Cholera in the Americas

Guidelines for the Clinician

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UNTIL recently, cholera has been rare in the Americas. However, epidemic cholera appeared in Peru in January 1991 and spread rapidly through Latin America. In the first year of this epidemic, 17 cases of cholera that were associated with travel to Latin America were reported in the United States. More cases are likely to be seen. Although no spread from these imported cases has occurred, it is possible that some areas of the United States with poor sanitary conditions may be at risk for limited continued transmission of cholera. This article reviews the methods for recognition, diagnosis, and treatment of cholera.

HISTORICAL BACKGROUND

Six pandemics of cholera spread throughout the world before the 20th century, including three successive waves of epidemic cholera that affected the United States in the 19th century until the “sanitary revolution” brought the modernization of water and sewage systems.¹ The current seventh pandemic began in Asia, probably in 1961, and spread to Africa, Europe, and Oceania, but spared the Western Hemisphere.²

From 1961 to 1990, 41 sporadic cases were reported among US travelers returning from countries with cholera (Centers for Disease Control [CDC], Atlanta, Ga, unpublished data, 1991). In 1973, a strain of Vibrio cholerae 01 unique to the Gulf of Mexico was identified.³ Since then, 65 cases associated with this endemic focus have been reported in the United States³⁴ (CDC, unpublished data, 1991). This strain has not caused epidemic cholera. Most of these cases were associated with consumption of raw or undercooked shellfish. In one instance, cholera spread on a Gulf Coast oil rig, where a cross-connection in water lines permitted contamination of food.⁴ No other subsequent spread from domestic or imported cases has been documented.

In late January 1991, toxigenic V cholerae 01, serotype Inaba, biotype El Tor, appeared nearly simultaneously in several coastal Peruvian cities; it was the first time in this century that cholera was reported in South America.⁶ The epidemic has spread rapidly. As of January 8, 1992, a total of 358,581 probable cholera cases and 3,871 deaths have been reported from 14 countries in North and South America⁷ (CDC, unpublished data, 1992).

In 1991, 17 cases associated with the epidemic in South America were reported among US residents. Six were in travelers to South America⁸⁻¹⁰ (CDC, unpublished data, 1992) and 11 were in persons who ate crabs brought back from Ecuador by other travelers.⁴⁻⁵ No secondary spread has been documented.

THE CAUSATIVE ORGANISM

Vibrio cholerae is a gram-negative bacterium of the family Vibrionaceae. It is a curved bacillus (vibrio is Greek for comma) with a polar flagellum. Although there are many serogroups, only serogroup O1 has exhibited the ability to cause epidemics. Vibrio cholerae O1 is divided into two serotypes, Inaba and Ogawa, and two biotypes, classic and El Tor. Strains that express both Inaba and Ogawa antigens are sometimes called serotype Hikojima. Cholera is caused by cholera toxin–producing (toxigenic) strains of the O1 antigenic serogroup. Nontoxigenic O1 strains and non-O1 strains of V cholerae can cause diarrhea and sepsis but do not cause epidemics.¹¹

The enterotoxin of V cholerae O1 is composed of an A subunit and five B subunits arranged in a circular form. The A subunit activates adenylate cyclase in intestinal mucosa, causing the secretion of water and electrolytes characteristic of cholera. The B subunit binds to cellular receptors but does not by itself cause diarrhea.¹²

Cholera is spread through contaminated water and food. Reported vehicles of transmission have included contaminated water, contaminated ice, raw oysters, improperly preserved fish, undercooked shellfish such as crabs, and rice or grain gruels that have been left unrefrigerated for many hours.¹³⁻¹⁵ Both waterborne and foodborne transmission has occurred in Latin America, with waterborne exposures accounting for most cases.¹⁶⁻¹⁸ The incubation period is typically 1 to 3 days, with a range of a few hours to 5 days. The organism is acid-sensitive and will not survive in foods with low pH. Persons with low gastric acidity are at increased risk for cholera infection.¹⁹⁻²⁰

CLINICAL PRESENTATION

Toxigenic V cholerae O1 causes a broad spectrum of clinical illness. Most infected persons display no symptoms, some have mild to moderate diarrhea, and a relatively small proportion (2% to 5%) have severe watery diarrhea, vomiting, and dehydration (cholera gravis). Individuals with mild to moderate illness may have symptoms indistinguishable from those of diarrhea of other causes. Persons with severe diarrhea may die within a few hours if no treatment is provided.¹⁹

The clinical characteristics of severe cholera are caused by massive fluid and
electrolyte loss. The illness begins as painless diarrhea without abdominal cramps or fever. The stools quickly lose their fecal character and become watery and colorless with small white flecks of mucus, classically described as “rice-water stools.” The stool is practically odorless except for a mild fishy smell. Vomiting is common and may be severe. Patients may lose up to 1 L of fluid per hour in the first 24 hours and may lose more than 10% of their body weight. These huge volume losses, in extreme cases, may lead to circulatory collapse and death in as few as 2 hours. More commonly, the diarrhea leads to severe dehydration with shock in 4 to 12 hours and death in 18 hours to several days.22 Dehydration may be manifested by hypotension with rapid, thready pulse, weakness, anuria, loss of skin turgor, sunken eyes, dry mucous membranes, and thirst. The mental status of patients is often altered; they may appear drowsy or even unconscious, but are usually arousable and think lucidly, even with extreme dehydration.21 Renal failure may follow hypovolemic shock, especially if volume replacement is not instituted quickly, if volume replacement is inadequate, or if prolonged or recurrent hypovolemia occurs.

The watery stools seen in severe cholera have high concentrations of sodium, potassium, and bicarbonate (Table 1). Clinical manifestations other than those from volume depletion are the result of severe electrolyte imbalances. Bicarbonate losses lead to acidosis, which may increase the frequency of vomiting. The vomiting hinders oral fluid and electrolyte replacement, thus allowing continued unincremented compensatory losses of bicarbonate to worsen acidosis. Hypokalemia may promote cardiac arrhythmia and renal failure and may be the cause of severe leg cramps. Unconsciousness and convulsions, especially in children, may indicate hypoglycemia, a rare complication in severe cholera.

**DIAGNOSIS**

Cholera is definitively diagnosed by isolation of toxigenic *V cholerae* O1 from a stool specimen or a rectal swab. Rectal swabs should be placed in transport medium such as Cary-Blair. Swabs can be refrigerated or stored at room temperature. If swabs and transport media are not available, a small amount of liquid stool can be collected and sealed in a jar or vial and refrigerated until plated, or stool can be placed on blotting paper and inserted into a leak-proof plastic bag for transport.

Because few laboratories in the United States routinely culture for *V cholerae* or other vibrios, clinicians should request that appropriate cultures be performed for clinically suspected cases. The specimen is plated on thiosulfate citrate bile salts sucrose (TCBS) agar either directly or after optional enrichment in alkaline peptone water. Because they ferment sucrose, *V cholerae* colonies will appear yellow on TCBS. Colonies are then biochemically identified and serologically confirmed as *V cholerae* O1. Diagnostic antisera are commercially available.22 Isolated specimens of *V cholerae* O1 should be sent to the state health department laboratory for confirmation and then sent to CDC for testing for production of cholera toxin. Antimicrobial susceptibility can be determined by disk diffusion or other standard methods.

Although strains of nontoxicogenic *V cholerae* O1 and non-O1 *V cholerae* may cause sporadic cases of diarrhea, they are not associated with epidemic illness. The diagnosis of cholera should not be based solely on the isolation of *V cholerae* from a stool specimen without confirming that it is serogroup O1 and produces cholera toxin.

Cholera should be suspected in patients presenting with severe watery diarrhea and vomiting, especially those with severe dehydration. Diarrhea from other causes is less common in adults than in infants and young children, so dehydration in an adult raises the suspicion of cholera. Deaths from diarrhea should be investigated. Clinical suspicion should be increased in cases of mild diarrheal illness in persons returning from areas known to have cholera or in persons who consumed raw or undercooked seafood in the 5 days preceding onset of illness.

Isolating *V cholerae* O1 from stool samples of cholera patients may be difficult if the samples are collected after antimicrobial therapy is begun or if samples are collected late in the course of illness. Vibrioalchial antibody titers peak 10 to 21 days after infection and can be used to confirm *V cholerae* O1 infection.25 A fourfold rise in vibrioalchial antibody titers between acute (first 5 days of illness) and early convalescent serum samples (10 to 21 days) or a fourfold decline in vibrioalchial antibody titers between early and late (>2 months) convalescent serum samples is considered diagnostic in clinically compatible cases.23 Vibrioalchial antibody titers can be determined on serum samples at CDC. Antitoxic antibody titers increase 2 to 4 weeks after onset and remain elevated, so a rise in antitoxic antibody titers may also be diagnostic. Because of the similarity of antitoxic antibodies and antibodies against *Escherichia coli* heat-labile toxin, which is a common infection in developing countries, highly specific reagents must be used.22,24

Clinicians should not wait for laboratory confirmation of cholera to begin treatment in clinically suspected cases.

**TREATMENT**

**Oral Rehydration**

Since cholera can kill by dehydration in as few as 2 hours and as many as 50% of untreated severe cases may be fatal, rapid administration of effective oral fluid replacement is the mainstay of therapy. Therefore, access to rehydration therapy is crucial to prevent death. With proper treatment, less than 1% of patients die.23,24 Treatment options depend on the resources available and the knowledge and training of the health care providers. The guidelines for the assessment of the degree of dehydration and rehydration treatment options detailed here are based on World Health Organization (WHO) recommendations.25 Other algorithms for evaluation and treatment of patients with diarrhea are also available.12,26,27

When a patient presents with diarrhea, the degree of dehydration (no, some, or severe signs of dehydration) should be determined rapidly (Table 2).26 Particular emphasis should be placed on the patient's mental status (eg, lethargic or unconscious), thirst, skin turgor (although not reliable in malnourished or obese individuals), and pulse. Treatment decisions may also be influenced if

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**Table 1.—Electrolyte Composition of Diarrheal Stools From Persons With Cholera and Composition of Rehydration Solutions**

<table>
<thead>
<tr>
<th></th>
<th>Na⁺, mEq/L</th>
<th>K⁺, mEq/L</th>
<th>Cl⁻, mEq/L</th>
<th>Base+, mEq/L</th>
<th>Glucose, g/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults</td>
<td>135</td>
<td>15</td>
<td>100</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Children</td>
<td>105</td>
<td>25</td>
<td>90</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Oral solutions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WHO-ORS</td>
<td>90</td>
<td>20</td>
<td>80</td>
<td>30</td>
<td>20</td>
</tr>
<tr>
<td>Rehydrate</td>
<td>75</td>
<td>20</td>
<td>65</td>
<td>30</td>
<td>25</td>
</tr>
<tr>
<td>Intravenous Ringer's</td>
<td>130</td>
<td>4</td>
<td>109</td>
<td>28</td>
<td></td>
</tr>
</tbody>
</table>

*Na⁺ indicates sodium; K⁺, potassium; Cl⁻, chloride; WHO, World Health Organization; and ORS, oral rehydration salts.

†Consists of bicarbonate, citrate, or lactate.
a patient experiences or exhibits anuria, altered breathing patterns (eg, rapid or deep), the passing of more than 10 watery stools per day, and severe vomiting.

If health workers are familiar with use of oral rehydration, patients with no or some dehydration can be treated largely or completely with oral solutions.23-25 Even patients with some vomiting absorb oral solutions taken in small sips. At present, WHO oral rehydration salt (ORS) packets (WHO-ORS, Johns Brothers, St. Louis, Mo) provide the best oral solution, containing the proper balance of electrolytes for treating cholera. Each packet contains glucose (20 g), sodium chloride (3.5 g), potassium chloride (1.5 g), and either trisodium citrate (2.9 g) or sodium bicarbonate (2.5 g) and is mixed with 1 L of water26 (Table 1).27 "Rehydration (Ross Laboratories, Colombus, Ohio) can be used if WHO-ORS is not available;28 however, its content of sodium is approximately 20% lower, so larger volumes are required for rehydration. WHO-ORS is available from the manufacturer; Rehydration is available over the counter.29 Other oral solutions with sodium concentrations less than 75 mEq/L are inappropriate for treatment of dehydration due to cholera. If ORS is not available, rehydration therapy should begin with intravenous fluids. The amount of ORS solution that should be given (in milliliters) within the first 4 hours to patients with some dehydration may be calculated by multiplying the patient's weight (in kilograms) by 100. For example, an infant who weighs 5 kg needs about 500 mL and a 15-year-old child who weighs 30 kg needs about 3000 mL within the first 4 hours of rehydration therapy. If the patient wants additional ORS solution, more may be given. For infants younger than 6 months who are not breast-fed, 100 to 200 mL of water should also be given during this period. Patients should be reevaluated after 4 hours, or sooner if they are not drinking sufficient ORS solution or if their condition appears to be deteriorating.25 Staff and family members should help facilitate adequate intake of ORS solution.

Average stool losses in the first 24 hours are 200 mL/kg and may reach 350 mL/kg in some persons. To compensate for ongoing fluid losses, ORS solution should also be given after each loose stool. Children younger than 24 months should be given 50 to 100 mL of ORS solution after each loose stool, children 2 to 10 years old, 100 to 200 mL, and persons over 10 years old, as much as desired. In addition to ORS solution, persons should drink water ad libitum.30

Patients with no dehydration should be given enough ORS solution to replace stool losses using the above guidelines. If, after observation, the patient's condition is stable enough to be treated at home, caretakers should be taught how to administer ORS solution and should be given a 2-day supply. The caretaker should be instructed to return with the patient if the patient develops an increased number of watery stools, experiences marked thirst, is unable to eat or drink adequately, or vomits repeatedly.

New cereal-based oral solutions have shown promise in treating patients with diarrhea in clinical trials by decreasing diarrheal volume, loss, rehydration, and shortening the duration of disease.27 However, the role of these solutions in treatment of cholera has not been defined.

**Intravenous Therapy**

Intravenous therapy is necessary for patients who are severely dehydrated (loss of 10% or more of body weight), especially those who are lethargic, unable to drink, or in hypovolemic shock. Even patients with no detectable pulse on arrival at the hospital may fully recover with rapid rehydration. The only intravenous solution readily available in the United States with the electrolyte composition needed for treating cholera is Ringer's lactate solution (Table 1). Normal saline is less effective for treatment because it contains no bicarbonate or potassium, should only be used if Ringer's lactate solution is unavailable, and should be accompanied by ORS solution as soon as the patient can drink. Plain glucose in water is ineffective and should not be used. Severely dehydrated adults will require several liters of fluid immediately to restore an adequate circulating volume. For example, a 60-kg person with a 10% loss of body weight will have a fluid deficit of 6 L. Half of this volume should be given immediately (over 30 minutes) using large-bore intravenous catheters (Table 3).32 Patients should be reassessed frequently. Overhydration can occasionally occur in infants and the elderly but is rare if guidelines are followed. Following rehydration, urine output usually resumes within 6 to 8 hours. Since Ringer's lactate solution contains no glucose and insufficient potassium for complete replacement, ORS solution should be given as soon as the patient can drink, even while the initial rehydration is being completed intravenously. Patients with cholera have substantial ongoing fluid losses that also need to be replaced. Replacement therapy should be changed from the intravenous to the oral route at the earliest practical moment. Patients should be encouraged to begin eating as soon as possible. A full liquid diet, ce-

<table>
<thead>
<tr>
<th>Examination</th>
<th>No Signs of Dehydration</th>
<th>Some Dehydration</th>
<th>Severe Dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Look at</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mental status</td>
<td>Well, alert</td>
<td>Restless, irritable†</td>
<td>Lethargic or unconscious; floppy infant†</td>
</tr>
<tr>
<td>Eyes</td>
<td>Normal</td>
<td>Sunken</td>
<td>Very sunken and dry</td>
</tr>
<tr>
<td>Tears</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Mouth/tongue</td>
<td>Moist</td>
<td>Dry</td>
<td>Very dry</td>
</tr>
<tr>
<td>Thirst</td>
<td>Drinks normally, not thirsty</td>
<td>Thirsty, drinks eagerly†</td>
<td>Drinks poorly or not able to drink†</td>
</tr>
<tr>
<td>Feel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin pinch</td>
<td>Goes back rapidly</td>
<td>Goes back slowly†</td>
<td>Goes back very slowly†</td>
</tr>
<tr>
<td>Pulse</td>
<td>Normal</td>
<td>Faster than normal†</td>
<td>Very fast, weak or nonpalpable†</td>
</tr>
<tr>
<td>Fontanelle</td>
<td>Normal</td>
<td>Sunken</td>
<td>Very sunken*</td>
</tr>
</tbody>
</table>

*Modified from reference 25. Important signs and symptoms for assessment of dehydration.

<table>
<thead>
<tr>
<th>Age</th>
<th>First Give</th>
<th>Then Give</th>
</tr>
</thead>
<tbody>
<tr>
<td>infants (under 12 mo)</td>
<td>1 h 20 mL/kg</td>
<td>5 h 70 mL/kg</td>
</tr>
<tr>
<td>Children and adults</td>
<td>30 min*</td>
<td>2.5 h</td>
</tr>
</tbody>
</table>

*Repeat if radial pulse is still very weak or not detectable.
Table 4.—Antibiotics for Treatment of Cholera*

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>Administration</th>
<th>Children</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First choices</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetracycline</td>
<td>Four times per day for 3 d</td>
<td>12.5 mg/kg</td>
<td>500 mg</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>One single dose</td>
<td>6 mg/kg</td>
<td>300 mg</td>
</tr>
<tr>
<td><strong>Alternatives</strong> (tetracycline-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>resistant strains)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Adults: four times per day</td>
<td>10 mg/kg</td>
<td>250 mg</td>
</tr>
<tr>
<td></td>
<td>for 3 d</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Children: three times per</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>day for 3 d</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TMP-SMX†</td>
<td>Two times per day for 3 d</td>
<td>5 mg/kg TMP,</td>
<td>160 mg TMP,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>25 mg/kg SMX</td>
<td>600 mg SMX</td>
</tr>
<tr>
<td>Furazolidone</td>
<td>Four times per day for 3 d</td>
<td>1.25 mg/kg</td>
<td>100 mg</td>
</tr>
</tbody>
</table>

*Modified from reference 20.
†TMP indicates trimethoprim; and SMX, sulfamethoxazole.

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real, and rice may be used initially. If possible, the patient should be observed until diarrhea stops or is infrequent and of small volume, especially any patient who presented with severe dehydration.

In summary, cholera causes a more severe form of diarrhea than physicians may be accustomed to treating. Large volumes of fluids are necessary, and rehydration should be aggressive. Oral rehydration therapy for cholera with the WHO-ORS solution has been successful in many countries. However, if health care workers are unfamiliar with mixing or administering ORS, or if ORS is unavailable, they should use the methods of rehydration that are most familiar to them.

**Antibiotics**

Although the mainstay of treatment is volume and electrolyte replacement, antibiotic treatment decreases the duration of illness, the requirements for fluid replacement, and the period of *Vibrio* excretion. In areas with poor sanitation, antibiotics also decrease the number of organisms introduced into the environment. Since vomiting usually stops within hours after beginning rehydration, antibiotics should be given orally. It is neither urgent nor additionally beneficial to use injectable antibiotics.

Tetracycline and doxycycline are the drugs of choice for treatment of cholera (Table 4). Doxycycline is easier to administer because only a single dose is required. If strains are resistant to tetracyclines or if tetracyclines are unavailable for use in children, erythromycin, the combination of trimethoprim and sulfamethoxazole, or furazolidone may be used. Chloramphenicol can also be used. Recently, norfloxacin was found to be effective for treatment of cholera in Calcutta, India. Since antibiotic-resistant *V cholerae* O1 has been documented in South America (CDC, unpublished data, 1991) and in other continents, it is important to determine the antibiotic susceptibility of newly isolated organisms and to be aware of antimicrobial susceptibility patterns in affected areas.

No other antidiarrheal, antispasmodic, cardiotonic, or corticosteroid drugs have any place in the treatment of patients with cholera.

**Treatment of Contacts**

In developing countries, if the household secondary attack rate is known to be high, family contacts of patients with cholera may be treated prophylactically with tetracycline or doxycycline to prevent illness. Ideally, prophylactic treatment should begin within 24 hours after the index case is identified. No documented instances of intrafamilial transmission have been reported in the United States since 1961 (CDC, unpublished data, 1991). Prophylactic treatment in the United States is not recommended unless unusual local sanitary and hygienic conditions make secondary transmission likely.

Families should be instructed to wash their hands with soap after defecating and before eating and preparing food, and to clean clothes and bed sheets contaminated by a patient’s feces with soap and bleach. The sanitary facilities in a cholera patient’s home should be inspected to make sure that the patient’s feces are disposed of via adequate sewage treatment or septic tank or are otherwise decontaminated. Patients with cholera can be treated in any hospital in which enteric precautions can be assured and sewage adequately treated.

**Epidemiologic Considerations**

**Reporting of Cases**

Cholera is one of three internationally notifiable diseases. All isolates of *V cholerae* O1 should be sent to state health departments and to CDC for confirmation and characterization. Cases confirmed by isolation of toxigenic *V cholerae* O1 or by testing acute and convalescent serum samples for vibriocidal or antitoxic antibodies are reported to WHO, which maintains global cholera surveillance.

**Avoid**

Unboiled or untreated water or ice
Food and beverages from street vendors
Raw or undercooked fish and shellfish including ceviche
Salads and raw vegetables

** Usually Safe**
Cooked foods that are still hot
Fruits peeled by the traveler
Carbonated bottled water (without ice)
Other carbonated beverages

**Table 5. — Advice for Travelers to Areas Affected by Epidemic Cholera**

**Epidemiologic Investigations**

Local and state health departments should investigate all possible cases of *V cholerae* O1. The source of infection should be determined to avert additional exposure and illness. The CDC can assist with these epidemiologic investigations.

**Mass Chemoprophylaxis**

Antibiotic treatment of an entire community, or mass chemoprophylaxis, has not been shown to limit the spread of cholera. It takes valuable resources away from effective control measures and may contribute to the emergence of antibiotic resistance. The only time mass treatment of a large group may be justified is when cholera occurs in a closed group that may have had a common exposure, such as aboard a ship.

**Vaccine**

One cholera vaccine, administered parenterally, is currently licensed in the United States. Field trials conducted in areas with endemic cholera have demonstrated only 50% efficacy in reducing the incidence of clinical illness. This protection lasts for only 3 to 6 months. It does not prevent *Vibrio* excretion or apparent infection and therefore is unlikely to prevent transmission of infection. In 1970, the US Public Health Service ended the vaccination requirement for persons returning to the United States from cholera-affected areas. The vaccine should not be administered to contacts of patients with cholera or used to control the spread of infection and is not recommended for international travelers.

Several experimental oral vaccines have shown somewhat higher efficacy with fewer side effects than the parenteral vaccine. However, their role in future efforts to control the spread of cholera has not been established.

**TRAVELERS**

The risk of acquiring cholera for US travelers in an affected area is thought to be less than one per 500,000 travelers; during the first 20 years of the seventh pandemic, only 10 cases of cholera were reported among US travelers. The six travelers who developed cholera after visiting South America in 1991 all con-
Travelers may want to bring WHO-ORS to affected areas for personal use. Travelers to areas with cholerawho develop watery diarrhea, especially if accompanied by vomiting, should seek medical attention immediately.

A CDC travelers' hotline is available in English and Spanish for persons planning travel to Central and South America; the telephone numbers are (404) 382-4559 (English) and (404) 380-3132 (Spanish).

**CONCLUSION**

With the reintroduction of cholera into Latin America, physicians should be prepared to diagnose and treat cholera. In severe cholera, rapid replacement of fluids and electrolytes is the only way to save lives.

Since this manuscript was submitted for publication, the authors have learned the following: Between January 1 and February 29, 1992, forty-two cases of cholera have been identified in travelers to and from Latin America, including at least 40 cases and one death among those who had all been passengers on the same airline flight from South America to the United States.

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