

Control of
**Communicable
Diseases** Manual



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BOTULISM

INTESTINAL BOTULISM, formerly

INFANT BOTULISM ICD-9 005.1; ICD-10A05.1

1. Identification--There are three forms of botulism--foodborne (the classic form), wound and intestinal (infant and adult) botulism. The site of toxin production is different for each of the forms but all share the flaccid paralysis that results from botulinum neurotoxin. Intestinal botulism has been proposed as the new designation for what had been called infant botulism. This new name has not been officially accepted as of mid-1999, but will be generally used in this chapter instead of infant botulism.

Foodborne botulism is a severe intoxication resulting from ingestion of preformed toxin present in contaminated food. The illness is characterized by acute bilateral cranial nerve impairment and descending weakness or paralysis. Visual difficulty (blurred or double vision), dysphagia and dry mouth are often the first complaints. These symptoms may extend to a symmetrical flaccid paralysis in a paradoxically alert person. Vomiting and constipation or diarrhea may be present initially. Fever is absent unless a complicating infection occurs. The case-fatality rate in the USA is 5%-10%. Recovery may take months.

In **wound botulism** the same clinical picture is seen after the causative organism contaminates a wound in which anaerobic conditions develop.

Intestinal (infant) botulism is the most common form of botulism in the USA; it results from ingestion of *Clostridium botulinum* spores with subsequent outgrowth and in-vivo toxin production in the large intestine. It affects infants under 1 year of age almost exclusively, but can affect adults who have altered GI anatomy and microflora. The illness typically begins with constipation, followed by lethargy, listlessness, poor feeding, ptosis, difficulty swallowing, loss of head control, hypotonia extending to generalized weakness (the "floppy baby") and, in some cases, respiratory insufficiency and arrest. Infant botulism has a wide spectrum of clinical severity, ranging from mild illness with gradual onset to sudden infant death; some studies suggest that it may cause an estimated 5% of cases of sudden infant death syndrome (SIDS). The case-fatality rate of hospitalized cases in the USA is less than 1%; without access to hospitals with pediatric intensive care units, more would die.

Diagnosis of foodborne botulism is made by demonstration of botulinum toxin in serum, stool, gastric aspirate or incriminated food; or by culture of *C. botulinum* from gastric aspirate or stool in a clinical case. Identification of organisms in a suspected food is helpful but not diagnostic because botulinum spores are ubiquitous; the presence of toxin in a suspected contaminated food source is more significant. The diagnosis may be accepted in a person with the clinical syndrome who had consumed a food item incriminated in a laboratory confirmed case. Wound botulism is diagnosed by toxin in serum or by positive wound culture. Electromyography with rapid repetitive stimulation can be useful in corroborating the clinical diagnosis for all forms of botulism.

The diagnosis of intestinal botulism is established by identification of *C. botulinum* organisms and/or toxin in patient's feces or in autopsy specimens. Toxin is rarely detected in the sera of patients.

2. Infectious agent--Foodborne botulism is caused by toxins produced by *Clostridium botulinum*, a spore forming obligate anaerobic bacillus. A few nanograms of the toxin can cause illness. Most human outbreaks are due to types A, B, E and rarely to type F. Type G has been isolated from soil and autopsy specimens but an etiologic role in botulism has not been established. Type E outbreaks are usually related to fish, seafood and meat from marine mammals.

Toxin is produced in improperly processed, canned, low acid or alkaline foods, and in pasteurized and lightly cured foods held without refrigeration, especially in airtight packaging. The toxin is destroyed by boiling; inactivation of spores requires much higher temperatures. Type E toxin can be produced slowly at temperatures as low as 3°C (37.4°F), which is lower than that of ordinary refrigeration.

Most cases of infant botulism have been caused by type A or B. A few cases (toxin types E and F) have been reported from neurotoxicogenic clostridial species *C. butyricum* and *C. baratii*, respectively.

3. Occurrence--Worldwide; sporadic cases, family and general outbreaks occur where food products are prepared or preserved by methods that do not destroy the spores and permit toxin formation. Cases rarely result from commercially processed products; outbreaks have occurred from contamination through cans damaged after processing. Cases of intestinal botulism have been reported from five continents: Asia, Australia, Europe, and North and South America. The actual incidence and distribution of intestinal botulism are unknown because physician awareness and diagnostic testing remain limited, as demonstrated by a review of intestinal botulism cases reported between 1976, when it was first recognized in California, and the beginning of 1999. Of the 1,700 cumulative global case total, over 1,400 were reported by the USA, with close to half of those cases reported by California. Internationally, about 150 cases have been detected in Argentina; less than 20 each in Australia and Japan; less than 15 in Canada; and about 30 from Europe (mostly Italy and the UK), with scattered reports from Chile, China, Israel and Yemen.

4. Reservoir--Spores are ubiquitous in soil worldwide; they are frequently recovered from agricultural products, including honey. Spores are also found in marine sediments and in the intestinal tract of animals, including fish.

5. Mode of transmission--Foodborne botulism is acquired by ingestion of food in which toxin has been formed, predominantly after inadequate heating during preservation and without subsequent adequate cooking. Most poisonings in the USA are due to home canned vegetables and fruits; meat is an infrequent vehicle. Several outbreaks have recently occurred following consumption of unviscerated fish. Cases associated with baked potatoes and improperly handled commercial potpies have been reported. One recent outbreak was attributed to sautéed onions, two others to minced garlic in oil. Some of these recent outbreaks originated in restaurants. Newer varieties of certain garden foods such as tomatoes, formerly considered too acidic to support growth of *C. botulinum*, may no longer be low hazard foods for home canning.

In Canada and Alaska, outbreaks have been associated with seal meat, smoked salmon and fermented salmon eggs. In Europe, most cases are due to sausages and smoked or preserved meats; in Japan, to seafood. These differences have been attributed in part to the greater use of sodium nitrite for preserving meats in the USA.

Wound botulism cases often result from contamination of the wounds by ground-in soil or gravel or from improperly treated open fractures. Wound botulism has been reported among chronic drug abusers (primarily in dermal abscesses from subcutaneous injection of heroin and also from sinusitis in cocaine "sniffers").

Intestinal botulism arises from ingestion of botulinum spores that then germinate in the colon, rather than by ingestion of preformed toxin. Possible sources of spores for infants are multiple, and include foods and dust. Honey, fed on occasion to infants, can contain *C. botulinum* spores.

6. Incubation period--Neurologic symptoms of foodborne botulism usually appear within 12-36 hours, sometimes several days, after eating contaminated food. In general, the shorter the incubation period, the more severe the disease and the higher the case-fatality rate. The incubation period of intestinal botulism in infants is unknown, since the precise time that the infant ingested the causal botulinum spores cannot be determined.

7. Period of communicability--Despite excretion of *C. botulinum* toxin and organisms at high levels (ca. 10^6 organisms/g) in the feces of intestinal botulism patients for weeks to months after onset of illness, no instance of secondary person to person transmission has been documented. Foodborne botulism patients typically excrete the

toxin and organisms for shorter periods.

8. Susceptibility and resistance--Susceptibility is general. Almost all patients hospitalized with intestinal botulism have been between 2 weeks and 1 year of age; 94% were less than 6 months, and the median age at onset was 13 weeks. Cases of intestinal botulism have occurred in all major racial and ethnic groups. Adults with special bowel problems leading to unusual GI flora (or with a flora unintentionally altered by antibiotic treatment for other purposes) may be susceptible to intestinal botulism.

9. Methods of control–

A. Preventive Measures:

- 1) Ensure effective control of processing and preparation of commercially canned and preserved foods.
- 2) Educate those concerned with home canning and other food preservation techniques regarding the proper time, pressure and temperature required to destroy spores, the need for adequately refrigerated storage of incompletely processed foods, and the effectiveness of boiling, with stirring, home canned vegetables for at least 10 minutes to destroy botulinum toxins.
- 3) *C. botulinum* may or may not cause container lids to bulge and the contents to have "off-odors." Other contaminants can also cause cans or bottle lids to bulge. Bulging containers should not be opened, and foods with off-odors should not be eaten or "taste tested." Commercial cans with bulging lids should be returned unopened to the vendor.
- 4) Although *C. botulinum* spores are ubiquitous, identified sources such as honey, should not be fed to infants.

B. Control of patient contacts and the immediate environment:

- 1) Report to local health authority: Case report of suspected and confirmed cases obligatory in most states and countries, Class 2A (see Communicable Disease Reporting); immediate telephone report indicated.
- 2) Isolation: Not required, but handwashing is indicated after handling soiled diapers.
- 3) Concurrent disinfection: The implicated food(s) should be detoxified by boiling before discarding, or the containers broken and buried deeply in soil to prevent ingestion by animals. Contaminated utensils should be sterilized by boiling or by chlorine disinfection to activate any remaining toxin.
Usual sanitary disposal of feces from infant cases. Terminal cleaning.
- 4) Quarantine: None.
- 5) Management of contacts: None for simple direct contacts. Those who are known to have eaten the incriminated food should be purged with cathartics, given gastric lavage and high enemas and kept under close medical observation. The decision to provide presumptive treatment with polyvalent (equine AB or ABE) antitoxin to asymptomatic exposed individuals should be weighed carefully: balance the potential protection when antitoxin is administered early (within 1-2 days after eating the implicated meal) against the risk of adverse reactions and sensitization to horse serum.
- 6) Investigation of contacts and source of toxin: Study recent food history of those ill, and recover all suspected foods for appropriate testing and disposal. Search for other cases of botulism to rule out foodborne botulism.

- 7) Specific treatment: Intravenous administration as soon as possible of 1 vial of polyvalent (AB or ABE) botulinum antitoxin, available from CDC, Atlanta, through state health departments is considered a part of routine treatment (the emergency telephone number at CDC for botulism calls during regular office hours is 404-639-2206; and after hours and on weekends is 404-639-2888). Serum should be collected to identify the specific toxin before antitoxin is administered, but antitoxin should not be withheld pending test results. Most important is immediate access to an intensive care unit so that respiratory failure, the usual cause of death, can be anticipated and managed promptly. For wound botulism, in addition to antitoxin, the wound should be debrided and/or drainage established, and appropriate antibiotics (e.g., penicillin) administered.

In intestinal botulism, meticulous supportive care is essential. Equine botulinum antitoxin is not used because of the hazard of sensitization and anaphylaxis. An investigational human derived botulinum immune globulin (BIG) is currently available for the treatment only of infant botulism patients under an FDA approved open-label Treatment Investigational New Drug protocol from the California Department of Health Services. Information on BIG for the "empiric" treatment of suspected intestinal botulism in infants can be obtained from the California Department of Health Services at 510-540-2646 (24-hour line). Antibiotics do not improve the course of the disease, and aminoglycoside antibiotics in particular may worsen it by causing a synergistic neuromuscular blockade. Thus, antibiotics should be used only to treat secondary infections. Assisted respiration may be required.

- C. Epidemic measures:** Suspicion of a single case of botulism should immediately raise the question of a group outbreak involving a family or others who have shared a common food. Home preserved foods should be the prime suspect until ruled out, although restaurant foods or widely distributed commercially preserved foods are occasionally identified as the source of intoxication and pose a far greater threat to the public health.

In addition, because recent outbreaks have implicated unusual food items, even theoretically unlikely foods should be considered. When any food is implicated by epidemiologic or laboratory findings, immediate recall of the product is necessary, as is immediate search for people who shared the suspected food and for any remaining food from the same source. Any remaining food may be similarly contaminated; such food, if found, should be submitted for laboratory examination. Sera, gastric aspirates and stool from patients and (when indicated) from others exposed but not ill should be collected and forwarded immediately to a reference laboratory before administration of antitoxin

- D. Disaster implications:** None.

- E. International measures:** Commercial products may have been distributed widely; international efforts may be required to recover and test implicated foods. International common source outbreaks have occurred.

- F. Bioterrorism measures:** Botulinum toxins can be easily used by terrorists. Although the greatest threat may be via aerosol use, the more common threat may be via its use in food and drink. The occurrence of even a single case of botulism, especially if there is no obvious source of an improperly preserved food should raise the possibility of deliberate use of botulinum toxin. All such cases should be reported immediately so that appropriate investigations can be initiated without delay.

DIARRHEA, ACUTE ICD-9 001–009; ICD-10 A00–A09

Diarrhea is often accompanied by other clinical signs and symptoms including vomiting, fever, dehydration and electrolyte disturbances. It is a symptom of infection by many different bacterial, viral and parasitic enteric agents. The specific diarrheal diseases—cholera, shigellosis, salmonellosis, *Escherichia coli* infections, yersiniosis, giardiasis, *Campylobacter* enteritis, cryptosporidiosis and viral gastroenteropathy—are each described in detail under individual listings elsewhere in this book. Diarrhea can also occur in association with other infectious diseases such as malaria and measles, as well as chemical agents. Change in the enteric flora induced by antibiotics may produce acute diarrhea by overgrowth and toxin production by *Clostridium difficile*.

Approximately 70%-80% of the vast number of sporadic diarrheal episodes in people visiting treatment facilities in less developed countries could be diagnosed etiologically if the complete battery of newer laboratory tests were available and utilized. In the USA, where 5 million cases per year are estimated to occur and approximately 4 million are seen by a health care provider, the comparable figure is about 45% of cases. In the USA, the majority of diarrheal illness is caused primarily by viral agents, and the most common cause of gastroenteritis is rotavirus. A smaller proportion of diarrheal disease in the USA is attributed to bacterial pathogens such as *E. coli*, *Salmonella* and *Shigella* species, *Vibrio* species, and *Cl. difficile*.

From a practical clinical standpoint, diarrheal illnesses can be divided into six clinical presentations:

- 1) simple diarrhea, managed by oral rehydration with solutions containing water, glucose and electrolytes, with its specific etiology not important in management;
- 2) bloody diarrhea (dysentery), caused by organisms such as *Shigella*, *E. coli* O157:H7 and certain other organisms;
- 3) persistent diarrhea that lasts at least 14 days;
- 4) severe purging as seen in cholera;
- 5) minimal diarrhea, associated with vomiting, typical of some viral gastroenteritides; and illness from the toxins, such as those of *Staphylococcus aureus*, *Bacillus cereus* or *Cl. perfringens*; and
- 6) hemorrhagic colitis, with watery diarrhea containing gross blood but without fever or fecal leukocytes.

The details pertaining to the individual diseases are presented in separate chapters.

DIARRHEA CAUSED BY *ESCHERICHIA COLI*

ICD-9 008.0;
ICD-10 A04.0-A04.4

Strains of *Escherichia coli* that cause diarrhea are of six major categories: 1) enterohemorrhagic; 2) enterotoxigenic; 3) enteroinvasive; 4) enteropathogenic; 5) enteroaggregative; and 6) diffuse-adherent. Each category has a different pathogenesis, possesses distinct virulence properties, and comprises a separate set of O:H serotypes. Differing clinical syndromes and epidemiologic patterns may also be seen.

I. DIARRHEA CAUSED BY ENTEROHEMORRHAGIC STRAINS

ICD-9 008.0; ICD-10 A04.3

(EHEC, Shiga toxin producing *E. coli* [STEC], *E. coli* O157:H7, Verotoxin producing *E. coli*) [VTEC]

1. Identification--This category of diarrheogenic *E. Coli* was recognized in 1982 when an outbreak of hemorrhagic colitis occurred in the USA and was shown to be due to an unusual serotype, *E. coli* O157:H7, that had not previously been incriminated as an enteric pathogen. The diarrhea may range from mild and nonbloody to stools that are virtually all blood but contain no fecal leukocytes. The most feared clinical manifestations of EHEC infection are the hemolytic uremic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP). Approximately 2-7% of subjects who manifest EHEC diarrhea progress to develop HUS. EHEC elaborate potent cytotoxins called Shiga toxins 1 and 2. Shiga toxin 1 is identical to Shiga toxin elaborated by *Shigella dysenteriae* 1; notably, HUS is also a well recognized severe complication of *S. dysenteriae* 1 disease. Previously, these toxins were called verotoxins 1 and 2 or Shiga-like toxins I and II. Elaboration of these toxins depends on the presence of certain phages carried by the bacteria. In addition, EHEC strains harbor a virulence plasmid that is involved in attachment of the bacteria to intestinal mucosa. Most EHEC strains have within their chromosome a pathogenicity island that contains multiple virulence genes encoding proteins that cause attaching and effacing lesions of the human intestinal mucosa.

In North America most strains of the most common EHEC serotype, O157:H7, can be identified in stool cultures by their inability to ferment sorbitol in media such as MacConkey-sorbitol (used to screen for *E. coli* O157:H7). Since it is now recognized that some EHEC strains ferment sorbitol, other techniques to detect EHEC must be employed. These include demonstrating the ability to elaborate Shiga toxins; serotyping to identify characteristic serotypes; or the use of DNA probes that identify the toxin genes, the presence of the EHEC virulence plasmid or specific sequences within the pathogenicity island. Lack of fever in most patients can help to differentiate this from shigellosis and dysentery caused by enteroinvasive strains of *E. coli* or by *Campylobacter*.

2. Infectious agent--While the main EHEC serotype in North America is *E. coli* O157:H7, other serotypes such as O26:H11, O111:H8, O103:H2, O113:H21, and O104:H21 have been implicated.

3. Occurrence--These infections are now recognized to be an important problem in North America, Europe, South Africa, Japan, the southern cone of South America and Australia. Their relative importance in the rest of the world is less well established. Serious outbreaks, including cases of hemorrhagic colitis, HUS, and some deaths, have occurred in the USA from inadequately cooked hamburgers, unpasteurized milk, apple cider (made from apples that were probably contaminated by cow manure) and alfalfa sprouts.

4. Reservoir--Cattle are the most important reservoir of EHEC; humans may also serve as a reservoir for person to person transmission. There is increasing evidence that in North America deer may also serve as a reservoir.

5. Mode of transmission--Transmission occurs mainly by ingestion of contaminated food; as with *Salmonella*, it is most often due to inadequately cooked beef (especially ground beef) and also raw milk and fruit or vegetables contaminated with ruminant feces. As with *Shigella*, transmission also occurs directly from person to person, in families, child care centers and custodial institutions. Waterborne transmission has also been documented; one outbreak was associated with swimming in a crowded lake and one was caused by drinking contaminated unchlorinated municipal water.

6. Incubation period--Typically relatively long, ranging from 2 to 8 days, with a median of 3-4 days.

7. Period of communicability--The duration of excretion of the pathogen, which is typically for a week or less in adults but 3 weeks in one third of children. Prolonged carriage is uncommon.

8. Susceptibility and resistance--The infectious dose is very low. Little is known about differences in susceptibility and immunity. Old age appears to be a risk factor, so hypochlorhydria may be a factor contributing to susceptibility. Children less than 5 years of age are at greatest risk of developing HUS.

9. Methods of control--

A. Preventive measures: The potential severity of this disease calls for early involvement of the local health authorities to identify the source and apply appropriate specific preventive measures. As soon as the diagnosis is suspected, it is of paramount importance to block person to person transmission by instructing family members in the necessity for frequent (and especially postdefecatory) handwashing with soap and water, disposal of soiled diapers and human waste, and prevention of food and beverage contamination. Measures likely to reduce the incidence of illness include the following:

- 1) Manage slaughterhouse operations to minimize contamination of meat by animal intestinal contents.
- 2) Pasteurize milk and dairy products.
- 3) Irradiate beef, especially ground beef.
- 4) Heat beef adequately during cooking, especially ground beef. The USDA Food Safety Inspection Service and the 1997 FDA Food Code recommend cooking ground beef to an internal temperature of 155°F (68°C) for at least 15-16 seconds. Reliance on cooking until all pink color is gone is not as reliable as using a meat thermometer.
- 5) Protect, purify and chlorinate public water supplies; chlorinate swimming pools.
- 6) Ensure adequate hygiene in childcare centers, especially frequent handwashing with soap and water.

B. Control of patient, contacts and the immediate environment:

- 1) Report to local health authority: Case report of *E. coli* O157:H7 infection is obligatory in many states (USA) and countries, Class 2B (see Communicable Disease Reporting). Recognition and reporting of outbreaks is especially important.
- 2) Isolation: During acute illness, enteric precautions. Because of the extremely small infective dose, infected patients should not be employed to handle food or to provide child or patient care until 2 successive

negative fecal samples or rectal swabs are obtained (collected 24 hours apart and not sooner than 48 hours after the last dose of antimicrobials).

- 3) Concurrent disinfection: Of feces and contaminated articles. In communities with a modern and adequate sewage disposal system, feces can be discharged directly into sewers without preliminary disinfection. Terminal cleaning.
- 4) Quarantine: None.
- 5) Management of contacts: When feasible, contacts with diarrhea should be excluded from food handling and the care of children or patients until the diarrhea ceases and 2 successive negative stool cultures are obtained. All contacts should be carefully indoctrinated in the need for thorough handwashing after defecation and before handling food or caring for children or patients.
- 6) Investigation of contacts and source of infection: Cultures of contacts should generally be confined to food handlers, attendants and children in child care centers and other situations where the spread of infection is particularly likely. Culture of suspected foods is relatively nonproductive in sporadic cases.
- 7) Specific treatment: Fluid and electrolyte replacement is important when diarrhea is watery or there are signs of dehydration (see Cholera, 9B7). The role of antibacterial treatment of infections with *E. coli* O157:H7 and other EHEC is uncertain. Some evidence suggests that treatment with TMP-SMX fluorquinolones and certain other antimicrobials may precipitate complications such as HUS.

C. Epidemic measures:

- 1) Report at once to the local health authority any group of acute bloody diarrhea cases, even in the absence of specific identification of the causal agent.
- 2) Search intensively for the specific vehicle (food or water) by which the infection was transmitted, evaluate potential for ongoing person to person transmission, and use the results of epidemiologic investigations to guide specific control measures.
- 3) Exclude use of and trace the source of suspected food; in large common-source foodborne outbreaks, prompt recall may prevent many cases.
- 4) If a waterborne outbreak is suspected, issue an order to boil water and chlorinate suspected water supplies adequately under competent supervision or do not use them.
- 5) If a swimming-associated outbreak is suspected, close pools or beaches until chlorinated or shown to be free of fecal contamination and until adequate toilet facilities are provided to prevent further contamination of water by bathers.
- 6) If a milkborne outbreak is suspected, pasteurize or boil the milk.
- 7) Prophylactic administration of antibiotics is not recommended.
- 8) Publicize the importance of handwashing after defecation; provide soap and individual paper towels if otherwise not available.

D. Disaster implications: A potential problem where personal hygiene and environmental sanitation are deficient (see Typhoid fever, 9D).

E. International measures: WHO Collaborating Centres.

II. DIARRHEA CAUSED BY ENTEROTOXIGENIC

STRAINS ICD-9 008.0; ICD-10 A04.1
(ETEC)

1. Identification--A major cause of travelers' diarrhea in people from industrialized countries who visit less developed countries, this bacterial disease is also an important cause of dehydrating diarrhea in infants and children in less developed countries. Enterotoxigenic strains may behave like *Vibrio cholerae* in producing a profuse watery diarrhea without blood or mucus. Abdominal cramping, vomiting, acidosis, prostration and dehydration can occur, and low grade fever may or may not be present; the symptoms usually last fewer than 5 days.

ETEC can be identified by demonstrating enterotoxin production, by immunoassays, bioassays or by DNA probe techniques that identify LT and ST genes (for heat labile and heat stable toxins) in colony blots.

2. Infectious agent--ETEC elaborate a heat labile enterotoxin (LT), a heat stable toxin (ST) or both toxins (LT/ST). The most common O serogroups include O6, O8, O15, O20, O25, O27, O63, O78, O80, O114, O115, O128ac, O148, O153, O159 and O167.

3. Occurrence--An infection primarily of developing countries. During the first 3 years of life, children in developing countries experience multiple ETEC infections which leads to the acquisition of immunity; consequently, illness in older children and adults occurs less frequently. Infection occurs among travelers from industrialized countries who visit less developed countries. Several outbreaks of ETEC infection have occurred recently in the USA.

4. Reservoir--Humans. ETEC infections are largely species specific; people constitute the reservoir for strains causing diarrhea in humans.

5. Mode of transmission--Contaminated food and, less often, contaminated water. Transmission via contaminated weaning foods may be particularly important in infection of infants. Direct contact transmission by fecally contaminated hands is believed to be rare.

6. Incubation period--Incubations as short as 10-12 hours have been observed in outbreaks and in volunteer studies with certain LT-only and ST-only strains. The incubation of LT/ST diarrhea in volunteer studies has usually been 24-72 hours.

7. Period of communicability--For the duration of excretion of the pathogenic ETEC, which may be prolonged.

8. Susceptibility and resistance--Epidemiologic studies and rechallenge studies in volunteers clearly demonstrate that serotype specific immunity is acquired following ETEC infection. Multiple infections with different serotypes are required to develop broad-spectrum immunity against ETEC.

9. Methods of control--

A. *Preventive measures:*

- 1) For general measures for prevention of fecal-oral spread of infection, see Typhoid fever, 9A.
- 2) For adult travelers going for short periods of time to high risk areas where it is not possible to obtain safe food or water, the use of prophylactic antibiotics may be considered; norfloxacin, 400 mg daily, has been shown to be effective. However, a much preferable approach is to initiate very early treatment, beginning with the onset of diarrhea (e.g., after the second or third loose stool). (See section 9B7, below.)

B. *Control of patient, contacts and the immediate environment:*

- 1) Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (see Communicable Disease Reporting).
- 2) Isolation: Enteric precautions for known and suspected cases.
- 3) Concurrent disinfection: Of all fecal discharges and soiled articles. In communities with a modern and adequate sewage disposal system, feces can be discharged directly into sewers without preliminary disinfection. Thorough terminal cleaning.
- 4) Quarantine: None.
- 5) Immunization of contacts: None.
- 6) Investigation of contacts and source of infection: Not indicated.
- 7) Specific treatment: Electrolyte-fluid therapy to prevent or treat dehydration is the most important measure (see Cholera, section 9B7). Most cases do not require any other therapy. For severe travelers' diarrhea in adults, early treatment with loperamide (Imodium®) (not for children) and an antibiotic such as a fluoroquinolone (ciprofloxacin PO 500 mg twice daily) or norfloxacin (PO 400 mg daily) for 5 days. Fluoroquinolones are used as initial therapy because many ETEC strains worldwide are resistant to a variety of other antimicrobials. However, if local strains are known to be sensitive, TMP-SMX (PO) (160 mg-800 mg) twice daily or doxycycline (PO) (100 mg) once daily, for 5 days are useful. Feeding should be continued, according to the patient's appetite.

C. *Epidemic measures:* Epidemiologic investigation may be indicated to determine how transmission is occurring.

D. *Disaster implications:* None.

E. *International measures:* WHO Collaborating Centres.

III. DIARRHEA CAUSED BY ENTEROINVASIVE STRAINS (EIEC)

ICD-9 008.0; ICD-10 A04.2

1. Identification--This inflammatory disease of the gut mucosa and submucosa caused by EIEC strains of *E. coli* closely resembles that produced by *Shigella*. The organisms possess the same plasmid dependent ability to invade and multiply within epithelial cells. However, clinically, the syndrome of watery diarrhea due to EIEC is much more

common than dysentery. The O antigens of EIEC may cross-react with *Shigella* O antigens. Illness begins with severe abdominal cramps, malaise, watery stools, tenesmus and fever; in less than 10% of patients, it progresses to the passage of multiple, scanty, fluid stools containing blood and mucus.

EIEC may be suspected by the presence of many fecal leukocytes visible in a stained smear of mucus, a finding also in shigellosis. Tests available in reference laboratories include an immunoassay that detects the plasmid encoded specific outer membrane proteins that are associated with epithelial cell invasiveness; a bioassay (the guinea pig-keratoconjunctivitis test) detects epithelial cell invasiveness; DNA probes detect the enteroinvasiveness plasmid.

2. Infectious agent--Strains of *E. coli* shown to possess enteroinvasiveness dependent on the presence of a large virulence plasmid encoding invasion plasmid antigens. The main O serogroups in which EIEC fail include O28ac, O29, O112, O124, O136, O143, O144, O152, O164 and O167.

3. Occurrence--EIEC infections are endemic in less developed countries, and cause about 1%-5% of diarrheal episodes among people visiting treatment centers. Occasional infections and outbreaks of EIEC diarrhea have been reported in industrialized countries.

4. Reservoir--Humans.

5. Mode of transmission--Scant available evidence suggests that EIEC is transmitted by contaminated food.

6. Incubation period--Incubations as short as 10 and 18 hours have been observed in volunteer studies and outbreaks, respectively.

7. Period of communicability--Duration of excretion of EIEC strains.

8. Susceptibility and resistance--Little is known about susceptibility and immunity to EIEC.

9. Methods of control--Same as for ETEC, above. For the rare cases of severe diarrhea with enteroinvasive strains, as for shigellosis, treat using antimicrobials effective against local *Shigella* isolates.

IV. DIARRHEA CAUSED BY ENTEROPATHOGENIC STRAINS ICD-9 008.0; ICD-10 A04.0 (EPEC, Enteropathogenic E coli enteritis)

1. Identification--This is the oldest recognized category of diarrhea producing *E. coli*, implicated in 1940s and 1950s studies in which certain O:H serotypes were found to be associated with infant summer diarrhea, outbreaks of diarrhea in infant nurseries, and community epidemics of infant diarrhea. Diarrheal disease in this category is virtually confined to infants less than 1 year of age in whom it causes watery diarrhea with mucus, fever and dehydration. EPEC cause dissolution of the microvilli of enterocytes and initiate attachment of the bacteria to enterocytes. The diarrhea in infants can be both severe and prolonged, and in developing countries may be associated with high case fatality.

EPEC can be tentatively identified by agglutination with antisera that detect EPEC O serogroups, but confirmation requires both O and H typing with high quality reagents. EPEC organisms exhibit localized adherence to HEp-2 cells in cell cultures, a property that requires the presence of an EPEC virulence plasmid. The EPEC adherence factor (EAF) DNA probe detects the EPEC virulence plasmid; there is a 98% correlation between the detection of localized

adherence and EAF probe positivity.

2. Infectious agent--The major EPEC O serogroups include O55, O86, O111, O119, O125, O126, O127, O128ab and O142.

3. Occurrence--Since the late 1960s, EPEC has largely disappeared as an important cause of infant diarrhea in North America and Europe. However, it remains a major agent of infant diarrhea in many developing areas, including South America, southern Africa and Asia.

4. Reservoir--Humans.

5. Mode of transmission--By contaminated infant formula and weaning foods. In infant nurseries, transmission by fomites and by contaminated hands can occur if handwashing techniques are compromised.

6. Incubation period--As short as 9-12 hours in adult volunteer studies. It is not known whether the same incubation applies to infants who acquire infection by natural transmission.

7. Period of communicability--Limited to the duration of excretion of EPEC, which may be prolonged.

8. Susceptibility and resistance--Although susceptibility to clinical infection appears to be confined virtually to young infants in nature, it is not known if this is due to immunity or to age related, nonspecific host factors. Since diarrhea can be induced experimentally in some adult volunteers, specific immunity may be important in determining susceptibility. EPEC infection is uncommon in breast fed infants.

9. Methods of control--

A. Preventive measures:

- 1) Encourage mothers to breast feed their infants exclusively from birth to 4-6 months of age. Provide adequate support for breast feeding. Help the mother to establish or reestablish breast feeding.
Where available, give newborns pasteurized donor breast milk until they go home if a mother's own breast milk is not available or sufficient. Infant formulas should be held at room temperature only for short periods. Cup feeding is preferred to bottle feeding as early as possible.
- 2) Practice rooming in for mothers and infants in maternity facilities, unless there is a firm medical indication for separating them. If mother or infant has a GI or respiratory infection, keep the pair together but isolate them from healthy pairs.
In special care facilities, separate infected infants from those who are premature or ill in other ways.
- 3) Provide individual equipment for each infant, include a thermometer, kept at the bassinet. No common bathing or dressing tables should be used, and no bassinet stands should be used for holding or transporting more than one infant at a time.
- 4) Prevention of hospital outbreaks depends on handwashing between handling babies and maintaining high sanitary standards in the facilities in which babies are held.

B. Control of patient, contacts and the immediate environment:

- 1) Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (see Communicable Disease Reporting). Two or more concurrent cases of diarrhea requiring treatment for these symptoms in a nursery or among those recently discharged are to be interpreted as an outbreak requiring investigation.
- 2) Isolation: Enteric precautions for known and suspected cases.
- 3) Concurrent disinfection: Of all fecal discharges and soiled articles. In communities with a modern and adequate sewage disposal system, feces can be discharged directly into sewers without preliminary disinfection. Thorough terminal cleaning.
- 4) Quarantine: Use enteric precautions and cohort methods (see 9C, below).
- 5) Immunization of contacts: None.
- 6) Investigation of contacts and source of infection: Families of discharged babies should be contacted for diarrheal status of the baby (see 9C, below).
- 7) Specific treatment: Electrolyte-fluid therapy (oral or IV) is the most important measure (see Cholera, 9B7). Most cases do not require any other therapy. For severe enteropathogenic infant diarrhea, oral TMP-SMX (10-50 mg/kg/day) has been shown to ameliorate the severity and duration of diarrheal illness; it should be administered in 3-4 divided doses for 5 days. However, since many EPEC strains are resistant to a variety of antibiotics, selection should be based on the sensitivity of local isolated strains. Feeding, including breast feeding, should be continued.

C. Epidemic measures: For nursery epidemics (see section 9B1, above) the following:

- 1) All babies with diarrhea should be placed in one nursery under enteric precautions. Admit no more babies to the contaminated nursery. Suspend maternity service unless a clean nursery is available with separate personnel and facilities; promptly discharge infected infants when medically possible. For the babies exposed in the contaminated nursery, provide separate medical and nursing personnel skilled in the care of infants with communicable diseases. Observe contacts for at least 2 weeks after the last case leaves the nursery; promptly remove each new case to one nursery ward used for these infants. Maternity service may be resumed after discharge of all contact babies and mothers, and thorough cleaning and terminal disinfection. Put into practice the recommendations of 9A, above, so far as feasible, in the emergency.
- 2) Carry out a thorough epidemiologic investigation into the distribution of cases by time, place, person and exposure to risk factors to determine how transmission is occurring.

D. Disaster implications: None.

E. International measures: WHO Collaborating Centres.

V. DIARRHEA CAUSED BY ENTEROAGGREGATIVE

E. COLI ICD-9 008.0; ICD-10 A04.4
(EaggEC)

This category of diarrhea producing *E. coli* is an important cause of infant diarrhea in less developed countries where it is the single most common cause of persistent diarrhea in infants. In animal models, these *E. coli* organisms evoke a characteristic histopathology in which EAggEC adhere to enterocytes in thick biofilm of aggregating bacteria and mucus. At present, the most widely available method to identify EAggEC is by the

HEp-2 assay, wherein these strains produce a characteristic "stacked brick" aggregative pattern as they attach to one another and to the HEp-2 cells; this is a plasmid dependent characteristic that is mediated by novel fimbriae. Most EAaggEC encode one or more cytotoxin/enterotoxin that are believed to be responsible for the watery diarrhea with mucus seen in infants and children infected with this pathogen. A DNA probe has been described. The incubation period is estimated to be 20-48 hours.

1. Identification--This category of diarrhea producing *E. coli* was first associated with infant diarrhea in a study in Chile in the late 1980s. It was subsequently recognized in India as being particularly associated with persistent diarrhea (diarrhea that continues unabated for at least 14 days), an observation that has since been confirmed by reports from Brazil, Mexico and Bangladesh.

2. Infectious agent--EAaggEC harbor a virulence plasmid required for expression of the unique fimbriae that encode aggregative adherence and many strains express a cytotoxin/enterotoxin. Among the most common EAaggEC O serotypes are O3:H2 and O44:H18. Many EAaggEC strains initially appear as rough strains lacking O antigens.

3. Occurrence--Reports associating EAaggEC with infant diarrhea, and particularly persistent diarrhea, have come from multiple countries in Latin America and Asia and from the Democratic Republic of Congo (DRC, formerly Zaire) in Africa. Reports from Germany and the United Kingdom suggest that EAaggEC may be responsible for a small proportion of diarrheal disease in industrialized countries as well.

VI. DIARRHEA CAUSED BY DIFFUSE-ADHERENCE

E. COLI ICD-9 008.0; ICD-10 A04.4
(DAEC)

A sixth category of diarrhea producing *E. coli* now recognized is diffuse-adherence *E. coli* (DAEC). The name derives from the characteristic pattern of adherence of these bacteria to HEp-2 cells in tissue culture. DAEC is the least well-defined category of diarrhea causing *E. coli*. Nevertheless, data from several epidemiologic field studies of pediatric diarrhea in less developed countries have found DAEC to be significantly more common in children with diarrhea than in matched controls; other studies have failed to find such a difference. Notably, preliminary evidence suggests that DAEC may be more pathogenic in children of preschool age rather than in infants and toddlers. Two DAEC strains failed to cause diarrhea when fed to volunteers and no outbreaks due to this category have yet been recognized. At present little is known about the reservoir, modes of transmission, host risk factors or period of communicability of DAEC.

FOODBORNE INTOXICATIONS

(Food poisoning)

Foodborne diseases, including foodborne intoxications and foodborne infections, are terms applied to illnesses acquired by consumption of contaminated food; they are frequently and inaccurately referred to as food poisoning. These terms include illnesses caused by chemical contaminants such as heavy metals and many organic compounds; however, the more frequent causes of foodborne illnesses are: (1) toxins elaborated by bacterial growth in the food before consumption (*Clostridium botulinum*, *Staphylococcus aureus*, and *Bacillus cereus*; scombroid fish poisoning, associated not with a specific toxin but with elevated histamine levels) or in the intestines (*Clostridium perfringens*); (2) bacterial, viral, or parasitic infections (brucellosis, *Campylobacter enteritis*, diarrhea caused by *Escherichia coli*, hepatitis A, listeriosis, salmonellosis, shigellosis, toxoplasmosis, viral gastroenteritis, taeniasis, trichinosis, and vibrios); and (3) toxins produced by harmful algal species (ciguatera fish poisoning, paralytic shellfish poisoning, neurotoxic shellfish poisoning, diarrhetic shellfish poisoning, and amnesic shellfish poisoning) or present in specific fish species (puffer fish poisoning).

This chapter deals specifically with toxin related foodborne illnesses (with the exception of botulism). Foodborne illnesses associated with infection by specific agents are covered in specific chapters dealing with these agents.

Foodborne disease outbreaks are recognized by the occurrence of illness within a usually short but variable period of time (from a few hours to a few weeks) after a meal, among individuals who have consumed foods in common. Prompt and thorough laboratory evaluation of cases and implicated foods is essential. Single cases of foodborne disease are difficult to identify unless, as in botulism, there is a distinctive clinical syndrome. Foodborne disease may be one of the most common causes of acute illness; many cases and outbreaks are unrecognized and unreported.

Prevention and control of these diseases, regardless of the specific cause, are based on the same principles: avoidance of food contamination, destruction or denaturation of the contaminants, and prevention of further spread or multiplication of contaminants. Specific problems and appropriate modes of intervention may vary from one country to another and depend on environmental, economic, political, technologic and sociocultural factors. Ultimately, prevention depends on educating food handlers about proper practices in cooking and storage of food and personal hygiene. Toward this end, WHO has developed "Ten Golden Rules for Safe Food Preparation." These are as follows:

1. Choose foods processed for safety.
2. Cook food thoroughly.
3. Eat cooked foods immediately.
4. Store cooked foods carefully.
5. Reheat cooked foods thoroughly.
6. Avoid contact between raw food and cooked food.
7. Wash hands repeatedly.
8. Keep all kitchen surfaces meticulously clean.
9. Protect food from insects, rodents and other animals.
10. Use safe water.

I. STAPHYLOCOCCAL FOOD INTOXICATION

ICD-9 005.0; ICD-10 A05.0

1. Identification--An intoxication (not an infection) of abrupt and sometimes violent onset, with severe nausea, cramps, vomiting and prostration, often accompanied by diarrhea, and sometimes with subnormal temperature and lowered blood pressure. Deaths are rare; duration of illness is commonly not more than a day or two, but the intensity of symptoms may require hospitalization and may result in surgical exploration in sporadic cases. Diagnosis is

easier when a group of cases is seen with the characteristic acute, predominantly upper GI symptoms and the short interval between eating a common food item and the onset of symptoms.

Differential diagnosis includes other recognized forms of food poisoning as well as chemical poisons. In the outbreak setting, recovery of large numbers of staphylococci (10^5 organisms or more per gram of food) on routine culture media or detection of enterotoxin from an epidemiologically implicated food item confirms the diagnosis. Absence of staphylococci on culture of a heated food does not rule out the diagnosis; a Gram stain of the food may disclose the organisms that have been heat killed. It may be possible to identify enterotoxin or thermonuclease in the food in the absence of viable organisms. Isolation of organisms of the same phage type from stools or vomitus of two or more ill persons also confirms the diagnosis. Recovery of large numbers of enterotoxin producing staphylococci from stool or vomitus from a single person supports the diagnosis. Phage typing and enterotoxin tests may help epidemiologic investigations but are not routinely available or indicated.

2. Toxic agent--Several enterotoxins of *Staphylococcus aureus*, stable at boiling temperature. Staphylococci multiply in food and produce the toxins.

3. Occurrence--Widespread and relatively frequent; one of the principal acute food intoxications in the USA. About 25% of people are carriers of this pathogen.

4. Reservoir--Humans in most instances; occasionally cows with infected udders, as well as dogs and fowl.

5. Mode of transmission--By ingestion of a food product containing staphylococcal enterotoxin. Foods involved are particularly those that come in contact with food handlers' hands, either without subsequent cooking or with inadequate heating or refrigeration, such as pastries, custards, salad dressings, sandwiches, sliced meat and meat products. Toxin has also developed in inadequately cured ham and salami, and in nonprocessed or inadequately processed cheese. When these foods remain at room temperature for several hours before being eaten, toxin producing staphylococci multiply and elaborate the heat stable toxin.

The organisms may be of human origin from purulent discharges of an infected finger or eye, abscesses, acneiform facial eruptions, nasopharyngeal secretions, or apparently normal skin; or of bovine origin, such as contaminated milk or milk products, especially cheese.

6. Incubation period--Interval between eating food and onset of symptoms is 30 minutes to 8 hours, usually 2-4 hours.

7. Period of communicability--Not applicable.

8. Susceptibility and resistance--Most people are susceptible.

9. Methods of control--

A. Preventive measures:

- 1) Educate food handlers about: (a) strict food hygiene, sanitation and cleanliness of kitchens, proper temperature control, handwashing, cleaning of fingernails; and (b) the danger of working with exposed skin, nose or eye infections and uncovered wounds.

- 2) Reduce food handling time (initial preparation to service) to an absolute minimum, with no more than 4 hours at ambient temperature. Keep perishable foods hot (greater than 60°C/140°F) or cold (below 10°C /50°F; best is less than 4°C/39°F) in shallow containers and covered, if they are to be stored for more than 2 hours.
- 3) Temporarily exclude people with boils, abscesses and other purulent lesions of hands, face or nose from food handling.

B. Control of patient contacts and the immediate environment:

- 1) Report to local health authority: Obligatory report of outbreaks of suspected or confirmed cases, Class 4 (see Communicable Disease Reporting).
- 2), 3), 4), 5) and 6) Isolation, Concurrent disinfection, Quarantine, Immunization of contacts and Investigation of contacts and source of infection: Not pertinent. Control is of outbreaks; single cases are rarely identified.
- 7) Specific treatment: Fluid replacement when indicated.

C. Epidemic measures:

- 1) By quick review of reported cases, determine time and place of exposure and the population at risk; obtain a complete listing of the foods served and embargo, under refrigeration, all foods still available. The prominent clinical features, coupled with an estimate of the incubation period, provide useful leads to the most probable etiologic agent. Collect specimens of feces and vomitus for laboratory examination; alert the laboratory to suspected etiologic agents. Interview a random sample of those exposed. Compare the attack rates for specific food items eaten and not eaten; the implicated food item(s) will usually have the greatest difference in attack rates. Most of the sick will have eaten the contaminated food.
- 2) Inquire about the origin of the incriminated food and the manner of its preparation and storage before serving. Look for possible sources of contamination and periods of inadequate refrigeration and heating that would permit growth of staphylococci. Submit any leftover suspected foods promptly for laboratory examination; failure to isolate staphylococci does not exclude the presence of the heat resistant enterotoxin if the food had been heated.
- 3) Search for food handlers with skin infections, particularly of the hands. Culture all purulent lesions and collect nasal swabs from all foodhandlers. Antibiograms and/or phage typing of representative strains of enterotoxin producing staphylococci isolated from foods and food handlers and from vomitus or feces of patients may be helpful.

D. Disaster implications: A potential hazard in situations involving mass feeding and lack of refrigeration facilities. A particular problem of air travel.

E. International measures: WHO Collaborating Centres.

II. CLOSTRIDIUM PERFRINGENS

FOOD INTOXICATION ICD-9 005.2; ICD-10 A05.2

(*C. welchii* food poisoning, Enteritis necroticans, Pigbel)

1. Identification--An intestinal disorder characterized by sudden onset of colic followed by diarrhea; nausea is common, but vomiting and fever are usually absent. Generally a mild disease of short duration, 1 day or less, and rarely fatal in healthy people. Outbreaks of severe disease with high case-fatality rates associated with a necrotizing enteritis have been documented in postwar Germany and in Papua New Guinea.

In the outbreak setting, diagnosis is confirmed by demonstration of *Clostridium perfringens* in semiquantitative anaerobic cultures of food ($10^5/g$ or greater) or patients' stool ($10^6/g$ or greater) in addition to clinical and epidemiologic evidence. Detection of enterotoxin in the stool of ill persons also confirms the diagnosis. When serotyping can be performed, the same serotype is usually demonstrated in different specimens; serotyping is done routinely only in Japan and the UK.

2. Infectious agent--Type A strains of *C. perfringens* (*C. welchii*) cause typical food poisoning outbreaks (they also cause gas gangrene); type C strains cause necrotizing enteritis. Disease is produced by toxins elaborated by the organisms.

3. Occurrence--Widespread and relatively frequent in countries with cooking practices that favor multiplication of clostridia to high levels.

4. Reservoir--Soil; also the GI tract of healthy people and animals (cattle, pigs, poultry and fish).

5. Mode of transmission--Ingestion of food that was contaminated by soil or feces and then held under conditions that permit multiplication of the organism. Almost all outbreaks are associated with inadequately heated or reheated meats, usually stews, meat pies, and gravies made of beef, turkey or chicken. Spores survive normal cooking temperatures, germinate and multiply during slow cooling, storage at ambient temperature, and/or inadequate rewarming. Outbreaks are usually traced to food catering firms, restaurants, cafeterias and schools that have inadequate cooling and refrigeration facilities for large-scale service. Heavy bacterial contamination (more than 10^5 organisms per gram of food) is usually required for clinical disease.

6. Incubation period--From 6 to 24 hours, usually 10-12 hours.

7. Period of communicability--Not applicable.

8. Susceptibility and resistance--Most people are probably susceptible. In volunteer studies, no resistance was observed after repeated exposures.

9. Methods of control--

A. Preventive measures:

- 1) Educate food handlers about the risks inherent in large scale cooking, especially of meat dishes. Where possible, encourage serving hot dishes while still hot from initial cooking.
- 2) Serve meat dishes hot, as soon as they are cooked, or cool them rapidly in a properly designed chiller and refrigerate until serving time; reheating, if necessary, should be thorough (internal temperature of at least $70^\circ\text{C}/158^\circ\text{F}$, preferably $75^\circ\text{C}/167^\circ\text{F}$ or higher) and rapid. Do not partially cook meat and poultry one day and reheat the next, unless it can be stored at a safe temperature. Large cuts of meat should be thoroughly cooked; for more rapid cooling of cooked foods, divide stews and similar dishes

prepared in bulk into many shallow containers and place in a rapid chiller.

B., C. and D. Control of patient, contacts and the immediate environment; Epidemic measures and Disaster implications: See Staphylococcal food intoxication (section 1, 9B, 9C and 9D, above).

E. International measures: None.

III. *BACILLUS CEREUS* FOOD

INTOXICATION

ICD-9 005.8; ICD-10 A05.4

1. Identification--An intoxication characterized in some cases by sudden onset of nausea and vomiting, and in others by colic and diarrhea. Illness generally persists no longer than 24 hours and is rarely fatal.

In the outbreak setting, diagnosis is confirmed by performing quantitative cultures with selective media to estimate the number of organisms present in the suspected food (generally more than 10^5 organisms per gram of the incriminated food are required). Diagnosis is also confirmed by isolation of organisms from the stool of two or more ill persons and not from stools of controls. Enterotoxin testing is valuable but may not be widely available.

2. Toxic agent--*Bacillus cereus*, an aerobic spore former. Two enterotoxins have been identified, one (heat stable) causing vomiting, and one (heat labile) causing diarrhea.

3. Occurrence--A well recognized cause of foodborne disease in the world; rarely reported in the USA.

4. Reservoir--A ubiquitous organism in soil and the environment commonly found at low levels in raw, dried and processed foods.

5. Mode of transmission--Ingestion of food that has been kept at ambient temperatures after cooking, permitting multiplication of the organisms. Outbreaks associated with vomiting have been most commonly associated with cooked rice that had subsequently been held at ambient room temperatures before reheating. Various mishandled foods have been implicated in outbreaks associated with diarrhea.

6. Incubation period--From 1 to 6 hours in cases where vomiting is the predominant symptom; from 6 to 24 hours where diarrhea is predominant.

7. Period of communicability--Not communicable from person to person.

8. Susceptibility and resistance--Unknown.

9. Methods of control--

A. Preventive measures: Foods should not remain at ambient temperature after cooking, since the ubiquitous *B. cereus* spores can survive boiling, germinate, and multiply rapidly at room temperature. Refrigerate leftover food promptly; reheat thoroughly and rapidly to avoid multiplication of microorganisms.

B., C. and D. Control of patient, contacts and the immediate environment; Epidemic measures and Disaster implications: See Staphylococcal food intoxication (section I, 9B, 9C and 9D, above).

E. International measures: None.

IV. SCOMBROID FISH

POISONING ICD-9 988.0; ICD-10 T61.1
(Histamine poisoning)

A syndrome of tingling and burning sensations around the mouth, facial flushing and sweating, nausea and vomiting, headache, palpitations, dizziness and rash that occur within a few hours after eating fish containing high levels of free histamine (more than 20 mg/100 g of fish); this occurs when the fish has undergone bacterial decomposition after capture. Symptoms resolve spontaneously within 12 hours and there are no long-term sequelae.

Occurrence is worldwide; the syndrome was initially associated with fish in the families Scombroidea and Scomberesocidae (tuna, mackerel, skipjack and bonito) which contain high levels of histidine that can be decarboxylated to form histamine by bacteria in the fish. However, nonscombroid fish, such as mahi-mahi (dolphinfish), bluefish and salmon, are commonly associated with illness. Risks appear to be greatest for fish imported from tropical or semitropical areas and fish caught by recreational fishermen, who may lack appropriate storage facilities for large fish. The diagnosis is confirmed by detection of histamine in epidemiologically implicated fish.

Adequate refrigeration or irradiation of caught fish prevents this spoilage. Symptoms usually resolve spontaneously. In severe cases, antihistamines may be effective in relieving symptoms.

While most often associated with fish, any food (such as certain cheeses) that contains the appropriate amino acids and is subjected to certain bacterial contamination and growth may lead to scombroid poisoning when ingested.

V. CIGUATERA FISH

POISONING ICD-9 988.0; ICD-10 T61.0

A syndrome of characteristic GI and neurologic symptoms may occur within 1 hour after eating tropical reef fish. GI symptoms (diarrhea, vomiting, abdominal pain) occur first, usually within 24 hours of eating implicated fish. In severe cases, patients may also be hypotensive, with a paradoxical bradycardia. Neurologic symptoms may occur at the same time as the acute symptoms or may follow 1-2 days later; they include pain and weakness in the lower extremities (a very characteristic symptom in the Caribbean) and circumoral and peripheral paresthesias, and may persist for weeks or months.

More bizarre symptoms, such as temperature reversal (ice cream tastes hot, hot coffee seems cold) and "aching teeth," are frequently reported. In very severe cases (particularly in the South Pacific), the neurologic symptoms may progress to coma and respiratory arrest within the first 24 hours of illness. Most patients recover completely within a few weeks, but intermittent recrudescence of symptoms over a period of months to years can occur.

This syndrome is caused by the presence in the fish of toxins elaborated by the dinoflagellate *Gambierdiscus toxicus* and other algae that grow on reefs under the sea. Fish eating the algae become toxic, and the effect is magnified through the food chain so that large predatory fish become the most toxic; this occurs worldwide in tropical areas.

Ciguatera is a significant cause of morbidity in areas in which consumption of reef fish is common--the Caribbean, southern Florida, Hawaii, the South Pacific and Australia. The incidence in the South Pacific has been estimated to be

in the range of 500 cases/100,000 population/year, with rates 50 times as high reported for some island groups. In the U.S. Virgin Islands, an incidence rate of 730 cases/100,000 population/year has been reported. More than 400 fish species are said to have the potential for becoming toxic. Worldwide, 50,000 cases of ciguatera occur per year. The diagnosis is confirmed by demonstrating ciguatoxin in epidemiologically implicated fish.

The consumption of large predatory fish should be avoided, especially in the reef area. In areas where assays for toxic fish are available (Hawaii), the risk of toxicity can be reduced by screening all large, "high risk" fish before their consumption. The occurrence of toxic fish is sporadic and not all fish of a given species or from a given locale will be toxic.

Intravenous infusion of mannitol (1 g/kg of a 20% solution, infused over 45 minutes) may have a dramatic effect on acute symptoms of ciguatera fish poisoning, particularly in severe cases; it has the most pronounced effect on neurologic symptoms and may be lifesaving in severe cases that have progressed to coma.

VI. PARALYTIC SHELLFISH

POISONING ICD-9 988.0; ICD-10 T61.2
(PSP)

Classic PSP is a syndrome of characteristic symptoms (predominately neurologic) with onset within minutes to several hours after eating bivalve molluscs. Initial symptoms include paresthesias of the mouth and extremities, frequently accompanied by GI symptoms. Symptoms usually resolve within a few days. In severe cases, ataxia, dysphonia, dysphagia and total muscle paralysis with respiratory arrest and death occur. In a retrospective review of PSP outbreaks that occurred in Alaska between 1973 and 1992, 29 (25% of 117 ill persons) required an emergency flight to a hospital, four (3%) required intubation, and one died. Recovery is complete, symptoms usually resolve within hours to days after shellfish ingestion.

This syndrome is caused by the presence in shellfish of saxitoxins produced by *Alexandrium* species and other dinoflagellates. Concentration of these toxins occurs especially during massive algae blooms known as "red tides," but can also occur in the absence of a recognizable algal bloom. PSP is particularly common in shellfish harvested from colder waters above 30°N and below 30°S latitude, but may occur in tropical waters as well. In the USA, PSP is primarily a problem in the New England states, Alaska, California, and Washington. Blooms of the causative *Alexandrium* species occur several times each year, primarily from April through October. Shellfish become toxic and remain toxic for several weeks after the bloom subsides; there are also some shellfish species that remain toxic constantly. Most cases occur in individuals or small groups who gather shellfish for personal consumption. Diagnosis is confirmed by detection of toxin in epidemiologically implicated food. On an experimental basis, it has been possible to demonstrate saxitoxins in serum during acute illness and in urine after acute symptoms resolve.

PSP neurotoxins are heat stable. Surveillance of high risk harvest areas in the USA is routinely conducted by state health departments, by using a standard mouse bioassay; areas are closed to harvesting when toxin levels in shellfish exceed 80 µg/100 g. When toxin levels exceed this value, warnings should be posted in shellfish growing areas, beaches and in the media.

VII. NEUROTOXIC SHELLFISH

POISONING ICD-9 988.0; ICD-10 T61.2

Neurotoxic shellfish poisoning is associated with blooms of *Gymnodinium breve*, which produce brevetoxin. Red tides caused by *G. breve* have occurred along the Florida coast for centuries, with associated fish kills and mortality in seabirds and marine mammals. Symptoms after eating toxic shellfish include circumoral paresthesias and paresthesias of the extremities, dizziness and ataxia, muscle aches, and gastrointestinal symptoms. Symptoms tend to be mild, and

resolve quickly and completely. In outbreaks which occurred in 1987 in North Carolina, median duration of illness was 17 hours (range 1-72 hours). Respiratory and eye irritation have also been reported in association with *G. breve* blooms, apparently due to aerosolization of the toxin by wind and wave action.

VIII. DIARRHETIC SHELLFISH POISONING

ICD-9 988.0; ICD-10 T61.2

Illness results from eating mussels, scallops, or clams that have been feeding on *Dinophysis fortii* or *Dinophysis acuminata*. Symptoms include diarrhea, nausea, vomiting, and abdominal pain. Case reports came initially from Japan; however, diarrhetic shellfish poisoning has occurred in France and other parts of Europe, Canada, New Zealand, and South America. There have been no confirmed USA cases, although the causative organisms have been identified in USA coastal waters.

IX. AMNESIC SHELLFISH POISONING

ICD-9 988.0; ICD-10 T61.2

Amnesic shellfish poisoning results from ingestion of shellfish containing domoic acid, produced by the diatom *Pseudonitzschia pungens*. A series of cases due to this toxin were reported in the Atlantic provinces of Canada in 1987. Symptoms included vomiting, abdominal cramps, diarrhea, headache, and loss of short term memory. On neuropsychological testing several months after the acute intoxication, patients were found to have severe antegrade memory deficits with relative preservation of other cognitive functions; patients also had clinical and electromyographic evidence of pure motor or sensorimotor neuropathy and axonopathy. Neuropathological studies in four patients who died demonstrated neuronal necrosis and loss, predominantly in the hippocampus and amygdala. Canadian authorities now analyze mussels and clams for domoic acid, and close shellfish beds to harvesting when levels exceed 20 µg/g.

In 1991, domoic acid was also identified in razor clams and Dungeness crabs on the Oregon and Washington coast, and it has been found in the marine food web along the Texas coast. While no clear cut human cases of amnesic shellfish poisoning have been identified outside of the original Canadian outbreaks, the clinical significance of ingestion of low levels of domoic acid (as may be occurring in persons eating shellfish and anchovies harvested from these and other areas where *Pseudonitzschia* species are present) is unknown.

X. PUFFER FISH POISONING (TETRODOTOXIN)

ICD-9 988.0; ICD-10 T61.2

Puffer fish poisoning is characterized by onset of paresthesias, dizziness, GI symptoms and ataxia, which often progresses rapidly to paralysis and death within several hours after eating. The case-fatality rate approaches 60%. The causative toxin is tetrodotoxin, a heat stable, nonprotein neurotoxin concentrated in the skin and viscera of puffer fish, porcupine fish, ocean sunfish, and species of newts and salamanders. More than 6,000 cases have been documented, mostly in Japan. Toxicity can be avoided by not consuming any of the species of fish or amphibians that produce tetrodotoxin.

GASTROENTERITIS, ACUTE

VIRAL

ICD-9 008.6; ICD-10 A08

Viral gastroenteritis presents as an endemic or epidemic illness in infants, children and adults. Several viruses (rotaviruses, enteric adenoviruses, astroviruses and caliciviruses including Norwalk-like viruses) infect children in their first years of life and cause a diarrheal illness that may be severe enough to produce dehydration requiring hospitalization for rehydration. Viral agents such as Norwalk-like viruses are also common causes of epidemics of gastroenteritis among children and adults. The epidemiology, natural history and clinical expression of enteric viral infections are best understood for type A rotavirus, in infants and Norwalk agent in adults.

I. ROTAVIRAL

ENTERITIS

ICD-9 008.61; ICD-10 A08.0

(Sporadic viral gastroenteritis, Severe viral gastroenteritis of infants and children)

1. Identification--A sporadic, seasonal, often severe gastroenteritis of infants and young children, characterized by vomiting, fever and watery diarrhea. Rotaviral enteritis is occasionally associated with severe dehydration and death in young children. Secondary symptomatic cases among adult family contacts can occur, although subclinical infections are more common. Rotavirus infection has occasionally been found in pediatric patients with a variety of clinical manifestations, but the virus is probably coincidental rather than causative in these conditions. Rotavirus is a major cause of nosocomial diarrhea of newborns and infants. Although rotavirus diarrhea is generally more severe than acute diarrhea due to other agents, illness caused by rotavirus is not distinguishable from that caused by other enteric viruses for any individual patient.

Rotavirus can be identified in stool specimens or rectal swabs by EM, ELISA, LA and other immunologic techniques for which commercial kits are available. Evidence of rotavirus infection can be demonstrated by serologic techniques, but diagnosis is usually based on the demonstration of rotavirus antigen in stools. False-positive ELISA reactions are common in newborns; positive reactions require confirmation by an alternative test.

2. Infectious agent--The 70-nm rotavirus belongs to the Reoviridae family. Group A is common, group B is uncommon in infants but has caused large epidemics in adults in China, while group C appears to be uncommon in humans. Groups A, B, C, D, E and F occur in animals. There are 4 major, and at least 10 minor, serotypes of group A human rotavirus, based on antigenic differences in the viral protein 7 (VP7) outer capsid surface protein, the major neutralization antigen. Another outer capsid protein, designated VP4, is associated with virulence and also plays a role in virus neutralization.

3. Occurrence--In both developed and developing countries, rotavirus is associated with about one third of the hospitalized cases of diarrheal illness in infants and young children under 5 years of age. Neonatal rotaviral infections are frequent in certain settings but are usually asymptomatic. Essentially all children are infected by rotavirus in their first 2-3 years of life, with peak incidence of clinical disease in the 6- to 24-month age group. Outbreaks occur among children in day care settings. Rotavirus is more frequently associated with severe diarrhea than other enteric pathogens; in developing countries, it is responsible for an estimated 600,000-870,000 diarrheal deaths each year.

In temperate climates, rotavirus diarrhea occurs in seasonal peaks during cooler months; in tropical climates, cases occur throughout the year, often with a less pronounced peak in the cooler dry months. Infection of adults is usually subclinical, but outbreaks of clinical disease occur in geriatric units. Rotavirus occasionally causes travelers' diarrhea in adults and diarrhea in immunocompromised (including AIDS) patients, parents of children with rotavirus diarrhea and

the elderly.

4. Reservoir--Probably humans. The animal viruses do not produce disease in humans; group B and group C rotaviruses identified in humans appear to be quite distinct from those found in animals.

5. Mode of transmission--Probably fecal-oral with possible contact or respiratory spread. Although rotaviruses do not effectively multiply in the respiratory tract, they may be encountered in respiratory secretions. There is some evidence that rotavirus may be present in contaminated water.

6. Incubation period--Approximately 24-72 hours.

7. Period of communicability--During the acute stage of disease, and later while virus shedding continues. Rotavirus is not usually detectable after about the eighth day of infection, although excretion of virus for 30 days or more has been reported in immunocompromised patients. Symptoms last for an average of 4-6 days.

8. Susceptibility and resistance--Susceptibility is greatest between 6 and 24 months of age. By age 3 years, most individuals have acquired rotavirus antibody. Immunocompromised individuals are at particular risk for prolonged rotavirus antigen excretion and intermittent rotavirus diarrhea. Diarrhea is uncommon in infected infants less than 3 months of age.

9. Methods of control--

A. Preventive measures

- 1) In August 1998, an oral, live, tetravalent, rhesus-based rotavirus vaccine (RRV-TV) was licensed for use among infants in the USA. This vaccine should be administered to infants between the ages of 6 weeks and 1 year. The recommended schedule is a 3-dose series, with doses to be administered at ages 2, 4 and 6 months. The first dose may be administered at ages 6 weeks to 6 months; subsequent doses should be administered with a minimum interval of 3 weeks between any two doses. The first dose should not be administered to children aged greater than or equal to 7 months because of an increased rate of febrile reactions after the first dose among older infants. Second and third doses should be administered before the first birthday. Routine use of this vaccine should prevent most physician visits for rotavirus gastroenteritis and at least 2/3 of hospitalizations and deaths related to rotavirus.

Intussusception (a bowel obstruction in which one segment of bowel becomes enfolded within another segment) was identified in prelicensure trials as a potential problem associated with RRV-TV. Because of continued reports of intussusception, CDC in July 1999, pending further studies, recommended postponing administration of RRV-TV to children scheduled to receive the vaccine before November 1999; this recommendation includes those who had already begun the RRV-TV series. All cases of intussusception which occur following administration of RRV-TV should be reported to the Vaccine Adverse Events Reporting System (VAERS, 800-822-7967);

www.fda.gov/cber/vaers/report.htm). The most current vaccine recommendations will be posted on the CDC immunization website: (<http://www.cdc.gov/nip>) and also on the CCDM website: (<http://www.ccdm.org>).

- 2) The effectiveness of other preventive measures is undetermined. Hygienic measures applicable to

diseases transmitted via the fecal-oral route may not be effective in preventing transmission. The virus survives for long periods on hard surfaces, in contaminated water and on hands. It is relatively resistant to commonly used disinfectants but is inactivated by chlorine.

- 3) In day care, dressing infants with overalls to cover diapers has been demonstrated to decrease transmission of the infection.
- 4) Prevent exposure of infants and young children to individuals with acute gastroenteritis in family and institutional (day care or hospital) settings by a high level of sanitary practices; exclusion from day care centers is not necessary.
- 5) Passive immunization by oral administration of IG has been shown to protect low birthweight neonates and immunocompromised children. Breast feeding does not affect infection rates, but may reduce the severity of the gastroenteritis.

B. Control of patient contacts and the immediate environment:

- 1) Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (see Communicable Disease Reporting).
- 2) Isolation: Enteric precautions, with frequent handwashing by caretakers of infants.
- 3) Concurrent disinfection: Sanitary disposal of diapers; place overalls over diapers to prevent leakage.
- 4) Quarantine: None.
- 5) Immunization of contacts: None.
- 6) Investigation of contacts and source of infection: Sources of infection should be sought in certain high risk populations and cohorted antigen excretors.
- 7) Specific treatment: None. Oral rehydration therapy with oral glucose-electrolyte solution is adequate in most cases. Parenteral fluids are needed in cases with vascular collapse or uncontrolled vomiting (see Cholera, 9B7). Antibiotics and antimotility drugs are contraindicated.

C. Epidemic measures: Search for vehicles of transmission and source on epidemiologic bases.

D. Disaster implications: A potential problem with dislocated populations.

E. International measures: WHO Collaborating Centres.

II. EPIDEMIC VIRAL

GASTROENTEROPATHY ICD-9 008.6, 008.8;
ICD-10 A08.1

(Norwalk agent disease, Norwalk-like disease, Viral gastroenteritis in adults, Epidemic viral gastroenteritis, Acute infectious nonbacterial gastroenteritis, Viral diarrhea, Epidemic diarrhea and vomiting, Winter vomiting disease, Epidemic nausea and vomiting)

1. Identification--Usually a self-limited, mild to moderate disease that often occurs in outbreaks, with clinical symptoms of nausea, vomiting, diarrhea, abdominal pain, myalgia, headache, malaise, low grade fever or a combination of these symptoms. GI symptoms characteristically last 24-48 hours.

The virus may be identified in stool by direct or immune EM or, for the Norwalk virus, by RIA or by reverse transcription polymerase chain reaction (RT-PCR). Serologic evidence of infection may be demonstrated by IEM or, for the Norwalk virus, by RIA. Diagnosis requires collection of a large volume of stool, with aliquots stored at 4°C

(39°F) for EM and at -20°C (-4°F) for antigen assays. Acute and convalescent sera (3-4-week interval) are essential to link particles observed by EM with disease etiology. RT-PCR seems to be more sensitive than IEM and can be used to examine links among widely scattered clusters of disease.

2. Infectious agents--Norwalk-like viruses are small, 27- to 32-nm, structured RNA viruses classified as caliciviruses; it has been implicated as the most common etiologic agent of the nonbacterial gastroenteritis outbreaks. Several morphologically similar but antigenically distinct viruses have been associated with gastroenteritis outbreaks; these include Hawaii, Taunton, Ditchling or W, Cockle, Parramatta, Oklahoma and Snow Mountain agents.

3. Occurrence--Worldwide and common; most often in outbreaks but also sporadically; all age groups are affected. Outbreaks in the USA are usually associated with consumption of raw shellfish. In one study in the USA, antibodies to Norwalk agent were acquired slowly; by the fifth decade of life, more than 60% of the population had antibodies. In most developing countries studied, antibodies are acquired much earlier. Seroreponse to Norwalk virus was detected in infants and young children in Bangladesh and Finland.

4. Reservoir--Humans are the only known reservoir.

5. Mode of transmission--Probably by the fecal-oral route, although contact or airborne transmission from fomites has been suggested to explain the rapid spread in hospital settings. Several recent outbreaks have strongly suggested primary community foodborne, waterborne and shellfish transmission, with secondary transmission to family members.

6. Incubation period--Usually 24-48 hours; in volunteer studies with Norwalk agent, the range was 10-50 hours.

7. Period of communicability--During acute stage of disease and up to 48 hours after Norwalk diarrhea stops.

8. Susceptibility and resistance--Susceptibility is widespread. Short-term immunity lasting up to 14 weeks has been demonstrated in volunteers after induced Norwalk illness, but long-term immunity was variable; some individuals became ill on rechallenge 27-42 months later. Levels of preexisting serum antibody to Norwalk virus did not correlate with susceptibility or resistance.

9. Methods of control--

A. Preventive measures: Use hygienic measures applicable to diseases transmitted via fecal-oral route (see Typhoid fever, 9A). In particular, cooking shellfish and surveillance of shellfish breeding waters can prevent infection from that source.

B. Control of patient contacts and the immediate environment:

- 1) Report to local health authority: Obligatory report of epidemics; no individual case report, Class 4 (see Communicable Disease Reporting).
- 2) Isolation: Enteric precautions.
- 3) Concurrent disinfection: None.

- 4) Quarantine: None.
- 5) Immunization of contacts: None.
- 6) Investigation of contacts and source of infection: Search for means of spread of infection in outbreak situations.
- 7) Specific treatment: Fluid and electrolyte replacement in severe cases (see Cholera, 9B7).

C. Epidemic measures: Search for vehicles of transmission and source; determine course of outbreak to define the epidemiology.

D. Disaster implications: A potential problem.

E. International measures: None.

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