- Bacterial food poisoning (BFP)

- Bacterial food poisoning (BFP) – polyethiological disease, meaning hit in the body of microbial agents and / or their toxins, followed by a sudden onset, acute evolving signs of impregnation infectious sindrome and digestive organ damage.

- Acute diarrhea is defined as a condition characterized by excess of 3 liquid stool per day in increased quantities (more than 300 gr.per day) lasting less than 14 days. Acute diarrrhea can be divided into infectious and non-infectious origin. Not all acute diarrrhea meet the criteria of BFP.
- Acute diarrhea  is the 2nd cause of death among infectious diseases worldwide. Annually there is about 4 billion cases of acute diarrhea(Mendel 2010).

- ***BFP by pathogenic mechanism* can be classified into:**
- **1) Infectious type**, characterized most often as acute diarrhea, caused by penetration of bacteria without or with their toxins, where the pathogenic mechanism are:

- *A_ Not-inflammatory* (caused by enterotoxins) - caracterised by watery diarrhea without the presence of leukocytes in the stool. Can be produced *by vibrios* (*Vibrio cholerae, V. vulnificus, V. parahaemolyticus some strains*) and *Enterobacteriaceae (some strains of Salmonella, E. coli enteropathogenic and enterotoxigenic).*
- *B _ Inflammatory* (invasive mechanism by producing cytotoxine), caracterised by presence of PMN leukocytes in stool with mucus and blood. Can be produced by *enterobacteria (Shigella, some strains of Salmonella enteritidis, Escherichia coli enterohaemorrhagic and enteroinvaziv), clostridia (Clostridium difficile, C. jejuni, C.perfringens A), some strains of Vibrio parahaemolyticus, Campylobacter, Yersinia intestinalis.*

- *C _ Bacteriemia* is caracterised by penetration of microbe of the intestinal mucosa and then entering in the bloodstream, spreading to organs and tissues, with the clinical appearance of enteric fever and the presence of mononuclear in stool. This form is characteristic for infections with *S.typhi and S.paratyphi*.
- **2) Toxic type** caused by bacterial toxins, often do not meet the requirements of acute diarrrhea, pathogenetic mechanism is characterized by the production of:
- *a. enterotoxin - some strains of Staph. aureus, Escherichia coli enterotoxigenic (ETEC), Bacillus cereus.*
- *b. cytotoxin - some strains of Staph. aureus, Clostridium perfringens.*
- *c. neurotoxin - Clostridium botulinum*.

- It is now found that the same pathogen (such as Salmonella, Staphylococcus, Campylobacter, clostridia) in some cases cause invasive diarrhea-type intestinal inflammatory manifestations; in other cases - from infection **with other strains of the same bacteria** producing enterotoxin - secretive type causes diarrhea.

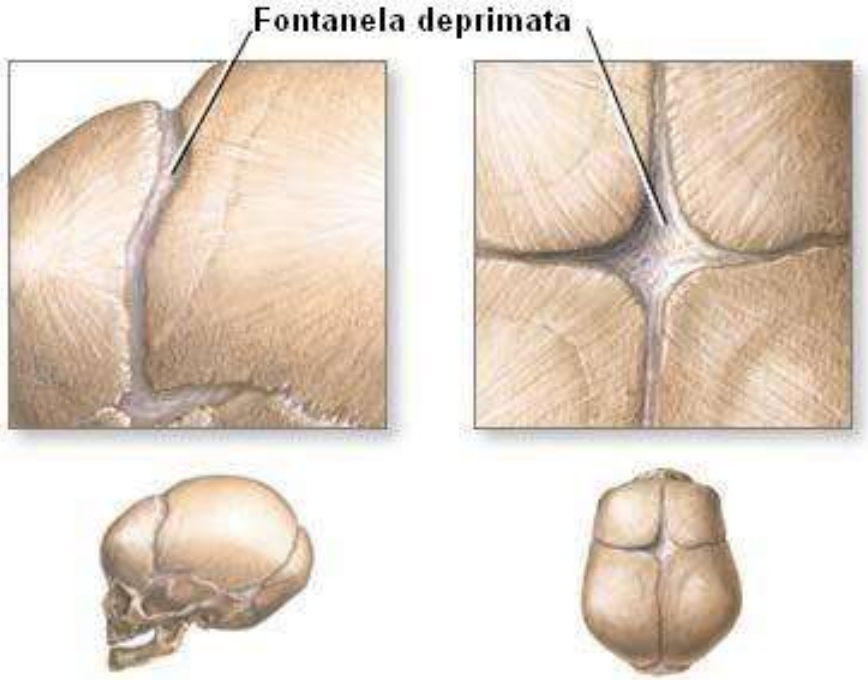
- For choosing of the treatment must by take into account osmotic component (fermentation), which can develop during illness.

## TABLE 93-2 Enteric Host Defenses

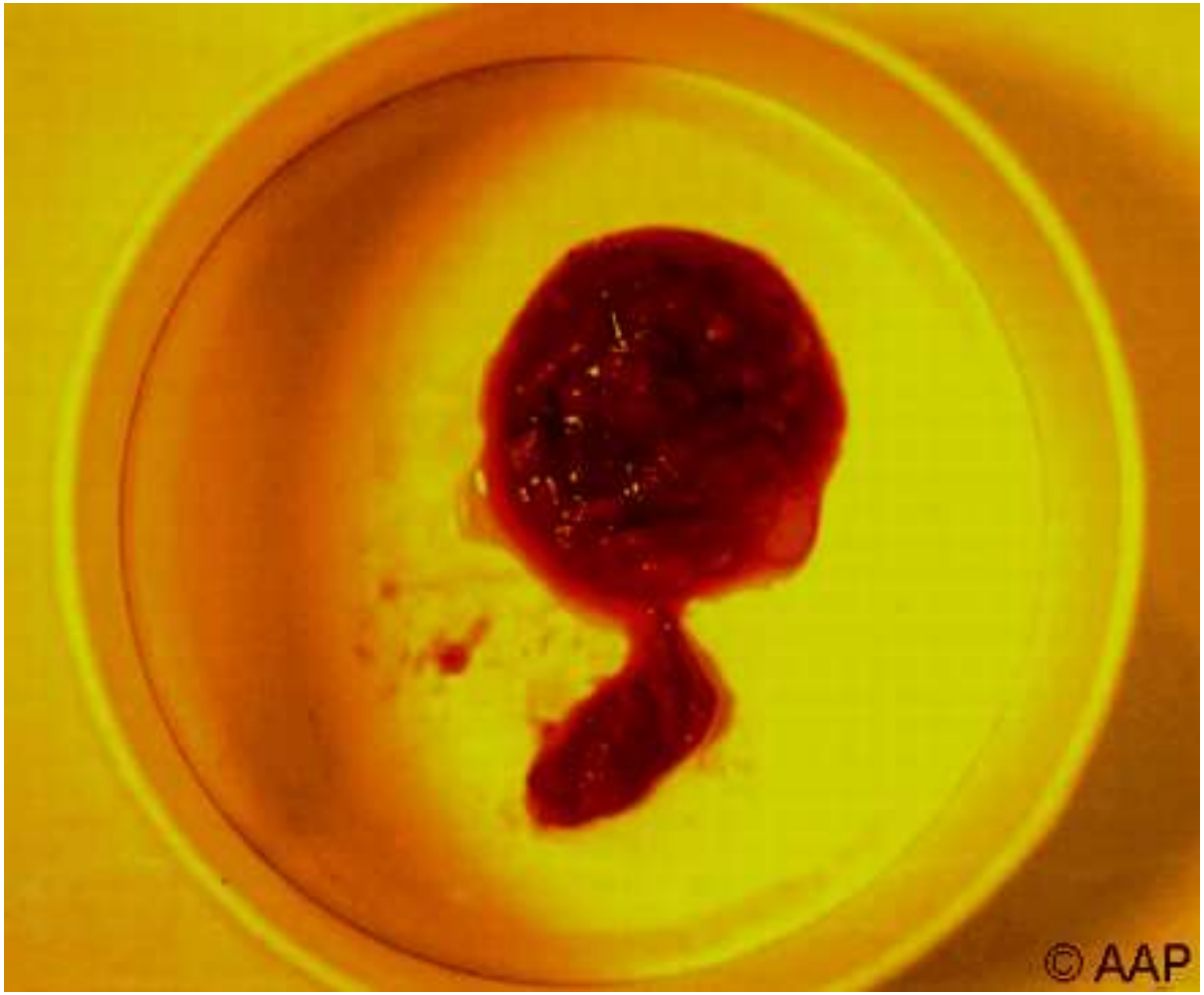
- Host species, genotype, and age factors
- Personal hygiene
- Gastric acidity and other physical barriers
- Intestinal motility
- Enteric microflora
- Immunity
  - Phagocytic
  - Humoral
  - Cell-mediated
- Nonspecific protective factors in human milk
- Intestinal receptors

- **Epidemiology.**
- Transmission mechanism of BFP and acute diarrhea is fecal-oral. Source of infection are the sick patients and healthy carriers of germs and animals - sick and carrier of germs.
- Foods most commonly implicated in food poisoning are eggs, milk, meat and their preparations. There are foods that contain abundant nutrients (mainly proteins) necessary for multiplication of germs.

- **Clinical**
- BFP is manifested by nausea, vomiting, abdominal pain and diarrhea. Dehydration can occur from 4 possible degree.

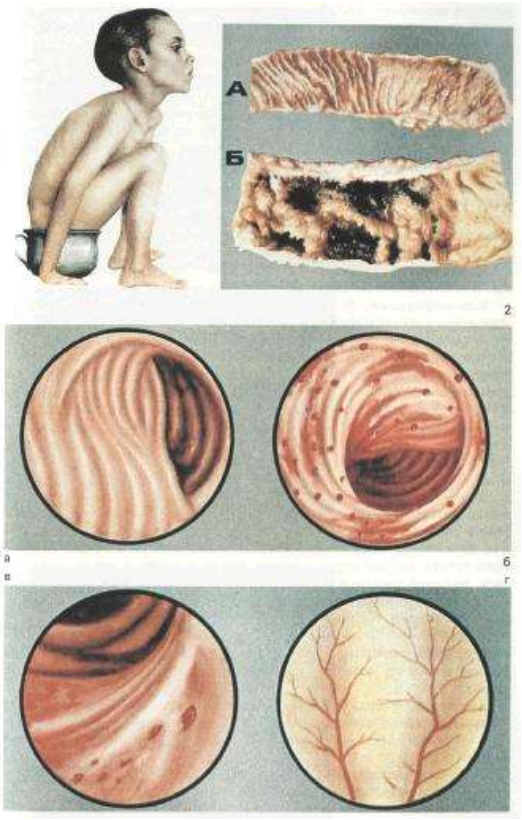



- Frequent liquid stools (10-25 times per day) - in small quantity, with a mixture of mucus and streaks of blood, purulent later.

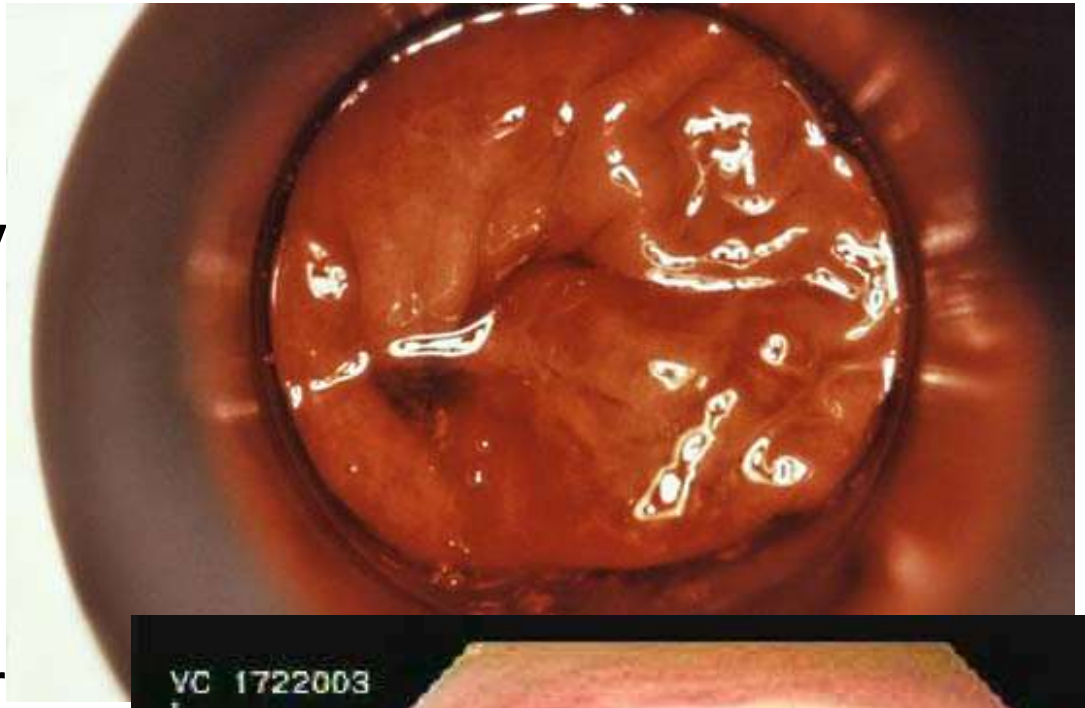


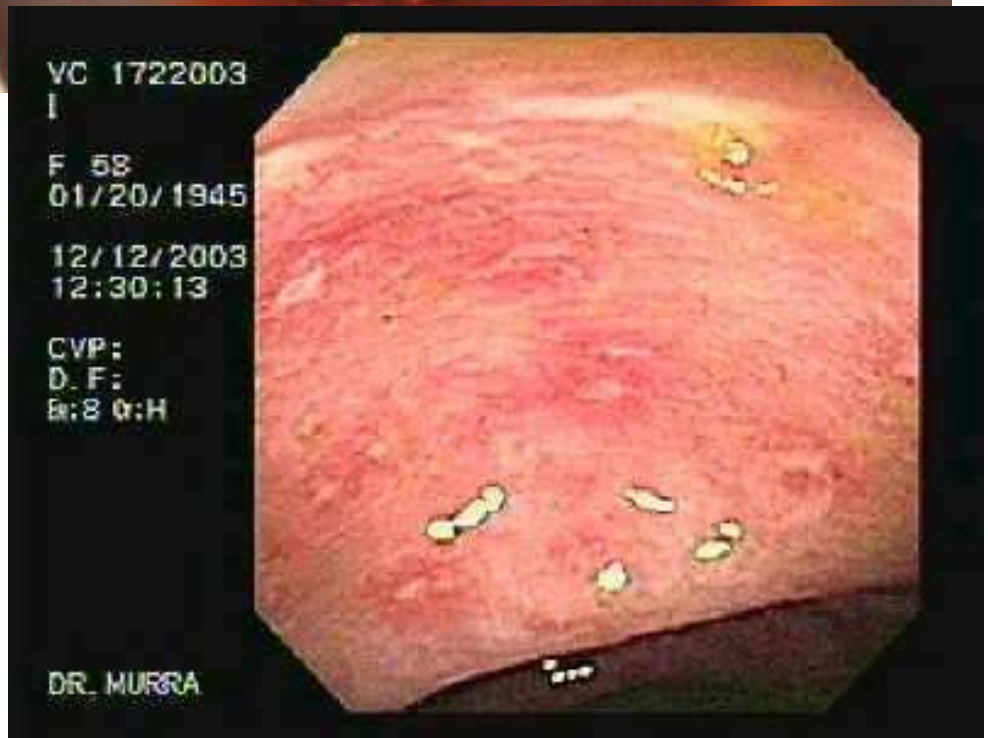
Morphological changes

Colonic mucosa lessions have extensive ulcerations of the surface of the epithelium, with exudate consisting of scaling colonic cells, polymorphonuclear leukocytes and erythrocytes, which may be found in microabscesses. In areas with  severe ulcerations, they appear as pseudomembranes. In chronic forms the reduction of the number of glands and collagen hyperplasia occurs.

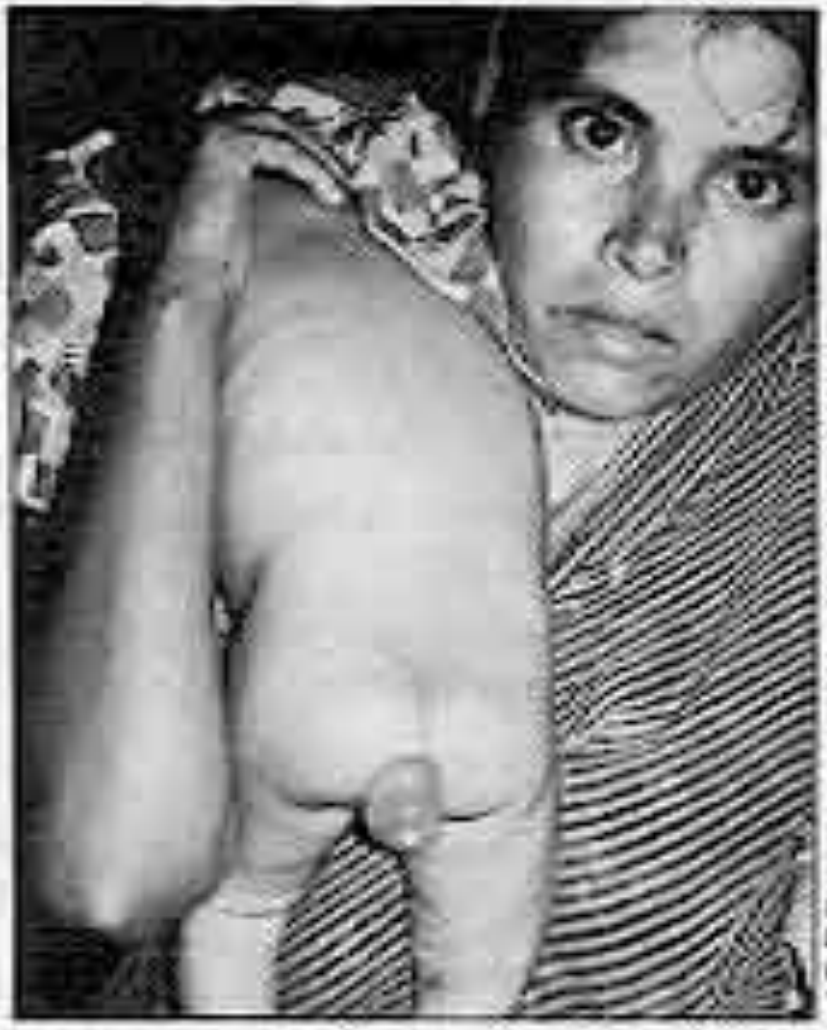


- Instrumental investigations - rectoromanoscopy : signs of inflammation of the distal colon (proctosigmoiditis in mild catarrhal or catarrhal-hemorrhagic form; erosive, ulcerative and fibrinous- in severe forms).





- Possible complications:
- In severe forms:
- - neurotoxicity and cerebral edema in children with toxic exicozis, toxico-infective shock, gastrointestinal bleeding, intestinal perforation, peritonitis, paraproctitis; colonic dysbacteriosis, urinary tract infections, toxic megacolon, hemolytic – uremic syndrome, rectal prolapse.



Inflammation and tissue damage causes painful straining to pass stools, which can lead to rectal prolapse.

- BFP, especially secretory type, can be complicated by hypovolemic shock. It is caused by the decrease of circulating blood volume, resulted in significant loss of fluid by vomiting and diarrhea.
- It is considered that the evolution of a patient in shock is in three phases defined more or less practical:
- A. shock onset (nonprogressive phase)
- B. progressive shock
- C. irreversible shock

- *Compensated shock*
- Can have a happy ending even without therapeutic intervention. Homeostasyc mechanisms do meet the requirements, and their reactions are not likely to cause further imbalances. It is accompanied by decreased blood pressure and decreased urine output.

- ***Progressive shock***
- Appears from tens of minutes or few hours after the initial disturbances, causing the shock, and is accompanied by:
- A. Heart functions' depression: caused by inadequate nutrition and oxygenation of the myocardium. Can be recognized clinically by heart rate reduction. If initially, with decreasing blood pressure heart rate greatly increases, when myocardial depression is installed, there is a crash which occurs to chnage both parameters. Is a bad sign, because the myocardium has great potential and great energy savings.
- B. Depression of sympathetic vasomotor centers of CNS: 10-20 minutes after the initial ischemic reaction, acidosis, hypoxia and catecholamine depletion inhibit the exhausted and almost any kind of sympathetic reaction.
- C.Intravascular coagulation:  initially there appears a slight agglutination of red blood cells in capillaries (so-called "sludged blood"), then the processes can get up to a syndrome of disseminated intravascular coagulation;

- ***Irreversible shock***
- It is the last phase of the shock showing the extreme gravity of the it. Any therapeutic intervention fails because all cellular mechanisms which should respond to it are paralyzed by energy depletion of receptors and effectors. In this phase, infusion of fluids to restore volume expansion produces no improvement in blood pressure and other vital functions. Death is imminent and occurs usually by respiratory arrest (by affecting the respiratory center) followed more or less by rapid cardiac arrest, the heart remains still the last to give up.

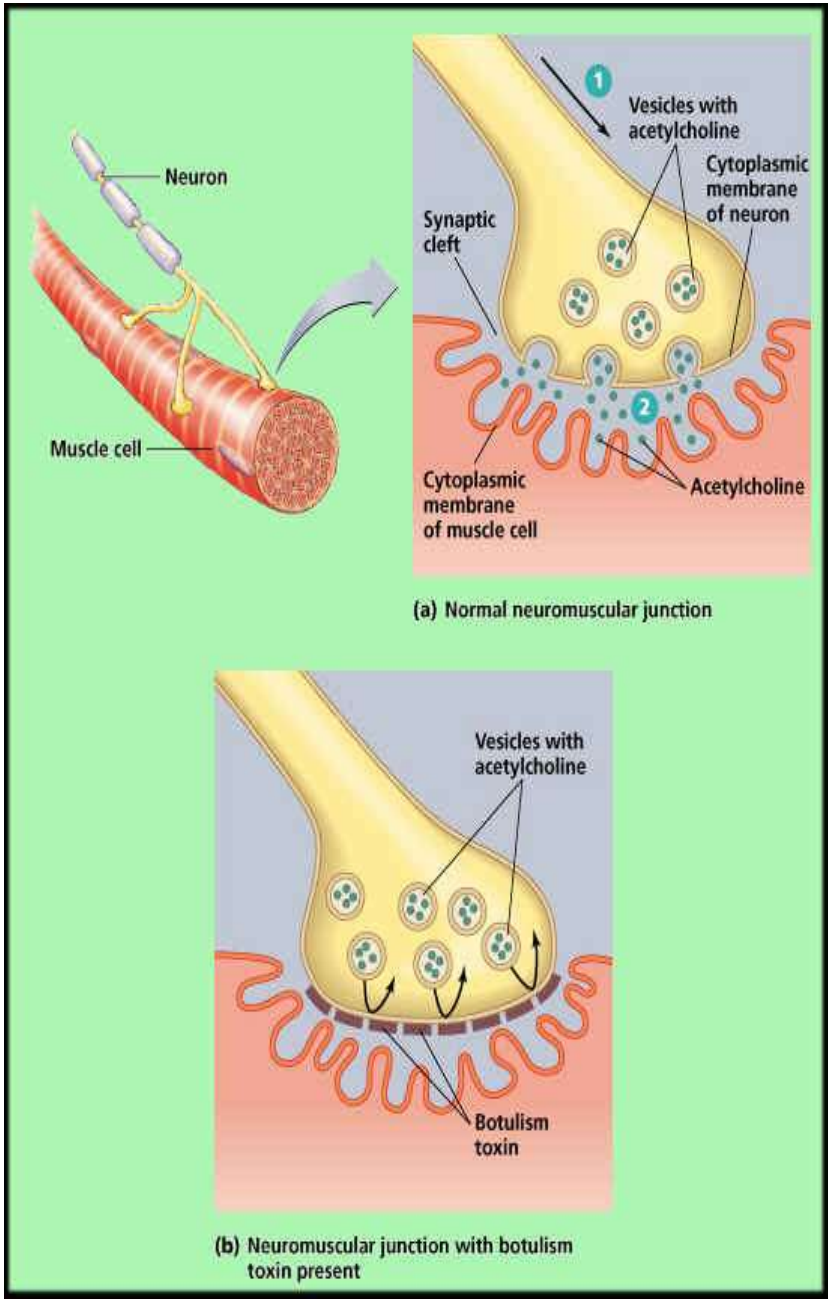
| | cholera | dysentery | salmonellosis |
|---|---|---|---|
| The chair | Aqueous, abundant, often colorless | Poor, with a mixture of mucus and streaks of blood | Green with undigested debris, sometimes colorless |
| Defecation | painless | tenesmus | Tenesmus can be |
| Pain in abdomen | - | + | + |
| Dehydration grade III-IV | + | - | - |
| Chill | - | + | + |
| Body temperature | Normal or low | grown | grown |
| Blood pressure | Low | Moderately low or normal | Moderately low or normal |
| Debut | With diarrhea | Often begins with vomiting, then diarrhea | Often begins with signs of infectious impregnation, then vomiting and diarrhea |
| Abdominal noise | + | - | - |
| Sigmoid painful spasms | - | + | + |
| Haemoconcentration | expressed | missing | missing |
| Oligoanuria | expressed | More often missing | More often missing |

# Clostridium botulinum food poisoning

- Botulism - is a severe and acute illness caused by botulinum neurotoxin, which is ingested with some contaminated food with Clostridium botulinum, but also develop at the level of infected wounds and is manifest by extensive peripheral paralysis with life-threatening respiratory paralysis. In survivors, the paralysis remains for a long time, also the disease leaves no immunity.

- The disease was first reported in Europe in the eighteenth century. It's spread was recognized in the late nineteenth century throughout Europe and then around the northern hemisphere. Botulism had a large expansions in the second half of the twentieth century, with hundreds of cases in China, Poland, Germany, USA, Japan. Nowadays, the disease has only a sporadic incidence.

- After ingestion the toxin is rapidly reabsorbed (still in the mouth). The mechanism includes rapid gut passage of the neurotoxin in the blood, but it was also detected after only 2 hours in the abdominal lymphatics, from where reaching the general circulation. Maximum absorption occurs in the proximal jejunum, but it was experimentally proved that all intestine allows absorption, including cecal mucosa. The irreversible fixing of synaptic knobs in neuromuscular plaque of motor neurons (terminal motor nerve synapse from the brain or spine), which will block the natural mediator - the acetilcholina.



The first manifestations occur in the cranial nerves, especially oculomotor. Patient has ptosis, diplopia, mydriasis, strabismus, accommodation palsy (by affecting the autonomic fibers of the third pair of cranial nerves, which innervates the ciliary muscle, which being paralyzed, leads to the swelling of the capacity of the lens for near vision).

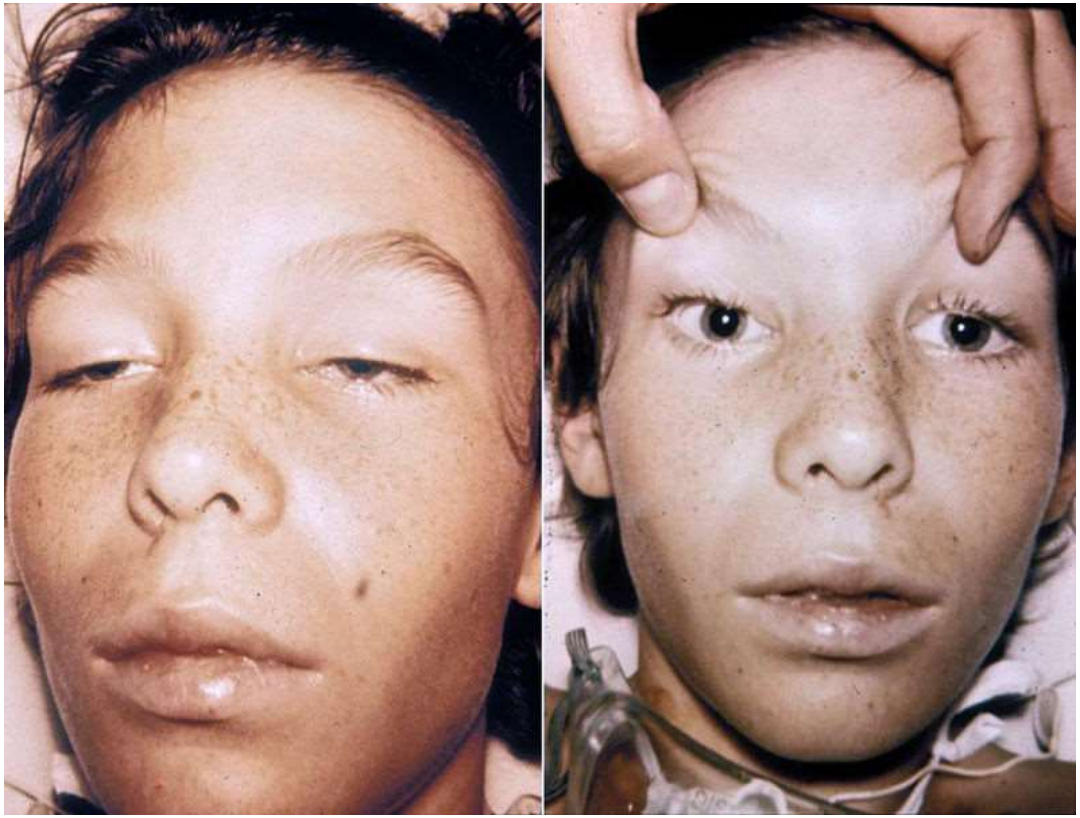
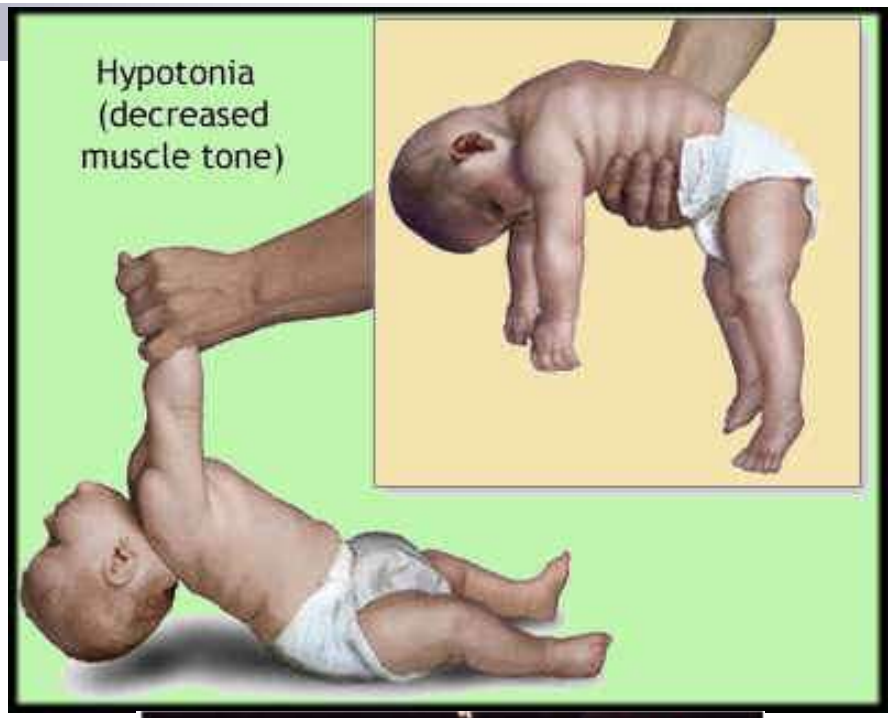


**Table 21.4** Frequency of symptoms in types A, B and E food-borne botulism[16]

| Symptoms | Type A disease (% of cases) | Type B disease (%) | Type E disease (%) |
|---|---|---|---|
| Dysphagia | 25–96 | 77–100 | 63–90 |
| Dry mouth | 26–83 | 96–100 | 55–88 |
| Diplopia | 50–90 | 57–100 | 85 |
| Dysarthria | 25–100 | 69–100 | 50 |
| Fatigue | 8–92 | 69–100 | Not known |
| Weakness of arm | 16–86 | 64–86 | Not known |
| Constipation | 73 | 17–100 | 25–38 |
| Weakness of leg | 16–76 | 64–86 | Not known |
| Dyspnea | 35–91 | 34 | 88 |
| Vomiting | 70 | 50–100 | 88–100 |
| Dizziness | 8–86 | 30–100 | 63 |
| Diarrhea | 35 | 8–14 | 10 |
| Paresthesiae | 20 | 12–14 | Not known |

- The infant botulism onset is characterized by cranial nerve damage: pupillary reaction to light are weak, ophthalmoplegia, dysphagia. Further development goes through a gradual biological degradation of the child, loss of appetite, flatulence and rebel constipation. During clynical status: worsening of onset signs, apathy until lethargy, weak cry, difficulty in sucking, hypotonia, until atony (child look flabby), loss of head control, lethargic feeling and asphyxia by ventilatory failure. Death may occur by respiratory stop or under assisted ventilation



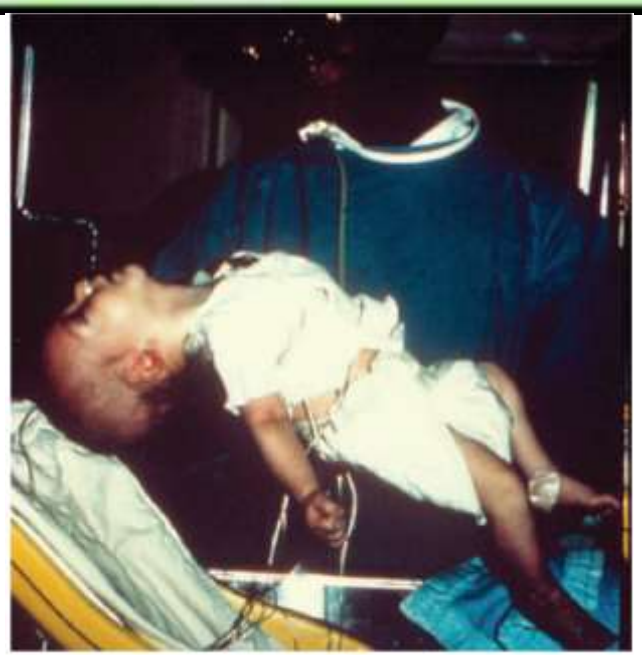



Figure 65.1 Infant with botulism. (From the U.S. Centers for Disease Control and Prevention Public Health Image Library at http://phil.cdc.gov/phil/home.asp.)

# FOODBORNE DESEASE'S TREATEMENT

- a) electrolyte and acido-basic rebalancing is the first therapeutic gesture, and should be started at the very beginning. In I-II dehydration degree, there will be used PERORAL SOLUTIONS (, which contains all the necessary electrolytes).

**Table 34.3** Composition of oral rehydration solution recommended by WHO

| Solute | Grams/liter | Mmol/liter |
|---|---|---|
| NaCl | 2.6 | Na 75; Cl 65 |
| Glucose | 13.5 | 7.5 |
| KCl | 1.5 | K 20 |
| Trisodium citrate | 2.9 | Citrate 10 |
| TOTALS | 20.5 | 245 osmolality |

**Table 15.9** Antibiotic Therapy for Acute Bacterial Diarrhea

| Pathogen | Therapy Recommendation |
|---|---|
| ***Campylobacter*** | Antibiotics only for severe disease or immunocompromised patients<br>azithromycin 500 mg PO qd × 3 days<br>*or*<br>ciprofloxacin 500 mg PO bid (regional resistance to quinolones exists) |
| ***Salmonella*** | Antibiotics only for severe disease or immunocompromised patients<br>ciprofloxacin, 500 mg PO bid × 5–7 days<br>*or*<br>azithromycin 1 g PO once, then 500 mg PO qd × 6 days |
| ***Shigella*** | Adults:<br>ciprofloxacin or levofloxacin, 500 mg PO bid × 3 days<br>*or*<br>TMP-SMX DS PO bid × 3 days<br>Children:<br>TMP-SMX 5/25 mg per kg PO qd × 3 days<br>Treat immunocompromised children and adults for 7–10 days |
| ***E. coli* shiga-toxin producing (*E. coli* 0157:H7)** | NO TREATMENT. Increased risk of HUS-TTP with antimicrobial and antimotility treatment. |
| ***Clostridium difficile*** | metronidazole 500 mg PO tid or 250 mg qid × 10–14 days<br>Severe disease: vancomycin 125 mg PO qid × 10–14 days |
| ***Yersinia enterocolitica*** | Antibiotics only for severe disease or immunocompromised patients<br>doxycycline 100 mg IV q 12h plus gentamicin or tobramycin 5 mg/kg q24h |
| ***Vibrio cholerae*** | ciprofloxacin 1g PO × 1 dose<br>Children or pregnant adults:<br>TMP-SMX DS PO bid × 2 days |
| ***Vibrio parahemolyticus*** | Generally supportive therapy is best.<br>Doxycycline 200 mg PO/IV bid × 3 days, then 100–200 mg PO bid × 14 days.<br>May consider fluoroquinolones or parenteral third-generation cephalosporins depending on organism sensitivity |
| **EIEC (Enteroinvasive *E. coli*)** | Generally supportive therapy is best<br>May treat severe disease with ciprofloxacin 500 mg PO bid × 3–5 days<br>*or*<br>TMP-SMX DS PO bid × 3–5 days |
| **EAEC (Enteroaggregative *E. coli*)** | Generally supportive therapy is best<br>May treat severe disease with ciprofloxacin 500 mg PO bid × 3–5 days<br>*or*<br>TMP-SMX DS PO bid × 3–5 days |
| **EHEC (Enterohemorrhagic *E. coli*)** | Antibiotics not recommended |

Adapted from Gilbert DN, Moellering RC, Eliopoulos GM, Sande MA. Sanford guide to antimicrobial therapy 2006, 36th ed. Sperryville, VA: Antimicrobial Therapy, 2006.
DS, double strength.