GYNECOLOGIC CAUSES OF THE ACUTE ABDOMEN AND THE ACUTE ABDOMEN IN PREGNANCY

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Evaluation of a female patient who presents with an acute abdomen must always include surgical and gynecologic disorders. Table 1 outlines the most common gynecologic causes of the acute abdomen. The two general considerations in the surgical evaluation of these conditions are laparoscopic approach versus the traditional laparotomy and preservation of reproductive capability.

Laparoscopy and pelviscopy have had a major impact on the surgical approach in gynecology. Most acute abdomens can now be approached laparoscopically. Certain conditions that are discussed require the traditional laparotomy.

Preservation of reproductive capability has a major impact on the wellness of a woman. In addition to childbearing, hormonal function and sexual health are important issues to be considered in surgically managing acute gynecologic problems.

ADNEXAL TORSION

Many of the pathologic diseases of the ovary and/or fallopian tube cause abnormal enlargement of the adnexa, and with this enlargement comes an increased risk of twisting of the adnexa upon its axis of the

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Table 1. GYNECOLOGIC CAUSES OF THE ACUTE ABDOMEN

Adnexal torsion
Hemorrhagic functional ovarian cysts
Pelvic inflammatory disease and tubo-ovarian abscess
Ruptured ectopic pregnancy

infundibulopelvic ligament. Torsion of the adnexa is an acute gynecologic surgical emergency because prolonged torsion can lead to infarction of the tube and ovary involved. If left untreated, peritonitis and death may ensue. In most cases, torsion is preceded by an enlarged functional ovarian cyst or neoplasm but can also be seen in patients with normal adnexa or have other causes such as tubo-ovarian abscess and hydrosalpinx. Usually the neoplasm or enlargement causing the torsion is approximately 10 to 12 cm; often solid, such as a dermoid cyst; and nonadherent. The heavy weight and slow-growing nature of these tumors create a long pedicle that is conducive to torsion upon change of position. Torsion is most common in women of reproductive age, although it can occur in females of any age, including neonates. It seems to be very rare in the postmenopausal patient. It is generally unilateral, with a slight preponderance of cases noted to the right side.[3] Reportedly this has been noted to be 3:2 over the left, possibly due to location of the sigmoid colon occupying the pelvic space on the left or the hypermobility of the cecum on the right. Although it is a relatively rare phenomenon, adnexal torsion should always be considered as part of the differential diagnosis of acute pelvic/abdominal pain in women, especially those with pelvic masses diagnosed by examination or ultrasonography. Early diagnosis is important because prompt surgical intervention can result in ovarian preservation by saving the ovary and adnexa from infarction.

The presenting complaint of adnexal torsion is pain that is often abrupt in nature, very severe, lateralized to the right or left lower quadrant of the abdomen, and usually causing nausea and vomiting. It is often described as sharp and "knifelike," although it can be colicky in nature. The pain is proportional to the degree of circulatory obstruction; that is, complete obstruction interrupting venous return results in sudden severe pain with nausea and vomiting developing rapidly. There may be a history of waxing and waning pain if the adnexa has been twisting and untwisting or has undergone partial torsion, causing vascular slowdown but not thrombosis.[56] There may also be a history of some sort of jarring or movement that has caused the torsion, such as exercise prior to the onset of the pain or even just turning over in bed. Low-grade temperature elevations may occur, but significant fevers are unlikely and, if present, may point to another cause of the pain. An elevation of the white blood cell (WBC) count may be present but is also not very predictive. If necrosis and infection of the twisted organ occur, then higher fever and leukocytosis may be present. Pelvic examination

usually reveals a tender mass on the affected side. If the patient had a normal adnexa prior to the torsion, she may not have a mass present until later in the course of the torsion when edema and swelling of the adnexa have set in. Therefore, serial examinations may be necessary in a patient suspected of torsion.

Because adnexal torsion produces no classic symptoms and there are no definitive diagnostic tests or studies, surgical exploration of the pelvis is required for definitive diagnosis. Traditionally, this was done by means of laparotomy; however, laparoscopy has become the preferred surgical approach for both diagnosis and management of adnexal torsion.[58]

Traditionally, adnexal torsion was treated aggressively with salpingo-oophorectomy of the involved side. Unwinding the torsion was condemned for fear of releasing a potentially fatal embolus.[35, 63] This fear, however, seems to be unfounded, and current conservative management involves unwinding the adnexa and assessing its viability. No emboli have been noted in several series using this approach.[47, 59] An embolic complication could have been encountered when adnexa/torsion was not treated promptly and twisted organs were found to be obviously gangrenous at laparotomy.

Once torsion is unwound, the adnexa shows one of the following: (1) no evidence of ischemia or mild ischemia with immediate and complete recovery; (2) severe ischemia with a dark red or black tube and ovary and partial recovery after the pedicle is untwisted; (3) gangrenous adnexa without recovery. Only the gangrenous adnexa needs complete removal of the adnexa; the first two situations can be conservatively treated with detorsion and preservation of the ovary, even after severe ischemia has occurred.[59] When an ovarian cyst is present, a complete ovarian cystectomy should be performed to obtain a histologic diagnosis.[47] Infarction may make accurate diagnosis difficult; therefore, the cyst should be completely removed. This may also prevent recurrence. Routine ovariopexy after detorsion does not seem warranted because the risk of retorsion is very low when a cause is found and treated.[47]

Hemorrhagic Functional Ovarian Cysts

Follicular and corpus luteum cysts of the ovary are functional cysts and benign growths of the ovary. A follicular cyst arises from a normal follicle that fails to undergo ovulation or does not undergo the normal atretic process. It is usually clear and fluid filled. Corpus luteum cysts are less common than follicular cysts but are more associated with clinical symptoms. Functional cysts of the ovary should not cause pain unless the cyst is accompanied by rupture, torsion, or hemorrhage. Corpus luteum cysts arise from the mature corpus luteum. They more frequently attain a larger size than the follicular cyst. Corpus luteum cysts often produce a delay in the onset of the menstrual period, and when it occurs it may be heavy in nature (Halban's syndrome). Because

the cysts are usually larger than follicular cysts and associated with intraluminal bleeding, pain may be a common complaint. The cysts usually regress on their own and resolve in 4 to 8 weeks.

Rupture of a follicular cyst may cause an acute onset of pain that is usually short lived. Corpus luteum cysts are very vascular, and severe life-threatening hemorrhage may occur when they rupture. The combination of a delayed menstrual period, acute pain, pelvic mass, and evidence of hemoperitoneum is strongly suggestive of a ruptured corpus luteum cyst. The acute pain associated with rupture of a blood-filled corpus luteum cyst is indistinguishable from that of a ruptured ectopic pregnancy. A serum beta–human chorionic gonadotropin (beta-hCG) level may be helpful in distinguishing these two entities. Pelvic examination usually reveals diffuse pelvic tenderness, often lateralized to the side of the cyst, and a mass may be palpated. If hemorrhage is severe, it may produce abdominal distention and shock.

If the patient is hemodynamically stable, pelvic and abdominal ultrasonography is valuable. Classic findings include a complex cystic adnexal mass with free fluid in the cul-de-sac. The identification of an intrauterine gestational sac essentially excludes the diagnosis of ectopic pregnancy and may permit expectant management even when intraperitoneal bleeding has occurred.[9]

If the patient is hemodynamically unstable or the diagnosis is in question, exploratory surgery is required. If rupture and bleeding do occur, diagnostic and therapeutic laparoscopy is appropriate. If the patient is hemodynamically unstable, then emergency laparotomy is indicated. After confirmation that the bleeding is secondary to a cyst, conservative therapy consisting of removing the cyst and coagulating its base is standard therapy. If it is necessary to remove a corpus luteum of early pregnancy (prior to 12 weeks), progesterone replacement is advisable following surgery. Appropriate volume and blood replacement support should be used as needed.

Pelvic Inflammatory Disease and Tubo-Ovarian Abscess

Despite an increase in the number of effective broad-spectrum antibiotics, pelvic inflammatory disease (PID) and the complications arising from the disease continue to reach epidemic proportions into the 1990s. Acute salpingitis and PID account for more than 350,000 hospital admissions and 150,000 surgical procedures per year.[87] The annual costs associated with the disease are projected to reach $10 billion by the year 2000.[77] In addition, some authors report that nearly one third of patients hospitalized for PID develop some degree of pelvic abscess.[43] Other sequelae such as ectopic pregnancy, salpingitis isthmica nodosa, tubal infertility, chronic pelvic pain syndromes, and pelvic adhesions are other consequences of PID. Tubo-ovarian abscess (TOA) is the most serious

manifestation of salpingitis because the intra-abdominal rupture of a TOA is potentially life-threatening, with mortality rates as high as 8.6%.[40]

Pelvic inflammatory disease and subsequent TOA may result whenever bacteria gain access to the upper female genital tract. Under normal circumstances the fallopian tubes and related pelvic structures are sterile. However, access of bacteria into the upper genital tract either via sexually transmitted diseases or through instrumentation of the uterus may inoculate the uterus with bacteria from the vagina, causing infection. It has been suggested that passive transport and vectors such as spermatozoa and *Trichomonas* assist in establishing the ascending infection from the polymicrobial vagina and cervix.[77] Once present in the upper genital tract in sufficient numbers and virulence, these bacteria initiate an inflammatory reaction (endometritis-salpingitis-peritonitis) that results in the signs and symptoms of PID. The rate of a TOA developing from typical PID has been reported to be between 1% and 4%.[69]

Tubo-ovarian abscess is usually a polymicrobial infection, whereas general pelvic infections may often be monomicrobial. Tubo-ovarian abscesses are usually a mixture of anaerobic, facultative anaerobic, and aerobic organisms, with the purest isolated generally being anaerobes. The most frequent isolates from TOAs include a variety of Enterobacteriaceae, such as *Escherichia coli* (37%), *Bacteroides fragilis* (22%), other *Bacteroides* species (26%), *Peptostreptococcus* (18%), and *Peptococcus* (11%).[40, 89] The sexually transmitted organisms such as *Neisseria gonorrhoeae* and *Chlamydia* are usually not present in the abscess but may be recovered from the cervix in one third of cases. The emergence and recognition of *Prevotella bivia* (formerly *Bacteroides bivius*) and *Prevotella disiens* as major pathogens in upper female genital tract infection, combined with data suggesting that increased concentration of anaerobic organisms in the vagina is a risk factor for PID, point toward an anaerobic-predominant mixed infection as a cause of PID and TOA. These anaerobic bacteria such as *Bacteroides* species and *Peptostreptococcus* species are commonly found in high concentrations in the vagina of women with bacterial vaginosis.[77]

Standardized diagnostic criteria for TOA do not exist. The clinical diagnosis of TOA has the same diagnostic difficulties of PID. Women presenting with PID and a pelvic mass may have a TOA, or it could be a hydrosalpinx, tubo-ovarian complex, or other complex adnexal mass.

Patients with TOAs typically present with a history of pelvic or abdominal pain and fever. A history of PID may be present in only 50% of patients. The majority of patients also have a leukocytosis.[43] A significant proportion of women with TOA are afebrile (20% to 30%) and have normal WBC counts.[43] Other laboratory studies that may help in diagnosis are an elevated erythrocyte sedimentation rate (ESR) and elevated C-reactive protein, which recently was found to be more sensitive than elevated WBC or ESR. Pelvic examination usually reveals extreme pelvic tenderness (cervical motion tenderness), and a mass may be present. If rupture has occurred, typical signs and symptoms of

peritonitis are present and may lead to shock and death if not treated immediately.

Ultrasonography is very helpful in the diagnosis of TOAs and in following TOAs that are managed conservatively. Ultrasonography has proven to be very reliable in the diagnosis of TOA.[42] The expected typical appearance of a TOA on ultrasonography is a complex or cystic adnexal mass with multiple internal echoes and septations. The "gold standard" for diagnosis has always been laparoscopy; however, as ultrasound technology continues to improve, laparoscopy may be reserved for patients in whom the diagnosis is questionable.

Indications for surgical intervention in the treatment of TOA include (1) questionable diagnoses, when another surgical emergency may exist (e.g., appendicitis); (2) rupture of abscess; and (3) failure of medical therapy with or without a drainage procedure. The first two are indications for immediate surgical intervention. Intraperitoneal rupture of a TOA represents a true surgical emergency. Delayed interventions may increase the risk of septic shock and even death. There is general agreement that acute rupture of a TOA requires immediate surgery, but the extent of the surgery required to achieve a cure is controversial.[42] Traditionally, aggressive surgical extirpation, usually consisting of total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO) and drainage of all pockets of infection, was the treatment of choice in TOAs. This radical approach was used largely because of the inadequacies of antibiotics of that time. This procedure dropped the mortality rate from 100% to 12%[38] and is probably the procedure of choice in a patient who has completed childbearing or in those who are postmenopausal. However, most women who present with a TOA are in the peak of their reproductive years and fertility is a major issue. Conservative therapy of an unruptured TOA consists of appropriate intravenous antibiotic therapy, close monitoring of the patient, and possible drainage of the abscess via posterior colpotomy,[67, 71] CT- or ultrasound-guided percutaneous drainage, or drainage via laparoscopy. The posterior colpotomy approach has largely been abandoned because of a high rate of complications, including peritoneal sepsis and death. Success rates of CT-guided percutaneous drainage have been from 77% to 94% in recent studies, and this technique may play more of a major role in the future.[49] Early drainage of abscess and irrigation via laparoscopy in addition to antibiotics achieved a success rate of 95% by Reich and McGlynn[66] in recent series of 21 patients. These latter approaches have shown initially promising results but still need to be studied prospectively in a controlled randomized fashion. It does appear that drainage of a TOA in combination with antibiotic therapy is much more successful than conservative management. It has been shown that 50% of patients treated with antibiotics alone eventually require surgical treatment for the disease.[43, 68] Others report a success rate of 70% when TOAs are treated with antibiotics alone.[77]

Approximately 19% of patients treated with conservative surgical therapy require reoperation at a later date.[26, 43, 68] In cases of grossly

apparent bilateral disease, a somewhat conservative approach of bilateral partial adnexectomy without hysterectomy may be performed. Patients without adnexa are still able to conceive via in vitro fertilization and donor eggs. One must always use clinical judgment, however, and in patients with severe pelvic disease, TAH-BSO may be necessary despite the patient's reproductive status. In patients who have completed childbearing, TAH-BSO is standard therapy. It should also be noted that when a TOA is present in a postmenopausal woman, associated underlying malignancies are found in 25% to 50% of cases and conservative surgical therapy has no role.[45] Antibiotic therapy should include a broad-spectrum cephalosporin such as cefoxitin or cefotetan. Anaerobic coverage with clindamycin or metronidazole should also be added, as these have been shown to have the best ability to penetrate an abscess.[42]

Ruptured Ectopic Pregnancy

Although the total number of pregnancies has declined over the past two decades, the rate of ectopic pregnancy has increased dramatically. The Centers for Disease Control reported that the number of ectopic pregnancies quadrupled from 17,800 in 1970 to 88,000 in 1989.[13] This is an increase in rate from 4.5 per 1000 to 16.8 per 1000. While the number of ectopic pregnancies has increased, the death rate from this disorder has steadily declined. It is an assumption that the decreased mortality rate is secondary to the effects of early detection and intervention. Despite this improvement, approximately 34 women die yearly of the complications of ectopic pregnancy. This accounts for 13% of all pregnancy-related deaths.[12] With the advent of conservative surgery, the emphasis on early diagnosis and increased awareness of this condition may be an important factor in reducing the morbidity and mortality of ectopic pregnancy.

It is important to be aware of the conditions and circumstances that put a patient at increased risk for an ectopic pregnancy. Understanding these risks may help in making an early diagnosis and therefore preventing rupture and hemorrhage, necessitating a surgical emergency. The overall risk is approximately 1 in 200 pregnancies but may be increased 20- to 100-fold in certain subsets of women. These risk factors include (1) previous laparoscopically proven PID, (2) previous tubal pregnancy, (3) current intrauterine device use, and (4) previous tubal surgery, including tubal sterilization.[46]

As many as 50% of uterine tubes removed because of an ectopic pregnancy show prior inflammatory disease. The increased incidence of PID is thought to be a major factor in the increased numbers of ectopic pregnancies.[19] Although the risk of pregnancy is very low with a tubal ligation, if a pregnancy does occur, there is a significantly higher risk of the gestation being an ectopic one. Of those pregnancies occurring after

tubal ligation, 10% to 50% are ectopic, which represents a 20- to 100-fold increased risk.[88]

In most cases, the diagnosis of a ruptured ectopic pregnancy may be very obvious, with an acute abdomen, hemorrhagic shock, and a positive pregnancy test; however, sometimes the presentation may be more subtle. Many times diagnosis of an ectopic pregnancy prior to rupture may not be practical because the patient may not even know she is pregnant. Most commonly, once the ectopic pregnancy ruptures through the tube, there is abrupt onset of pelvic pain that may be lateralized to the side of the pregnancy. The classic signs of hemoperitoneum can occur rapidly and include abdominal pain, shoulder pain from diaphragmatic irritation, an urge to defecate, and syncope even in the absence of hypovolemia. If the rupture is only slight or has tamponaded itself off, the findings may be much more subtle. Pain is the most common symptom and can be extremely variable. It is initially described as dull and/or cramping. The woman usually seeks treatment because of a sudden change in the character of the pain, which usually occurs around the time of rupture. A history of menstrual abnormality is almost always present. Subjective symptoms of pregnancy may also be present, such as breast tenderness and emesis gravidarum. The classic triad of pelvic pain, amenorrhea, and vaginal bleeding may not always be present. As previously stated, if the patient does not present in a hemodynamically unstable state with an acute abdomen, the diagnosis of a ruptured ectopic pregnancy can be much more challenging, especially if the pregnancy is desired. The mainstays of diagnosis are serum beta-hCG assays and pelvic ultrasonography. Rapid urine hCG assays are available for immediate detection of a pregnancy, which may be the only test needed to confirm the diagnosis in a patient with an acute abdomen secondary to a hemoperitoneum. The urine hCG assay is sensitive to 25 mIU/mL or less, and more than 95% of patients with ectopic pregnancies have a positive test result.[11] Endovaginal ultrasound scanning has replaced transabdominal scanning for ectopic pregnancy diagnosis and early screening for an intrauterine pregnancy because it can visualize an intrauterine sac at an earlier gestational age. A gestational sac should always be seen in the patient with a viable intrauterine pregnancy when the hCG titer reaches a level of 2000 mIU/mL. In many cases the gestational sac can be seen at a level of about 1000 mIU/mL.[9,79] Ultrasonography is also very helpful in diagnosing blighted ovum or threatened abortions, which may be part of the differential diagnosis. Following beta-hCG titer (which should double every 48 hours in a normal, viable pregnancy) has no role in a patient with a suspected ruptured ectopic pregnancy, as that patient needs immediate surgical attention. Most patients with ectopic pregnancies have some abnormality on the sonographic scan.[70] These abnormal findings include a cystic or complex adnexal mass (60% to 90%) and free fluid in the peritoneal cavity (25% to 35%, higher in a ruptured ectopic pregnancy). However, the findings are nonspecific, and not visualizing an ectopic pregnancy on ultrasonography can never definitely exclude it as a possible diagnosis.

Other methods to assist in the diagnosis include culdocentesis, suction curettage, and laparoscopy. The presence of nonclotting blood within the peritoneal cavity in association with a positive pregnancy test is highly suggestive of an ectopic pregnancy. However, only about 50% of patients with a positive culdocentesis have a ruptured tube.[86] If a pregnancy has been previously determined to be nonviable by serum beta-hCG assays or if it is an undesired pregnancy, endometrial sampling by suction curettage may be performed to determine whether an intrauterine pregnancy is present. If chorionic villi are obtained from the uterine cavity, the presence of a concurrent ectopic pregnancy along with the intrauterine one is highly unlikely. The reported incidence of coexistent pregnancies in the general population (i.e., intrauterine and extrauterine) is between 1 in 4000 and 1 in 30,000.[5, 46] Sampling the endometrium with biopsy instruments such as the pipelle does not obtain an adequate sample for diagnosis and should not be used. The gold standard for diagnosis is still laparoscopy, but it should be remembered that approximately 3% of ectopic pregnancies are not visualized by laparoscopy. Typically, these are very early gestations. For patients who are hemodynamically stable, diagnostic laparoscopy is an excellent tool for both diagnosis and treatment.

A ruptured ectopic pregnancy is not, in and of itself, an indication for laparotomy. A laparoscopic approach may be used for hemodynamically stable patients. This decision must be made at the time of surgery. The choice of laparoscopic surgery versus laparotomy should be based on clinical experience, equipment availability, and the patient's physical status.[46] In an experienced laparoscopic surgeon's hand (with adequate equipment), all of the treatment traditionally performed via laparotomy for ectopic pregnancy can be completed via laparoscopic surgery. Clearly, if clinically possible, the patient is better served with a laparoscopic approach with reduced morbidity, recovery time, costs, and equivalent future fertility rates compared with laparotomy.[46, 79, 83] A patient who is hemodynamically unstable requires emergency laparotomy for surgical treatment, and laparoscopy has no role. There is also currently no role for medical therapy (i.e., methotrexate) for the treatment of a ruptured ectopic pregnancy even if the patient is hemodynamically stable. Once a diagnosis of ruptured ectopic pregnancy has been made by laparoscopy or laparotomy, treatment consists of removal of the ectopic gestation. It is very important to be aware of the patient's desire for future fertility, as this may play a role in intraoperative management. In the past, recommendations were made that included ipsilateral oophorectomy and cornual resection at the time of surgery. Today neither is recommended treatment.[36, 46] In women desiring future fertility, conservative tube-sparing surgery has been recommended.[46] Tube-sparing salpingostomy has been compared with salpingectomy, and no increase in the rate of ectopic pregnancies has been found and no difference in the subsequent intrauterine pregnancy rate.[20] Tube-sparing surgery is accomplished by removal of the ectopic pregnancy from the fallopian tube via linear salpingostomy by making an incision

on the antimesenteric portion of the tube over the bulge of the ectopic pregnancy, removing the pregnancy, achieving hemostasis, and allowing the tube to heal by secondary intention. There are no differences in subsequent pregnancy rates, adhesion formations, or fistula formation with or without closure of the incision site.[20, 57] Complications of salpingostomy include hemorrhage and persistent trophoblastic tissue. Trophoblastic tissue persists in approximately 5% of patients.[85] Therefore, all patients must undergo follow-up beta-hCG levels. Fimbrial expression consists of "milking" the pregnancy out of the tube. This technique has been associated with complications such as persistent trophoblastic tissue and postoperative bleeding and probably should be reserved for ectopic pregnancies located at or very near the fimbria itself.

Salpingectomy is the procedure of choice if the woman has no desire for further pregnancies. It also may be necessary for hemostatic control of an attempted conservative approach with salpingostomy or with a tube that appears unable to be salvaged. Salpingectomy is the standard procedure in a patient who is hemodynamically unstable. It has also been suggested that women with a previous history of infertility may be better served with salpingectomy; as it has been shown in that subset of patients, treatment with salpingectomy resulted in equivalent pregnancy rates and a decrease in recurrent ectopic pregnancy.[60]

THE ACUTE ABDOMEN IN PREGNANCY

Nonobstetric abdominal surgery in the pregnant patient can be both diagnostically and technically challenging. Hancock in 1848 described the drainage of an appendiceal abscess in a pregnant patient, and since that time there has been much debate over the proper evaluation and care of surgical disease in pregnant patients.[21] Advances in laparoscopic surgery in recent years have added even more fuel to this issue. Advances in laparoscopic technology and surgical techniques have overcome the technical difficulty of an enlarged gravid uterus; however, our enthusiasm should be tempered by the fact that many of the physiologic effects of laparoscopic surgery in pregnancy are still unknown.[7] Whatever the outcome of this debate is, surgical disease will continue to persist in the pregnant population. What is well known is that delays in diagnosis and definitive treatment represent the most significant risk for poor outcome in both the mother and the fetus.[23] Diagnostic delays tend to occur with a pregnant patient for several reasons: first, and probably most important, the patient and her doctors attribute many signs and symptoms of disease to her pregnancy. Second, the pregnant abdomen can be difficult to examine and may "hide" or change what would be classic findings in many disease states. Lastly, many physicians tend to be more cautious and conservative with pregnant patients, and this may actually lead to more harm by causing a delay in diagnosis and treatment. In 1908 it was reported that the mortality of appendicitis complicating pregnancy is the mortality of delay.[6] This holds true for any

condition that would cause an acute abdomen in pregnancy; however, because surgical disease in pregnancy is a rare event, there remains a lack of data on the indications for operation, the approach of the operation, and the risks to both mother and fetus.[39]

In two separate large institutional reviews, nonobstetric intra-abdominal surgery was reported to have a frequency of 1 in 451 to 1 in 635 deliveries.[1, 39] Both series confirmed that intra-abdominal surgery during pregnancy carries an acceptable risk to the mother and fetus and that complications are related to disease severity and operative delay rather than the operative procedure itself.[1] Mazze and Kallen[52] reported on a very large series of 778 cases of appendectomy during pregnancy which agreed with the above authors' conclusions. There was no increase in the number of stillborn infants and no increase in the number of congenital malformations.

Most series in the literature showed an increased risk of preterm labor with nonobstetric operations. We do need to be careful interpreting the data because many studies do not define preterm labor (i.e., is the definition used by authors uterine contractions alone or uterine contractions associated with cervical changes?). The issue of whether or not prophylactic perioperative tocolytic agents prevent preterm labor has not been resolved.[16] There has been no randomized prospective trial evaluating this, and it is doubtful that there will ever be because it would be a large undertaking to obtain the numbers for statistical significance.

The overall risk of preterm labor has been reported to be between 4% and 6% with pelvic or lower abdominal surgery.[32, 37] Others have reported this risk to be as high as 15% to 20%[1] or even up to 38%.[39] Allen et al[1] and Kort et al[39] both showed that the actual preterm delivery rate was approximately 12%; however, many of these patients did not receive tocolysis prophylactically or as treatment. In Allen's series they did show that once premature labor was diagnosed postoperatively, 16 of 16 patients were successfully tocolyzed with cessation of their labor; all 16 of these patients went on to deliver at term.[1]

The prophylactic use of beta-adrenergic tocolytic agents has not been recommended because their potential complications of vasodilation are not outweighed by any clinical effect.[8, 37] If premature contractions ensue postoperatively, tocolytic therapy with beta-adrenergic agents may be used; a single dose of terbutaline sulfate, 0.25 mg SC or IV, is often enough to stop contractions. If labor ensues, then standard IV magnesium sulfate should be used and has been shown to be effective.[1, 39] It is important to note that preterm labor is a significant risk for up to 7 days postoperatively[39] and that the entire effect of an appendectomy on shortening the length of pregnancy was caused by preterm delivery within 1 week postoperatively.[51] Therefore, one must maintain a cautious postoperative course with close follow-up if the patient is discharged from the hospital.

The issue of using progesterone postoperatively to prevent spontaneous abortions seems to have been resolved. In 1938 investigators made

a plea for the prophylactic use of progesterone postoperatively.[41] This "plea" was based on nine patients without discussion of gestational age or type of operation. In 1974 the benefit of progestational agents preventing abortion postoperatively after adnexal operation was refuted.[30] The authors found that progesterone had no effects on the spontaneous abortion rate; however, patients receiving different therapies were grouped together. Therefore, the literature was still inconclusive. In the series of Kort et al,[39] the patients given progesterone had a higher fetal loss rate (10% versus 0%) and preterm delivery rate (40% versus 0%). Therefore, the current data do not support the use of progestational agents beyond the first trimester. It certainly seems that progesterone may still play an important role postoperatively in the first trimester if the surgery involves the adnexa and/or corpus luteum. However, by the seventh week the placenta has taken over the role of producing progesterone to support the pregnancy. In the first trimester, progesterone is given at an intramuscular dose of 100 mg.[55]

Fetal Loss Rates

The percentages of fetal loss reported in the literature to date have covered a wide range. This is a very important issue because the fear of endangering the fetus is a legitimate concern and is probably the cause of much of the delay associated with operating on a pregnant patient. It is important to again stress that the literature supports the fact that the fetal mortality rates seem to be related to the severity of maternal disease and not to the operation itself. Early reviews reported a fetal loss rate between 5% and 12% (120 patients). A concerning report by Saunders and Milton[73] in 1974 reported a fetal loss rate of 26% (6 of 23) in their series of patients undergoing appendectomy. Five of the 6 deaths occurred in women who delivered within 1 week of surgery; 4 of the women were between 14 and 16 weeks of gestation. More recent studies have shown lower rates of perinatal mortality consistent with the baseline risk of the general population. Allen et al[1] and Mazze and Kallen[51] showed a fetal mortality rate of 2% (2 of 88) and 1.8% (14 of 778), respectively. Both studies supported the fact that most losses occur and the fetus is at greatest risk within 1 week of surgery, mostly owing to the increased risk of preterm labor and preterm delivery. There was no increased risk of stillbirth or congenital malformations in 5405 women who underwent nonobstetric operations in Sweden during the years 1973 to 1981.[52] Kort et al[39] in 1993 showed a fetal loss rate of 3.8% (3/78), compared with 2.0% in the general population for the same time period. They did note an increase in fetal loss rate of 11% versus 1.6% for surgery on adnexal masses compared with other types of surgery. This trend has been previously reported.[73] These reports show significant improvement in mortality rates since Babler[6] originally reported maternal and fetal mortality rates of 24% and 40%, respectively, in a review of more than 200 cases. It has been shown many times that early

operative intervention is paramount, as the risk of fetal loss is dramatically higher in the presence of perforation.

Role of Laparoscopy

Not long ago, laparoscopy was considered an absolute contraindication in pregnancy.[81] Recently, however, with the advancements in technology and skill in laparoscopic surgery, in particular the advent of laparoscopic cholecystectomy and appendectomy, the role of laparoscopy in pregnancy seems to be undergoing redefinition. Diagnostic and therapeutic laparoscopy during pregnancy is being performed at an increasing rate. There have been at least 50 reported cases of laparoscopic cholecystectomy[44] and case reports and case series of laparoscopic appendectomy. The first series of 12 patients reported by Spirtos et al[78] reported the use of diagnostic laparoscopy to aid in the diagnosis of appendicitis. All patients went on to laparotomy for removal of their appendix. More recently, there have been reports of laparoscopic appendectomy in pregnant women without maternal or fetal complications.[74]

The move for general surgeons to begin doing laparoscopic surgery in pregnancy was prompted by the fact that gynecologic surgeons for many years have been routinely performing diagnostic laparoscopy for the evaluation of adnexal masses and potential ectopic pregnancies.[72] Diagnostic laparoscopy in the first trimester did not seem to have any adverse effects upon the pregnancy if no ectopic pregnancy or disease was found. It has even been suggested that laparoscopic surgery may result in less fetal loss by avoiding the physiologic trauma of open surgery.[61] There have been numerous case reports of laparoscopic surgery for the removal of adnexal masses. Parker et al[61] in 1995 reviewed the literature and found 29 cases of laparoscopic adnexal procedures performed during pregnancy, with all pregnancies proceeding without adverse effect. More recently, Moore and Smith[55] reported on a series of eight patients with laparoscopic removal of adnexal mass in the second trimester of pregnancy without any adverse effects on the fetus or mother. This has been the largest series to date on the laparoscopic removal of adnexal masses in pregnancy. However, Shalev and Peleg[75] in 1993 reported on simple detorsion of torsed ovaries in 10 pregnant patients via laparoscopy, again without adverse fetal effects.

All of these reports seem to be supported by the fact that a recent national survey conducted by Reedy et al[65] reported 413 laparoscopic procedures performed in pregnancy. The most common laparoscopic surgery in pregnancy in this survey was cholecystectomy at 48.1%; 28% were adnexal surgeries, 16.2% appendectomies, and 7.5% miscellaneous. The majority of cases were in the second trimester (54%). This survey again supported the view that laparoscopic surgery appears to be safe in pregnancy because no adverse fetal or maternal complications seemed to be above the baseline for the general population. Five spontaneous abortions were reported, all in the first trimester, and this was consistent

with the rate of spontaneous abortion in the general population. There were five other postoperative complications reported which included one preterm labor successfully treated, one repeat ovarian torsion requiring reoperation, one pancreatitis, one common duct stone, and one postoperative hemorrhage from a trocar site. All of these pregnancies proceeded to term deliveries without complications.

We do have to be careful, however, because these surveys and retrospective reports do not all carry the same scientific significance as a prospective randomized study. Such a study would be an enormous undertaking to obtain enough data to support a definitive conclusion; it will not happen anytime soon. We also must remember that although the technical limitations of the gravid uterus have been overcome and laparoscopy has been proven safe and preferred in the nongravid patient, the physiologic effects of the pneumoperitoneum upon the uterus and fetus are still unknown. The benefits of laparoscopic surgery should equally apply to the pregnant patient provided that they outweigh any potential adverse effects. These possible adverse effects have not yet been totally determined. Recently one group of authors elected to abandon laparoscopic surgery during pregnancy secondary to a small adverse experience they have had with laparoscopic surgery in pregnancy.[2] Seven patients underwent laparoscopic procedures for emergent nonobstetric conditions (three gallstone pancreatitis, three appendicitis, one acute cholecystitis). There were four fetal deaths among the patients (three during the first postoperative week and one 4 weeks postoperatively). Although no conclusions can be drawn from such a small number, it underscores the need for additional studies, especially in the area of the effects of the pneumoperitoneum on the gravid uterus and fetus.

Bordelon and Hunter[7] identified three issues central to laparoscopy and pregnancy: (1) safe laparoscopic access with a gravid uterus, (2) modifications of trocar sites to allow smooth conduct of the procedure in the presence of an enlarged uterus, and (3) identifying the possible adverse effects of a sustained CO_2 pneumoperitoneum upon fetal physiology and blood flow. The first two issues are technical in nature and have been solved fairly easily by open laparoscopy and a more caudad approach for secondary trocar sites.[56] The third issue is the major and most important difference between proven open surgical procedures in pregnancy and the laparoscopic approach.

The safety of a CO_2 pneumoperitoneum in healthy adults has been studied extensively, with no adverse effects on cardiac output or acid-base status reported.[50, 56] These studies were, however, on healthy young individuals with brief procedures, and it has been shown to be more important in patients who are not as healthy, especially with a longer CO_2 pneumoperitoneum.[90] The pneumoperitoneum affects the fetus in two ways: (1) directly increasing the pressure on the uterus, and (2) altering maternal hemodynamics and acid-base balance. It is well known that a gravid uterus puts increased pressure on the vena cava, thus decreasing venous return. A pneumoperitoneum may aggravate this. Therefore, the right hip should be elevated to 30 degrees to help alleviate

this effect. It is known that a release of epinephrine and cortisol with an increase in intra-abdominal pressure occurs.[33] The question is whether this affects fetal blood flow. Chiu[14] showed a 60% decrease in maternal renal blood flow, resulting in a 50% decrease in urine output that is maintained for 1 hour after evacuation of CO_2. This may be secondary to an increased release of antidiuretic hormone and aldosterone from peritoneal stretch receptors.[33]

Hunter et al,[34] in studying pregnant ewes, have given us some valuable information on these issues as well as acid-base balance in the maternal-fetal unit with laparoscopic surgery. Removal of fetal CO_2 by the placenta is rapid, and the fetus normally maintains a slight respiratory acidosis.[34] Although this slight acidosis is normal and beneficial, the effects of moderate acidosis for 1 to 2 hours during laparoscopic surgery are unknown. Hunter looked at physiologic alterations in maternal and fetal blood pressure, pulse, and acid-base balance in response to a CO_2 pneumoperitoneum in pregnant ewes. He showed that no adverse effect resulted from the increase in pressure alone. There is, however, maternal hypercarbia and acidosis with a CO_2 pneumoperitoneum. This subsequently causes a fetal acidosis (7.35 to 7.25) and hypercarbia. Hypercarbia is known to cause tachycardia and hypertension in adults and has been seen in experiments with fetuses.[34] There is, however, no change in fetal P_{O_2}; therefore, maternal oxygenation and maternal and fetal cardiac output are maintained. Maternal respiratory acidosis is easily corrected by the anesthesiologist, but the end-tidal CO_2 may not reflect true P_{CO_2} and acid-base balance in the fetus. Alterations in ventilator settings based on maternal end-tidal CO_2 resulted in late and incomplete correction of respiratory acidosis; it has therefore been suggested that one follow arterial blood gases for correct monitoring, especially because the end-tidal CO_2 significantly underestimates maternal P_{CO_2} by 15 mm Hg and lags behind it.[34]

In summary, Hunter et al[34] showed fetal hypercarbia, acidosis, possible tachycardia, and an increase in fetal arterial pressure with the use of a CO_2 pneumoperitoneum. Again, the long-term effects of these physiologic alterations on the fetus are unknown but should be avoided if possible by close monitoring of maternal indices. Interestingly, an N_2O pneumoperitoneum showed none of the above changes, but this gas has traditionally not been used because of combustion concerns.[34]

Appendicitis

Appendicitis is the most common nongynecologic cause of acute abdomen during pregnancy, with an estimated frequency of one case of acute appendicitis per 1500 pregnancies.[51,76] Acute appendicitis can occur at any point during gestation but is most common in the first and second trimesters.[23] Significant complications were recognized early on, as Bobler[6] in 1932 reported maternal and fetal mortality rates of 24% and 40% in a review of more than 200 cases. Earlier in his career he made

famous the statement, "The mortality of appendicitis complicating pregnancy is the mortality of delay." Although maternal mortality rates have improved significantly, a perforated appendix still carries a high risk of fetal mortality.[23, 76]

Diagnosis of appendicitis in pregnancy is difficult, as in other abdominal surgical conditions. The symptoms are nonspecific and most often are attributed to the pregnancy itself. These symptoms include anorexia, nausea, vomiting, and abdominal pain. The studies of Baer et al[4] in 1932 are well known, showing the migration of the appendix progressively upward in the right lower and upper quadrants through the pregnancy. This migration causes a shift in the point of maximal tenderness superiorly and laterally. This displacement, when associated with a retrocecal appendix, can result in flank or back pain, which can be confused with a urinary tract infection, stone, or pyelonephritis. Additionally, the gravid uterus lifts the abdominal wall away from the abdominal viscera, and findings associated with inflammation of the parietal peritoneum may be absent.[17] In the majority of cases, the patient's temperature is less than 38°C, and right lower quadrant pain, rebound tenderness, and guarding are not as specific in a pregnant patient. The WBC count increases normally during pregnancy and can reach levels of 16,000 mm^3; therefore, a leukocytosis must be interpreted carefully and in and of itself may not be helpful in the diagnosis. However, a leukocytosis with a neutrophil count greater than 80% has been shown to be present at a significantly higher rate among patients with acute appendicitis than among those with a normal appendix.[39] Others have also shown a left shift present in 75% of patients with appendicitis. They have also seen that 50% of patients without appendicitis develop a left shift as well.[82] A catheterized urine specimen should be obtained to help rule out the most common differential diagnosis—pyelonephritis. However, pyuria can also be commonly seen with appendicitis. Clinical judgment based on overall presentation is still the gold standard for deciding which patients require surgical intervention.

Because of the difficulty in clinically diagnosing acute appendicitis, the negative laparotomy rate is much higher in the pregnant than the nonpregnant patient. An accepted rate of normal appendices in nonpregnant patients undergoing laparotomy for suspected appendicitis is 15%. This has been much higher in pregnant patients, with most larger series having a misdiagnosis rate between approximately 20% and 35%.[51, 76]

It may, however, be important to have a higher negative laparotomy rate in pregnant patients with suspected appendicitis secondary to the grave consequences of missing the diagnosis. The fetal mortality rate is dramatically higher if abscess or perforation of the appendix is present. Fetal loss occurs in 3% to 5% of cases of acute appendicitis but increases to 20% with abscess or perforation.[48] The risk to the mother and fetus is minimal if appendicitis is not found.[82] An aggressive surgical approach is therefore justified. Broad consensus exists that the incidence of perforation is associated with delay in removing the appendix after the diagnosis of appendicitis has been made.[28] Tamir et al[82] reported a 66%

incidence of perforation in patients when surgery was delayed by more than 24 hours (n = 35) and 0% incidence of patients taken to surgery within 24 hours of presentation.

There is no difference in the risk of preterm labor with a negative laparotomy versus an early appendectomy; both carry a risk of 10% to 15%[23, 39]; however, the risk can last for up to 7 days postoperatively. Mazze and Kallen[52] reported a series of 778 patients undergoing appendectomy in pregnancy. Then they noted an increase in the risk of preterm delivery in the first week after appendectomy when the operation was performed after 23 weeks' gestation. Beyond the first week, the rates of premature delivery were no different than in the general population. They emphasized the need for close follow-up postoperatively. Laparotomy has been the gold standard for diagnosis of appendicitis, but, as discussed earlier in the article, laparoscopy may have an emerging role in diagnosis and treatment of appendicitis in pregnancy. Diagnostic laparoscopy can potentially prevent an unnecessary appendectomy in the pregnant patient as well as prevent a delay in the diagnosis of appendicitis. Recently case reports have described both diagnostic and therapeutic laparoscopy for appendicitis in pregnant patients with no maternal or fetal complications.[74, 78]

Cholecystitis

Hormonal changes that occur during pregnancy seem to predispose the patient to gallstone formation; however, despite the marked increases in these hormones during pregnancy, marked increase in the incidence of symptomatic gallstones or cholecystitis has not been noted.[18] The key hormones during pregnancy are progesterone and estrogen. Progesterone is a smooth muscle relaxant that also inhibits cholecystokinin. This results in decreased gallbladder emptying and an increase in gallbladder residual volume. Estrogen as well as progesterone leads to biliary cholesterol hypersaturation, which increases the chance of forming gallstones. In more than 90% of cases, cholelithiasis is the cause of cholecystitis in pregnancy.[76] It is estimated that acute cholecystitis occurs at a frequency of 1 to 6 per 10,000 pregnancies.[44] It is the second most frequent nonobstetric emergency of pregnancy after acute appendicitis[18] and therefore makes gallbladder disease in pregnancy a significant clinical entity. This includes biliary colic, acute cholecystitis, and gallstone pancreatitis.

The symptoms of biliary disease are essentially the same in pregnant as in nonpregnant patients. These include anorexia, nausea and vomiting, dyspepsia, and intolerance to or avoidance of certain foods that can cause exacerbation of pain, typically fatty foods. The pain of biliary colic is usually acute in onset and is colicky or stabbing pain that begins over the midepigastrium and radiates to the back, right scapula, or shoulder. The pain of acute cholecystitis is more constant right upper quadrant pain associated with fever as well. Murphy's sign (tenderness

under the right costal margin on deep inspiration) is less commonly present in cases of cholecystitis in pregnancy.[78] It should be remembered that appendicitis occurring later in pregnancy can have a similar presentation.

Biliary colic has been defined as right upper quadrant pain with documented cholelithiasis. Acute cholecystitis includes fever (>38.5°C) and leukocytosis greater than 13,000 mm^3 in addition to the above, and gallstone pancreatitis includes elevated amylase or lipase in association with documented epigastric pain and cholelithiasis.[80] Serum levels of direct bilirubin and transaminases may be elevated. Alkaline phosphatase is less helpful because it is normally elevated in pregnancy.

Ultrasound evaluation is the diagnostic imaging tool of choice.[23] In patients with symptoms suggestive of biliary tract disease, sonography is very accurate at identifying stones, signs of acute or chronic inflammation such as thickening of the wall, and gallbladder edema or fluid around the gallbladder and at determining whether there is any dilation of the diameter of the common bile duct.[23]

Until recently, symptomatic gallbladder disease was treated with the conservative techniques of intravenous hydration, enteric rest with nasogastric suction, analgesia, and antibiotics. This was considered necessary to avoid surgery in the pregnant patient and the presumed fetal morbidity associated with surgery. As we have gained more experience with surgery in pregnancy and understand that the morbidity is usually associated with the disease and not the surgery, a more aggressive surgical approach, especially in the second trimester, has been recently recommended by several authors.[18, 22, 80] It has been pointed out that traditional conservative management has potential problems and risks. These include nutritional problems, extended hospital stays, and the need for supplemental alimentation in some cases.[17] There has also been a high incidence of readmission for gallbladder disease. Dixon et al[22] noted a 58% recurrence rate among 44 women managed conservatively. This has been confirmed by further studies.[18, 80]

Swisher et al,[80] in a retrospective study comparing conservative versus surgical management of gallbladder disease in 72 patients, agreed with Dixon's conclusions. Furthermore, patients managed conservatively had relapses resulting in a higher rate of premature labor and the need to induce labor in 10 patients. In contrast, no patient who underwent surgery required induction of labor or readmission to the hospital because of recurrence. They concluded that surgery in the second trimester reduced the relapse rate and the need to induce labor without increasing fetal or maternal morbidity. Another, more serious complication of conservative management was the risk of developing gallstone pancreatitis and the fact that a fetal loss rate as high as 60% has been reported with this entity.[64]

Recent review articles indicate that surgery should be considered as possible primary management in pregnant women with symptomatic gallbladder disease, especially in the second trimester.[18, 80] Most authors still recommend conservative nonoperative management in the first and

third trimesters. This more aggressive surgical approach has had no associated increased risk in fetal morbidity or mortality. In the past, surgery was saved for the worst cases of biliary disease. The increased losses seen in older series probably represent the fact that surgery was withheld until the disease was very severe. It may also be related to the significant number of women undergoing elective pregnancy termination from fear of first trimester radiation exposure. With modern ultrasonography this risk is alleviated.

Laparoscopic cholecystectomy has become the procedure of choice for most cholecystectomies. However, many authors have considered pregnancy to be an absolute or relative contraindication to laparoscopic cholecystectomy.[24] Recently a growing number of case reports have shown successful completion of laparoscopic cholecystectomy in the pregnant patient without maternal or fetal morbidity. A recent review of 46 cases reported in the world literature to date supported this approach.[65] Thirty-eight of these cases were in the second trimester of pregnancy (the ideal time for the laparoscopic cholecystectomy in the pregnant patient).

Bowel Obstruction in Pregnancy

The incidence of small and large bowel obstruction in pregnancy has varied in the literature from 1 in 1,500 to 1 in 66,431.[53] The higher rates are consistent with more recent studies. This is thought to be secondary to an increase in abdominal surgeries in young women since 1940, causing an increase in intra-abdominal adhesions. It is well documented that adhesions are the most common causes of bowel obstruction in pregnancy.[27, 62] Most adhesions are probably secondary to previous appendectomy or gynecologic surgery. They also may form following an inflammatory process such as pelvic inflammatory disease.

Although adhesions are the leading cause of obstruction in pregnant patients (55%), intestinal volvulus has a much higher incidence in pregnancy and is the cause of obstruction in 25% of pregnant women versus approximately 4% in the nonpregnant population.[15] Other causes (intussusception, hernia, and cancer) are rare. The uterus may compress the sigmoid colon, and it also displaces the bowel out of the pelvis.[53] The incidence of volvulus increases with the length of gestation; the most common location is the sigmoid colon. Surgical intervention with resection is almost always indicated because nonoperative management is rarely successful.[15] The incidence of cecal volvulus is significantly increased in the pregnant patient, and this is thought to be secondary to the enlarged uterus raising the mobile cecum out of the pelvis and into the right upper quadrant. This change in position, along with the enlarged uterus, causes partial obstruction and proximal distention. The loop of distended bowel is now more cephalad in the abdomen, and the superior mesenteric vessels become a point of fixation around which torsion can occur. The mechanism in the puerperium is similar except

that the sudden decrease in the size of the uterus causes changes in the position of abdominal organs. Three periods during the gestation are associated with an increased risk of obstruction. During the three periods there is a rapid change in uterine size—from 16 to 20 weeks, when the uterus becomes an intra-abdominal organ; from 32 to 36 weeks, when there is descent of the fetal head or "lightening"; and in the immediate postpartum period.[27] It also has been shown that patients are at an increased risk for obstruction in their first pregnancy following abdominal surgery, as this the first time that adhesions, if formed, are tested.[53]

The mortality rate of intestinal obstruction is much higher during pregnancy than in the general population. Goldthorp[27] noted a maternal mortality rate of 12% in 1996, and a more recent series by Perdue et al,[62] which reviewed the literature between 1966 and 1991, reported four maternal deaths (6%) in 66 cases of bowel obstruction in pregnancy. Fetal mortality rates are significantly higher with maternal bowel obstruction and have remained so through the years. Fetal mortality rates have been reported as high as 26% to 50%.[27, 53, 62] There is a dramatic progression of an increase in fetal mortality as the pregnancy progresses, with a mortality rate as high as 64% in the third trimester.[23] Higher rates of necrotic bowel requiring resection are also seen. All of these factors (i.e., high fetal mortality rates, increased maternal morbidity and mortality) may be secondary to pregnancy itself, causing a delay in diagnosis or a more complicated clinical picture, but it again may also be secondary to practitioners treating pregnant patients more conservatively, and this, in many cases, delays definitive management of obstruction.

The diagnosis of obstruction during pregnancy is based on the same triad of symptoms found in the general population: (1) abdominal pain, (2) obstipation, and (3) vomiting (which is not uncommon during a normal pregnancy, especially in the first trimester). However, nausea and vomiting that persist or present later in pregnancy should be suspicious and evaluated thoroughly. Patients who suffer from obstruction usually have undergone abdominal surgery. Obstruction presents with pain 85% to 98% of the time.[53] The patient usually experiences colicky pain that may radiate to the back. Typically no abdominal tenderness is present unless the obstruction is associated with underlying peritoneal irritation. The presence of abdominal tenderness in association with obstruction is an indication of intestinal ischemia requiring operative intervention. Emesis that is feculent and foul-smelling may be evidence of a complete obstruction for 48 to 72 hours because prolonged stasis allows microbial colonization of intraluminal contents, and large fluid volumes in the bowel may neutralize the pH and allow the succus to act as a culture medium.[15] Laboratory values are not reliable, but electrolyte imbalances should be looked for.

There should be no delay in ordering radiologic studies because delays can lead to increased complications. In the series of Perdue et al,[62] 82% of patients showed radiographic evidence of obstruction. Simple kidney-ureter-bladder and upright films were used to make the diagnosis 91% of the time. A dose of 1 rad (i.e., 10 times the typical dose

for two abdominal films) presents a risk of congenital malformation of less than 1 per 1000. The natural incidence of congenital malformations is estimated to be 30 per 1000.[54] A barium enema exposes the fetus to approximately 1 rad (809 millirad). Radiation doses to the fetus from 0 to 5 rad have no reported associated malformation; however, any radiation carries a potential for oncogenesis and an increased cancer risk. Therefore, any radiation exposure during gestation needs to be undertaken with care, with appropriate shielding if possible. Again, if a bowel obstruction in pregnancy is part of a differential diagnosis, it is worth the risk to the fetus for diagnostic radiography, even if serial radiographs are needed.

There is consensus in the literature that once the diagnosis of bowel obstruction is made, the only role of conservative therapy, including nasogastric decompression, is to prepare the patient for surgery.[27, 53, 62] The appropriate management for an intestinal obstruction in pregnancy is a true surgical emergency for both mother and fetus. Aggressive monitoring and fluid resuscitation are necessary, as there are fluid losses from vomiting, nasogastric suction, bowel wall edema, intraluminal losses, and free peritoneal fluid. Attention should be paid to the correction of electrolyte abnormalities and the stabilization of vital signs. A Foley catheter should be placed for the close monitoring of urine output. If obstruction occurs past 24 weeks (fetal viability), then continuous fetal monitoring is appropriate, as is delivery via cesarean section for distress, which would precede evaluation and treatment of the bowel obstruction.

References

1. Allen JR, Helling TS, Langerfeld M: Intra-abdominal surgery during pregnancy. Am J Surg 158:567, 1989
2. Amos DJ, Schorr SJ, Norman PF: Laparoscopic surgery in pregnancy. Am J Surg 171:435, 1996
3. Anspach BM: The torsion of tubal enlargement with special reference to pyosalpinx. Am J Obstet 66:553, 1912
4. Baer JL, Reis RA, Aren RA: Appendicitis in pregnancy with changes in position and axis of the normal appendix in pregnancy. JAMA 98:1359, 1932
5. Bello GV, Schonholz D, Moshirpur J, et al: Combined pregnancy: The Mount Sinai experience. Obstet Gynecol Surv 41:603, 1986
6. Babler EA: Perforative appendicitis complicating pregnancy. JAMA 51:1310, 1908
7. Bordelon BM, Hunter JG: Laparoscopy in the pregnant patient. *In* Ballantyne, Leahy, Modlin (eds): Laparoscopic surgery. Philadelphia, WB Saunders, 1992, pp 69–76
8. Borden TP, Peter JB, Merkatz IR: Ritodrine hydrochloride. A betamimetic agent for use in preterm labor. I. Pharmacology, clinical history, administration, side effects and safety. Obstet Gynecol 56:1, 1980
9. Bree RL, Edwards M, Bohm VM, et al: Transvaginal sonography in the evaluation of normal early pregnancy with hCG level. AJR 53:75, 1989
10. Burnett LS: Gynecologic causes of the acute abdomen. Surg Clin North Am 68:385, 1988
11. Cartwright PS: Diagnosis of ectopic pregnancy. Obstet Gynecol Clin North Am 18:22, 1991
12. Centers for Disease Control: Ectopic pregnancy in the United States 1988–89. MMWR 41:592, 1992

13. Centers for Disease Control: Ectopic pregnancy surveillance in the United States, 1970–1987. MMWR 39:11, 1990
14. Chiu: Oliguria with laparoscopy. J Am Coll Surg 181:397, 1991
15. Connolly MM, Unti JA, Nora PF: Bowel obstruction in pregnancy. Surg Clin North Am 75:101, 1995
16. Cox SM, Sherman L, Leveno KJ: Randomized investigation of magnesium sulfate for prevention of pre-term birth. Am J Obstet Gynecol 163:767, 1990
17. Cunningham FG, McCubbin JH: Appendicitis complicating pregnancy. Obstet Gynecol 45:415, 1977
18. Davis A, Katz VL, Cox R: Gallbladder disease in pregnancy. J Reprod Med 40:759, 1995
19. DeCherney AH: Ectopic pregnancy. Rockford, MD, Aspen Publishers, 1986, pp 1, 81, 89, 103
20. DeCherney AH, Jones EE: Ectopic pregnancy. Clin Obstet Gynecol 28:365, 1985
21. DeCherney A, Kase N: The conservative surgical management of unruptured ectopic pregnancy. Obstet Gynecol 54:451, 1979
22. Dixon NP, Faddis DM, Silberman H: Aggressive management of cholecystitis in pregnancy. Am J Surg 154:292, 1987
23. Fallon WF, Newman JS, Fallon GF, et al: The surgical management of intra-abdominal inflammatory conditions during pregnancy. Surg Clin North Am 75:15, 1995
24. Gadacz TR, Talamini MA, Lillemoe KD, et al: Laparoscopic cholecystectomy. Surg Clin North Am 70:1249, 1990
25. Gianopoulos JG: Establishing the criteria for anesthesia and other precautions for surgery during pregnancy. Surg Clin North Am 75:33, 1995
26. Ginsberg DS, Stern JL, Hamod KA, et al: Tubo-ovarian abscess, a retrospective review. Am J Obstet Gynecol 138:1055, 1980
27. Goldthorp WO: Intestinal obstruction during pregnancy and puerperium. BJ Clin Pract 20:368, 1966
28. Halvorsen AC, Brandt B, Anderson JJ: Acute appendicitis in pregnancy: Complications and subsequent management. Eur J Surg 158:603, 1992
29. Hancock H: Disease of the appendix coeci cured by operation. Lancet 2:380, 1848
30. Hill LM, Johnson CE, Lee RA: Ovarian surgery in pregnancy. Am J Obstet Gynecol 122:565, 1975
31. Hill LM, Johnson CE, Lee RA: Prophylactic use of progesterone in abdominal surgery during pregnancy: A retrospective evaluation. Obstet Gynecol 46:287, 1975
32. Hunt MG, Martin JN Jr, Martin RW, et al: Perinatal aspects of abdominal surgery for non-obstetric disease. Am J Perinatol 6:412, 1989
33. Hunter JG: Laparoscopic pneumoperitoneum: The abdominal compartment syndrome revisited. J Am Coll Surg 181:469, 1991
34. Hunter JG, Swanstrom L, Thornburg K: Carbon dioxide pneumoperitoneum induces fetal acidosis in a pregnant ewe model. Surg Endosc 9:272, 1995
35. James DF, Barber HRK, Graber EA: Torsion of uterine adnexa in children: Report of 3 cases. Obstet Gynecol 35:226, 1970
36. Kalchman GG, Meltzer RM: Interstitial pregnancy following homolateral salpingectomy: Report of 2 cases and review of the literature. Am J Obstet Gynecol 96:1139, 1966
37. Kammerer WD: Non-obstetric surgery in pregnancy. Med Clin North Am 71:551, 1987
38. Kaplan AL, Jacobs WM, Ehresman JB: Aggressive management of pelvic abscesses. Am J Obstet Gynecol 98:487, 1967
39. Kort B, Katz VL, Watson WJ: Effect of non-obstetrical operation during pregnancy. Surg Gynecol Obstet 177:371, 1993
40. Krivak T, Propst A, Horowitz G: Tubo-ovarian abscess. Principles of contemporary management. Fem Patient 22:27, 1997
41. Lackner JE, Tulsky AS: Abortion as a complication of operation in the pregnant woman. A plea for the prophylactic use of progesterone. Am J Surg 46:362, 1939
42. Landers DV: Tubo-ovarian abscess complicating pelvic inflammatory disease. *In* Landers DV, Sweet RL (eds): Pelvic Inflammatory Disease. New York, Springer Verlag, 1996, p 94
43. Landers DV, Sweet RL: Tubo-ovarian abscess: Contemporary approach to management. Rev Infect Dis 5:876, 1983

44. Lanzafame RJ: Laparoscopic cholecystectomy during pregnancy. Surg 118:627, 1995
45. Lavy G, Hilsenrath R: Management of tubo-ovarian abscess. Infert Reprod Clin North Am 3:821, 1992
46. Ling FW, Stovall TG: Update on the management of ectopic pregnancy. Adv Obstet Gynecol 1:57, 1994
47. Mage G, Canis M: Laparoscopic management of adnexal torsion. A review of 35 cases. J Reprod Med 34:521, 1989
48. Mahmoodian S: Appendicitis complicating pregnancy. South Med J 85:19, 1992
49. Mann WJ, Stovall TG: Gynecologic surgery. New York, Churchill Livingstone, 1996, p 668
50. Marshall RL, Jebson PJR, Davie LT, Scott DB: Circulatory effects of carbon dioxide insufflation of the peritoneal cavity for laparoscopy. Br J Anaesth 44:680, 1972
51. Mazze RI, Kallen B: Appendectomy during pregnancy: A Swedish Registry Study of 778 cases. Obstet Gynecol 77:836, 1991
52. Mazze RI, Kallen B: Reproductive outcome following anesthesia and operation during pregnancy: A registry study of 5,405 cases. Am J Obstet Gynecol 1611:1178, 1989
53. Meyerson S, Holtz T, Ehrinpreis M, et al: Small bowel obstruction in pregnancy. Am J Gastroenterol 90:299, 1995
54. Mole RH: Radiation effects on prenatal development and their radiologic significance. Br J Radiol 52:101, 1979
55. Moore RD, Smith WG: Laparoscopic removal of ovarian masses in the pregnant patient [abstract]. *In* Abstracts of New England Association of Gynecologic Oncologists Annual Meeting, York, ME, 1996
56. Mote WM, Ovankovich AD, Bienarz J, et al: Cardiovascular effects and acid-base and blood gas changes during laparoscopy. Am J Obstet Gynecol 115:1002, 1973
57. Nelson LM, Margara RA, Winston RML: Primary and secondary closure of ampullary salpingostomy compared in the rabbit. Fertil Steril 45:292, 1986
58. Nichols DH, Julian PJ: Torsion of the adnexa. Clin Obstet Gynecol 28:375, 1985
59. Oelsner G, et al: Long-term follow up of the twisted ischemic adnexa managed by torsion. Fertil Steril 60:976, 1993
60. Ory SJ, Nnadi E, Hermann R, et al: Fertility after ectopic pregnancy. Fertil Steril 60:231, 1993
61. Parker J, Robinson H, Byrne D: Laparoscopic adnexal surgery during pregnancy: A case of heterotopic tubal pregnancy treated by laparoscopic salpingectomy. Aust NZ J Obstet Gynaecol 35:208, 1995
62. Perdue PW, Johnson HW, Stafford PW: Intestinal obstruction complicating pregnancy. Am J Surg 164:384, 1992
63. Powell JL, Foley FP, Llorens AS: Torsion of the fallopian tube in postmenopausal women. Am J Obstet Gynecol 28:375, 1985
64. Printen KJ, et al: Cholecystectomy during pregnancy. Am Surg 44:432, 1978
65. Reedy MB, Galan HL, Richards WE: Laparoscopy during pregnancy: A survey of laparoendoscopic surgeons. J Reprod Med 42:33, 1997
66. Reich H, McGlynn F: Laparoscopic treatment of tuboovarian and pelvic abscesses. J Reprod Med 32:747, 1987
67. Rivlin ME: Clinical outcome following vaginal drainage of pelvic abscess. Obstet Gynecol 61:169, 1983
68. Rivlin ME, Hunt JA: Ruptured tubo-ovarian abscess: Is hysterectomy necessary? Obstet Gynecol 50:518, 1977
69. Roberts W, Dockery JL: Management of tubo-ovarian abscess due to pelvic inflammatory disease. S Med J 77:7, 1984
70. Romero R, Kadar N, Castro D, et al: The value of adnexal sonographic findings in the diagnosis of ectopic pregnancy. Am J Obstet Gynecol 158:52, 1988
71. Rubenstein PR, Mishell DR, Ledger WJ: Colpotomy drainage of pelvic abscess. Obstet Gynecol 48:142, 1976
72. Samuellson S, Sjovall A: Laparoscopy in suspected ectopic pregnancy. Acta Obstet Gynecol Scand 51:31, 1972
73. Saunders P, Milton PJD: Laparotomy during pregnancy: An assessment of diagnostic accuracy and fetal wastage. Br Med J 3:165, 1973

74. Schriber JH: Laparoscopic appendectomy in pregnancy. Surg Endosc 4:100, 1990
75. Shalev E, Peleg D: Laparoscopic treatment of adnexal torsion. Surg Gynecol Obstet 176:448, 1993
76. Sharp HT: Gastrointestinal surgical conditions during pregnancy. Clin Obstet Gynecol 37:306, 1994
77. Soper DE: Pelvic inflammatory disease. Adv Obstet Gynecol 1:85, 1994
78. Spirtos NM, Eisenkop SM, Spirtos TW, et al: Laparoscopy: A diagnostic aid in cases of suspected appendicitis. Its use in women of reproductive age. Am J Obstet Gynecol 156:90, 1987
79. Stovall TG: Diagnosis and surgery for ectopic pregnancy. *In* Mann WJ, Stovall TG (eds): Gynecologic Surgery. New York, Churchill Livingstone, 1996, p 592
80. Swisher SG, Schmit PJ, Hunt KK: Biliary disease in pregnancy. Am J Surg 168:576, 1994
81. Talamini MA: Controversies in laparoscopic cholecystectomy; contraindications, cholangiography, pregnancy and avoidance of complications. Baillers Clin Gastroenterol 7:881, 1993
82. Tamir IL, Bongard FS, Klein SR: Acute appendicitis in the pregnant patient. Am J Surg 160:571, 1990
83. Thorburn J, Philipson M, Lindblom B: Fertility after ectopic pregnancy in relation to background factors and surgical treatment. Fertil Steril 50:5803, 1988
84. Thornton JG, Wells M: Ovarian cysts in pregnancy: Does ultrasound make traditional management inappropriate? Obstet Gynecol 69:717, 1987
85. Vancaillie TG: Salpingectomy in ectopic pregnancy. Obstet Gynecol Clin North Am 18:111, 1991
86. Vermesh M, Graczykowski JW, Saver MV: Reevaluation of the role of culdocentesis in the management of ectopic pregnancy. Am J Obstet Gynecol 162:411, 1990
87. Walker CE, Landers DV: Pelvic abscesses: New trends in the management of tuboovarian abscesses. Obstet Gynecol Surv 46:615, 1991
88. Weinstein L, Morris MB, Dotters D, et al: Ectopic pregnancy, a new surgical epidemic. Obstet Gynecol 61:698, 1983
89. Wiesenfeld HC, Sweet RL: Progress in the management of tuboovarian abscesses. Clin Obstet Gynecol 138:433, 1993
90. Wittgen CM, Andrus CH, Fitzgerald SD, et al: Analysis of the hemodynamic and ventilatory effects of laparoscopic cholecystectomy. Arch Surg 126:997, 1991

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