



Gastrointestinal surgery and pregnancy

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Gastrointestinal disorders that require surgery occur in 0.5% to 1% of pregnancies [1]. The diagnosis and timely management of these conditions can be challenging because the signs and symptoms of these infrequent diseases can be similar to more frequent conditions that do not require surgery and occur during a normal pregnancy, particularly in the first trimester. Therefore, proper evaluation requires clinical acumen and close attention to avoid a delay in diagnosis, which can be devastating to the patient and fetus. This article reviews the physiologic, gastrointestinal changes that take place during pregnancy, and the gastrointestinal disorders during pregnancy that require surgery, with a focus on how pregnancy alters the clinical presentation and management of these disorders.

General considerations

Anatomic and physiologic changes

Various anatomic and physiologic changes occur during normal pregnancy, which can alter the presentation of conditions that require surgery. Anatomic displacement of intraperitoneal organs by the gravid uterus can affect the clinical presentation. These displacements can alter the location of abdominal pain and tenderness, as well as influence incision placement. As the gravid uterus grows, it can exert pressure on the inferior vena cava in the supine position that can decrease venous return of blood and exacerbate the effects of blood loss or dehydration [2].

Physiologic changes during pregnancy, particularly in the circulation, alter the maternal response to stress. Cardiac output increases by 30% to 50% by the end of the second trimester, at which time it plateaus, and then decreases to more normal levels [3]. The heart rate increases progressively to become

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approximately 15% above normal in the last trimester. Heart rate and stroke volume increase because of the expanded blood volume during pregnancy and a direct inotropic effect of estrogen [3]. Arterial blood pressure, particularly the diastolic pressure, decreases slightly during pregnancy, whereas venous blood pressure increases as pregnancy advances. Plasma volume and erythrocyte volume increase, but the plasma volume increases relatively more, thereby producing a physiologic anemia of pregnancy. The physiologic increase in blood volume can mask the pathologic effects of intravascular volume loss and dehydration. Because of these changes, cardiac work increases despite decreased systemic vascular resistance. Patients with normal cardiac function adapt well to these changes, but patients with little myocardial reserve can exhibit significant dysfunction during pregnancy.

Minute ventilation increases progressively during pregnancy to become approximately 50% above normal near term [4]. Diaphragmatic displacement by intra-abdominal organs caused by the growing gravid uterus results in a decrease in residual lung volume and functional residual capacity. The pregnant patient is consequently more vulnerable to arterial oxygen desaturation. Alveolar ventilation increases by way of an increase in tidal volume, because of direct stimulation by progesterone, and an increase in respiratory rate [5]. These respiratory changes are associated with a reduction in $p\text{CO}_2$ and an increase in $p\text{O}_2$, with minimal change in oxygen saturation. Other respiratory parameters, such as vital capacity and maximum breathing capacity, remain normal. Pulmonary artery pressures remain normal, but pulmonary vascular resistance decreases because of changes in blood volume and cardiac output [3,5].

The increased serum progesterone level early in pregnancy decreases gastrointestinal smooth muscle activity [6]. Gastric acidity increases somewhat, gastric emptying is slightly delayed, and gastric dysrhythmias associated with nausea can occur [6]. The enlarging, gravid uterus compresses abdominal viscera which results in decreased intestinal motility, a diminished response to peritoneal irritation, and altered pain perception.

The urinary collecting system gradually dilates, sometimes producing urinary stasis that can predispose to pyelonephritis. Endocrinologic changes during pregnancy include an increase in the serum levels of glucocorticoids and aldosterone. During pregnancy, basal metabolic rate and oxygen consumption progressively increase, and water, salts, and proteins are retained [4].

Laboratory studies

Various laboratory studies are affected by pregnancy. Along with a decreased hematocrit, fibrinogen and plasma proteins increase. Higher levels of clotting factors, particularly factors VII, VIII, X, and XII, result in a hypercoagulable state [7]. The maternal blood oxygen dissociation curve is shifted to the right, which promotes oxygen release from maternal hemoglobin into the fetal bloodstream, whereas the fetal blood oxygen dissociation curve is shifted to the left, which promotes fetal oxygen retention. Serum

sodium levels generally decrease slightly, and other commonly measured serum cations may also decrease. Blood pH remains unchanged.

The leukocyte count generally increases to approximately 12,000/ μ L during pregnancy and can increase to more than 20,000/ μ L during labor [2,8]. The platelet count may decrease because of the dilutional effect of increasing blood volume. Serum alkaline phosphatase and transaminase levels increase slightly.

Symptoms and signs

Various symptoms and signs that normally occur during pregnancy can be confused with acute abdominal diseases. Nausea and occasional vomiting affect 50% or more of women during pregnancy, particularly in the first trimester [6]. Abdominal pain and dyspepsia frequently occur [6].

The most common symptoms of acute abdominal illnesses during pregnancy are abdominal pain, nausea, vomiting, and abdominal distention. The usual signs of an acute abdomen are present during pregnancy, but may be altered because of displacement of intra-abdominal organs by the enlarging, gravid uterus. Radiologic studies, such as abdominal ultrasound remain useful to establish a diagnosis, but use of traditional radiographs and computed tomography may be limited by radiation teratogenicity.

Perioperative care

Normal variations in pregnancy may require correction before surgery, particularly restoration of intravascular volume and electrolyte abnormalities. Hypovolemic shock may respond to simple maneuvers, such as placing the pregnant patient in a left lateral decubitus position to avoid obstruction of venous return through the inferior vena cava that is caused by uterine compression [2]. This maneuver can also help restore uterine perfusion. Other abnormalities that are sometimes associated with pregnancy, such as hyperglycemia or hypertension, should be corrected. Fetal monitoring should be performed, in conjunction with an obstetrician, whenever an acute surgical condition is suspected and fetal heart tones are detectable [9]. Tocolytic use depends upon the fetal effects of the acute disease and the therapy. Routine prophylactic administration of tocolytics is not recommended because of scant evidence of beneficial maternal or fetal effects [10].

Abdominal incisions generally heal well during pregnancy. Incision location depends on the abdominal disorder, the stage of pregnancy, and anatomic changes caused by the enlarged, gravid uterus. Laparoscopy can be performed during pregnancy with less morbidity and greater patient acceptance than laparotomy under appropriate circumstances. Laparoscopy is relatively contraindicated during the third trimester, however. Laparoscopy in early pregnancy is minimally affected provided the technique is modified [11]. The initial trocar insertion should be done under direct vision rather

than blindly [9]. The periumbilical trocar is often moved to a supraumbilical position, and subsequent trocar placements may be modified because of anatomic alterations. A lower insufflation pressure (≤ 12 mm Hg) should be used to avoid reduced placental perfusion.

Arterial oxygen saturation, the end tidal partial pressure of carbon dioxide, and routine vital signs, should be monitored in the perioperative period [4,9]. Induction of general anesthesia should be preceded by administration of 100% oxygen for at least 3 minutes, and the concentration of oxygen in the anesthetic mixture should be 40% or more. Precautions are taken to avoid respiratory obstruction and hypoventilation. Five hundred to 1000 mL of lactated Ringer's solution should be administered before anesthetic induction to avoid hypotension. Regional anesthesia (eg, spinal or epidural anesthesia) can eliminate autonomic compensatory vasoconstriction, can cause a greater decline in arterial blood pressure than occurs in the nonpregnant state, and can cause hypotension which is dangerous to mother and fetus [4]. General anesthesia can be hazardous under emergency situations because of the risk of aspiration of gastric contents. Properly administered anesthetic agents should minimally affect perioperative maternal outcome.

Historically, abdominal operations that were performed under general anesthesia during pregnancy were associated with an increased risk of spontaneous abortion or premature labor [9]. This risk is least during the second trimester; elective operations should be delayed until then, if possible. Recent reports suggest that the risk of fetal wasting and teratogenicity from gastrointestinal operations during pregnancy are minimal [12–15].

Specific diseases

Acute appendicitis

Acute appendicitis is the most common gastrointestinal disorder that requires surgery during pregnancy [15]. Acute appendicitis is no more frequent during pregnancy than at other times, and occurs once in every 1500 to 2000 pregnancies [16]. Most cases occur during the first two trimesters, although it can occur at any time during pregnancy.

The symptoms and signs of acute appendicitis are similar to those in nonpregnant patients. They include anorexia, nausea, vomiting, and periumbilical abdominal pain that eventually migrates to the right lower quadrant. As discussed earlier, some symptoms can be confused with normal pregnancy-related conditions, particularly during the first trimester. The occurrence of these symptoms after the first trimester, the sudden onset of new symptoms, or the exacerbation of symptoms requires prompt evaluation.

Nearly all patients with appendicitis develop right-sided abdominal pain. Changes in appendiceal location because of increasing uterine size may affect the location of abdominal pain and tenderness (Fig. 1) [17]. During the first trimester, the appendix is usually not displaced from its normal

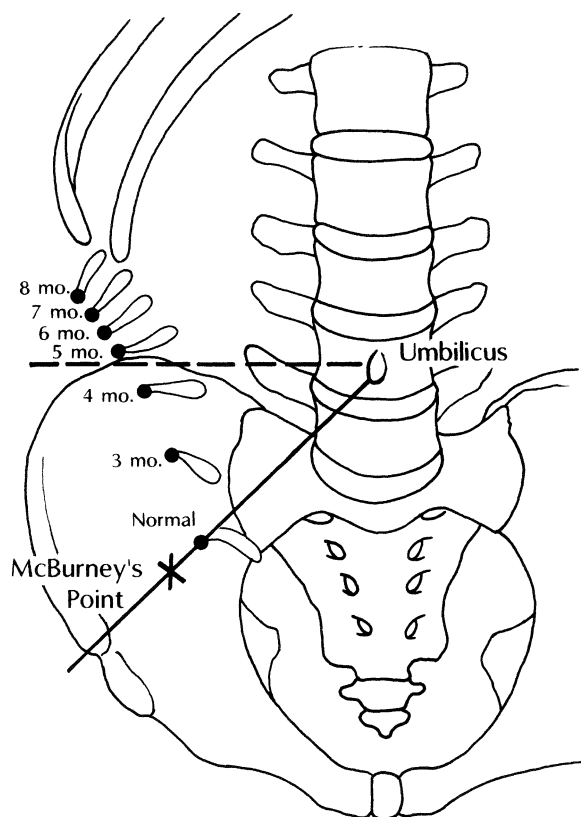


Fig. 1. Changes of appendix position during pregnancy. (Adapted from Reis RA, Arena RA. Appendicitis in pregnancy with changes in position and axis of normal appendix in pregnancy. JAMA 1932;98:1359; with permission.)

anatomic location. During the second trimester, the appendix is gradually displaced upward and laterally so that the appendiceal tip becomes located above McBurney's point. By the third trimester, the appendiceal tip is usually located in the right upper quadrant. This displacement affects the location of the pain from acute appendicitis. Later in pregnancy, the appendix may no longer be in contact with parietal peritoneum. This can alter the pain pattern and could result in delayed diagnosis [18,19].

Abdominal guarding, rebound tenderness, and referred tenderness occur in 60% to 70% of patients with acute appendicitis [20]. Guarding is a less reliable sign during late pregnancy because of laxity of the abdominal wall muscles. Approximately 25% of patients with acute appendicitis have pyrexia, and 15% have tachycardia. Rectal or pelvic tenderness occurs in a minority of patients, and becomes less common as the pregnancy progresses and the appendix migrates from its normal pelvic location.

Because of the physiologic leukocytosis of pregnancy, a moderate leukocytosis is nonspecific. More than 80% neutrophils on a leukocyte differential helps distinguish pregnant women with acute appendicitis from those with abdominal pain who have a normal appendix at operation [21]. Adjunctive radiologic testing may help exclude various other disorders in the differential diagnosis during pregnancy (Box 1). Abdominal CT utilizing intravenous, oral, and sometimes rectal contrast reduces the false positive appendectomy rate in nonpregnant patients. [22] CT can be used to diagnose appendicitis during pregnancy, but its applicability may be limited by radiation teratogenicity. Abdominal ultrasound is helpful in the first trimester, but test accuracy declines during later pregnancy as the appendix migrates.

Once appendicitis is diagnosed, immediate surgery is recommended to avoid appendiceal perforation. Laparoscopic appendectomy can be done

Box 1. The differential diagnosis of acute appendicitis during pregnancy

Gastrointestinal diseases

Acute cholecystitis
Biliary colic
Gastroenteritis
Intestinal obstruction
Mesenteric adenitis
Pancreatitis

Gynecologic diseases

Degenerating leiomyoma
Endometriosis
Ovarian torsion
Pelvic inflammatory disease
Ruptured ovarian cyst

Obstetric diseases

Chorioamnionitis
Ectopic pregnancy
Placental abruption
Ruptured corpus luteum cyst

Urologic diseases

Pyelonephritis
Urolithiasis

safely during the first two trimesters, but is generally difficult to perform after the uterus projects above the umbilicus [9,12,15]. Diagnostic laparoscopy is helpful in equivocal cases, and has reduced the false positive appendectomy rate to less than 15% [23]. In an open operation, a standard muscle splitting incision on the right side of the abdomen is performed. The incision should be placed over the point of maximal tenderness, which results in a gradual cephalad movement of the incision as the pregnancy progresses. When diffuse peritonitis is present, a midline incision should be performed; laparoscopic appendectomy is contraindicated in this situation because of a high complication rate [24].

Appendectomy is delayed only during active labor, in which case it is performed immediately following delivery. If labor is prolonged or appendiceal perforation is suspected, cesarean section should be performed followed immediately by appendectomy. Maternal mortality is unusual, but the mortality increases with advanced gestation and appendiceal perforation. Maternal mortality is as high as 4% in patients with perforated appendicitis, and is related to more frequent generalized peritonitis, because of the diminished ability of omentum to localize the perforation as uterine size increases. Fetal mortality ranges from zero to 1.5% in patients with nonperforated appendicitis, but increases to as much as 20% when perforation is present [12,14–16]. The incidence of preterm delivery and other complications are similar between open and laparoscopic approaches [12].

An appendiceal abscess can be treated by percutaneous drainage together with antibiotics [25]. Successful management may be followed by interval appendectomy, 6 weeks to 3 months later.

Patients with acute appendicitis should receive perioperative antibiotics. A second-generation cephalosporin, extended-spectrum penicillin, or carbapenem are safe choices [26]. Antimicrobial therapy should include anaerobic coverage because more than 95% of patients grow *Bacteroides fragilis* on culture. A perioperative prophylactic antibiotic can be administered either as a single dose or for up to 24 hours unless perforation has occurred, in which case therapy should continue until fever has disappeared, bowel function has returned, and the leukocyte count has normalized. Skin closure should be avoided in the presence of appendiceal perforation or gangrene because of a higher rate of wound infection.

Biliary tract diseases

Biliary tract diseases represent the second most common gastrointestinal disorder that requires surgery during pregnancy [15,27]. Pregnancy predisposes to gallstone formation because of increased bile lithogenicity and decreased gallbladder contractility caused by the effects of estrogen [18,28]. Gallstones occur in approximately 3% of pregnant women, but most patients are asymptomatic [29]. The incidence of acute cholecystitis is one to eight cases per 10,000 pregnancies [27,30,31].

The symptoms of biliary tract diseases are similar to those in nonpregnant patients and are usually caused by intermittent cystic duct obstruction. Common symptoms include anorexia, nausea, and vomiting. The precipitation of these symptoms by fatty foods, accompanied by right upper quadrant or epigastric pain, is suggestive of gallbladder disease. The pain may radiate to the back or right shoulder. Biliary colic usually begins abruptly and lasts from 15 minutes to several hours. The most important signs are right upper quadrant tenderness and Murphy's sign. Pyrexia, tachycardia, and leukocytosis usually indicate that acute cholecystitis is present. Serum alkaline phosphatase is often elevated. Jaundice and abnormal hepatic transaminases suggest possible complicated biliary tract disease, particularly choledocholithiasis [32]. Hyperamylasemia usually indicates that gallstone pancreatitis is present because serum amylase levels are not affected by pregnancy [33].

The differential diagnosis includes other acute abdominal inflammatory disorders such as appendicitis, pancreatitis, peptic ulcer disease, spontaneous hepatic rupture, and pyelonephritis. Pre-eclamptic and hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndromes can also mimic gallbladder disease [32].

A right upper quadrant ultrasound should be performed for suspected gallbladder disease. Ultrasonography has an accuracy of 97% or higher in detecting cholelithiasis [34]. Ultrasound seems to have no adverse maternal or fetal effects [35]. Findings such as gallbladder wall thickening, pericholecystic fluid, common bile duct dilation, or the presence of gallstones signify biliary tract disease. The pancreas and liver can also often be evaluated by ultrasound, which can aid in the diagnosis.

The initial management of biliary tract diseases is medical, including discontinuing oral intake, intravenous fluid replacement, analgesia, and administration of antibiotics when signs of infection are present. Cefazolin or an extended spectrum penicillin are effective against the common biliary pathogens and are relatively safe during pregnancy [26]. Patients who have evidence of gallstone pancreatitis should have serial monitoring of serum amylase.

Patients who fail to respond to medical therapy or who develop recurrent symptoms should undergo cholecystectomy. Early operation is advocated in these situations to avoid biliary complications and fetal loss because biliary colic commonly recurs during pregnancy [36]. Cholecystectomy should be deferred until the second trimester whenever possible, because of an increased rate of fetal loss when it is performed during the first trimester [36,37]. When symptoms are mild or the patient is in the third trimester, cholecystectomy can often be delayed until postpartum. Operation should be performed early for patients with gallstone pancreatitis, choledocholithiasis, or unresolving acute cholecystitis, regardless of gestational age.

Laparoscopic cholecystectomy can be performed in most pregnant patients, particularly during the first two trimesters [12,13,37]. Advantages

of laparoscopic cholecystectomy include less abdominal pain, fewer incisional complications, and shorter hospitalization. The fetus should be closely monitored intraoperatively. If fetal distress occurs, temporary desufflation of the pneumoperitoneum or conversion to an open procedure should be strongly considered.

Routine, intraoperative cholangiography is not recommended during pregnancy, and is reserved for patients who have evidence of common bile duct obstruction, a history of pancreatitis, or persistently abnormal serum transaminase levels. If cholangiography is necessary, the fetus should be shielded to minimize irradiation, particularly during the first trimester. The presence of choledocholithiasis can alternatively be evaluated by intraoperative ultrasound, when available [38]. When discovered intraoperatively, choledocholithiasis can be removed by laparoscopic or open common bile duct exploration, or by intraoperative or postoperative endoscopic retrograde cholangiopancreatography (ERCP) [16,39,40].

Choledocholithiasis is rare during pregnancy. ERCP with endoscopic sphincterotomy can be used for stone extraction or stent insertion during pregnancy [70]. The safety of ERCP during pregnancy is considered in detail elsewhere in this issue. Patients who have successful clearance of stones from the common bile duct by ERCP can often have cholecystectomy delayed until postpartum [9].

Maternal and fetal complications are uncommon and the likelihood of preterm delivery is minimized when elective operation is done in the second trimester [37]. Fetal mortality increases when acute infection or other complications are present, when pancreatitis occurs, or when surgery is inappropriately delayed.

Pancreatitis

Pancreatitis during pregnancy is usually caused by gallstones. Patients present with severe epigastric or upper abdominal pain, which can radiate to the mid-back and is usually accompanied by hyperamylasemia. Failure of the serum amylase to rapidly normalize, or the presence of two or more unfavorable prognostic signs of pancreatitis indicate complicated disease [84]. When complicated pancreatitis is suspected, abdominal CT should be performed. CT should be avoided in uncomplicated cases.

Pancreatitis not caused by gallstones can usually be managed medically. Laparotomy with débridement of pancreatic necrosis is indicated for pancreatic infection or a deteriorating condition. A pancreatic pseudocyst that is larger than 6 cm in diameter should be treated by internal or external drainage.

Spontaneous hepatic rupture

Spontaneous hepatic rupture is a rare and frequently lethal complication of pregnancy. The incidence is estimated at one per 45,000 pregnancies [32].

Hepatic rupture is a pathologic process that is unique to pregnancy. Ninety percent of cases are associated with pre-eclampsia or the HELLP syndrome [41]. Patients with HELLP syndrome have a 1% incidence of subcapsular hematoma [40,41]. Other risk factors for spontaneous hepatic rupture include maternal hypertension, and poor prenatal care. It occurs most often in older, multiparous women, usually in the third trimester of pregnancy, or at term.

A subcapsular hematoma is a precursor to hepatic rupture. The most common symptom of subcapsular hepatic hemorrhage is epigastric or right upper quadrant abdominal pain, often radiating to the right shoulder [41]. Patients may experience pain for several days or weeks before presentation. Physical examination usually reveals right upper quadrant tenderness. Abdominal distention, shock, and signs of peritonitis are usually present when rupture occurs.

Laboratory evaluation usually shows mildly elevated hepatic transaminases, hyperbilirubinemia, thrombocytopenia, low hematocrit, and an abnormal coagulation profile [40,41]. Abdominal ultrasound can usually distinguish hepatic rupture from biliary tract disease, and can be diagnostic for a subcapsular hematoma. Abdominal CT can also be diagnostic. Rupture occurs more commonly in the right hepatic lobe [4].

An asymptomatic hematoma should be treated with bedrest, correction of coagulation abnormalities, and treatment of underlying medical conditions, such as hypertension or eclampsia. Continuous fetal monitoring and serial evaluation of the hematoma by abdominal CT scan or ultrasound are mandatory. Delivery is often curative. Some patients undergo cesarean section to evaluate the extent of the hematoma.

When signs of hepatic rupture or hematoma expansion are present, the patient should undergo immediate cesarean section. The hematoma is generally treated by hepatic packing, with subsequent reoperation to remove the packing [42]. Other measures to control bleeding, such as hepatic resection or hepatic artery ligation, are infrequently used [43].

Hepatic rupture is associated with a poor outcome. Maternal mortality is high and fetal mortality exceeds 50% [85]. Patients with frank rupture do not survive without operation [76].

Intestinal obstruction

Intestinal obstruction has a reported incidence of between one in 1500 to 17,000 pregnancies [44–46]. As in nonpregnant patients, adhesions are the most common cause, but volvulus is a prominent cause during pregnancy (see Table 1) [47]. Intestinal obstruction most commonly occurs during the second and third trimesters [46].

The diagnosis and treatment of intestinal obstruction during pregnancy is similar to that in the nonpregnant state. Common symptoms include nausea and vomiting [48]. Abdominal distention with hyperactive bowel sounds are

Table 1
Causes and mortality from intestinal obstruction during pregnancy

Cause	Incidence (%)	Mortality (%)	
		Fetal	Maternal
Adhesions	59	28	5
Volvulus	23	20	13
Intussusception	5		
Hernia	3		
Cancer	1		
Acute appendicitis	1		
Overall		26	6

Adapted from Perdue PW, Johnson HW, Stafford PW. Intestinal obstruction complicating pregnancy. *Am J Surg* 1992;164:384; with permission of Excerpta Medica Inc.

frequent signs. The presence of bilious vomiting increases the likelihood that intestinal obstruction is present; feculent vomiting is rarely due to another cause [49].

When intestinal obstruction is suspected, supine and upright abdominal radiographs are useful to establish the diagnosis, and expose the fetus to minimal radiation. Early radiographic evaluation is mandatory because of significant maternal and fetal risks if the diagnosis is delayed.

Fluid and electrolyte shifts commonly accompany intestinal obstruction. Initial treatment includes nasogastric decompression, replacement of fluid and electrolyte deficits, and administration of moderate levels of analgesia. Colonic volvulus may be treated initially by decompression by way of colonoscopy or a rectal tube [50]. Persistent tachycardia, pyrexia, localized abdominal pain, leukocytosis, or failure of symptoms to resolve within 48 hours are indications for surgery [48]. Surgery is mandatory when intestinal ischemia or perforation is suspected.

Hernias are an unusual cause of intestinal obstruction during pregnancy. Diaphragmatic hernias have an increased risk of complications during pregnancy because of increased intra-abdominal pressure from the enlarging uterus. An incarcerated diaphragmatic hernia incidentally discovered during surgery should be treated by elective repair because of the risk of gastrointestinal strangulation with an associated high maternal mortality [83].

Exploration is done through a midline celiotomy, and treatment varies according to the underlying cause of obstruction. Maternal mortality ranges from zero to 6% and fetal mortality ranges from 25 to 40% [51]. Preterm delivery occurs in most cases. The mortality is greater when volvulus or intestinal ischemia is present.

Intestinal pseudo-obstruction

Intestinal pseudo-obstruction (Ogilvie's syndrome) is characterized by gaseous distention of the colon without mechanical obstruction. This condition

usually occurs following delivery [45]. Nausea, constipation, and abdominal distention are common symptoms and signs. The diagnosis is made by the presence of diffuse colonic distension on abdominal radiograph in the absence of any focal lesion. Treatment consists of nasogastric suction, intravenous fluid replacement, and decompression of the colon. Neostigmine or colonoscopic decompression often helps resolve this condition without the need for operation [52].

A colonic diameter that is larger than 10 to 12 cm is associated with a higher incidence of colonic perforation [45]. The mortality of patients with postpartum intestinal pseudo-obstruction was reported to be as high as 8% for patients without perforation and 70% when perforation occurs [31]. Operative decompression is indicated when colonoscopic decompression or neostigmine are unsuccessful, or when signs of intestinal ischemia occur. At surgery, tube cecostomy is often effective, with colonic resection reserved for patients with perforation or gangrene [31,45].

Splenic artery aneurysm

Splenic artery aneurysms are four times more likely in women than men, and are more likely during pregnancy [53]. Approximately 25% of splenic artery aneurysm ruptures occur during pregnancy [54]. These aneurysms are usually caused by fibromuscular dysplasia of the medial layer of the arterial wall. Female reproductive hormones affect the elastic properties of the arterial wall, and may predispose the patient to this disorder [55]. Most patients with splenic artery aneurysms are multiparous. Rupture occurs in less than 2% of patients.

Rupture often produces left upper quadrant or epigastric pain. If untreated, aneurysmal rupture can progress to hemorrhagic shock and death. Plain abdominal roentgenograms often show a rim of vascular calcification in the left upper quadrant [56]. Abdominal CT scan usually confirms that this calcification is caused by a splenic artery aneurysm, and can help assess whether localized rupture has occurred. Aneurysms that are smaller than 2 cm in diameter rarely rupture. Maternal and fetal mortality from rupture exceed 70%. These aneurysms can be treated by excision with splenectomy, exclusion of the aneurysm, or angiographic embolization [57,58].

Hemorrhoids

Hemorrhoids are a common problem in adults [59]. Their occurrence is greater during pregnancy because of increased intra-abdominal pressure, straining from constipation, and changes in rectal venous drainage and pressure. Nearly one third of women complain of hemorrhoids during pregnancy, and approximately 10% require treatment [60]. Pruritis is the most common complaint, along with perianal discomfort and rectal bleeding. Hemorrhoidal thrombosis can cause severe pain.

Mild hemorrhoidal disease usually responds to treatment with increased dietary fiber, medicinal fiber preparations such as psyllium, stool softeners, increased fluid intake, and hemorrhoidal suppositories. Severe hemorrhoids can be treated with elastic band ligation [61]. Although generally safe, banding is associated with a small risk of acute necrotizing perianal infection. Hemorrhoidectomy under local anesthesia for acute thrombosis is associated with little maternal or fetal risk [62]. As with nonpregnant patients, rectal bleeding that is attributed to hemorrhoids can be a sign of underlying colorectal cancer.

Inflammatory bowel disease

Most patients with Crohn's disease and ulcerative colitis can be treated medically during pregnancy. Approximately 0.1% of women of child-bearing age are affected by these diseases [30]. Pregnancy usually minimally affects the course of inflammatory bowel disease. Patients who have active disease at conception are more likely to have persistent symptoms during pregnancy [63,64]. Flexible sigmoidoscopy and colonoscopy are useful to establish the diagnosis and present minimal risk to the pregnant patient and fetus [39,65]. Endoscopy during pregnancy is considered in detail elsewhere in this issue.

Surgery is indicated when medical treatment fails or an acute emergency develops (eg, toxic megacolon, colonic perforation, gastrointestinal obstruction, or gastrointestinal bleeding). Surgery for inflammatory bowel disease entails an increase in maternal and fetal mortality, possibly because of the operation being performed or the suboptimal conditions under which surgery is needed. Up to 60% of pregnant women who undergo total colectomy spontaneously abort because of intraoperative manipulation of the uterus [63]. Inflammatory bowel disease during pregnancy is discussed in the article by Kane (see elsewhere in this issue).

Gastrointestinal cancer

Colorectal and gastric cancer are the most common gastrointestinal malignancies during pregnancy. With a trend towards pregnancy occurring later in life, these problems will likely occur more frequently. Colorectal cancer occurs in approximately 1 per 10,000 to 15,000 pregnancies [66]. Approximately 5% of colorectal cancers occur in patients under 40 years of age.

Most cancers of the large intestine during pregnancy are located in the rectum [67]. This distribution differs from that in nonpregnant women and has not been explained. This distribution might be the result of bias from detection of rectal tumors during pelvic and rectal examinations in the prenatal period [68].

As in the nonpregnant population, the most common clinical findings that are associated with colorectal cancer are rectal bleeding, abdominal pain, abdominal distention, and unexplained anemia. Because these

symptoms are nonspecific, they can be easily ignored which results in a delayed diagnosis. For example, rectal bleeding can be attributed to hemorrhoids. Woods et al [66] reported that 29 of 32 colon cancers that were discovered during pregnancy were diagnosed after the 20th week of gestation.

When colorectal cancer is suspected, colonoscopy should be done to evaluate for synchronous lesions. Endorectal ultrasound is useful to stage rectal cancers. Magnetic resonance imaging may be preferable to abdominal CT to evaluate metastatic or regional disease. Serum carcinoembryonic antigen (CEA) levels are not affected by pregnancy and should be measured [69].

Colorectal cancer treatment depends upon the tumor stage, the gestational age of the fetus, the patient's desire for future pregnancy, and the personal views of the patient. Complications such as intestinal obstruction, gastrointestinal bleeding, or colonic perforation also impact upon the treatment.

When the cancer is discovered during the first half of pregnancy, resection should be performed. When discovered during the second half of pregnancy, resection is generally best deferred until after delivery [69]. This may require induction of labor, when the fetus is determined to be viable. When a rectal tumor is large and obstructs the birth canal or is located on the anterior rectal wall such that birth trauma or an episiotomy would result in entering the tumor, cesarean section should be performed. Otherwise, vaginal delivery is not contraindicated.

Adjuvant chemotherapy is often recommended for patients with Stage III colorectal cancer. Most regimens include 5-fluorouracil and leucovorin. Chemotherapy should be delayed until the second trimester, after which time fetal loss or developmental abnormalities are not increased [81].

Adjuvant radiation therapy has been advocated for Stage II and III rectal cancers. Although the benefits of preoperative radiation therapy, particularly for Stage III rectal cancer, are well-recognized, pelvic radiotherapy is inadvisable during pregnancy because of fetal teratogenicity. Therefore, radiation therapy should be delayed until the pregnancy is completed [69].

The maternal outcome of colorectal cancer that is discovered during pregnancy correlates with tumor stage, and is similar to that in the general population. These cancers usually pose no threat to the fetus; however, Woods et al [66] reported that only 78% of women with colorectal cancer during pregnancy delivered liveborn infants. This subject is discussed in detail elsewhere in this issue.

Gastric cancer is much less common than colorectal cancer during pregnancy. Unfortunately, these patients tend to present with advanced disease and have a worse survival rate than nonpregnant women or men of comparable age [71,72].

Trauma

Approximately 6% to 7% of all pregnancies are complicated by trauma [73]. Regardless of the injury, care and resuscitation of the mother is empha-

sized during the initial evaluation. Attention is focused on the treatment of hypovolemia and hypoxia. Because of the expanded blood volume during pregnancy, one third of the blood volume may be lost without a change in blood pressure or heart rate [7]. The gravid uterus can compress the inferior vena cava in the supine position in the late second and third trimester; turning the patient to the left side can displace the uterus and increase cardiac output by up to 30% [2]. Although this maneuver violates the principle of maintaining the patient in a supine position, precautions against spinal movement can be taken. This may result in transportation of the patient in the left lateral decubitus position, or rotation of the backboard to the right.

The initial assessment and resuscitation of the injured pregnant patient is the same as for a nonpregnant patient. The initial assessment focuses on airway patency, adequacy of breathing, and hemodynamic evaluation. Tachycardia or hypotension must be treated aggressively because these are signs of shock. Lactated Ringer's solution should be administered intravenously. If uncrossmatched red blood cells are needed, type O, Rh-negative red blood cells should be transfused to avoid Rh isoimmunization [7,9,74]. A urine test for pregnancy should be performed routinely in all women of childbearing age. Pregnant patients should have fetal ultrasound done early during the evaluation.

Maternal death and fetal or uterine injury are associated with a high fetal mortality [75]. In addition, a high injury severity score, the amount of fluid administered, and metabolic acidosis are associated with a poor fetal outcome [9,76].

Blunt abdominal trauma should be evaluated by standard diagnostic procedures including chest and pelvic roentgenograms. Focused abdominal ultrasonography for trauma can be used in pregnant patients to assess for intra-abdominal bleeding [7]. Diagnostic peritoneal lavage (DPL) can be performed, if necessary, with minimal fetal risk [77]. If DPL is necessary, the initial incision should be placed cephalad to the uterus to avoid inadvertent uterine puncture. Abdominal CT scan is also effective for the diagnosis of intra-abdominal injury but results in an absorbed radiation dose of approximately 0.2 cGy.

When intra-abdominal injury is suspected and surgery is necessary, operation should be performed immediately. Pregnant patients have decreased sensitivity to peritoneal irritation, and the usual physical findings that are associated with abdominal injury can be masked. Exploratory laparotomy has not been demonstrated to be an independent risk factor for fetal mortality [76]. The indications for exploratory laparotomy, including intra-abdominal bleeding and intestinal injury, are associated with a worse fetal outcome [78,82].

Emergency cesarean section should be reserved for when the estimated gestational age is greater than 26 weeks and fetal heart tones are present. Cesarean section that is performed in these circumstances results in a 75%

infant survival [79]. Survival is rare when fetal heart tones cannot be detected.

Fetal loss occurs in up to 15% of all pregnancies that are complicated by severe trauma [74]. Major transplacental hemorrhage is a predictor of poor fetal outcome. Placental abruption can occur after minor trauma, and results in fetal demise in 50% of cases. Minor degrees of placental separation are compatible with fetal survival. The presence of a hard, tender uterus that is larger than expected for the stage of pregnancy, especially if accompanied by fetal distress or demise, supports a clinical diagnosis of placental abruption [74]. Vaginal bleeding is not uniformly present. Disseminated intravascular coagulation can occur within a few hours after placental abruption.

When penetrating abdominal injury occurs, immediate surgery is required. If the uterus is injured, cesarean section should be done if the pregnancy is near term. Uterine repair can be performed for stab wounds when the fetus is viable without evident fetal injury on ultrasound [7].

The major focus in the care of a pregnant trauma patient should be on saving the mother, which will improve the fetal prognosis. In cases of severe maternal trauma or late death from injury, perimortum cesarean section can be performed with delivery of a viable infant [80].

Summary

Gastrointestinal disorders during pregnancy that require surgery often mimic the symptoms and signs of conditions that do not require surgery. Anatomic and physiologic changes of pregnancy can alter the usual clinical presentation of gastrointestinal disorders that require surgery. These alterations can be a challenge to diagnosis. Prompt treatment is critical to successful management. Most elective and urgent operations can be performed during pregnancy with minimal maternal and fetal risk. The condition of the mother should always take priority because proper treatment of the mother usually benefits the fetus as well.

References

- [1] Varner MW. General medical and surgical diseases in pregnancy. In: Scott JR, Disaia PJ, Hammond CB, et al, editors. *Danforth's obstetrics and gynecology*. 7th edition. Philadelphia: JB Lippincott; 1994. p. 456.
- [2] Martin C, Varner MW. Physiologic changes in pregnancy: surgical implications. *Clin Obstet Gynecol* 1994;37:241–55.
- [3] Gonik B. Intensive care monitoring of the critically ill pregnant patient. In: Creasy RK, Resnick R, editors. *Maternal-fetal medicine: principles and practice*. 2nd edition. Philadelphia: WB Saunders; 1989. p. 845–74.
- [4] Hammond CB. The female reproductive system. In: Townsend CM, Beauchamp RD, Evers BM, et al, editors. *Textbook of surgery*. 16th edition ed. Philadelphia: W.B. Saunders; 2001. p. 1619–48.

- [5] Hume RF, Killiam AP. Maternal physiology. In: Scott JR, KiSaia J, Hammon DB, et al, editors. *Obstetrics and gynecology*. Philadelphia: JB Lippincott; 1990. p. 93–100.
- [6] Broussard CN, Richter JE. Nausea and vomiting of pregnancy. *Gastroenterol Clin North Am* 1998;27:123–51.
- [7] Rozycki GS, Champion HR. Definitive care phase: trauma in pregnancy. In: Greenfield LJ, editor. *Surgery: scientific principles and practice*. Philadelphia: J.B. Lippincott; 1993. p. 344–8.
- [8] Delgado I, Neubert R, Dudenhausen J. Changes in white blood cells during parturition in mothers and newborns. *Gynecol Obstet Invest* 1994;38:227–35.
- [9] Firstenberg MS, Malangoni MA. Gastrointestinal surgery during pregnancy. *Gastroenterol Clin North Am* 1998;27:73–88.
- [10] Kort B, Katz VL, Watson WJ. The effect of nonobstetric operation during pregnancy. *Surg Gynecol Obstet* 1993;177:371–6.
- [11] Eichenberg BJ, Vanderlinden J, Miguel C, et al. Laparoscopic cholecystectomy in the third trimester of pregnancy. *Am Surg* 1996;62:874–7.
- [12] Affleck DG, Handrahan DL, Egger MJ, et al. The laparoscopic management of appendicitis and cholelithiasis during pregnancy. *Am J Surg* 1999;178:523–9.
- [13] Barone JE, Bears S, Chen S, et al. Outcome study of cholecystectomy during pregnancy. *Am J Surg* 1999;177(3):232–6
- [14] Tracey M, Fletcher HS. Appendicitis in pregnancy. *Am Surg* 2000;66:555–9.
- [15] Visser BC, Glasgow RE, Mulvihill KK, et al. Safety and timing of nonobstetric abdominal surgery in pregnancy. *Dig Surg* 2001;18:409–17.
- [16] Al-Mulhim AA. Acute appendicitis in pregnancy: a review of 52 cases. *Int Surg* 1996;81:295–7.
- [17] Reis RA, Arens RA. Appendicitis in pregnancy with changes in position and axis of normal appendix in pregnancy. *JAMA* 1932;98:1359.
- [18] DeVore GR. Acute abdominal pain in the pregnant patient due to pancreatitis, acute appendicitis, cholecystitis, or peptic ulcer disease. *Clin Perinatol* 1980;7:349–69.
- [19] Weingold AB. Appendicitis in pregnancy. *Clin Obstet Gynecol* 1983;26:801–9.
- [20] Wagner JM, McKinney P, Carpenter JL. Does this patient have appendicitis? *JAMA* 1996;276:1589–94.
- [21] Masters K, Levine BA, Gaskill HV, et al. Diagnosing appendicitis during pregnancy. *Am J Surg* 1984;148:768–71.
- [22] Rao PM, Rhea JT, Novelline RA, et al. Effect of computed tomography of the appendix on treatment of patients and use of hospital resources. *N Engl J Med* 1998;338:141–6.
- [23] Gurbuz AT, Peetz ME. The acute abdomen in the pregnant patient: is there a role for laparoscopy? *Surg Endosc* 1997;11:98–102.
- [24] Paik PS, Towson JA, Antbone GJ, et al. Intra-abdominal abscesses following laparoscopic and open appendectomies. *J Gastrointest Surg* 1997;1:188–93.
- [25] Fabiszewski NL, Sumkin JH, Johns CM. Contemporary radiologic percutaneous abscess drainage in the pelvis. *Clin Obstet Gynecol* 1993;36:445–56.
- [26] Briggs GG, Freeman RK, Yaffe SJ. *Drugs in pregnancy and lactation*. 4th edition. Baltimore: Williams & Wilkins; 1994.
- [27] McKellar DP, Anderson CT, Boynton CJ. Cholecystectomy during pregnancy without fetal loss. *Surg Gynecol Obstet* 1992;174:465–8.
- [28] Scott LD. Gallstone disease and pancreatitis in pregnancy. *Gastroenterol Clin North Am* 1992;21:803–15.
- [29] Stauffer RA, Adams A, Wygal J, et al. Gallbladder disease in pregnancy. *Am J Obstet Gynecol* 1982;144:661–4.
- [30] Kammerer WS. Nonobstetric surgery in pregnancy. *Med Clin North Am* 1987;71:551–60.
- [31] Sharp HT. Gastrointestinal surgical conditions during pregnancy. *Clin Obstet Gynecol* 1994;37:306–15.
- [32] Mayer IE, Hussain H. Abdominal pain during pregnancy. *Gastroenterol Clin North Am* 1998;27:1–36.

- [33] Ordorica SA, Frieden FJ, Marks F, et al. Pancreatic enzyme activity in pregnancy. *J Reprod Med* 1991;36:359–62.
- [34] Cooperberg PL, Gibney RG. Imaging of the gallbladder, 1987. *Radiology* 1987;163:605–13.
- [35] Reece EA, Assimakopoulos E, Zheng XZ, et al. The safety of obstetric ultrasonography: concern for the fetus. *Obstet Gynecol* 1990;76:139–46.
- [36] Dixon NP, Faddis DM, Silberman HL. Aggressive management of cholecystitis during pregnancy. *Am J Surg* 1987;154:292–4.
- [37] Curet MJ, Allen D, Josloff RK, et al. Laparoscopy during pregnancy. *Arch Surg* 1996; 131:546–50.
- [38] Machi J, Sigel B, McGraph EC, et al. Operative ultrasonography in the biliary tract during pregnancy. *Surg Gynecol Obstet* 1985;160:119–23.
- [39] Cappell MS. The safety and efficacy of gastrointestinal endoscopy during pregnancy. *Gastroenterol Clin North Am* 1998;37–71.
- [40] Sungler P, Heinerman PM, Steiner H, et al. Laparoscopic cholecystectomy and interventional endoscopy for gallstone complications during pregnancy. *Surg Endosc* 2000;14:267–71.
- [41] Smith LG, Moise KJ Jr, Dildy GA III, et al. Spontaneous rupture of liver during pregnancy: current therapy. *Obstet Gynecol* 1991;77:171–5.
- [42] Stevenson JT, Graham DJ. Hepatic hemorrhage and the HELLP syndrome: a surgeon's perspective. *Am Surg* 1995;61:756–60.
- [43] Terasaki KK, Quinn MF, Lundell CJ. Spontaneous hepatic hemorrhage in preeclampsia: treatment with hepatic arterial embolization. *Radiology* 1990;174:1039–41.
- [44] Ballantyne GH. Review of surgical volvulus: clinical patterns and pathogenesis. *Dis Colon Rectum* 1982;25:823–30.
- [45] Davis MR, Bohon CJ. Intestinal obstruction in pregnancy. *Clin Obstet Gynecol* 1983; 26:832–42.
- [46] Meyerson S, Holtz T, Ehrinpreis M, et al. Small bowel obstruction in pregnancy. *Am J Gastroenterol* 1995;90:299–302.
- [47] Lord SA, Boswell WC, Hungerpiller JC. Sigmoid volvulus in pregnancy. *Am Surg* 1996;62:380–2.
- [48] Malangoni MA, Times ML, Kozik D, et al. Admitting service influences the outcomes of patients with small bowel obstruction. *Surg* 2001;130:706–13.
- [49] Connolly MM, Unti JA, Nora PF. Bowel obstruction in pregnancy. *Surg Clin North Am* 1995;75:101–13.
- [50] Brothers TE, Strodel WE, Eckhauser FE. Endoscopy in colonic volvulus. *Ann Surg* 1987;206:1–4.
- [51] Perdue PW, Johnson HW, Stafford PW. Intestinal obstruction complicating pregnancy. *Am J Surg* 1992;164:384–8.
- [52] Ponec RJ, Saunders MD, Kimmey MD. Neostigmine for the treatment of acute colonic pseudo-obstruction. *N Engl J Med* 1999;341:137–41.
- [53] Messina LM, Stanley JC. Visceral artery aneurysms. *Surg Clin North Am* 1997;77:425–42.
- [54] Stanley JC, Messina LM, Zelenock GB. Splanchnic and renal artery aneurysms. In: Moore WS, editor. *Vascular surgery: a comprehensive review*. 4th edition. Philadelphia: WB Saunders Co.; 1993. p. 435.
- [55] Stanley JC, Fry WJ. Pathogenesis and clinical significance of splenic artery aneurysms. *Surgery* 1974;76:898–909.
- [56] Mattar SG, Lumsden AB. The management of splenic artery aneurysms: experience with 23 cases. *Am J Surg* 1995;169:580–4.
- [57] Carr SC, Pearce WH, Vogelzang RL, et al. Current management of visceral artery aneurysms. *Surg* 1996;120:627–33.
- [58] Prbulos AM, Chen HH, Rodis JF, et al. Angiographic embolization of a ruptured renal artery aneurysm during pregnancy. *Obstet Gynecol* 1997;90:630–5.
- [59] Johanson JF, Sonnenberg A. The prevalence of hemorrhoids and chronic constipation: an epidemiologic study. *Gastroenterology* 1990;98:380–6.

- [60] Medich DS, Fazio VW. Hemorrhoids, anal fissure and carcinoma of the colon, rectum and anus during pregnancy. *Surg Clin North Am* 1995;75:77–88.
- [61] Bayer I, Myslovaty B, Picovsky BM. Rubber band ligation of hemorrhoids: convenient and economic treatment. *J Clin Gastroenterol* 1996;23:50–2.
- [62] Saleeby RG Jr, Rosen L, Stasik JJ, et al. Hemorrhoidectomy during pregnancy: risk or relief? *Dis Colon Rectum* 1991;34:260–1.
- [63] Korelitz BI. Inflammatory bowel disease during pregnancy. *Gastroenterol Clin North Am* 1998;27:213–24.
- [64] Mogadam M, Korelitz BI, Ahmed SW, et al. The course of inflammatory bowel disease during pregnancy and postpartum. *Am J Gastroenterol* 1981;75:265–9.
- [65] Cappell MS, Colon VJ, Sidhom OA. A study at 10 medical centers of the safety and efficacy of 48 flexible sigmoidoscopies and 8 colonoscopies during pregnancy with follow-up of fetal outcome and with comparison to control groups. *Dig Dis Sci* 1996;41:2353–61.
- [66] Woods JB, Martin JN Jr, Ingram FH, et al. Pregnancy complicated by carcinoma of the colon above the rectum. *Am J Perinatol* 1992;9:102–10.
- [67] Bernstein MA, Madoff RD, Caushaj PF. Colon and rectal cancer in pregnancy. *Dis Colon Rectum* 1993;36:172–8.
- [68] Parry BR, Tan BK, Chan WB, et al. Rectal carcinoma during pregnancy. *Aust NZ J Surg* 1994;64:618–20.
- [69] Walsh C, Fazio VW. Cancer of the colon, rectum, and anus during pregnancy: the surgeon's perspective. *Gastroenterol Clin North Am* 1998;27(1):257–67.
- [70] Axelrad AM, Fleischer DE, Strack LL, et al. Performance of ERCP for symptomatic choledocholithiasis during pregnancy: techniques to increase safety and improve patient management. *Am J Gastroenterol* 1994;89:109–12.
- [71] Furukawa H, Iwanaga T, Hiratsuka M, et al. Gastric cancer in young adults: growth accelerating effect of pregnancy and delivery. *J Surg Oncol* 1994;55:3–6.
- [72] Maeta M, Yamashiro H, Oka A, et al. Gastric cancer in the young, with special reference to 14 pregnancy-associated cases: analysis based on 2,325 consecutive cases of gastric cancer. *J Surg Oncol* 1995;58:191–5.
- [73] Mighty H. Trauma in pregnancy. *Crit Care Clin* 1994;10:623–34.
- [74] Udekwo AO, Gammie JS, Schwab CW. Care of the pregnant trauma patient. In: Peitzman AB, Rhodes M, Schwab CW, et al, editors. *The trauma manual*. Philadelphia: Lippincott-Raven; 1998. p. 443–50.
- [75] McAnena OJ, Moore EE, Marx JA. Initial evaluation of the patient with blunt abdominal trauma. *Surg Clin North Am* 1990;70:495–515.
- [76] Henny CP, Lim AE, Brummelkamp WH, et al. A review of the importance of acute multidisciplinary treatment following spontaneous rupture of the liver capsule during pregnancy. *Surg Gynecol Obstet* 1983;156:593–8.
- [77] Rothenberger D, Quattlebaum FW, Perrfy JF, et al. Blunt maternal trauma: a review of 103 cases. *J Trauma* 1978;18:173–9.
- [78] Esposito TJ, Gens DR, Smith LG, et al. Evaluation of blunt abdominal trauma occurring during pregnancy. *J Trauma* 1989;29:1628–32.
- [79] Morris JA, Rosenbower TJ, Jurkovich GJ, et al. Infant survival after cesarean section for trauma. *Ann Surg* 1996;223:481–8.
- [80] Pearlman MD, Tintinalli JE, Lorenz RP. Blunt abdominal trauma during pregnancy. *N Engl J Med* 1990;323:1609–13.
- [81] Avilés A, Diaz-Maqueo JC, Talavera A, et al. Growth and development of children of mothers treated with chemotherapy during pregnancy: current status of 43 children. *Am J Hematol* 1991;36:243–8.
- [82] Hoff WS, D'Amelio LF, Tinkoff GH, et al. Maternal predictors of fetal demise in trauma during pregnancy. *Surg Gynecol Obstet* 1991;172:175–80.
- [83] Kurzel RB, Naunheim KS, Schwartz RA. Repair of symptomatic diaphragmatic hernia during pregnancy. *Obstet Gynecol* 1988;71:869–71.

- [84] Ranson JH, Rifkind KM, Roses DF, et al. Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet* 1974;139:69–81.
- [85] Sibai BM, Ramadan MK, Usta I, et al. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). *Am J Obstet Gynecol* 1993;169:1000–6.