</td>
</tr>
<tr>
<td>Enterotomy and gallstone removal</td>
<td>2</td>
<td>3.8</td>
</tr>
<tr>
<td>Anterior rectum resection</td>
<td>4</td>
<td>7.7</td>
</tr>
<tr>
<td>Total colectomy</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Prosthetic mesh repair incisional hernia</td>
<td>3</td>
<td>5.8</td>
</tr>
<tr>
<td>Umbilical hernia repair</td>
<td>1</td>
<td>1.9</td>
</tr>
<tr>
<td>Prosthetic mesh repair groin hernia</td>
<td>2</td>
<td>3.8</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2.** Fifty-two acute intestinal obstructions: surgical interventions.
In our experience, all intestinal obstructions have been treated with urgent surgery. Various types of surgical procedures have been employed based on intraoperative pathological findings. They should be useful to underline that the choice of the urgent treatment allowed the resolution of majority of adhesion-related obstructions and small bowel volvulus with the surgical procedure of adhesiolysis and intestinal derotation (42.3%). The intestinal resection has been performed in six cases (11.5%). Ogilvie’s syndrome required cecostomy; colorectal malignancies have been treated with primary tumors resection and delayed intestinal anastomosis. Our surgical interventions are reported in Table 2.

5. Pathophysiology of simple intestinal obstruction

In simple obstruction, important and progressive alterations take place and develop in the gut above the obstruction. Accumulation of gas and liquids with progressive distension in intestinal segments upstream the obstruction and the blockage of content progress change the bacteriological content. Also there are damages of blood circulation in the distended bowel wall. The accumulation of fluid and electrolytes in the obstructed gut and their loss in the general circulation take an important place in the pathophysiology of intestinal obstruction [2, 3].

5.1. Distension

The distension of obstructed bowel above the obstacle is due to the accumulation of fluid and gas. The intestinal gas that normally progresses by peristaltic movements shows the following composition at the start of occlusion: swallowed air, carbon dioxide (it comes from neutralization of bicarbonates), and later on gas bacterial fermentation. The carbon dioxide that forms in large quantities in the intestinal cavity participates minimally gaseous accumulation because it is largely absorbed by the intestinal mucosa.

<table>
<thead>
<tr>
<th>Gas</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrogen $\text{N}_2$</td>
<td>70%</td>
</tr>
<tr>
<td>Oxygen $\text{O}_2$</td>
<td>10–12%</td>
</tr>
<tr>
<td>Carbon dioxide $\text{CO}_2$</td>
<td>6–9%</td>
</tr>
<tr>
<td>Hydrogen $\text{H}_2$</td>
<td>1%</td>
</tr>
<tr>
<td>Methane $\text{CH}_4$</td>
<td>1%</td>
</tr>
<tr>
<td>Hydrogen disulphide</td>
<td>1–10%</td>
</tr>
</tbody>
</table>

Table 3. Intestinal obstruction: composition of intestinal gas.

On the contrary, the swallowed air significantly contributes to gaseous bowel distension because it contains a high rate of nitrogen content, which is not absorbed: in fact about 70% of the intestinal gas is constituted by nitrogen.
For this reason, the nasogastric aspiration in the patients with intestinal distension should be relevant and useful.

A little advantage can be added to gastric aspiration by the administration of pure oxygen to distended patients because increasing the pressure of a gas increases its solubility [4].

The common composition of intestinal gas in obstruction is reported in Table 3 [5, 6].

### 5.2. Fluid loss

In the distended bowel above, the obstruction gastrointestinal secretions accumulate in large amounts. This occurs for two reasons: deprivation of the absorptive activity of intestine beyond the obstruction and also damage in fluid and electrolyte exchange in the wall of the obstructed and distended gut [7].

<table>
<thead>
<tr>
<th>Source</th>
<th>Volume (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saliva</td>
<td>1500</td>
</tr>
<tr>
<td>Gastric secretion</td>
<td>2500</td>
</tr>
<tr>
<td>Bile</td>
<td>500</td>
</tr>
<tr>
<td>Pancreatic juice</td>
<td>700</td>
</tr>
<tr>
<td>Intestinal mucosa secretion</td>
<td>3000</td>
</tr>
<tr>
<td>Total</td>
<td>8200</td>
</tr>
<tr>
<td>Normal plasmatic volume</td>
<td>3500</td>
</tr>
</tbody>
</table>

Table 4. Volume of digestive secretion per day.

The saliva, gastric secretion, bile, pancreatic juice, and small intestinal secretion accumulate the total volume of about 8000 ml in 24 h as reported in more detail in Table 4. These fluid secretions are isotonique with the plasma, except for gastric secretion, which has minor sodium concentration (Figure 3) [8].

Therefore, normally water and electrolytes absorption is almost complete in the colon. In small bowel obstruction, the function of the colon cannot develop and the total intestinal secretions accumulate in the obstructed gut.

Beside the decreased absorption in the obstructed patients, there is also, above the obstacle, increased secretion into the bowel lumen.

In the pathophysiology of intestinal obstruction, the fluid and electrolytes loss plays a very important role. The progressive accumulation of gas and fluid in the intestinal lumen allows the increase in the endoluminal pressure to very high values: in the small bowel the pressure can reach 15 cm H$_2$O, whereas in the colon it can reach 25 cm H$_2$O. In the initial period of mechanical obstruction in the intestinal segments above the obstacle, active peristalsis causes further pressure rise to 20–30 cm H$_2$O [9]. In the large bowel, the intraluminal pressure can reach 50 cm H$_2$O because the pressure increase is based on the product of the pressure value...
multiplied by bowel diameter. The major detrimental effect of the progressive gut distension and intraluminal pressure increase is the impairment of the intramural circulation of the bowel.

Figure 3. Electrolytes composition in blood plasma and digestive secretion.

5.3. Pathophysiology of circulatory changes in the distended bowel

The experimental studies demonstrated the linear connection between bowel obstruction, increase in intraluminal pressure, gut wall distension, changes in blood flow [10]. In the clinical situations, there are some difficulties to connect the degree of intraluminal pressure, intestinal distension, and the damage of parietal blood perfusion.

In the pathological setting, the increase in the intraluminal pressure should develop slowly and should not reach high degree [11, 12].

The intestinal wall distension in the obstructed patients causes increased distensibility of the gut wall that becomes more vulnerable to a further increment of distension. In this way, a small rise in the intraluminal pressure and the wall distension allows considerable tension in the intestinal wall and increased resistance in the capillaries with damage bowel blood flow.

The ischemic necrosis of obstructed bowel should be caused by progressive thinning of gut wall, reduction of the lumen of vessels, and finally interruption of the blood supply [4].

In the pathogenetic sequence, start a self-handing mechanism because the parietal distension increases the intestinal secretions with further intraluminal fluid accumulation, increased wall distension, impaired parietal blood flow, and finally hypovolemia.

The intestinal secretions enhance because the capillary leak increases the fluid flux to intestinal lumen [12, 13].
Contrast studies, such as water soluble contrast material or contrast fluoroscopy, have indications and purposes rather limited in the obstruction diagnosed as complete and persistent.

Management

The first approach in the management of intestinal obstruction includes the correction of physiologic impairment caused by obstruction. Some measures can be required: the use of a bladder catheter to monitoring urine output, adequate intravenous access, arterial canalization, and CVP monitoring.

The purpose of the therapeutic approach is the correction of hypovolemia and electrolytes depletion with volume resuscitation. The development of fluid-electrolyte replacement and the adequacy of resuscitation should be guided by the degree of systemic impairment and the reaction of the patient to therapy. Therefore, aggressive replacement of fluid and electrolytes can be employed after restoration of renal function. The use of nasogastric tube for the control of severe intestinal distension can be helpful.

Antibiotics should be started at the confirmation of diagnosis of intestinal obstruction, mostly if fever, and leucocytosis is present.

The aim of the use of antibiotics is based on the control and treatment of intestinal overgrowth of bacteria and their translocation across the bowel wall [44]. Antibiotics, based on the particular type of bacterial overgrowth in the obstruction, could have more coverage against anaerobes and Gram-negative bacteria. The main objective of the therapeutic program of bowel obstruction is to remove the obstacle. Surgery is the leading option.

Three criteria guide this therapeutic choice:

- Degree of impairment of general conditions due to complications: intestinal ischemia, necrosis, perforation, and peritonitis;
- Etiology of obstructive syndrome (hypothesized or confirmed);
- Type of intestinal obstruction diagnosed (hypothesized or confirmed):
  - Complete versus incomplete
  - Small or large bowel obstacle site
  - Strangulation occlusion

Peritonitis and abdominal sepsis caused by complications of obstructive syndrome (perforation, ischemia, necrosis, etc.) prescribe urgent surgical intervention. The choices of the surgical procedures are conditioned by pathological findings, sometimes intraoperative.

Clinical instability, diagnostic uncertainly, unexplained leucocytosis and metabolic acidosis, and consequently the doubt of perforation or abdominal sepsis justify the abdominal surgical intervention.
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Antibiotics should be started at the confirmation of diagnosis of intestinal obstruction, mostly if fever, and leucocytosis is present. Antibiotics, based on the particular type of bacterial overgrowth in the obstruction, could have more coverage against anaerobes and Gram-negative bacteria. The main objective of the therapeutic program of bowel obstruction is to remove the obstacle. Surgery is the leading option.

Three criteria guide this therapeutic choice:

• Degree of impairment of general conditions due to complications: intestinal ischemia, necrosis, perforation, and peritonitis;
• Etiology of obstructive syndrome (hypothesized or confirmed);
• Type of intestinal obstruction diagnosed (hypothesized or confirmed):
  • Complete versus incomplete
  • Small or large bowel obstacle site
  • Strangulation occlusion

Peritonitis and abdominal sepsis caused by complications of obstructive syndrome (perforation, ischemia, necrosis, etc.) prescribe urgent surgical intervention. The choices of the surgical procedures are conditioned by pathological findings, sometimes intraoperative. Clinical instability, diagnostic uncertainty, unexplained leucocytosis and metabolic acidosis, and consequently the doubt of perforation or abdominal sepsis justify the abdominal surgical intervention.
Contrast studies, such as water soluble contrast material or contrast fluoroscopy, have indications and purposes rather limited in the obstruction diagnosed as complete and persistent.

**10. Management**

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exploration. Also the suspicion of strangulation occlusion, based on continuous and severe abdominal pains, should prompt surgery.

The surgical choice for irreducible or strangulated hernia of abdominal wall is very obvious. In the case of high suspicion for digestive malignancy, most frequently in large bowel, surgical intervention should be performed. In these cases, surgical procedures should contemporary treat both diseases, intestinal obstruction and digestive neoplasm: primary resection followed by temporary diversion (Hartmann procedure) or immediate reconstruction.

In summary, for the complete and permanent intestinal obstruction, the surgical intervention should be the first-line option.

In the management of intestinal obstructions, there are some issues under discussion with no simple solution.

The treatment of acute small bowel obstruction should be a common clinical challenge. The choice of operative management within the first 12–24 h from the onset can be followed by nontherapeutic laparotomy with the unfortunate results of further adhesions and postoperative morbidities [45]. Nasogastric decompression, fluid-electrolytes replacement, and careful clinical reassessment can have a considerable success rate in the approach of small bowel obstruction. Unfortunately, failure to acknowledge or late recognition of strangulation obstruction cause increased morbidity and mortality [46–48]. In this complicated setting, the solution is the selection of the patients. Of course, as stated previously, the cases with clinical and/or instrumental evidence of peritoneal phlogosis or perforation are excluded from this evaluation. Some criteria have been proposed to identify the patients with alleged simple small bowel obstruction for immediate operative treatment. Clinical appearance of fast onset of abdominal pain, continuous pain, not colicky, abdominal tenderness localized, or diffuse on physical examination suggest the choice of immediate surgical approach. There are also specific findings on abdominal CT: free intraperitoneal fluid, mesenteric edema, thickened wall, pneumatosis intestinalis, “small bowel feces signs” (gas bubbles and debris within the lumen of obstructed small bowel) [49]. On the other hand, the selected patients’ choice for nonoperative management should be characterized by the following criteria: the absence of abdominal wall hernias, previous abdominal pelvic surgery, previous abdominal malignancies, history and diagnosis of IBD (especially Crohn disease), colicky pain, absent abdominal tenderness on clinical assessment, and finally hemodynamic stability and absence of impairment of general conditions.

In summary, clinically stable patients with partial obstruction can be treated by conservative management [50].

Conservative management includes intestinal intubation and decompression, aggressive intravenous rehydration, and antibiotics [51].

The results of the conservative management of acute mechanical small bowel obstruction are uncertain and not conclusive, from the data of literature [52]. There are high success rates in the stable patients with incomplete obstruction [53, 54]. Among the patients with adhesive small bowel obstruction, 24.6% of patients are treated with nonoperative management,
without surgery or readmission [55]. On the other hand, high rates of recurrence and the risk of complications including vascular impairment were reported [47]. Therefore, among the patients managed conservatively to start with, the operative rates were very high because of the diagnostic difficulty to distinguish simple from strangulation obstruction on clinical and instrumental examinations [56].

The international guidelines [34, 57] for the evaluation and management of small bowel obstruction confirm and summarize the data from the literature.

The guidelines in evidence are as follows:

- The instrumental diagnosis should be based on the CT scan of abdomen because it can clarify the grade, severity, and etiology of small bowel obstruction.

- Urgent surgical approach is the first option for small bowel obstructions with evidence of peritonitis or clinical deterioration (fever, tachycardia, leucocytosis, and metabolic acidosis).

- Patients with partial or complete small bowel obstruction and stable general conditions and without physical and instrumental signs of peritoneal phlogosis can undergo initial nonoperative management.

- Water-soluble contrast study can be useful in partial small bowel obstruction not resolved within 48 h based on the improvement of water-soluble contrast on bowel function.

- After 3–5 days of conservative management, the patients with small bowel obstruction should undergo surgery;

- Laparoscopic treatment can be a safe and possible procedure for small bowel obstruction, but not commonly employed. In fact, its use requires some selection criteria: proximal obstruction, localized distension on radiography, no sepsis, and mild abdominal distension [58–60].

Large bowel obstruction due to colorectal cancer requires the treatment of malignancy and abdominal urgency. The first objective should be the control and management of malignancy. Several factors may influence the therapeutic choice: the location of the tumor (proximal-distal colon), the degree of colonic distension and the impairment of blood flow of intestinal wall, the involvement of the general conditions of the patient with organ failure, dehydration, hypovolemia, and sepsis.

In this scenario, variable and complex, it is very difficult to establish a well-defined and unequivocal line therapy in relation to the surgical procedure to be used. There are several proposals: at the beginning resolution of the occlusive complication only with colostomy (two-stage procedure) followed, sometime later, by resection of neoplastic lesion (with radical surgical criteria or palliative). On the other hand, a one-stage procedure with resection of the tumor (radical or palliative), followed by temporary colostomy (Hartmann’s procedure) or primary anastomosis, can be employed.

Endoscopic colonic stents have been proposed in neoplastic obstruction of distal colon for palliation or as a bridge to surgery [61]. With the palliative intent, the colorectal stent can be
used as an alternative to colostomy, whereas in the hospitals it can be employed as a bridge to elective surgery with specific expertise [62].

The tumor site along the colon is an important factor for the therapeutic choice. Right colectomy can be defined as the treatment of choice for right-sided colon cancer in obstruction setting. It is a safe technique for one-stage resection and anastomosis [63]. The guidelines of World Society of Emergency Surgery suggest some recommendations on obstructive left colon carcinoma [62]. Hartmann’s procedure should be the preferred choice in the patients with impaired blood supply of intestinal wall and high surgical risk compared to loop colostomy. Hartmann’s procedure shows overlapped survival results compared to segmental colonic resection with primary anastomosis.

The patients submitted to primary resection and anastomosis have similar mortality-morbidity rates with total or subtotal colectomy and segmental colectomy. The immediate results are similar in these patients (with primary resection and anastomosis) related to the choice of intraoperative colonic irrigation or manual decompression.

11. Intestinal pseudo-obstruction

Intestinal pseudo-obstruction is a syndrome characterized by a complete intestinal dilatation, generally of large bowel, without mechanical obstacle. The proposal nosography of intestinal pseudo-obstruction is reported in Table 8.

<table>
<thead>
<tr>
<th>Intestinal pseudo-obstruction</th>
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<tbody>
<tr>
<td>Acute</td>
</tr>
<tr>
<td>Hydro electrolytes alterations</td>
</tr>
<tr>
<td>Anticholinergic, ganglionic blocker drugs</td>
</tr>
<tr>
<td>Laparotomy</td>
</tr>
<tr>
<td>Intraperitoneal diseases (perforation, appendicitis, cholecystitis, pancreatitis)</td>
</tr>
<tr>
<td>Extraperitoneal diseases (hematoma, vertebral trauma, pneumonia, myocardial infarct)</td>
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<tr>
<td>Sclerosing therapy of esophagus</td>
</tr>
<tr>
<td>Acute colonic pseudo-obstruction</td>
</tr>
<tr>
<td>Chronic</td>
</tr>
<tr>
<td>Secondary</td>
</tr>
<tr>
<td>Endocrine disease (diabetes, hypothyroidism)</td>
</tr>
<tr>
<td>Collagenopathies (scleroderma, dermatomyositis)</td>
</tr>
<tr>
<td>Neuropathies (Parkinson’s disease, multiple sclerosis)</td>
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<tr>
<td>Degenerative diseases (amyloidosis)</td>
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<tr>
<td>Elevated blood levels of prostaglandin A, E, F</td>
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<tr>
<td>Idiopathic</td>
</tr>
<tr>
<td>Hereditary hollow visceral myopathy</td>
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<tr>
<td>Familial neuronal disease</td>
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</tbody>
</table>

Table 8. Intestinal pseudo-obstruction: nosography.
The intestinal pseudo-obstruction can affect small or large bowel and it may be possible to differentiate the syndromes with acute or chronic onset and evolution.

Paralytic ileus can be inserted in the broader field of acute pseudo-obstructions that are based on pathophysiological impairment of intestinal peristalsis (intestinal paresis). The acute pseudo-obstruction can be caused by severe fluid electrolytes disorders (hypokalemia, hypocalcemia), medications with anticholinergics or opiates, abdominal interventions (postoperative ileus), inflammatory and septic abdominal diseases as peritonitis, pancreatitis, perforations, intestinal ischemia, and retroperitoneal trauma.

In this area, the acute colonic pseudo-obstruction (ACPO) (Ogilvie’s syndrome) should be highlighted. The Ogilvie’s syndrome has been diagnosed in association with various pathologies such as cholecystitis, acute pancreatitis, retroperitoneal traumatic hematoma, Parkinson disease, and so on. The impaired colonic motility should be caused by imbalance in the autonomic nervous system: increase in sympathetic tone and decrease in parasympathetic tone [64]. Increased sympathetic tone to the colon results in the inhibition of colonic motility [65]. The pathologic findings, more evident in the right colon, are distension, fluid-gaseous accumulation, and increased endoluminal pressure. Severe blood circulatory impairment can occur in the large bowel wall with damage of venous return, edema, trophic mucosal alteration to serous, sometimes necrosis, and perforation. The cecum is more dilated colonic section. According to Laplace’s law, the cecum, with its larger diameter, requires less pressure to increase in size and in wall tension. Ischemia, longitudinal splitting of serosa, and herniation of the mucosa and perforation, the so-called “diastasis breaking” of cecum, is caused with the increased wall tension.

Clinical features are obvious abdominal distension as earliest sign, no tenderness, hyperresonance, or tympany to percussion throughout the abdomen, and no peristalsis and bowel sounds to auscultation. The symptoms are similar to large bowel obstruction and develop over 3–7 days.

Imaging examinations, plain radiography, CT scan should help to exclude mechanical bowel obstruction. The size of the cecum (more than 8–10 mm) on abdominal films could be useful to decide colonic decompression, surgical or endoscopic, because of the risk of perforation [66].

In the ACPO, conservative treatment can be proposed: no oral intake, nasogastric decompression, correction of fluid and electrolytes disorders, and discontinuance of drugs that inhibit gastrointestinal motility [67].

In the conservative option, the use of pharmacologic agents can be added to increase colonic motility. Several drugs have been employed with nonunique and uncertain results, erythromycin, cisapride, metoclopramide, and neostigmine [68, 69]. The invasive therapeutic approach of Ogilvie’s syndrome includes colonoscopic decompression and surgical intervention. The endoscopic decompression should be a safe and effective procedure for ACPO and has been associated with high success rates (77–86%) [70].

Recurrence rates of colonoscopy decompression are also high, ranging from 20 to 60% [71]. Clinical signs of ischemia, abdominal sepsis, perforation, or failure of conservative manage-
ment require surgery. The choice of surgical procedure is indicated by intraoperative pathological findings: tube cecostomy, subtotal colectomy, etc.

Chronic intestinal pseudo-obstruction can be subdivided into secondary and idiopathic. In the first cases, the chronic pseudo-obstructions are part of severe systemic diseases: endocrine diseases (diabetes and hypothyroidism) and collagenopathies (scleroderma, amyloidosis, dermatomyositis, and lupus erythematosus). The chronic intestinal idiopathic pseudo-obstructions are frequently familiar diseases. Pathophysiology is not completely defined and should be based on derangement of autonomic nervous system with alteration of intestinal motility. The pathophysiological findings are hereditary hollow visceral myopathy, familiar neuronal visceral disease, lesion of myenteric plexus, and alimentary tract ganglioneuromatosis.

The role of interstitial cells of Cajal has been hypothesized in the pathogenesis of idiopathic chronic intestinal pseudo-obstruction. Electron microscopy and immunochemistry studies showed a decreased number of interstitial cells of Cajal in the intestinal wall and alterations in interstitial cells of Cajal network [72].

According to clinical features, the cramping abdominal pain is more frequent; nausea, vomiting, and abdominal distension are occasional, sporadic. There are also alternating diarrhea and constipation and weight loss. Finally, the clinical evolution of the disease is chronic.

The results of the therapy of secondary forms are scanty and uncertain; usually, the therapy is connected with the treatment of the serious systemic diseases. Drugs that stimulate the smooth muscles (acetylcolinesterase inhibitors) can be employed in the idiopathic forms. Surgical procedures are indicated for the treatment of severe and continuous symptomatology that can be related to a portion of the digestive tract or for complete failure of conservative therapy [73].

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References


