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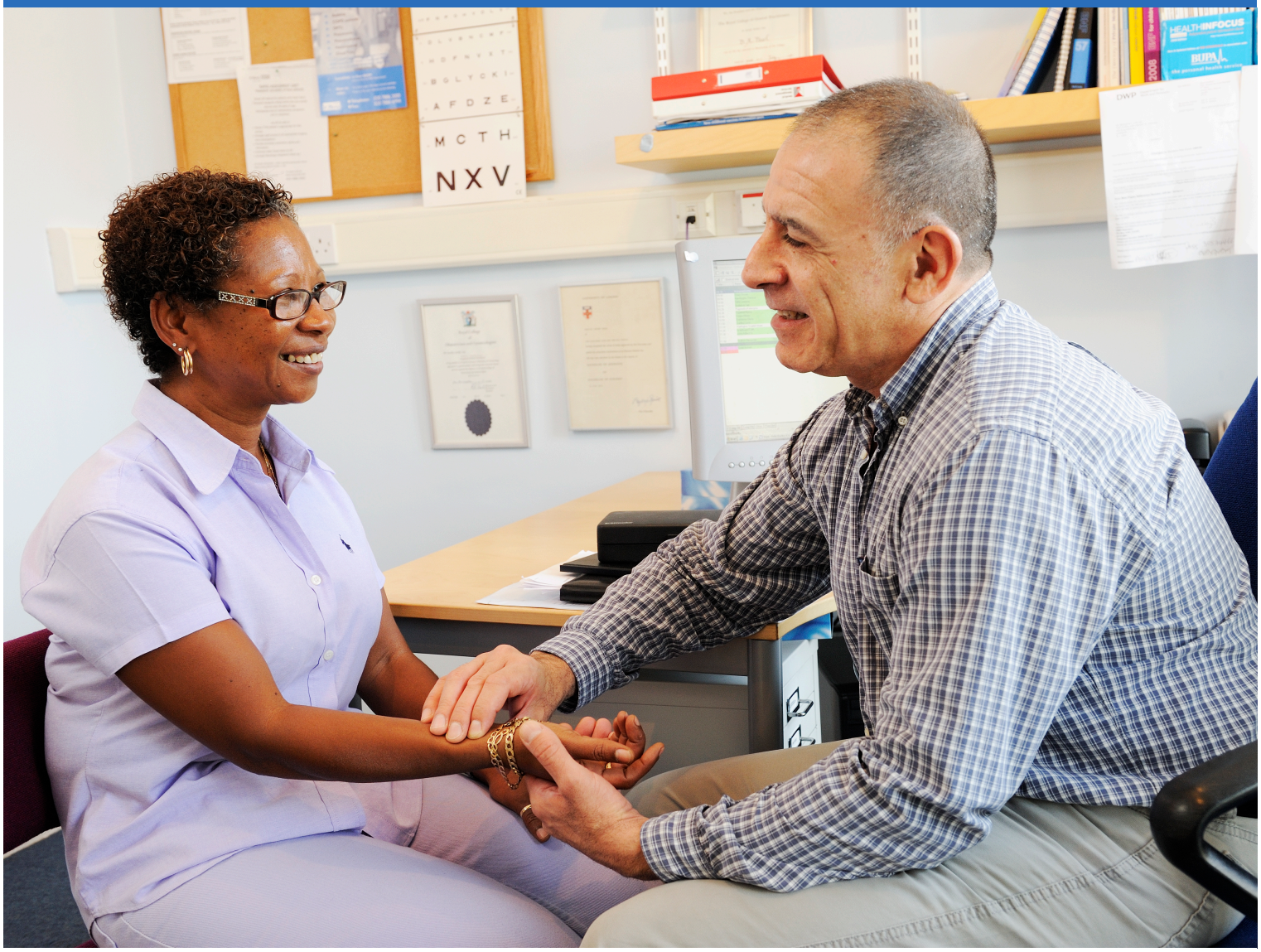
Assessment of acute diarrhoea

Straight to the point of care



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Summary

Diarrhoea can be defined as the passage of:[1] [2]

- three or more loose or liquid stools per 24 hours, and/or
- stools that are more frequent than what is normal for the individual lasting <14 days.

Based on duration, diarrhoea is classified as:[2] [3] [4] [5]

- acute (≤14 days),
- persistent (>14 days), or
- chronic (>4 weeks).

Epidemiology

Globally, there are almost 1.7 billion cases of childhood diarrhoeal disease each year.[1] Death due to diarrhoea disproportionately affects postneonatal children aged <5 years.[4] However, with implementation of improved access to oral rehydration therapy (ORT), vitamin supplementation, rotavirus vaccines, and education on feeding and weaning, it is estimated that mortality has dropped from 3.9 million deaths in 1967 to 526,000 in 2015.[6] Additionally, new antisecretory agents have shown promise in conjunction with ORT to reduce diarrhoea volume burden.[7] Despite the improvement, diarrhoea remains the third leading cause of death in children 1-59 months of age, with approximately 88% of these deaths due to water, sanitation and hygiene issues.[1] [8]

Around 47.8 million cases of acute diarrhoeal infection occur each year in the US, with an estimated cost of at least US$150 million to the healthcare economy.[9] [10] This compares to England and Wales, where infectious intestinal disease causes 300 deaths and 35,000 hospital admissions annually.[11] There are an estimated 17 million cases and 1 million consultations with a general practitioner attributed to acute infectious diarrhoea in the United Kingdom every year.[12]

Basic pathophysiology

Normally approximately 10 litres of fluid consisting of ingested food and drink, in addition to secretions from the salivary glands, stomach, pancreas, bile ducts, and duodenum, enters the gastrointestinal tract every day. The small intestine is the major site for re-absorption. Overall, about 99% of the fluid is re-absorbed, leaving 0.1 litre to be excreted in the faeces. Diarrhoea occurs when various factors interfere with this normal process, resulting in decreased absorption or increased secretion of fluid and electrolytes, or increase in bowel motility.

Improved understanding of the pathophysiology of infectious diarrhoea, and the factors that promote the spread of causative infectious agents, will lead to practical approaches for preventing and responding to outbreaks.[13]

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Aetiology

Acute diarrhoea can be classified based on pathophysiology or aetiology.[14]

Pathophysiological classification of diarrhoea

A commonly used pathophysiological classification divides diarrhoea into two categories.

Inflammatory diarrhoea

- This indicates the presence of an inflammatory process, which can be due to bacterial, viral, or parasitic infection, or may develop early in the course of bowel ischaemia, radiation injury, or inflammatory bowel disease.
- It is usually associated with mucoid and bloody stool, tenesmus, fever, and severe crampy abdominal pain.
- Infectious inflammatory diarrhoea is usually small in volume, with frequent bowel movements. It therefore does not usually result in volume depletion in adults, but may do so in children or older adults.
- The most common cause of infectious diarrhoea in the US is bacterial infection: mainly *Campylobacter* , *Salmonella* , *Shigella* , *Escherichia coli* , or *Clostridium difficile* . Viruses are more common among children who attend day care centres. Protozoa and parasites are common causes of acute diarrhoea in developing countries.
- Examination of the stool may show leukocytes, and tests for faecal occult blood may be positive. The test for faecal leukocytes is plagued by a high rate of false-negative results leading to low sensitivity, but a positive test is very informative.
- Histology of the gastrointestinal (GI) tract is abnormal in inflammatory diarrhoea.

Non-inflammatory diarrhoea

- This is usually watery, large-volume, frequent stool (>10 to 20 per day).
- Volume depletion is possible due to high volume and frequency of bowel movements.
- There is no tenesmus, blood in the stool, fever, or faecal leukocytes.
- Histologically the GI architecture is preserved.

Non-inflammatory diarrhoea can be subdivided into:

A) Secretory diarrhoea

There is an altered transport of ions across the mucosa, which results in increased secretion and decreased absorption of fluids and electrolytes from the GI tract, especially in the small intestine. Secretory diarrhoea tends not to decrease by fasting. Examples of causes are:

- Enterotoxins: these can be from infection such as *Vibrio cholerae* , *Staphylococcus aureus* , enterotoxigenic *E coli* , and possibly HIV and rotavirus.
- Hormonal agents: vaso-active intestinal peptide, small-cell cancer of the lung, and neuroblastoma.
- Laxative use, intestinal resection, bile salts, and fatty acids.

It is also seen in chronic diarrhoea with coeliac sprue, collagenous colitis, hyperthyroidism, and carcinoid tumours.

B) Osmotic diarrhoea

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Stool volume is relatively small (compared with secretory diarrhoea), and diarrhoea improves or stops with fasting. It results from the presence of unabsorbed or poorly absorbed solute (magnesium, sorbitol, and mannitol) in the intestinal tract that causes an increased secretion of liquids into the gut lumen. Measuring stool electrolytes shows an increased osmotic gap (>50), but the test has very limited practical value. Stool (normal or diarrhoea) is always isosmotic (260 to 290 mOsml/L).

Osmotic diarrhoea can be subdivided into:

- Maldigestion - refers to impaired digestion of nutrients within the intestinal lumen or at the brush border membrane of mucosal epithelial cells. It can be seen in pancreatic exocrine insufficiency and lactase deficiency.
- Malabsorption - refers to impaired absorption of nutrients. It can be seen in small bowel bacterial overgrowth, in mesenteric ischaemia, post bowel resection (short bowel syndrome), and in mucosal disease (coeliac disease).

Aetiological classification of acute diarrhoea

Diarrhoea can be classified into two broad categories based on aetiological factors: infectious and non-infectious.

Infectious diarrhoea

- The most common cause of acute diarrhoea worldwide is infection (viruses, bacteria, and parasites). Most are acquired through the faecal-oral route, from contaminated water or food. Most infections are self-limiting or treated easily. Specific investigations are warranted when resources are available in moderate to severe disease, or if there is a public health risk such as high risk for spreading disease to others.[2] Worldwide, most cases of acute infectious gastroenteritis are viral, as indicated by the observation that bacterial stool cultures in patients with acute diarrhoea are positive in only 1.5% to 5.6% of patients.[15] However, bacterial infections are much more likely to be responsible for severe cases of diarrhoea.
- Bacterial infections
    - *Escherichia coli* : this is a more common cause of diarrhoea in developing than in developed countries. It is the most common cause of infectious diarrhoea leading to hospitalisations in developing countries.[16] It usually occurs in epidemics in the summer season. Sources of infection include: beef, pork, fast food restaurants (undercooked hamburger), apple cider, leaf lettuce, milk, cheese, spinach, and sprouts. It is most common in very young or old people and can affect the small intestine (enterotoxigenic and enteropathogenic *E coli*). It is a common cause of traveller's diarrhoea (enterotoxigenic) and diarrhoea in children and can also affect the colon (enteroinvasive and enterohaemorrhagic, enteroaggregative *E coli*). It is complicated by dysentery in the enteroinvasive subtype. Enterohaemorrhagic *E coli* (most notably *E coli* O157:H7) causes haemolytic uraemic syndrome with associated high mortality.[17] Some studies have suggested that use of antibiotics to treat *E coli* O157:H7 leads to a greater incidence of haemolytic uraemic syndrome or mortality.[18] [19] However, this finding is not consistent through the few trials, and overall there is no clear evidence as to whether antibiotics are beneficial or detrimental.[20] [21]
    - *Campylobacter* : infection is generally acquired from undercooked contaminated poultry in developed countries.[22] *Campylobacter* is one of the two most commonly documented

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foodborne diseases in the US (the other being salmonella). Diarrhoea can be watery or bloody and is frequently associated with crampy abdominal pain. It has been linked to serious complications such as reactive arthritis and Guillain-Barre syndrome.[23] [24]

- *Salmonella* : non-typhoidal salmonellosis is the joint leading cause of food-borne disease and a common cause of diarrhoea leading to outpatient care in developed countries.[22] *Salmonella* is most commonly associated with ingestion of poultry, eggs, and milk products. The patient can become an asymptomatic carrier.
- *Shigella* : this is the classic cause of colonic or dysenteric diarrhoea. *Shigella* continues to be a major problem in day care centres and institutional settings. It presents with bloody stools, fever, abdominal cramps, and tenesmus.
- *Clostridioides difficile* : this is one of the most common hospital-acquired (nosocomial) infections and is a frequent cause of morbidity and mortality among older hospitalised patients. *C difficile* colonises the human intestinal tract and after the normal microbiota has been altered by antibiotic therapy it can lead to pseudomembranous colitis. Recurrent disease is common and thought to be due to altered host immunity. *C difficile* produces toxins, which are implicated in the disease. Leukaemoid reaction and hypoalbuminaemia, renal failure, and shock are seen in severe disease. Colectomy is necessary in severe cases. Diagnosis is by detection of toxins A and B or B alone, cell cytotoxicity assay, or detection of toxigenic *C difficile* in the stool.[25]
- *Yersinia* : infection is usually from eating pork meat or pig intestine. It causes acute or chronic colitis and can mimic Crohn's disease or acute appendicitis.
- *Aeromonas* : this is a common isolate in asymptomatic patients, but has been implicated as a cause of diarrhoea, mainly traveller's diarrhoea.[26]
- *Plesiomonas* : this has been documented in outbreaks of diarrhoea associated with contaminated water and oysters containing the micro-organism.[27]
- *Listeria* : is relatively rare; there are 0.1 to 10 cases of listeriosis per 1 million people per year, depending on the country/region.[28] It is usually transmitted by contaminated dairy or water. It can grow at refrigerator temperature.
- *Staphylococcus aureus* : leads to vomiting, and in some instances diarrhoea, within 4 to 8 hours following the ingestion of food contaminated with pre-formed toxin.
- *Bacillus cereus* : heat-stable pre-formed toxins cause symptoms within 6 hours of ingestion. In rare cases, infection causes acute liver necrosis.[29]
- *Clostridium perfringens* : causes watery diarrhoea secondary to pre-formed toxins. Ingestion of *C perfringens* spores is usually from the consumption of poultry, meat, and gravy. It can rarely result in a serious complication, enteritis necroticans, a haemorrhagic necrosis of the jejunum.[30]
- *Vibrio cholerae* : the hallmark of this infection is severe, toxin-induced, large-volume, non-bloody, secretory, dehydrating diarrhoea. It can be asymptomatic; present as mild disease indistinguishable from gastroenteritis; or present as severe disease (cholera gravis) in which a healthy individual can deteriorate quickly to a gravely unwell patient. It is diagnosed by detecting the bacteria or choleratoxin in stool. It is a vaccine-preventable disease.[5] [31]
- *Klebsiella oxytoca* : this has been associated with some cases of *C difficile* -negative antibiotic-associated haemorrhagic colitis.[32]

- Viral infections

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- Rotavirus: the leading known cause of severe viral gastroenteritis in infants and young children worldwide. It is a vaccine-preventable disease.[33] It causes diarrhoea that results in volume depletion in children and young adults. This infection peaks during cooler weather.
- Norovirus: this is a major cause in epidemic viral gastroenteritis. Noroviruses are the most common cause of outbreaks of non-bacterial gastroenteritis in the US.[34] Surveillance studies of food-borne diseases show that two-thirds of all food-related illnesses are due to noroviruses.[35] It is becoming the leading cause of medically attended acute gastroenteritis in countries with high rotavirus vaccine coverage.[36]
- Adenovirus: enteric adenovirus is second to rotavirus in causing diarrhoea, especially in day care centres.
- Astrovirus: responsible for 4% to 7% of diarrhoeas in day care centres, and is a known cause of nosocomial disease in young children.[37] [38] Astrovirus also causes illness in immunocompromised people and older institutionalised patients.[39] Unlike norovirus, astrovirus is an uncommon cause of epidemic gastroenteritis.
- COVID-19 (severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2]) infection: gastrointestinal symptoms are common in COVID-19 infection and diarrhoea may be the only presenting symptom. COVID-19-associated diarrhoea may be severe, but it is usually mild and self-limiting.[40]

- Parasites/protozoa infections

  - *Entamoeba histolytica* : worldwide, approximately 40 to 50 million people develop colitis or extraintestinal disease annually, with 40,000 deaths.[41] It is commonly asymptomatic. Clinical amoebiasis generally has a subacute onset, usually over 1 to 3 weeks. Symptoms range from mild diarrhoea to severe dysentery, producing abdominal pain (12% to 80%), diarrhoea (94% to 100%), and bloody stools (94% to 100%). Weight loss is present in just under 50% of patients.[42] Fever is also seen.
  - *Giardia lamblia* : causes both epidemic and sporadic disease, and is an important aetiology of water-borne and food-borne diarrhoea, day care centre outbreaks, and diarrhoea in international travellers and adoptees.[43] It is seen more commonly in patients with immunoglobulin A deficiency. Diagnosis is by stool *Giardia* antigen test (higher sensitivity than stool ova and parasite).
  - *Cryptosporidium* : has been known since 1976, but has become more prevalent with the increased prevalence of HIV/AIDS, with the increase in transplants and need for immunosuppression, and with an ageing population.[44] The diarrhoea may be acute or chronic; transient, intermittent, or continuous; and scant or voluminous with up to 25 L/day of watery stool.
  - Microsporidiosis: this is a less well known cause of traveller's diarrhoea in normal hosts. It is also associated with chronic diarrhoea in immunosuppressed patients.
  - *Cyclospora* : this organism is a cause of prolonged traveller's diarrhoea.[45] Infected patients may have a single self-limiting episode, but a prolonged waxing and waning course of GI symptoms lasting for weeks or months is common.[46]
  - Helminthic parasites (worms) (except *Strongyloides* in immunocompromised hosts) rarely cause diarrhoea.

Non-infectious diarrhoea

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- Drugs: a number of drugs are associated with acute diarrhoea. These include, but are not limited to, antacids containing magnesium, anti-arrhythmics (e.g., quinidine), antibiotics (as a primary cause or by causing *C difficile* infection), anti-hypertensives (beta-blockers, hydrochlorothiazide), anti-inflammatories (e.g., non-steroidal anti-inflammatory drugs [NSAIDs], gold salts), antineoplastic agents (including immune checkpoint inhibitors, which may lead to diarrhoea and colitis), antiretroviral drug, acid-reducing agents (e.g., H2 antagonists, proton-pump inhibitors), colchicine, prostaglandin analogues (e.g., misoprostol), theophylline, vitamins and mineral supplements, herbal drugs, heavy metals, and overuse of drugs for constipation.[47] [48] The mechanism differs between drug classes. Most of these drugs are thought to cause secretory diarrhoea.
- Acute diarrhoea can be seen as an initial presentation of chronic diarrhoea, such as seen in inflammatory bowel disease, bowel ischaemia, and radiation injury.

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Urgent considerations

(See **Differentials** for more details)
Certain complications of acute diarrhoea require urgent evaluation and treatment.

Volume depletion and electrolyte disturbances

These are the most common complications of acute diarrhoea. Children and older adults are at high risk. With children, the carer may not be replacing the fluid loss in a timely manner. Volume depletion manifests with increased thirst, decreased urinary output with dark urine, inability to sweat, and orthostatic symptoms. In severe cases, it may lead to acute renal failure and mental status changes (confusion and drowsiness). Cholera gravis in *Vibrio cholerae* infection may cause severe volume depletion, electrolyte disturbance, and arrhythmias. Prompt correction of hydration is required using low-osmolarity oral rehydration solution and isotonic intravenous fluid if oral or nasogastric intake is impaired.[5] In children, zinc supplementation is recommended as an adjunct to oral rehydration.[49] The use of the antisecretory agent racecadotril adjunctively with oral rehydration solution has been shown to diminish stool output safely, when studied in children up to the age of 10 years.[7]

Colonic perforation

This occurs principally in infants or severely malnourished patients and can be seen with *Clostridioides difficile* , *Salmonella* , and *Shigella* infections.[50] Urgent surgery is the treatment of choice.

Toxic megacolon

The pathogenesis of this complication is unclear; it occurs in the setting of pancolitis. Broad-spectrum antibiotic use may lead to *C difficile* infection with the associated complications of toxic megacolon, sepsis, perforation, and death. Toxic megacolon can also be seen with *Shigella* , cytomegalovirus (CMV), or *Yersinia* infection, and in ulcerative colitis and Crohn's disease.[51] In patients with known ulcerative colitis, toxic megacolon can be precipitated by superimposed infection. Treatment consists of supportive therapy with intravenous fluids, bowel rest, and total parenteral nutrition. Specific targeted therapy based on the underlying aetiology is given, such as corticosteroids in ulcerative colitis, antiviral agents in CMV colitis, and *C difficile* active antibiotics, or colectomy, in *C difficile* infection.

Intestinal obstruction and complications

This can occur in *Shigella* infections, helminth infections, and opportunistic infections in patients with AIDS. Crohn’s disease can also present with acute intestinal obstruction, intestinal perforation, peritonitis, and intra-abdominal abscess formation. The presenting symptoms are of crampy abdominal pain, nausea, vomiting, and abdominal distension. Treatment consists of nasogastric suction, strict nothing by mouth, and intravenous fluids. Antiparasitics and antibiotics may be needed, and surgery is considered if medical management fails.

Complications in other organs, bacteraemia, and sepsis

This can occur in severe infection. The normal microbiota may translocate across an inflamed colonic epithelium into the bloodstream. Endocarditis and osteomyelitis may complicate *Salmonella* infection. Myocarditis, glomerulonephritis, liver failure, peritonitis, and suppurative appendicitis may complicate *Yersinia* infection. *Yersinia* infection may also be mistaken for acute appendicitis. *C difficile* infection may lead to profound bowel necrosis, multiple organ failure, and death. Antibiotic treatment and supportive care is

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indicated in cases of severe infectious diarrhoea especially when additional complications arise. Specifically, colon resection is sometimes necessary in severe *C difficile* -induced bowel necrosis.

Sepsis is a spectrum of disease, where there is a systemic and dysregulated host response to an infection.[52] Presentation ranges from subtle, non-specific symptoms (e.g., feeling unwell with a normal temperature) to severe symptoms with evidence of multi-organ dysfunction and septic shock. It is important to consider the possibility of sepsis in any patient with symptoms or signs that indicate possible infection (e.g., acute diarrhoea).[53] Patients may have signs of tachycardia, tachypnoea, hypotension, fever or hypothermia, poor capillary refill, mottled or ashen skin, cyanosis, newly altered mental state or reduced urine output.[53] Sepsis and septic shock are medical emergencies.

Risk factors for sepsis include: age under one year, age over 75 years, frailty, impaired immunity (due to illness or drugs), recent surgery or other invasive procedures, any breach of skin integrity (e.g., cuts, burns), intravenous drug misuse, indwelling lines or catheters, and pregnancy or recent pregnancy.[53]

Early recognition of sepsis is essential because early treatment improves outcomes.[53] [54] [Evidence C] [Evidence C] However, detection can be challenging because the clinical presentation of sepsis can be subtle and non-specific. A low threshold for suspecting sepsis is therefore important. The key to early recognition is the systematic identification of any patient who has signs or symptoms suggestive of infection and is at risk of deterioration due to organ dysfunction. Several risk stratification approaches have been proposed. All rely on a structured clinical assessment and recording of the patient's vital signs.[53] [56] [57][58] [59] It is important to check local guidance for information on which approach your institution recommends. The timeline of ensuing investigations and treatment should be guided by this early assessment.[58]

Treatment guidelines have been produced by the Surviving Sepsis Campaign and remain the most widely accepted standards.[54] [60] Recommended treatment of patients with suspected sepsis is:

- Measure lactate level, and remeasure lactate if initial lactate is raised (>2 mmol/L)
- Obtain blood cultures before administering antibiotics
- Administer broad-spectrum antibiotics (with methicillin-resistant *Staphylococcus aureus* [MRSA] coverage if there is high-risk of MRSA) for adults with possible septic shock or a high likelihood for sepsis
- For adults with sepsis or septic shock at high-risk of fungal infection, empiric antifungal therapy should be administered
- Begin rapid administration of crystalloid fluids for hypotension or lactate level ≥4 mmol/L. Consult local protocols
- Administer vasopressors peripherally if hypotensive during or after fluid resuscitation to maintain mean arterial pressure (MAP) ≥65 mmHg, rather than delaying initiation until central venous access is secured. Noradrenaline (norepinephrine) is the vasopressor of choice
- For adults with sepsis-induced hypoxemic respiratory failure, high flow nasal oxygen should be given
- Ideally these interventions should all begin in the first hour after sepsis recognition.[60]

For adults with possible sepsis without shock, if concern for infection persists, antibiotics should be given within 3 hours from the time when sepsis was first recognised.[54] For adults with a low likelihood of infection and without shock, antibiotics can be deferred while continuing to closely monitor the patient.[54]

For more information on sepsis, please see Sepsis in adults and Sepsis in children .

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Neurological problems

Seizure is the most common neurological complication. It is a well-recognised complication of *Shigella* infection. Encephalopathy with lethargy, confusion, and headache can be seen. Obtundation or coma and abnormal neurological signs, including posturing, are rare. In cases of fatal encephalopathy, cerebral oedema has been found at autopsy. Delirium and coma may be present in *Salmonella* infection. Guillain-Barre syndrome may be seen as a late complication in *Campylobacter* enteritis. *Listeria* infection can cause meningitis.

Reactive arthritis

This may be seen alone or in association with conjunctivitis and urethritis, a triad formerly known as Reiter's syndrome.[61] It may be seen in *Shigella* , *Salmonella* , *Campylobacter* , and *Yersinia* infections. The arthritis is a sterile inflammatory arthritis. Treatment is usually supportive with non-steroidal anti-inflammatory drugs.

Haemolytic uraemic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP)

HUS presents with haemolysis and acute renal failure, while TTP presents with fever, haemolytic anaemia, thrombocytopenia, renal failure, and neurological changes. They can occur in infections with enterohaemorrhagic *E coli* and, less commonly, *Shigella* , particularly in young children and older adults who are exposed to antibiotics and antidiarrhoeal agents. Rare cases of HUS with *Campylobacter* and *Aeromonas* have also been reported. Treatment is supportive, but plasma exchange and glucocorticoids could be considered. Antibiotics should be avoided in infections secondary to enterohaemorrhagic *E coli* , as it is unclear if they increase risk of HUS.[20]

Hepatic necrosis

This is rarely associated with *Bacillus cereus* infection.[62]

Enteritis necroticans

Clostridium perfringens can cause enteritis necroticans (pigbel).[63]

Listeria infection in pregnancy

Listeriosis should be suspected in pregnant women with presumptive *Listeria* exposure based on ingestion of high-risk foods such as unpasteurised dairy products, unwashed fruits, improperly heated hot dogs or deli meats, and symptoms (including myalgia, abdominal or back pain, nausea, vomiting, or diarrhoea with or without fever ≥38.1°C [100.6°F]). The incidence of listeriosis associated with pregnancy is approximately 10 to 20 times higher than in the general population and it is diagnosed most commonly during the third trimester.[64] [65] Outcomes of Listeria infection in pregnant women are typically good, but it can lead to fetal death, premature birth, or infected newborns. Early treatment has been shown to improve fetal and neonatal outcomes; consult local protocols for guidance.[65]

Gangrenous bowel

Ischaemic colitis may result in gangrenous bowel. The patient may develop severe volume depletion and shock, and require surgical intervention.

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Approach

The diagnosis and differential diagnosis rely heavily on the patient's history. Most cases of acute diarrhoea are self-limiting, and further evaluation is not needed. However, the American College of Gastroenterologists (ACG) highlights that this approach may be a barrier to providing appropriate directed therapies that can result in more rapid symptom resolution and potentially prevent postinfectious complications.[2] On a practical level, many centres do not have access to the rapid tests advocated by the ACG.

Diagnostic testing is indicated in severe diarrhoea (hypovolaemia, large volume/bloody diarrhoea, fever), persistent diarrhoea, and immunocompromised or older patients.[2] [5]

History

In the vast majority of people, the diagnosis of acute diarrhoea is made exclusively by history and physical examination because it is impractical to collect and weigh the stool expelled over 24 hours. A clinically pertinent definition of diarrhoea is passage of three or more loose or liquid stools per 24 hours, or more frequently than what is normal for the individual. A full history and physical examination are very helpful in contemplating the specific cause of diarrhoea and deciding on the need for further laboratory or endoscopic evaluation. Characteristics of the diarrhoea provide clues to the diagnosis. Specific points of the history include the following.

- Onset of diarrhoea. Symptoms that begin within 6 hours of ingestion of contaminated food suggest a pre-formed toxin of *Staphylococcus aureus* or *Bacillus cereus* as the cause.
- Frequency of stool passage. Diarrhoea due to infectious causes tends to have more frequent stool passage.
- Amount of the stool. Toxin-induced diarrhoea tends to be large-volume (e.g., cholera) and osmotic diarrhoea smaller-volume.
- Consistency of the stool. Watery diarrhoea tends to be associated with non-invasive and toxin-producing pathogens.
- Blood in the stool. Suggests invasive pathogens or severe inflammation (e.g., ulcerative colitis).
- Mucus or pus in the stool. Seen usually in colonic involvement with inflammatory processes or infective pathogens.
- Fever. If present, suggests infection with invasive bacteria (e.g., *Salmonella* , *Shigella* , or *Campylobacter*), enteric viruses, or a cytotoxic organism such as *Clostridioides difficile* or *Entamoeba histolytica* .
- Recent travel. A history of travel to endemic regions may point to a specific pathogen. Infection with *Giardia* , *Cryptosporidium* , and *Cyclospora* can occur in Russia, Nepal, eastern Europe, or mountainous regions.
- Dietary history with recent types of food (meat, seafood, eggs, dairy) and water (well-water) ingestion, recent picnic, or barbecue may all be suggestive of infectious causes (e.g., *Campylobacter* , *Salmonella* , *Shigella* , *Escherichia coli* , or *C difficile*).
- Exposure to pets or cattle.
- Associated symptoms. Abdominal pain (e.g., invasive organisms), nausea (e.g., *Cryptosporidium*), vomiting (e.g., preformed toxins), bloating, flatus (e.g., *Giardia*), fever, tenesmus (left-sided colitis), anal itch.
- Drugs, specifically recent use of antibiotics or laxatives.
- Past medical or surgical history.
- Social history. Sexual practice, drug use, alcohol use.

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- Occupational history. Workers in day care centres, hospitals, mental institutions, and nursing homes may be exposed to *Giardia* , *Cryptosporidium* , and norovirus.

Physical examination

Physical examination is used to assess the severity of the diarrhoea, but rarely helps to determine its cause. In most people, diarrhoea is a self-limiting disease, so physical examination may be entirely normal.

Important parameters that can help in assessing fluid balance include:

- General appearance of the patient (i.e., whether unwell or well, and nutritional status)
- Pulse
- Skin turgor
- Whether or not mucous membranes appear dry
- Capillary refill time (usually <3 seconds, but may be increased in dehydration)
- Blood pressure
- Orthostatic changes (e.g., symptomatic orthostatic hypotension).

Careful abdominal examination may reveal clues to some diagnoses. Patients may have hyperactive, normal, or absent bowel sounds, localised or generalised abdominal tenderness, rebound tenderness, abdominal distension, enlarged liver (in *Salmonella* , amoebic liver abscess), or an abdominal mass.

Rectal examination can help in characterising stool and content, presence of mucus, or blood and faecal occult blood testing.

Indications for diagnostic testing

A close, positive working relationship between the physician and the microbiologist is needed to determine the best use of laboratory testing in cases of acute diarrhoea.[66] According to the American College of Gastroenterology, diagnostic evaluation is indicated in patients with relatively severe illness, as suggested by one or more of the following:[2]

- Dysentery
- Moderate-to-severe disease (severe = total disability due to diarrhoea; moderate = able to function but with forced change in activities)
- Symptoms lasting >7 days
- At high risk for spreading disease to others

According to guidelines for the management of diarrhoea in children with or without vomiting, diagnostic testing is indicated by one or more of the following:[67]

- History of blood with or without mucus in the stool
- Combination of abrupt onset of diarrhoea with more than 4 stools per day and no vomiting pre-diarrhoea
- Temperature >40°C (104°F)
- Five or more stools in the previous 24 hours
- Systemically unwell, severe or prolonged diarrhoea

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- History suggestive of food poisoning
- Recent history of travel overseas

Stool tests

Stool for faecal leukocytes is one of the initial stool tests in suspected inflammatory diarrhoea. The sensitivity and specificity of faecal leukocytes for inflammatory diarrhoea are 73% and 84%, respectively.[68] False-negative and false-positive results are possible. The presence of faecal leukocytes and a positive occult blood test support the diagnosis of invasive or inflammatory diarrhoea such as inflammatory bowel disease.[69] Although the faecal leukocyte test is routinely recommended as an initial test for the evaluation of acute diarrhoea, it is rarely done in practice due to the suboptimal specificity.

Faecal lactoferrin assays were developed in response to the limitation of faecal leukocyte testing. Faecal lactoferrin sensitivity and specificity range from 90% to 100% in distinguishing inflammatory diarrhoea (bacterial colitis, inflammatory bowel disease) from non-inflammatory diarrhoea (irritable bowel syndrome).[70] Calprotectin is another marker of faecal inflammation and can differentiate inflammatory bowel disease from functional disorders.[70] [71]

Stool culture, which most often tests for *E coli*, *Campylobacter*, *Shigella*, *Salmonella*, and *Yersinia*, (if specifically requested), is usually done in the following circumstances:[66]

- Immunocompromised patients, including those infected with HIV
- Patients with multiple comorbidities
- Patients with severe inflammatory diarrhoea (bloody diarrhoea)
- Patients with underlying inflammatory bowel disease in whom the distinction between a flare and superimposed infection is critical
- The test for stool leukocytes is positive
- Some employees, such as food handlers, who occasionally require negative stool cultures to return to work
- Outbreak investigations

Stool for ova and parasites is rarely helpful early in the evaluation of acute diarrhoea, but it can be useful in cases of persistent diarrhoea (14 to 30 days' duration). Specific indications to obtain stool for ova and parasites in patients with persistent diarrhoea include:

- History - suggests infection with a specific parasite
- Following travel to endemic or developing areas of the world
- Exposure to infants in day care centres (associated with *Giardia* and *Cryptosporidium*)
- Men who have sex with men or a patient with AIDS (associated with *Giardia*, *Cryptosporidium*, and *Entamoeba histolytica* and a variety of other parasites)
- A community waterborne outbreak (associated with *Giardia* and *Cryptosporidium*)
- Bloody diarrhoea with few or no faecal leukocytes (associated with intestinal amoebiasis)

Stool *Giardia* and *Cryptosporidium* antigen testing has a higher sensitivity than stool ova and parasites and should be requested if either is suspected from the history.

Tests for *C difficile* detect either the organism itself (i.e., nucleic acid amplification tests [NAAT], such as stool polymerase chain reaction (PCR), glutamate dehydrogenase [GDH] enzyme immunoassay, or toxigenic culture) or its major toxins (i.e., toxin A and B enzyme immunoassays, cell culture cytotoxicity neutralisation assay) directly in the stool. Molecular testing, the most common diagnostic method used, does

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not differentiate between infection and colonisation. It is highly sensitive with low/moderate specificity.[72] Stool cultures are the most sensitive test available, but are labour intensive, require an appropriate culture environment, and take 48 to 96 hours for results. They are not typically used in practice.[73]

In the interests of good diagnostic stewardship, it is recommended that *C difficile* testing is limited to patients with unexplained, new-onset diarrhoea (defined as 3 or more unformed stools in 24 hours) who are not receiving laxatives in the last 48 hours; however, this recommendation is based on very low-quality evidence.[72]

- If hospital and laboratory personnel agree on this stool submission criteria, NAAT alone is recommended as this is the most sensitive method of diagnosis in stool specimens from patients who are likely to have *C difficile* infection based on clinical symptoms.
- However, if there are no pre-agreed institutional criteria for patient stool submission, a stool toxin test as part of a multi-step algorithm is recommended (e.g., GDH plus toxin; GDH plus toxin, arbitrated by NAAT; or NAAT plus toxin) rather than NAAT alone.
- Repeat testing should not be performed within 7 days during the same episode of diarrhoea, and asymptomatic patients should not be tested.

European guidelines for *C difficile* testing recommend a two-step algorithm, starting with a highly sensitive test (e.g., NAAT, or GDH enzyme immunoassay) and, if positive, confirmation with a highly specific test (toxin A/B enzyme immunoassay). Alternatively, samples can be screened with both a GDH and toxin A/B enzyme immunoassay.[74]

Multiplex PCR tests have been developed and used to yield quicker and broader identification of viral, bacterial, and protozoal pathogens. These technological advances are frequently used now, although they are still unavailable in some settings despite their ability to diagnose gastrointestinal (GI) infections more rapidly and sensitively than other tests. While multiplex PCR tests increase the diagnostic yield, there are some drawbacks; the significance of detected organisms that these tests detect is not always clear with, for example, asymptomatic carriage of potential pathogens, multi-organism identification, and their inability to discriminate between viable and non-viable organisms. In addition, microscopy is still necessary for identification of some helminth and intestinal protozoa in tropical settings.[75] These culture and microscopy-independent diagnostic tests are recommended as an adjunct to the more traditional diagnostic methods.[2] Additionally, there are limited data available on the cost effectiveness of the use of GI multiplex panels as the only diagnostic tool.

Laxative screen can be performed when laxative use or misuse is suspected.

Stool weight, electrolytes, and osmotic gap are rarely if ever done in the evaluation of acute diarrhoea. Faecal fat is also rarely helpful.

Blood tests

Diagnostic tests that may be considered in the diagnostic evaluation of acute diarrhoea in people with severe illness are:

- FBC. This can help with assessing the severity of the diarrhoea (look for haemoconcentration, anaemia, and leukocytosis with left shift)
- Serum chemistry. Electrolytes, urea nitrogen, and creatinine (look for hypokalaemia, acidosis, and renal dysfunction). Serum albumin and lactic acid (look for low albumin or raised lactic acid)

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- Antibody testing. May be considered for inflammatory bowel disease as an adjuvant to endoscopic and radiographic evaluation.

Radiological studies

Radiological studies are not needed in all patients. Studies including abdominal x-ray, or computed tomography scan of the abdomen and pelvis, are useful to identify some of the complications of acute diarrhoea, such as ileus, perforation, or megacolon.

Toxic megacolon or perforation may be found in patients with *C difficile* or *Yersinia* infection, ulcerative colitis, and Crohn's disease.

Endoscopic evaluation

If all other tests are negative, endoscopic intervention (i.e., proctoscopy, flexible sigmoidoscopy, colonoscopy, oesophagogastroduodenoscopy, or video capsule endoscopy) will help in visualising the GI tract and obtaining biopsy (for histology and culture) as well as fluids for analysis and culture. Colonoscopy rather than sigmoidoscopy should be used in immunocompromised patients.[76] Endoscopy can be considered to:

- Distinguish inflammatory bowel disease from infectious diarrhoea.
- Rapidly diagnose *C difficile* infection by the presence of a pseudomembrane; the widespread use of enzyme-linked immunosorbent assay for *C difficile* toxins A and B, and PCR, has reduced the time for *C difficile* results to become available and therefore decreased the need for endoscopic evaluation.
- Evaluate for opportunistic infections such as cytomegalovirus and herpes simplex virus infection, as well as graft-versus-host disease in immunocompromised patients, and ischaemic colitis: findings vary and are non-specific; confirmation with a biopsy is required.
- Asses for pathognomonic changes of ischemic colitis.

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Differentials overview

Common
Rotavirus
Norovirus
Enteric adenovirus
Campylobacter enteritis (Campylobacter jejuni and Campylobacter coli)
Shigella
Salmonella
Escherichia coli (enterotoxigenic, enteropathogenic, enteroinvasive, enterohaemorrhagic, enteroaggregative)
Clostridioides difficile
Vibrio cholerae
Staphylococcus aureus
Bacillus cereus
Clostridium perfringens
Listeria
Giardia
Entamoeba histolytica
Microsporidiosis
Drugs
Ulcerative colitis
Crohn's disease
Irritable bowel syndrome

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Uncommon
Astrovirus
Yersinia
Aeromonas
Plesiomonas
Cryptosporidium
Cyclospora
Cystoisospora belli
Klebsiella oxytoca
Bowel ischaemia
Radiation injury

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Differentials

Common

Rotavirus

History	Exam	1st Test	Other tests
common in children from day care centres; may infect adults, immunocompromised people; fever, vomiting; diarrhoea: watery, yellow, no blood or mucus	normal with mild disease; signs of severe volume depletion seen with prolonged or acute diarrhoea	»**no testing needed:** history usually supports diagnosis History is typically diagnostic. Testing is reserved at discretion of physician.	»**faecal leukocytes:** may be positive »**FBC:** normal »**blood chemistry:** raised urea and creatinine »**stool enzyme-linked immunosorbent assay or latex agglutination:** positive »**stool polymerase chain reaction:** positive

◊ Norovirus

History	Exam	1st Test	Other tests
consumption of shellfish, prepared foods, salads, sandwiches, fruit; symptoms: nausea, abdominal cramps, followed by diarrhoea and vomiting; diarrhoea is watery, moderate, contains no blood or mucus; fever, malaise, myalgia, headache common	normal	»**faecal leukocytes:** negative	»**stool polymerase chain reaction:** positive

◊ Enteric adenovirus

History	Exam	1st Test	Other tests
common in infants, older adults, and immunocompromised people; secondary to contaminated food and water; common in	normal	»**stool for viral culture:** positive Patient may continue to shed virus for weeks	»**adenovirus-specific enzyme-linked immunosorbent assay or immunofluorescence assay:** positive

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Common

◊ Enteric adenovirus

History	Exam	1st Test	Other tests
day care centres and institutions; symptoms: mild, self-limiting diarrhoea, no fever		after cessation of viral infection.	Non-specific.

Campylobacter enteritis (Campylobacter jejuni and Campylobacter coli)

History	Exam	1st Test	Other tests
ingestion of undercooked poultry, raw milk, or cheese; diarrhoea either watery profuse or bloody with mucus, usually self-limiting, and resolves after 5 to 7 days; associated severe crampy periumbilical abdominal pain and fever; bloody diarrhoea can be present from third day of illness	fever; abdominal examination: generalised or localised tenderness, right lower quadrant tenderness may resemble acute appendicitis; rarely toxic secondary to toxic megacolon	»**faecal leukocytes:** positive Indicates inflammation and mucosal invasion.	»**FBC:** normal or raised WBC count with high percentage bands »**stool culture:** isolation of *Campylobacter* species Confirmatory test. »**stool polymerase chain reaction:** positive

Shigella

History	Exam	1st Test	Other tests
usually in children attending day care centres; also acquired from eating vegetables; sexual transmission increasingly reported, particularly among gay, bisexual, and other men who have sex with men; fever, then develops diarrhoea, watery becoming mucoid and bloody; stool: small in amount, ranges from 10 to 12 stools/day; fever and tenesmus in one third of patients	may be normal; fever; abdominal examination: generalised tenderness, distension, or absent bowel sounds, dependent on severity; rectal examination: tender, and rarely rectal prolapse	»**faecal leukocytes:** positive Indicates inflammation and mucosal invasion.	»**stool culture:** positive Confirmatory test, best done using mucoid stool. »**stool polymerase chain reaction:** positive

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Common

Salmonella

History	Exam	1st Test	Other tests
source usually food (beef, pork, poultry, eggs, raw milk, ice cream, orange juice) and faecally contaminated water; common with pets (pet ducklings, reptiles, and lizards); symptoms of nausea, vomiting, fever, diarrhoea (can persist for 10 days), cramping; usually self-limiting	usually normal, might present with mild volume depletion; abdominal examination: usually benign, abdominal tenderness may be localised to lower abdomen; enlargement of liver and spleen not uncommon	»**faecal leukocytes:** positive	»**FBC:** anaemia, leukopenia, or leukocytosis »**blood and stool culture:** positive »**stool#polymerase chain reaction:** positive

Escherichia coli (enterotoxigenic, enteropathogenic, enteroinvasive, enterohaemorrhagic, enteroaggregative)

History	Exam	1st Test	Other tests
occurs in travellers, children; dysentery; source usually contaminated food; depending on site and strain of infection, symptoms include profuse watery diarrhoea or bloody diarrhoea; abdominal pain but no fever	can be normal; minority of patients dehydrated with low blood pressure and high heart rate; mild diffuse or lower abdominal tenderness common	»**faecal leukocytes:** positive (invasive strain)	»**FBC:** anaemia in haemolytic uraemic syndrome »**stool culture:** positive Confirmatory test. »**blood chemistry:** renal dysfunction in haemolytic uraemic syndrome »**stool polymerase chain reaction:** positive

Clostridioides difficile

History	Exam	1st Test	Other tests
history of antibiotic use, hospitalisation, chemotherapy, development of diarrhoea in 2 to 3 weeks (up to 3 months); acute watery diarrhoea; milder disease: 3 to	signs of mild volume depletion; may be hypotensive, hypothermic, or have high fever; diffuse abdominal tenderness, abdominal distension,	»**stool polymerase chain reaction (PCR):** positive Recommended as sole investigation if hospital and laboratory personnel agree on	»**stool culture:** positive The most sensitive test, and is essential for epidemiological studies, but is not clinically practical.[73]

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Common

Clostridioides difficile

History	Exam	1st Test	Other tests
4 stools/day, mild lower abdominal pain; severe disease with pseudomembranous colitis: profuse diarrhoea, up to 15 times/day, lower abdominal crampy pain; fulminant colitis: fever, chills, diffuse abdominal pain, volume depletion	and absent bowel sounds in severe cases	the following stool submission criteria: unexplained, new-onset diarrhoea (defined as three or more unformed stools in 24 hours) in patients who are not receiving laxatives in the last 48 hours.[72] Recommended as part of multi-step algorithm if there are no pre-agreed institutional criteria for patient stool submission.[72] [74] Highly sensitive with low/moderate specificity. »**stool immunoassay for glutamate dehydrogenase:** positive Detects the presence of *Clostridioides difficile* in the bowel, although it does not provide confirmation of infection. Recommended as part of multi-step algorithm if there are no pre-agreed institutional criteria for patient stool submission.[72] [74] Has high sensitivity and specificity for the diagnosis of *C difficile* infection.[79]	»**FBC:** WBC count is raised, especially in severe disease »**sigmoidoscopy or colonoscopy:** pseudomembrane »**radiological imaging (CT or plain abdominal films):** toxic megacolon or perforation Indicated in patients with significant abdominal distension, worsening pain, or absent bowel sounds.

DIAGNOSIS

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Common

Clostridioides difficile

History	Exam	1st Test	Other tests
		»**stool immunoassay for toxins A and B:** positive Recommended as part of multi-step algorithm if there are no pre-agreed institutional criteria for patient stool submission.[72] [74] Results available within a few hours with sensitivity of 65% to 85% and specificity of 95% to 100%.[80] Can be repeated up to 3 times to improve sensitivity, although evidence for repeat ELISA testing has not been conclusive.[81] Enzyme immunoassays detect toxin A, toxin B, or both A and B. Most laboratories perform a toxin B-only or toxin A and B assay due to concerns over toxin A-negative and B-positive strains causing disease.[73]	

Vibrio cholerae

History	Exam	1st Test	Other tests
mild disease cannot be distinguished from other forms of gastroenteritis, can present as watery	normal to acutely ill; severe disease, signs of severe volume depletion, including	»**stool culture:** curved gram-negative rods	»**FBC:** haemoconcentration »**electrolytes:** acidosis, hypokalaemia, renal dysfunction

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Common

Vibrio cholerae

History	Exam	1st Test	Other tests
diarrhoea; severe cases: diarrhoea with massive volume loss, termed rice water stool (due to flakes of mucus), abdominal cramps	hypotension and mental status changes		»**stool polymerase chain reaction:** positive

◊ Staphylococcus aureus

History	Exam	1st Test	Other tests
ingestion of beef (hamburger), pork, poultry, eggs 4 to 6 hours prior to symptom onset; nausea and vomiting, later associated watery diarrhoea; no fever or abdominal pain	normal	»**clinical diagnosis; no routine testing:** testing stool or vomitus for toxin is done only in suspected outbreaks	

Bacillus cereus

History	Exam	1st Test	Other tests
classic history of eating reheated rice; within 6 hours develops nausea, vomiting, later followed by diarrhoea; also seen with consumption of beef, pork and vegetables; no fever present	normal	»**none:** clinical diagnosis	

Clostridium perfringens

History	Exam	1st Test	Other tests
nausea and vomiting followed by diarrhoea 8 to 12 hours after consumption of beef, pork (chitterlings), poultry, and home	normal	»**none:** clinical diagnosis	

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Common

Clostridium perfringens

History	Exam	1st Test	Other tests
foods; no associated fever or abdominal pain			

Listeria

History	Exam	1st Test	Other tests
consumption of beef, pork, poultry, milk, cheese, coleslaw, hot dogs, or potato salad; seen in pregnancy, neonates, immunocompromised people; watery diarrhoea, moderate frequency, nausea and vomiting, mild abdominal pain, fever; may be asymptomatic	volume depletion uncommon; febrile; abdominal examination may show mild tenderness; obtundation in central nervous system disease	»**blood culture:** positive Ordered if high suspicion of bacteraemia exists (positive epidemiological risk factors) or with the presence of fever and rigors.	»**FBC:** raised WBC count, may have low platelets Leukocytosis may be the first indication of an invasive bacterial infection. Thrombocytopenia may indicate impending disseminated intravascular coagulation associated with sepsis. »**polymerase chain reaction#bf blood:** positive May be ordered when there is high suspicion of listeriosis and cultures are negative. Blood cultures may be negative after antibiotic treatment, reducing their sensitivity. »**cerebrospinal fluid culture:** positive Ordered in immunocompromised patients and if meningitis is suspected.

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Common

Giardia

History	Exam	1st Test	Other tests
travel to endemic areas; spread: person-to-person or contaminated food, water; may be asymptomatic; sudden-onset watery diarrhoea, abdominal bloating, and cramps, nausea, vomiting, foul-smelling fatty stool, and flatulence; symptoms usually prolonged	normal or mild volume depletion; abdominal examination: increased bowel sound, no tenderness or localising sign	»**stool microscopy (ova and parasite):** positive for trophozoites »**direct fluorescence antibody test:** presence of green, glowing ovoid objects indicates Giardia cysts Gold standard for diagnosing giardiasis.[82] When compared with ova and parasite examination, this test is more sensitive and specific and has enhanced sensitivity and faster turnaround time than conventional stool microscopy methods.[83] »**enzyme-linked immunosorbent assay (ELISA):** positive for parasite antigens and/or cyst wall ELISA can detect soluble stool antigens.[82] When compared with ova and parasite examination, this test is more sensitive and specific and has enhanced sensitivity and faster turnaround time than conventional stool microscopy methods.[83]	»**duodenal aspirate/ biopsy:** usually normal, trophozoite can sometimes be identified Duodenal biopsy may show flattened villi but is usually normal.[84] Duodenal mucosa is normal in 96% in a study.[84]

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Common

Giardia

History	Exam	1st Test	Other tests
		»**nucleic acid amplification test, polymerase chain reaction of stool sample:** positive More sensitive than ova and parasite examination.[83] Does not confirm viable parasite infection.	

Entamoeba histolytica

History	Exam	1st Test	Other tests
gradual onset over 1-3 weeks; abdominal pain; a minority of patients have fever and haematochezia	abdominal tenderness; enlarged liver with tenderness; signs of volume and electrolyte imbalance in patients with profuse diarrhoea; in patients with extraintestinal disease there may be signs associated with brain, lung, and liver abscesses	»**stool wet mounts or trichrome stain:** visualisation of cysts and trophozoites Stool samples which are positive on microscopy for *E histolytica* should be sent for confirmatory PCR testing.[85] »**stool polymerase chain reaction (PCR):** amplification of amebic DNA Where available, PCR is now the method of choice for diagnosis of *E histolytica* .[85] [86] However, protocols (DNA extraction and primer-probe sets) are not fixed, and only limited institutes can return reliable results. Also, cost is a barrier to its use as a routine test in many endemic areas, meaning that laboratory	»**serum antibodies:** positive for anti-amoebic antibodies »**radiological studies:** extraintestinal disease such as liver abscess, empyema, brain abscess

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Common

Entamoeba histolytica

History	Exam	1st Test	Other tests
		diagnosis in many countries still relies on antigen-detection or microscopy for ova and parasites. Stool samples which are positive on microscopy for *E histolytica* should be sent for confirmatory PCR testing.[85]	

◊ Microsporidiosis

History	Exam	1st Test	Other tests
immunocompetent people: self-limiting, mild or rarely chronic diarrhoea; chronic diarrhoea in older people; immunocompromised people: diarrhoea, non-bloody, watery, may be continuous or intermittent, can be associated with crampy abdominal pain; weight loss, wasting, nausea and vomiting, and malabsorption; fever is rare	may be normal; immunocompromised patient may present with fever, abdominal tenderness, and wasting	»**stool or tissue specimen:** spores	»**biopsy:** spores Less sensitive than stool or tissue specimen, and can be negative because the infection is patchy. »**transmission electron microscopy:** microspora Most definitive test, but expensive and time-consuming. »**modified trichome stain (urine, stool, mucus, or tissue):** spores More commonly used than transmission electron microscopy. »**stool culture:** positive

DIAGNOSIS

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Common

Drugs

History	Exam	1st Test	Other tests
recently started drug, followed by development of diarrhoea within couple of days to weeks; important to inquire about antibiotics, antacids, proton-pump inhibitors, laxatives, non-steroidal anti-inflammatory drug use; diarrhoea watery, no mucus or blood	usually normal, mild volume depletion not uncommon; abdominal examination normal	»**none:** clinical diagnosis	»**stool for Clostridioides difficile toxins:** may be positive in antibiotic-related infection »**laxative screen:** positive if laxative-related diarrhoea

Ulcerative colitis

History	Exam	1st Test	Other tests
blood in stools; may present with toxic megacolon	normal in mild disease; severe disease: toxic, volume depletion; abdominal examination: variable degree of tenderness; rectal examination: mucus, blood; extra-intestinal manifestations: joint, eye, mucous membrane, skin involvement	»**faecal calprotectin:** raised Faecal calprotectin is raised when there is bowel inflammation and correlates with endoscopic and histological gradings of disease severity. Testing is useful in supporting clinicians in the differential diagnosis of irritable bowel syndrome (IBS)/inflammatory bowel disease (IBD) and can prevent unnecessary referrals for colonoscopy. »**flexible sigmoidoscopy/ colonoscopy:** variable degree of inflammatory mucosa	»**FBC:** may show raised WBC count, anaemia »**erythrocyte sedimentation rate and C-reactive protein:** may be raised »**comprehensive metabolic panel (including LFTs):** non-specific ranging from normal to hypoalbuminaemia to significant electrolyte abnormalities in toxic megacolon »**radiological imaging (x-ray, barium enema, CT):** may show toxic megacolon, perforation Utilised in acute presentation.

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Common

Ulcerative colitis

History	Exam	1st Test	Other tests
		Histology may help confirm diagnosis.	

Crohn's disease

History	Exam	1st Test	Other tests
abdominal pain, fever, weight loss; diarrhoea: more gradual-onset, large-volume watery diarrhoea suggests small-bowel involvement, frequent small-bowel movements with tenesmus suggest colonic involvement; blood may be seen in stools	normal in mild disease; can present with acute intestinal obstruction, intestinal perforation, peritonitis, and intra-abdominal abscess formation; severe disease: toxic, volume depletion, variable degree abdominal tenderness; mucus, blood per rectum, perianal fistulas, perirectal abscess; extra-intestinal manifestations: for example, joint, eye, mucous membrane, skin involvement	»**faecal calprotectin:** raised Faecal calprotectin is raised when there is bowel inflammation and correlates with endoscopic and histological gradings of disease severity. Testing is useful in supporting clinicians in the differential diagnosis of IBS/IBD and can prevent unnecessary referrals for colonoscopy. »**ileocolonoscopy:** normal rectum, small discrete aphthous ulcer, serpiginous and linear ulcers, skip lesions (normal mucosa and areas of erythema), isolated terminal ileum involvement Histology may help confirm diagnosis.	»**FBC:** WBC count may be normal or raised; anaemia can be seen »**erythrocyte sedimentation rate and C-reactive protein:** usually increased »**comprehensive metabolic panel (including LFTs):** non-specific ranging from normal to hypoalbuminaemia to significant electrolyte abnormalities in toxic megacolon »**radiological imaging (abdominal x-ray, small bowel follow through and enema, CT):** toxic megacolon, fistula, abscess, or perforation

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Common

◊ Irritable bowel syndrome

History	Exam	1st Test	Other tests
diarrhoea: small frequent loose stools, small to moderate volume, preceded by urgency, common after meals, no midnight waking, alternates with constipation; may be abdominal discomfort, pain, and bloating; may see mucus; blood is rare	usually normal; may show abdominal distension and mild abdominal tenderness	»**none:** clinical diagnosis made by exclusion	»**faecal calprotectin:** <50 micrograms/g makes IBD unlikely (and IBS more likely)

Uncommon

◊ Astrovirus

History	Exam	1st Test	Other tests
common in children attending day care centres; winter months; usually a water-borne disease; symptoms: loose watery stool, up to 20 stools/day	usually normal; may show mild signs of volume depletion	»**none:** clinical diagnosis	

Yersinia

History	Exam	1st Test	Other tests
ingestion of contaminated pork, beef, milk, cheeses; diarrhoea insidious onset, watery, 5 to 10 times/day; classically abdominal pain localised to the right lower quadrant; can cause mesenteric adenitis mimicking acute appendicitis and Crohn's disease; associated with iron overload syndrome	may be normal or present as acutely unwell patient; abdominal examination will demonstrate right lower quadrant rebound tenderness and signs of peritonitis in severe cases	»**stool culture:** positive Confirmatory (may remain positive for weeks after the infection subsides).	»**FBC:** normal »**serology test (agglutination and enzyme-linked immunosorbent assay):** positive »**stool#polymerase chain reaction:** positive »**radiological imaging (CT):** toxic megacolon or perforation Should be ordered when toxic megacolon

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Uncommon

Yersinia

History	Exam	1st Test	Other tests
			or perforation is suspected. Can help distinguishing from acute appendicitis.

Aeromonas

History	Exam	1st Test	Other tests
self-limiting; variety of presentations including acute secretory diarrhoea, acute dysentery type with blood and mucus, chronic diarrhoea, choleric and traveller's diarrhoea (endemic in Southeast Asia); worse in children, older people, and immunocompromised people	usually normal	»**stool culture:** positive	

◊ Plesiomonas

History	Exam	1st Test	Other tests
consumption of raw or undercooked shellfish and contaminated water; watery secretory diarrhoea or dysentery type with severe abdominal pain; usually self-limiting, can progress to chronic diarrhoea; vomiting uncommon	usually normal; patient may be febrile	»**stool culture:** positive	

Cryptosporidium

History	Exam	1st Test	Other tests
asymptomatic, mild diarrhoeal illness	may be normal; signs of volume depletion,	»**stool ova and parasites:** positive	»**abdominal ultrasound:** enlarged

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Uncommon

Cryptosporidium

History	Exam	1st Test	Other tests
or severe enteritis with or without biliary involvement; malaise, nausea, anorexia, and abdominal pain; diarrhoea: acute, chronic, transient, intermittent, or continuous, scanty, or large volume up to 20 times/day; in immunocompromised people the disease may be frequent and severe	abdominal tenderness; patient with chronic diarrhoea may present with weight loss; immunocompromised patients present with significant wasting	Oocysts seen on stained microscopy with acid-fast, fluorescent, or direct immunofluorescent stains. Does not differentiate species. »**Cryptosporidium antigen detection:** positive for cryptosporidium antigen Enzyme immunoassays are reportedly superior to acid-fast microscopy and comparable to direct immunofluorescence microscopy in terms of sensitivity and specificity. Rapid immunochromatographic assays are comparable to acid-fast microscopy. »**stool#polymerase chain reaction:** positive Detection of DNA by PCR-based methods may also be helpful for the detection of other lifecycle stages in specimen types other than stool (intestinal fluid, broncho-alveolar washings, antral washings, tissue samples, biopsy specimens) from	gall bladder with thickened wall, dilated intra- and extra- hepatic bile ducts »**CT scan:** enlarged gall bladder with thickened wall, dilated intra- and extra-hepatic bile ducts Confirmatory test.

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Uncommon

Cryptosporidium

History	Exam	1st Test	Other tests
		immunocompromised patients. Availability of PCR testing varies.	

◊ Cyclospora

History	Exam	1st Test	Other tests
ingestion of infectious oocysts; may be asymptomatic; mild diarrhoeal illness and flu-like symptoms; flatulence, burping, malaise, and weight loss have been described; disease self-limiting, but in immunocompromised people might be prolonged	usually normal; in HIV/AIDS, may present with volume depletion and wasting	»**stool ova and parasites:** *Cyclospora* oocyst May be positive in asymptomatic patient.	»**small bowel biopsy:** several stages of development of the parasite

◊ Cystoisospora belli

History	Exam	1st Test	Other tests
self-limiting in immunocompetent people, but chronic cases have been seen; diarrhoea, steatorrhoea, abdominal pain, nausea and vomiting, fever, malaise, volume depletion, and weight loss; in immunocompromised people: chronic diarrhoea, can relapse after treatment; severe watery diarrhoea and weight loss	usually normal; fever, abdominal tenderness, signs of volume depletion, weight loss, and wasting	»**stool ova and parasites:** *Cystoisospora* oocyst Not seen in routine ova and parasite test of the stool; therefore, acid-fast staining or specific fluorescent techniques should be used.	»**FBC:** high eosinophils Seen only with *Cystoisospora* (no other protozoa).

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Uncommon

◊ Klebsiella oxytoca

History	Exam	1st Test	Other tests
has been associated with some cases of *Clostridioides difficile* -negative antibiotic-associated haemorrhagic colitis	signs of mild volume depletion; may be hypotensive, hypothermic, or have high fever; diffuse abdominal tenderness, abdominal distension, and absent bowel sounds in severe cases	»**stool culture:** positive	

Bowel ischaemia

History	Exam	1st Test	Other tests
history of coronary artery disease, aorto-iliac surgery, cardiopulmonary bypass, cardiac embolism, myocardial infarction, atrial fibrillation, haemodialysis, mesenteric vein thrombosis, mechanical injury, or patient presenting with shock or cardiopulmonary compromise; abdominal pain, passage of bloody loose stool	initial phase: minimal abdominal tenderness over affected side; discrepancy between degree of abdominal pain described by patient and abdominal examination; progresses to abdominal distension, severe abdominal tenderness, and absent bowel sounds; severe cases: volume depletion, shock	»**CT scan of abdomen with contrast/CT angiogram:** bowel thickening, bowel dilation, pneumatosis intestinalis, portal venous gas, occlusion of the mesenteric vasculature, bowel wall thickening with thumbprinting sign suggestive of submucosal oedema or haemorrhage Obtain the scan early if acute or chronic mesenteric ischaemia.is suspected; prompt diagnosis (and intervention) is essential to improve the clinical outcome. »**WBC:** raised Indicates intestinal tissue damage. »**liver function tests:** may be raised Derangement may be a consequence of septic	»**abdominal x-ray:** bowel distension and pneumatosis Examination may reveal non-specific findings. »**sigmoidoscopy or colonoscopy:** pale mucosa and petechial bleeding, bluish haemorrhagic nodule, and mucosal ulceration Should be avoided with suspicion of bowel perforation. It permits biopsy. »**mesenteric angiography:** proximal defect of a mesenteric vessel or vasoconstriction of all mesenteric arcades Historically, mesenteric angiography has been the definitive test for diagnosing mesenteric ischaemia. In current practice it is usually preceded by positive

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Uncommon

Bowel ischaemia

History	Exam	1st Test	Other tests
		shock or concomitant with bowel ischaemia »**arterial blood gas and serum lactate:** acidosis Assists with making the diagnosis and determining the severity of the illness	CT angiography in the acute setting. »**amylase:** may be raised Raised serum amylase is found in approximately half of patients with acute mesenteric ischaemia.[87]

◊ Radiation injury

History	Exam	1st Test	Other tests
history of radiation to the abdominal region for different reasons (e.g., prostate cancer); abdominal pain, nausea, vomiting, and diarrhoea; intestinal obstruction and/or intestinal stricture; might present with new lactose intolerance	usually normal; some might present with mild abdominal tenderness, abdominal distension, and increased bowel sounds; might present with signs of bowel obstruction	»**upper gastrointestinal series:** stricture and the extent of the injury	»**enteroclysis:** submucosal thickening, stenosis, adhesion, or fistula Radiographic examination after barium introduced into the small intestine for contrast. »**CT scan or MRI enteroclysis:** thickening of bowel segment Superior to CT scan. »**CT scan of the abdomen:** thickening of bowel segment »**colonoscopy:** pale, friable mucosa, telangiectasia Biopsy is non-specific.

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Guidelines

United Kingdom

Diarrhoea and vomiting caused by gastroenteritis in under 5s: diagnosis and management (https://www.nice.org.uk/guidance/cg84)

Published by: National Institute for Health and Care Excellence
Last published: 2009

International

Acute diarrhoea in adults and children: a global perspective (http://www.worldgastroenterology.org/guidelines/global-guidelines)

Published by: World Gastroenterology Organisation
Last published: 2012

North America

Guide to utilization of the microbiology laboratory for diagnosis of infectious diseases: 2024 update by IDSA/ASM (https://www.idsociety.org/practice-guideline/practice-guidelines/#/+/0/date_na_dt/desc)

Published by: Infectious Diseases Society of America; American Society for Microbiology
Last published: 2024

Clinical practice guideline: 2021 focused update guidelines on management of Clostridioides difficile infection in adults (https://www.idsociety.org/practice-guideline/practice-guidelines/#/+/0/date_na_dt/desc)

Published by: Infectious Diseases Society of America (IDSA) and Society for Healthcare Epidemiology of America (SHEA)
Last published: 2021

Clinical practice guidelines for Clostridium difficile infection in adults: 2017 update (https://www.idsociety.org/practice-guideline/practice-guidelines/#/+/0/date_na_dt/desc)

Published by: Infectious Diseases Society of America; Society for Healthcare Epidemiology of America
Last published: 2018

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North America

2017 Infectious Diseases Society of America clinical practice guidelines for the diagnosis and management of infectious diarrhea (https://www.idsociety.org/practice-guideline/practice-guidelines/#/+/0/date_na_dt/desc)

Published by: Infectious Diseases Society of America
Last published: 2017

ACG clinical guideline: diagnosis, treatment, and prevention of acute diarrheal infections in adults (https://gi.org/guidelines)

Published by: American College of Gastroenterology
Last published: 2016

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Evidence tables

What are the effects of early versus late initiation of empiric antimicrobial treatment in adults with or at risk of developing sepsis or severe sepsis?[55]

This table is a summary of the analysis reported in a guideline (underpinned by a systematic review) that focuses on the above important clinical question.

View the full source guideline (https://www.nice.org.uk/guidance/ng51/evidence)

Evidence C *	Confidence in the evidence is very low or low where GRADE has been performed and the intervention may be more effective/beneficial than the comparison for key outcomes, however this is uncertain and new evidence could change this in the future.

Population: Adults with or at risk of developing sepsis or severe sepsis
Intervention: Early initiation of empiric antimicrobial treatment
Comparison: Late initiation of empiric antimicrobial treatment

Outcome	Effectiveness (BMJ rating)†	Confidence in evidence (GRADE)‡
<1 hour versus >1 hour		
Mortality [a]	Favours intervention	Very Low
Mortality - Intensive Care Unit (ICU) setting	Favours intervention	Very Low
Mortality - Emergency Department (ED) setting	No statistically significant difference	Very Low
<2 hours versus >2 hours		
Mortality [a]	No statistically significant difference	Very Low
Mortality -ICU setting	Favours intervention	Very Low
Mortality - ED setting	No statistically significant difference	Very Low
<3 hours versus >3 hours		
Mortality [a]	Favours intervention	Very Low
Mortality -ICU setting	No statistically significant difference	Very Low

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Outcome	Effectiveness (BMJ rating)†	Confidence in evidence (GRADE)‡
Mortality - ED setting	Favours intervention	Very Low
<4 hours versus >4 hours		
Mortality - ED setting	No statistically significant difference	Very Low
<5 hours versus >5 hours		
Mortality - ED setting	No statistically significant difference	Very Low
<6 hours versus >6 hours		
Mortality [a]	Favours intervention	Very Low
Mortality - ICU setting	No statistically significant difference	Very Low
Mortality - ED setting	Favours intervention	Very Low

Recommendations as stated in the source guideline

The guideline committee recommends that adults, children and young people over the age of 12 who have suspected sepsis and one or more high risk criteria, should be given a broad-spectrum antimicrobial at the maximum recommended dose without delay (within 1 hour of establishing they meet high risk criteria in an acute hospital setting).[b] See guideline for details on criteria for different levels of risk.

Note

Results in this table are based on observational studies only.

[a] Includes overall mortality in intensive care and emergency department settings.

[b] This guideline recommends that all people with suspected sepsis have a face-to-face assessment and a risk stratification tool is used to determine risk of severe illness and death from sepsis. Recommendations depend on the presence and number of high, moderate to high and low risk criteria.

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What are the effects of early versus late initiation of empiric antimicrobial treatment in children with or at risk of developing sepsis or severe sepsis?[55]

This table is a summary of the analysis reported in a guideline (underpinned by a systematic review) that focuses on the above important clinical question.

View the full source guideline (https://www.nice.org.uk/guidance/ng51/evidence)

Evidence C *	Confidence in the evidence is very low or low where GRADE has been performed and the intervention may be more effective/beneficial than the comparison for key outcomes, however this is uncertain and new evidence could change this in the future.

Population: Children with or at risk of developing sepsis or severe sepsis
Intervention: Early initiation of empiric antimicrobial treatment
Comparison: Late initiation of empiric antimicrobial treatment

Outcome	Effectiveness (BMJ rating)†	Confidence in evidence (GRADE)‡
<1 hour versus >1 hour [a]		
Paediatric Intensive Care Unit (PICU) mortality	No statistically significant difference	Very Low
<2 hours versus >2 hours [a]		
PICU mortality	No statistically significant difference	Very Low
<3 hours versus >3 hours [a]		
PICU mortality	Favours intervention	Very Low
<4 hours versus >4 hours [a]		
PICU mortality	Favours intervention	Very Low

Recommendations as stated in the source guideline
For children aged 5–11 years who have suspected sepsis and 1 or more high-risk criteria, give a broad-spectrum antimicrobial [b] at the maximum recommended dose without delay (within 1 hour of identifying that they meet any high-risk criteria in an acute hospital setting).

Note
The guideline group noted that the direct evidence in children came from one small (n=130), single-centre retrospective study of children in PICU with severe sepsis and septic shock. Therefore, they also extrapolated from the indirect evidence in adults to make the same recommendation for all age groups (including children aged under 5 years and 5-11 years).

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[a] Time from sepsis recognition to initial treatment and first appropriate treatment.

[b] See full guideline for more information.

* Evidence levels

The Evidence level is an internal rating applied by BMJ Best Practice. See the EBM Toolkit (https://bestpractice.bmj.com/info/evidence-tables/) for details.

Confidence in evidence

A - High or moderate to high

B - Moderate or low to moderate

C - Very low or low

† Effectiveness (BMJ rating)

Based on statistical significance, which demonstrates that the results are unlikely to be due to chance, but which does not necessarily translate to a clinical significance.

‡ Grade certainty ratings

High	The authors are very confident that the true effect is similar to the estimated effect.
Moderate	The authors are moderately confident that the true effect is likely to be close to the estimated effect.
Low	The authors have limited confidence in the effect estimate and the true effect may be substantially different.
Very Low	The authors have very little confidence in the effect estimate and the true effect is likely to be substantially different.

BMJ Best Practice EBM Toolkit: What is GRADE? (https://bestpractice.bmj.com/info/toolkit/learn-ebm/what-is-grade/)

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Key articles

- Riddle MS, DuPont HL, Connor BA. ACG clinical guideline: diagnosis, treatment, and prevention of acute diarrheal infections in adults. Am J Gastroenterol. 2016 May;111(5):602-22. Full text (https://journals.lww.com/ajg/fulltext/2016/05000/acg_clinical_guideline__diagnosis,_treatment,_and.14.aspx) Abstract

- World Gastroenterology Organisation. Acute diarrhea in adults and children: a global perspective. 2012 [internet publication]. Full text (http://www.worldgastroenterology.org/guidelines/global-guidelines/acute-diarrhea/acute-diarrhea-english)

- Shane AL, Mody RK, Crump JA, et al. 2017 Infectious Diseases Society of America clinical practice guidelines for the diagnosis and management of infectious diarrhea. Clin Infect Dis. 2017 Nov 29;65(12):e45-80. Full text (https://academic.oup.com/cid/article/65/12/e45/4557073) Abstract

- McDonald LC, Gerding DN, Johnson S, et al. Clinical practice guidelines for clostridium difficile infection in adults and children: 2017 update by the Infectious Diseases Society of America (IDSA) and Society for Healthcare Epidemiology of America (SHEA). Clin Infect Dis. 2018 Mar 19;66(7):e1-48. Full text (https://academic.oup.com/cid/article/66/7/e1/4855916) Abstract

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Figure 1 – BMJ Best Practice Numeral Style

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