



# Constipation, diarrhea, and symptomatic hemorrhoids during pregnancy

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Many physicians, however, are uncomfortable when dealing with these conditions during pregnancy. In general, this is true not because physiologic changes associated with pregnancy may predispose these disorders, but because of concern about fetal toxicity. In particular, the physician may have little experience with the impact of pregnancy on these disorders. This article reviews the known physiologic changes in pregnancy that predispose constipation and hemorrhoids and reviews the management of constipation, hemorrhoids, and diarrhea during pregnancy.

## Constipation in pregnancy

There are few published surveys of constipation during pregnancy and none is published recently. Two studies reported an incidence of approximately one third in the third trimester [1,2], whereas an Israeli study reported an incidence of only 11% [3]. In the latter study, only 1.5% of women required laxatives for the constipation. Constipation may occur de novo during pregnancy, or chronic constipation may increase in severity during pregnancy. In part, this occurs because of physiologic changes or coexistent conditions associated with pregnancy, as summarized in Box 1.

Gastrointestinal motility is believed to be somewhat inhibited during pregnancy. This inhibition may directly promote constipation or indirectly exacerbate underlying disorders of bowel habits. Because studies of pregnant women are restricted greatly by ethical considerations, most data are derived from animal studies, which have suggested that female sex

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**Box 1. Factors contributing to constipation during pregnancy***Dietary*

Poor fluid intake with nausea and vomiting  
Iron supplements

*Behavioral*

Decreased physical activity  
Psychosocial stress

*Hormonal changes (slowed gastrointestinal transit)*

Increased progesterone  
Increased estrogen  
Decreased motilin

*Other*

Enlarging gravid uterus  
Painful hemorrhoids

hormones, particularly progesterone, decrease smooth muscle contractility and slow gastrointestinal transit [4]. Working with canine colonic smooth muscle strips, investigators demonstrated a dose-dependent and reversible inhibition of circular and longitudinal muscle contractility by progesterone, an inhibition that was reversed by increasing extracellular calcium concentrations [5]. Similarly, colonic transit was prolonged in pregnant rats and in those treated with estrogen and progesterone after undergoing ovariectomy as compared with nonpregnant or ovariectomy control animals [6].

Human studies of the effects of female sex steroid hormones on small intestine and colonic transit have been limited to measurements of orocecal transit using breath  $H_2$  techniques and of colonic transit using radio-opaque marker studies in nonpregnant women. In one study of menstruating women, orocecal transit was significantly longer in the luteal phase, when progesterone levels are high, than in the follicular phase, when progesterone levels are low [7]. Similarly, orocecal transit was significantly slower (by approximately one third) in the third trimester of pregnancy, when estradiol and progesterone levels are elevated, as compared with the postpartum period [8]. This finding was confirmed in another study [9]. In this study, orocecal transit slowed in direct proportion to increases in serum progesterone levels.

One study demonstrated that plasma motilin levels are reversibly depressed during pregnancy, perhaps via inhibition by progesterone [10]. This depressed level inhibits fasting migrating motor complex activity, which is expressed as slowed orocecal transit during the fasting stage.

Colonic transit in pregnant women has not been studied. The few studies of colonic transit as a function of gender or of the menstrual cycle suggest that female sex hormones minimally affect colonic transit. Hinds et al found that women tend to have somewhat slower colonic transit than men, but this difference was not statistically significant; these findings are similar to another study [11]. Colonic transit did not significantly vary, moreover, during different phases of the menstrual cycle or in women using oral contraceptives. These findings do not preclude changes during pregnancy, but suggest that any slowing may be of minimal clinical importance. Thus, the causes and treatment of constipation during pregnancy are similar to those of chronic constipation in the general population. Extensive investigation of constipation is unnecessary in pregnant women except under extraordinary circumstances [12].

### **Treatment of constipation during pregnancy**

Patient education includes reassurance and an explanation as to normal bowel habits. Patients are advised to increase fluid and fiber intake, to perform moderate exercise, and to use postprandial increases in colonic motility by defecating in the morning and after meals, when colonic motor activity is highest. Dietary fiber and bulk laxatives, such as psyllium, methylcellulose, or polycarbophil, with adequate fluids are the most physiologic and safest medical therapy for constipation during pregnancy. In general, stimulant laxatives should be reserved for patients who do not respond to these initial measures [13].

Dietary fiber is that portion of plant food that escapes digestion. It consists of both insoluble and soluble components. In general, fibers in cereals possess cell walls that resist digestion and retain water within their cellular structures, whereas fibers in citrus fruits and legumes stimulate the growth of colonic flora and increase fecal mass. Wheat bran is a highly effective fiber laxative, with a clear dose-dependent effect on fecal output. Particle size may be important, as the large particle size of cereal products seems to enhance colonic transit [13].

Patients with poor dietary habits may add 2 to 6 tablespoons of bran to each meal, followed by a glass of water or another beverage. The laxative effect may be delayed for three to five days. Relief of chronic constipation may require several weeks. Vegetables and fruits contain soluble fiber but are not always adequate substitutes for bran. Pregnant women should be warned that consuming a large amount of bran can cause abdominal bloating or flatulence and that sufficient fluids should be consumed with it. These

symptoms can be alleviated by starting with small amounts and gradually increasing as tolerated to the desired effect.

### Laxatives with onset of action in one to three days

Psyllium, calcium polycarbophil, and methylcellulose are more refined and concentrated than bran, but more expensive (Table 1). These agents should be diluted to ensure adequate mixing with food and are generally consumed before meals or at bedtime. They increase fecal water content and volume, decrease colonic transit time, increase stool weight, and improve stool consistency.

Sorbitol and lactulose are poorly absorbed sugars that are partly hydrolyzed to lactic, acetic, and formic acids by coliform bacteria. These acidic metabolites stimulate fluid accumulation in the colon by an osmotic effect and usually produce soft, well-formed stools. A large dose of these agents is required to overcome the metabolism of colonic bacteria. Sorbitol is less expensive than lactulose. In one well-controlled study, sorbitol 70% administered at bedtime was as effective as lactulose for constipation [14]. These agents can cause side effects of abdominal bloating and flatulence, which may limit their use. In addition, lactulose should be used with caution in diabetic patients and avoided in patients who require a low galactose diet, because lactulose contains both galactose and lactose. In addition, it should not be used by pregnant women with nausea because it can exacerbate this symptom.

Polyethylene glycol (PEG) has been approved recently as an osmotic agent for chronic constipation in daily doses of 8 to 25 g [15]. This agent

Table 1  
Preferred laxatives for constipation during pregnancy

Laxative	Usual adult dose*
Bulk-forming laxatives	
Natural (eg, psyllium)	7 g/d
Synthetic (eg, methylcellulose, polycarbophil)	4–6 g/d
Hyperosmolar laxatives	
Polyethylene glycol	8–25 g/day
Lactulose	15–30 mL/day
Sorbitol (70%)	15–30 mL/day
Glycerine	3 g suppository
Diphenylmethanes	
Bisacodyl	30 mg; 10 mg suppository
Anthraquinones	
Senna	17–34 mg
Cascara sagrada	2–5 mL

\* Oral, except where indicated otherwise.

is prescribed in powder form. Patients are instructed to initially mix 17 g of PEG powder with any fluid and to adjust the amount ingested based on clinical response. Abdominal bloating and flatulence are not as problematic as they are with fiber or poorly absorbed sugars, because colonic bacteria do not hydrolyze PEG. Salt and water absorption are not a problem because this solution contains no electrolytes. Although safety during pregnancy is not established (Federal Drug Administration [FDA] Pregnancy Category C), PEG is inert, absorption is minuscule, and toxicity theoretically is unlikely.

Docusates soften stool by reducing surface tension; this permits intestinal fluids to penetrate into the fecal mass. Docusates are of questionable efficacy and marginal value, however, for treating chronic constipation. One case was reported of neonatal hypomagnesemia associated with maternal oral administration of sodium docusate [16].

Mineral oil softens stool as a result of its emollient action. It is effective particularly in enemas for softening fecal impactions. Aspiration, resulting in lipoid pneumonia, is a hazard of oral administration, especially in patients with impaired swallowing. Repeated use is associated with decreased maternal absorption of fat-soluble vitamins, neonatal hypoprotrombinemia, and hemorrhage [17].

### **Stimulant laxatives**

Stimulant laxatives may be considered in patients who fail to respond to bulk or osmotic laxatives (see Table 1). These agents affect fluid and electrolyte transport, gastrointestinal motility, or both. Some physicians recommend that they be taken for no longer than several weeks, but this seems unduly restrictive. Although long-term daily use can produce severe diarrhea that causes hyponatremia, hypokalemia, and dehydration, use for two to three times per week is safe and effective [13].

Of the anthraquinone laxatives, senna is safe and effective in pregnancy [17–19]. When combined with psyllium or other bulk forming agents, smaller amounts of senna may be administered. Senna is administered best at bedtime with fluids three times per week if defecation does not occur spontaneously. Cascara is milder and produces little or no colic. In contrast, aloe (casanthranol) often produces colic and has been associated with congenital malformations [20].

With the removal of phenolphthalein from the market, bisacodyl is the only diphenylmethane laxative available for nonprescription use. Only 5% is absorbed and then excreted in the urine. Bisacodyl, although safe for use in pregnancy, tends to produce more colic than the anthraquinone laxatives, especially when administered orally [13]. It also seems to be effective when administered by suppository.

Laxatives to be avoided during pregnancy include castor oil, because it may initiate premature uterine contractions [20], and saline hyperosmotic

agents, such as magnesium laxatives and phosphosoda, because they promote sodium and water retention, which is inadvisable during pregnancy.

Constipation rarely is complicated by fecal impaction. This may present with “spurious diarrhea” from seepage of liquid stool around the impaction or with symptoms of colonic obstruction. A scybalous impacted mass should be fragmented digitally, followed by administration of a small tap water enema containing mineral oil to soften the mass. Tap water enemas or retention enemas can be administered safely to achieve disimpaction. This can be followed by a bisacodyl suppository to empty the left colon before resuming normal constipation therapy.

### **Hemorrhoids in pregnancy**

Hemorrhoids are common in the adult population. They are estimated to affect approximately 50% of the population at some time. They often first become symptomatic during pregnancy, when they may present with bleeding, pain, or pruritus [21]. Although the pathogenesis is not well understood, hemorrhoids may be caused by prolapse of anal canal cushions, which consist of redundant rectal mucosa, smooth muscle, connective tissue, and blood vessels [21]. Clinical manifestations arise when prolapse leads to congestion, engorgement, and bleeding. Hemorrhoids above the dentate line are internal, whereas those below the dentate line are external. The former arise from the superior hemorrhoidal plexus and are covered by columnar epithelium, whereas the latter arise from the inferior hemorrhoidal plexus and are covered by squamous epithelium.

Multiple factors have been implicated in the development of hemorrhoids, including straining at defecation, vascular engorgement from increased intraabdominal pressure, absence of valves in hemorrhoidal vessels and draining veins, chronic constipation, and, perhaps, genetic factors. As mentioned previously, during pregnancy constipation is increased in frequency or exacerbated, the circulating blood volume increases by 25% to 40%, which can increase venous dilation and engorgement, and venous stasis may be increased by the enlarging gravid uterus or increased pelvic floor laxity [21].

External hemorrhoids require treatment predominantly for acute thrombosis. They rarely bleed unless the thrombosis causes necrosis of overlying skin. In contrast, internal hemorrhoids most often present with painless bleeding or prolapse with discomfort, pruritus, or pain associated with incarceration of the hemorrhoids.

Internal hemorrhoids are classified by degree: first-degree hemorrhoids bleed but do not prolapse and can be visualized only by anoscopy; second-degree hemorrhoids protrude during defecation or with straining but return to their internal position when straining stops; third-degree hemor-

rhoids are continuously prolapsed but can be manually reduced with little effort; and fourth-degree hemorrhoids are irreducibly prolapsed [22].

### **Treatment of hemorrhoids during pregnancy**

External hemorrhoids require no treatment unless they acutely thrombose. If the pain is beginning to remit by the time the patient is evaluated, conservative treatment, consisting of stool softeners, mild analgesics, and several warm sitz baths daily, often suffices. If the pain is severe or unremitting, surgical excision under local anesthesia is safe and effective during pregnancy [21]. Parturition and fetal viability are not affected. Most women require no further treatment after delivery. In contrast, clot incision and removal is an inadequate therapy, because the thrombosis usually recurs [23].

Internal hemorrhoids generally are managed conservatively with anticonstipation measures, such as fiber supplements, fluids, and a high-fiber diet. Pregnant women administered iron supplements should switch to a slow-release formula, which may be less constipating. Minor symptoms, such as anal pruritus and discomfort, can be treated with various nonprescription products, such as skin protectants after each defecation, and topical local anesthetics containing benzocaine, dibucaine, or pramoxine. Products containing epinephrine or phenylephrine, which contract blood vessels thereby reducing hemorrhoidal swelling, should be used cautiously during pregnancy, especially in women with hypertension, diabetes mellitus, or fluid overload. Astringents, such as witch hazel or hydrocortisone, may help relieve pruritus.

For patients who have internal hemorrhoids with refractory symptoms, various office-based procedures are available, such as band ligation, injection, sclerotherapy, and coagulation. Cryotherapy is less effective and more painful and, therefore, an unacceptable alternative in view of better therapies.

Rubber band ligation is effective for first-, second-, or third-degree internal hemorrhoids. Bands are applied in duplicate, to avoid slippage of a single band, at least 1 cm above the dentate line. Complications are minimal in experienced hands. There is a small risk of acute necrotizing perianal sepsis.

Injection sclerotherapy, using 5% phenol in almond oil, quinine, sodium morrhuate, or ethanolamine oleate, is safe and effective during pregnancy with rare recurrences [21,22]. These sclerosing agents are injected into the bases of first- or second-degree hemorrhoids. Infrared photocoagulation or laser coagulation is safe and effective in nonpregnant patients with first- and second-degree hemorrhoids and theoretically is safe in pregnant women [21].

Surgical hemorrhoidectomy is reserved for internal hemorrhoids that prolapse and incarcerate or when office-based procedures fail. Closed

excisional hemorrhoidectomy using local anesthesia has been reported safe and effective during pregnancy [23] and in the puerperium [24,25].

### Diarrhea in pregnancy

Few studies have investigated the prevalence of diarrhea in pregnancy. There is no convincing evidence that the pathogenesis of diarrhea in pregnant women differs from that of the nonpregnant population. Indeed, as discussed previously, pregnancy promotes constipation, not diarrhea. Nevertheless, acute and chronic diarrhea may occur in pregnant women; in such cases, the differential diagnosis is similar to that of nonpregnant women. Most cases of acute diarrhea are caused by infection with a viral, bacterial, or parasitic enteropathogen usually transmitted by the fecal-oral route [26]. In patients with acute self-limited diarrhea and upper gastrointestinal symptoms, viral agents, such as rotavirus and Norwalk virus, are the most frequent cause. Viral agents tend to cause large volume and watery diarrhea, with negative routine analyses of stool. The remaining causes often are bacterial, including *Campylobacter*, *Shigella*, *Escherichia coli*, and *Salmonella*. Bacterial pathogens tend to produce inflammatory diarrhea with frequent stools of small volume, abdominal pain, occasional fever, and blood or leukocytes in the stool. In such cases, diagnostic testing and antibiotic therapy may be beneficial. Noninfectious causes most commonly include medications, followed by functional diarrheas, food intolerances, sugar substitutes such as sorbitol and mannitol, and inflammatory bowel disease. Clinical issues during pregnancy include the use of diagnostic tests and treatments that do not harm the fetus [1,27].

### Treatment of diarrhea during pregnancy

As most episodes of acute diarrhea are mild and self-limited, extensive diagnostic evaluation generally is unnecessary during pregnancy [26]. Evaluation often is warranted if diarrhea is profuse and leads to dehydration, if fever exceeds 38.3°C, if stools are grossly bloody, or if the illness persists for more than 48 hours without improvement. A suggested algorithm, based on clinical features, is shown in Fig. 1. Studies show that flexible sigmoidoscopy is safe during pregnancy and may be employed when clinically indicated [28]. Endoscopy generally is not useful for most otherwise healthy patients with acute diarrhea, but is useful for patients with persistent or chronic diarrhea.

The pregnant woman with uncomplicated self-limited acute diarrhea should receive supportive treatment including restoration of fluid losses, maintaining hydration, correction of electrolyte abnormalities, dietary changes, and symptomatic relief. The belief that diarrhea should be permitted to run its natural course because it is a response that purges the body of

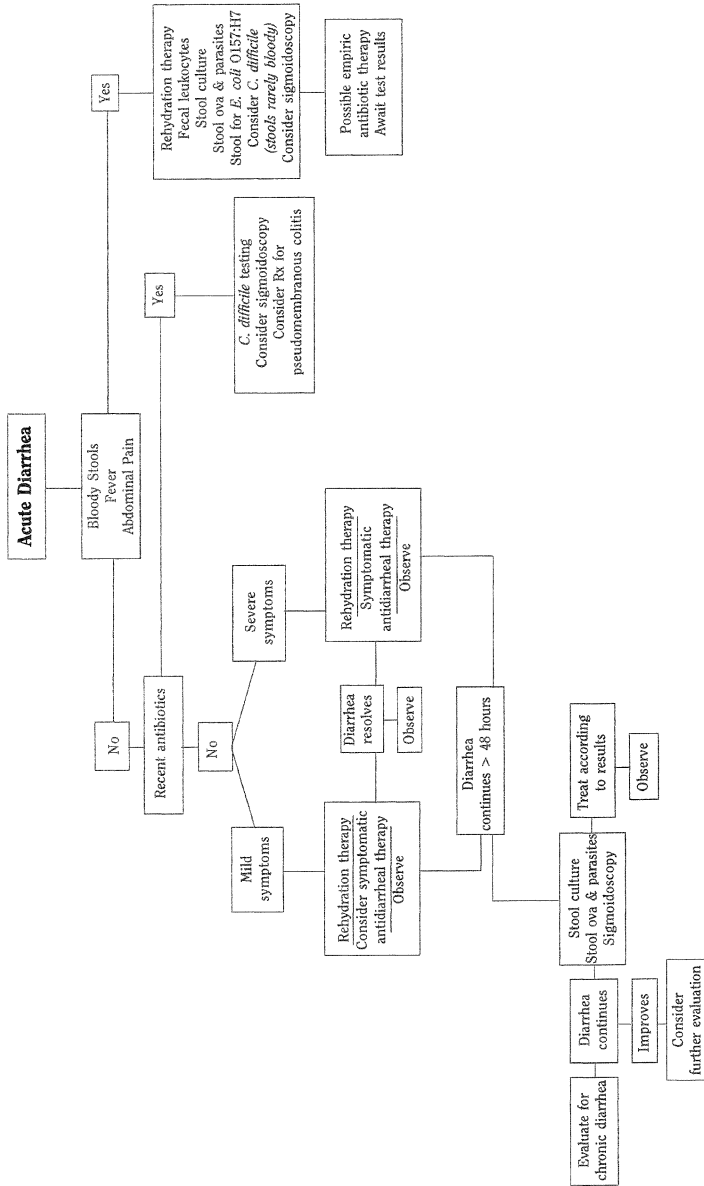


Fig. 1. Algorithm for evaluation and treatment of acute diarrhea (From: Brandt LJ, editor. Acute and chronic diarrhea: a primer on diagnosis and treatment. American college of Gastroenterology. p. 38; with permission.)

infectious enteropathogens now is considered invalid [29]. Many, but not all, antidiarrheal agents are considered safe during pregnancy.

Oral rehydration in adults can be administered at home using juices, non-caffeinated beverages, and nondietetic drinks that do not contain mannitol or sorbitol. Orange juice and bananas provide potassium, and salted crackers and broths provide needed electrolyte replacement. For high-volume diarrhea, the World Health Organization recommends a solution consisting of 1 liter of water, 1 level teaspoon of salt, and 8 level teaspoons of sugar [30].

Maintenance of oral food intake in adults with acute diarrhea is controversial. Small, frequent meals with little fat, caffeine, and artificial sweeteners are best. Solutes in ingested food enhance net fluid and electrolyte absorption, but may stimulate defecation via the gastrocolonic response.

Antidiarrheals are divided into adsorbents, bulk agents, opioids, and bismuth containing compounds.

### *Adsorbents*

Adsorbents are inert substances, such as kaolin, that bind water thereby decreasing fecal water and producing better formed stools. They do not, however, seem to reduce fluid loss and seem to have little clinical value compared with opioid agents [17].

### *Bismuth subsalicylate*

This agent has been used to treat patients with traveler's diarrhea and other acute diarrheas associated with nausea and vomiting. Bismuth seems to have antimicrobial activity, whereas subsalicylate inhibits prostaglandin synthetase and decreases intestinal fluid secretion. Orally administered salicylate, however, is absorbed partly and has been found to be teratogenic in animals [20]. Salicylates also have been associated with decreased birth weight, neonatal hemorrhage, and increased perinatal mortality [1]. They are not recommended during the second and third trimesters of pregnancy.

### *Opioids*

Most opiate antidiarrheal agents act centrally and peripherally to decrease gastrointestinal transit, thereby increasing the time available for water and electrolyte absorption and decreasing the bowel frequency. Loperamide, however, does not cross the adult blood-brain barrier, acts only peripherally, has fewer central side-effects than other opioids, and has little potential for abuse. This accounts for its availability without prescription, in contrast to the other opiate antidiarrheal agents.

In one study, loperamide and codeine were superior to diphenoxylate with atropine in reducing rectal urgency and producing a solid stool [31]. All

the drugs were equivalent in reducing the frequency of bowel movements. Diphenoxylate with atropine use during the second or third trimester was found to be teratogenic in animals and humans [20,32]. Loperamide is, therefore, the drug of choice as an antidiarrheal agent during pregnancy. Although there is no data in humans, loperamide has been reported not to be harmful to animal fetuses at doses up to 30 times the maximum human recommended dose [20].

In summary, the management of acute diarrhea in the pregnant woman consists of oral rehydration, dietary changes, and, if symptoms are disabling, small amounts of loperamide. Bismuth subsalicylate may be safe during the first trimester, but clinically is less effective than loperamide. Loperamide probably is safe during pregnancy, but untested in humans.

#### *Treatment of bacterial and parasitic diarrheas*

Antibiotics generally are indicated when a treatable pathogen is identified in stool samples or when a bacterial pathogen is suspected clinically, as in traveler's diarrhea. Many antibiotics are, however, contraindicated or inadvisable during pregnancy, as listed in Box 2. For example, metronidazole is

#### **Box 2. Risk of selected antibiotics during pregnancy [35,36]**

##### *Class B: No risk in controlled animal studies*

Metronidazole (after first trimester)  
All cephalosporins (except moxalactam)  
All erythromycins (except estolate)  
Sulfa antibiotics (before third trimester)

##### *Class C: Small risk in controlled animal studies*

All fluoroquinones  
Trimethoprim/sulfamethoxazole  
Vancomycin<sup>a</sup>

##### *Class D: Strong evidence of risk to human fetus*

Metronidazole (first trimester)<sup>b</sup>  
Sulfa antibiotics (third trimester)  
All tetracycline antibiotics  
Erythromycin estolate

<sup>a</sup> Oral compound poorly absorbed.

<sup>b</sup> See reference [33] for new evidence of safety.

**Box 3. Treatment of diarrhea during pregnancy***Effective and safe*

Oral fluid replacement

Dietary changes

Loperamide

Antibiotics

Erythromycin stearate: *Campylobacter jejuni*Cephalosporins: *Shigella*Ampicillin: *Salmonella* (moderate/severe)*Effective and low risk*

Codeine, opium

Metronidazole (second and third trimesters, possibly first trimester): *Giardia lamblia*, *Clostridium difficile*, *Entameba histolytica*Vancomycin (oral): *Clostridium difficile*

Fluoroquinolones

*Effective and high risk – AVOID*

Diphenoxylate with atropine

Trimethoprim/sulfamethoxazole (third trimester)

Erythromycin estolate

an effective therapy for *Clostridium difficile*, *Giardia lamblia*, and *Entameba histolytica* infection, but may be teratogenic during the first trimester in humans. Its use is discouraged during the first trimester [1,27], although some evidence suggests it may be safe even during early pregnancy [33]. Similarly, vancomycin is categorized as a FDA Pregnancy Category C drug, with no animal data and relatively few human data available. It should have a better safety profile when administered orally than intravenously because of minimal gastrointestinal absorption. Ampicillin and erythromycin stearate seem to be safe during pregnancy. The latter antibiotic is the antibiotic of choice for *Campylobacter jejuni* infection. In view of ampicillin resistance in shigellosis and of concerns regarding ciprofloxacin and trimethoprim-sulfamethoxazole teratogenicity, second or third generation cephalosporins, such as cefuroxime and cefixime, are safe and effective for acute shigellosis [34]. Travel to third world countries, where the risk of traveler's diarrhea is significant, is discouraged during pregnancy. The safety and efficacy of various therapies for diarrhea during pregnancy are summarized in Box 3.

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