An overview of gastrointestinal illness in children

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Abstract
This article aims to provide a concise overview of gastrointestinal (GI) conditions in children. GI conditions in children can be classified into various disease groups. This article is designed to focus on the functional, organic and infectious gastrointestinal conditions that are prevalent in children of all age groups. GI conditions in children result in emotional and physical pain to the child and tremendous distress to the caregiver. A review of GI conditions in recent and established research reveals novel methods of diagnosing and managing such conditions. Included in the article is up-to-date diagnostic criteria and management of functional gastrointestinal conditions (FGIDs) as well as information on recent treatment options for infectious gastrointestinal conditions, as adapted from the Essential Medicines List (South Africa).

Introduction
Infants and children are predisposed to various gastrointestinal conditions throughout their lives. It is estimated that more than 50% of all children will experience some form of gastrointestinal condition or disturbance within the first few months of life. Conditions that clinicians find daunting to diagnose include FGIDs, which show no structural or biochemical abnormalities. These conditions have prompted the development of consistently updated guidelines that are used by clinicians universally in diagnosing infants and adolescents. Other childhood conditions include organic and infectious diseases. Functional diseases are complicated by an incomplete pathophysiological understanding of the diseases, which makes them challenging to treat. On the contrary, organic conditions are more readily diagnosed due to the presence of structural and/or biochemical abnormalities. Established tests, rather than symptomatic diagnosis, can be used by clinicians to effectively diagnose these morbid conditions.

In developing countries one of the leading causes of death in children under the age of five years is diarrhoea, mainly due to dehydration and electrolyte loss in liquid stools. In South Africa (SA) and Africa, 19% and 46%, respectively, of deaths in children younger than five years are due to diarrhoea. The majority of diarrhoeal cases in SA are caused by bacterial or viral pathogens; in HIV-infected individuals it is more likely to be due to a protozoan pathogen. It is believed to be the second leading cause of death in children under the age of five, accounting for 9.2% of deaths globally. In 2012 a study was conducted by the National Burden of Disease where it was found that diarrhoea accounted for 3.7% of total years of healthy lives lost, being the third leading cause of death after Human Immune Deficiency Virus (HIV) and low birth weight in SA. Deaths in children due to diarrhoea occurred mostly (80%) in children under the age of two, and, in developing countries, children younger than three years old experience diarrhoea on average three times a year. In SA, statistics according to the General Household Survey (GHS) recorded deaths due to diarrhoea in children under five years to be between 8–13%.

Functional gastrointestinal disorders (FGIDs) in children
In infants and toddlers, the occurrence of FGIDs is frequently observed. In the first few months of life FGIDs can occur in up to 50% of paediatric patients. FGIDs include a variable combination of often age-dependent, chronic or recurrent symptoms not explained by structural or biochemical abnormalities. They are multifactorial conditions that have been observed to be caused by several varied pathophysiological mechanisms which include altered motility, visceral hyperalgesia, brain-gut disturbance, genetic, environmental and psychological factors. The clinical expression of an FGID varies with age and depends on an individual’s stage of development, particularly with regard to physiological, autonomic, affective and intellectual development. Although in most cases GI symptoms are transient and with spontaneous resolution in infancy, multiple dietary changes and pharmacological therapies are often initiated despite the lack of evidence-based data.

The Rome diagnostic criteria
The Rome criterion are a set of criteria which provides symptom-based guidelines by which child and adolescent FGID can
be diagnosed. This classification highlighted that patients report symptoms despite the lack of chemical, radiological or physiological abnormalities. The Rome classification scheme was initiated in 1988 when a group of international experts met in Rome. The primary reason for the convention was to categorise FGIDs using a symptom-based classification scheme. This eventually led to the inception of Rome I in 1992. The success and effectiveness of Rome I was assessed several years later which led to the publishing of Rome II in 1999. In one of the studies conducted, the diagnostic accuracy of the Rome I criteria was evaluated in 339 irritable bowel syndrome patients. The study reported that the diagnosis of FGIDs using Rome I had a sensitivity of 85% and a specificity of 71%. In 2006 Rome III was published. The most memorable recommendation made in Rome III was that there should be “no evidence” for organic disease which may have prompted a focus on testing. This means that an organic disease had to be tested for and ruled out in order to diagnose and classify a disease as an FGID. In 2016, Rome IV was published in which a recommendation was made based on reviewed literature and compiled evidence which stated that there was no need to test for organic diseases. FGIDs can be diagnosed based on symptom-based evidence. If the clinician finds the symptom presentation of a patient consistent with FGID diagnostic criteria, the patient may be classified as suffering from an FGID and be treated accordingly.

**Functional gastrointestinal disorders in infants and toddlers**

The current absence of specific biological markers for FGIDs has created a predicament for clinicians in the diagnosis and treatment of FGIDs. The diagnosis of FGIDs is clinically based on a set of symptoms with the exclusion of warning signs. Table I was designed by the Rome foundation to clinically diagnose FGIDs without the need to perform any tests. The table shows the

<table>
<thead>
<tr>
<th>FGID</th>
<th>Rome IV diagnostic criteria</th>
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<tbody>
<tr>
<td><strong>G1. Infant regurgitation</strong></td>
<td>Must include both of the following in otherwise healthy infants three weeks to 12 months of age: 1. Regurgitation two or more times per day for three or more weeks 2. No retching, haematemesis, aspiration, apnoea, failure to thrive, feeding or swallowing difficulties, or abnormal posturing</td>
</tr>
<tr>
<td><strong>G2. Rumination syndrome</strong></td>
<td>Must include all of the following for at least two months: 1. Repetitive contractions of the abdominal muscles, diaphragm, and tongue 2. Effortless regurgitation of gastric contents, which are either expelled from the mouth or re-chewed and re-swallowed 3. Three or more of the following: a. Onset between three and eight months b. Does not respond to management for gastroesophageal reflux disease and regurgitation c. Unaccompanied by signs of distress d. Does not occur during sleep and when the infant is interacting with individuals in the environment</td>
</tr>
<tr>
<td><strong>G3. Vomiting syndrome</strong></td>
<td>1. Two or more periods of unremitting paroxysmal vomiting with or without retching, lasting hours to days within a six-month period 2. Episodes are stereotypical in each patient 3. Episodes are separated by weeks to months with return to baseline health between episodes of vomiting</td>
</tr>
<tr>
<td><strong>G4. Infant colic</strong></td>
<td>For clinical purposes, must include all of the following: 1. An infant who is &lt; 5 months of age when the symptoms start and stop 2. Recurrent and prolonged periods of infant crying, fussing, or irritability reported by caregivers that occur without obvious cause and cannot be prevented or resolved by caregivers 3. No evidence of infant failure to thrive, fever, or illness</td>
</tr>
<tr>
<td><strong>G5. Functional diarrhoea</strong></td>
<td>Must include all of the following: 1. Daily painless, recurrent passage of four or more large, unformed stools 2. Symptoms last more than four weeks 3. Onset between six and 60 months of age 4. No failure to thrive if caloric intake is adequate</td>
</tr>
<tr>
<td><strong>G6. Diagnostic criteria for infant dyschezia</strong></td>
<td>Must include: 1. An infant &lt; 9 months of age 2. At least 10 minutes of straining and crying before successful or unsuccessful passage of soft stools 3. No other health problems</td>
</tr>
<tr>
<td><strong>G7. Functional constipation</strong></td>
<td>Must include one month of at least two of the following in infants up to four years of age: 1. Two or fewer defecations per week 2. History of excessive stool retention 3. History of painful or hard bowel movements 4. History of large-diameter stools 5. Presence of a large faecal mass in the rectum In toilet-trained children, the following additional criteria may be used: 6. At least one episode/week of incontinence after the acquisition of toileting skills 7. History of large-diameter stools that may obstruct the toilet</td>
</tr>
</tbody>
</table>
latest Rome IV classification which was updated and adopted to be used by all clinicians that face the predicament of diagnosing and treating FGIDs.²

It is essential for general practitioners and paediatricians to have significant knowledge and understanding of FGIDs. This will assist them in the selection and exclusion of pharmacological and non-pharmacological management of these frequently occurring conditions.¹ It is beneficial that pharmacological treatment is not offered to all patients as there is minimal evidence of the effectiveness of such in infants and adolescents.² There are different approaches to managing FGIDs which may include biological management, as well as the need for a biopsychosocial approach. According to this model, symptoms are both physiologically multi-determined and modifiable by sociocultural and psychosocial influences.¹ ¹² Table II shows the management of FGIDs especially in infants, and the outcome of specific management approaches.

**The prevalence of FGIDs in children**

Meta-analysis studies have reported a high prevalence of FGIDs in children and adolescents. Studies from the USA, Germany, United Kingdom, China, Sri Lanka, El Salvador, Panama, Ecuador and Colombia have found a high prevalence of FGIDs in school-age children (range 20–29%).¹³ Even though there is a high prevalence of FGIDs in children, they are not dangerous when the symptoms and caregiver’s concerns are managed and attended to.¹ Several FGID studies have been conducted in children and adolescents but there are only two studies to date that have used the Rome III criteria to diagnose FGIDs in toddlers and infants.¹³ Diagnosing symptoms in infants and toddlers is a daunting task as, in their early years, children cannot accurately report symptoms such as nausea or pain; the infant and pre-school child cannot discriminate between emotional and physical distress.² ¹³ Incorrect diagnosis and inappropriate treatment of functional symptoms could be the cause of needless physical and emotional suffering.² ¹³ To avoid this, clinicians need to accurately interpret the reports of the parents, who are more familiar with their children’s behaviour.¹ ² ¹³

**Organic gastrointestinal conditions in children**

The term *organic* disease is used as a broad term to classify a variety of illnesses. It can be used to classify diseases that affect a specific part of the body, or those that affect numerous organ systems.⁴ Organic disease is used to describe observable and measurable disease processes such as inflammation or tissue damage; such diseases may be inherited or may be influenced by external factors.⁵

**Lactase deficiency**

In most cases, the carbohydrates that we ingest are still in their disaccharide form. One such disaccharide that tends to cause most gastrointestinal distress is lactose. It is broken down to glucose and galactose by the enzyme lactase.¹⁴ Once lactase is produced it moves to the microvilli, and breaks down the lactose into simple carbohydrates that can be absorbed by the intestines.¹⁴ The amount of lactase produced is highest in infants and decreases as we grow older.¹⁴ The normal flora in the intestines ferment the lactose producing methane, hydrogen ions and carbon-

### Table II. Prevalence and treatment of functional gastrointestinal disorders in neonates and toddlers¹

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Age</th>
<th>Prevalence (%)</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant regurgitation</td>
<td>Three-weeks to 12 months</td>
<td>41–67 (peak at four months of age)</td>
<td>Education, smaller feedings, feeding thickening, positioning</td>
<td>Resolves in 90% of cases by 12 months of age</td>
</tr>
<tr>
<td>Infant rumination syndrome</td>
<td>Three to eight months</td>
<td>1.9</td>
<td>Behavioural interventions, improved nurturing</td>
<td>Recovery with nurturing</td>
</tr>
<tr>
<td>Cyclic vomiting syndrome</td>
<td>Wide range</td>
<td>3.4</td>
<td>Prevention of triggers, prophylactic medications, abortive medications, supportive measures</td>
<td>Usually resolves as child gets older but may continue or change to abdominal migraine or migraine headache</td>
</tr>
<tr>
<td>Infant colic</td>
<td>Early infancy to five months</td>
<td>5–19</td>
<td>Reassurance</td>
<td>Resolves by five months of age</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>No evidence that pharmacological interventions are useful</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>There is inadequate evidence whether elimination of cow’s milk protein, probiotics, or herbal interventions provide viable and effective treatments</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>These approaches remain problematic and controversial</td>
<td></td>
</tr>
<tr>
<td>Functional diarrhoea</td>
<td>6–60 months</td>
<td>6–7</td>
<td>Education, dietary changes</td>
<td>Usually resolves by 60 months of age</td>
</tr>
<tr>
<td>Infant dyschezia</td>
<td>Birth to nine months</td>
<td>2.4</td>
<td>Education and reassurance, avoidance of anal stimulations and laxatives</td>
<td>Resolves in most cases by nine months of age</td>
</tr>
<tr>
<td>Functional constipation</td>
<td>Birth to adulthood</td>
<td>3–27</td>
<td>Education, behavioural interventions, laxatives</td>
<td>Successful long-term treatment in 80% after first year, and increases over time</td>
</tr>
</tbody>
</table>
dioxide which are responsible for producing symptoms of gas and bloating. Along with these gases, short-chain fatty acids are produced e.g. propionate, acetate and butyrate.15 Both the lactose and the products of fermentation increase osmotic pressure, which drives water into the intestines causing diarrhoea. In rare cases lactase deficiency may be congenital16; this is an autosomal recessive disorder in which infants cannot break down lactose, have problems with breast milk and develop diarrhoea from birth.14 Sometimes infection or inflammation of the small intestines may lead to a temporary lactose intolerance due to insult of the microvilli; however once the small intestines heal everything normalises.14

**Dysphagia**

Dysphagia is derived from the Greek word meaning “disordered eating”. It refers to difficulty in eating as a result of disturbances in the swallowing process.17 Dysphagia can pose a serious risk to health as it can increase the risk of developing aspiration pneumonia, malnutrition, dehydration, weight loss and airway obstruction.17 In children, prematurity and impaired neurological development (e.g. cerebral palsy) are common causes of dysphagia. Symptoms include but are not limited to the following: choking or coughing with swallowing, food stuck in the throat, recurrent pneumonia, nasal regurgitation and unexplained weight loss.19 In most cases patients with dysphagia may not ingest fluids, which puts them at risk of dehydration.15 These patients' hydration status must therefore be closely monitored. Treatment goals for managing dysphagia are to maintain adequate nutrition and to protect the airway. Medications used in the treatment of dysphagia include botulinum toxin type A, diltiazem, nitrates and cysteine-depleting therapy with cysteamine.19

**Inflammatory bowel disease (IBD)**

Inflammatory bowel disease consists of two conditions: Crohn's disease which may affect any part of the GI tract causing potential scarring or strictures; and ulcerative colitis, which is confined to the large intestines.20 Crohn's disease affects 58/100 000 children in the United States. Crohn's disease causes vitamin (including fat soluble vitamins) and mineral malabsorption, resulting in deficiency in vitamins K, D and E, and iron, folate, calcium and vitamin B12.20

Ulcerative colitis has a lower prevalence than Crohn's disease, with one-third of children with ulcerative colitis having at least one severe attack before reaching the age of 15.21 It is reported that more than half of paediatric patients suffering from ulcerative colitis will present with weight loss. Many of the patients will have multiple macro- and micronutrient imbalances due to loss/deficiency in magnesium, zinc, and potassium.21 To date there is no cure for IBD. Pharmacological treatment is aimed at inducing remission of the disease, maintaining remission, minimising the side-effects of the prescribed medication and improving quality of life. Therapeutic preparations include mesalamine preparations, corticosteroids, immunomodulatory therapy, biological agents and anti-α₄ therapy.22

**Coeliac disease**

Coeliac disease is an autoimmune disorder that occurs in genetically susceptible patients.23 It is triggered by a well-identified environmental factor (gluten and related prolamins that are found in wheat, rye, and barley).23 The disease normally affects the small intestines, where it progressively leads to flattening of the small intestinal mucosa. Paediatrics present with abdominal distention (bloating), diarrhoea and failure to thrive.24 Immunoglobulin A (IgA) for both transglutaminase antigen (tTg) and endomysial tTg can be effective ways to determine if a patient has coeliac disease, especially in severe cases.24 Symptoms resolve when patients adopt a gluten-free diet. Even though patients may adopt a gluten-free diet, some patients are at risk of developing a refractory disease like small bowel cancer or T-cell lymphoma due to chronic inflammation or immune activation over a period of time.25 Avoiding food products that contain gluten is the cornerstone of treatment in patients with coeliac disease. Corticosteroids are used to control severe symptoms.21,26 Corticosteroids may also be used in cases where the disease does not respond to dietary modifications.26

**The incidence of infectious diarrhoea in children**

Diarrhoea in SA is mostly caused by bacterial or viral pathogens and in HIV-infected individuals it is more likely to be due to a protozoan pathogen. In the winter months, there seems to be an increase due to the rotavirus, whereas in summer bacterial enteropathogens are more common. Rotavirus is the leading cause of diarrhoea in children under the age of five years in both developed and developing countries.6

Viral pathogens such as the rotavirus are responsible for 70–80% of all acute diarrhoeal episodes and the remaining causes of acute diarrhoea become difficult to diagnose due to the self-limiting nature of the disease. In both of these cases the use of antibiotics is not advised.27

Diarrhoea caused by *Escherichia coli* is usually self-limiting within a couple of days of treatment with oral rehydration solution (ORS) and not antibiotics – contrary to popular belief. Antibiotic use in diarrhoea is mainly recommended for dysentery due to the *Shigella* species or infection caused by *Campylobacter bacteria or Vibrio cholerae*.28 Figure 1 depicts the most common causes of diarrhoeal disease in children of all ages.27

**Preventative measures to minimise the incidence of infectious diarrhoea**

The following options are available:

**Vaccination**

The administration of the rotavirus vaccine was introduced into SA’s national extended programme for immunisation (EPI-
SA) in August 2009, making SA the first country in Africa where national implementation is supported by government structures. The impact of the rotavirus vaccine in paediatrics has shown a remarkable reduction in death associated with diarrhoea caused by the rotavirus, due to the reduction in prevalence and frequency of rotavirus-associated diarrhoea.\textsuperscript{29,30} The rotavirus vaccine should be administered to children at ages six weeks and 14 weeks, and it is of utmost importance that both these doses are received at the correct time.\textsuperscript{5}

**Breastfeeding**

Exclusive breastfeeding has been proven to protect the infant against environmental contaminations and intestinal infections, which could lead to diarrhoea.\textsuperscript{31} The occurrence of paediatric diarrhoea is reduced in breastfed infants as compared to those not exclusively breastfed.\textsuperscript{32} Breastfeeding also reduces the frequency of infants being exposed to contaminated fluids and foods.\textsuperscript{32}

**Sanitation**

The provision of safe and clean drinking water, adequate sanitation, education on hand-washing routines before and after food preparation, and safe disposal of waste plays a role in reducing the occurrence of childhood diarrhoea.\textsuperscript{33} Adequate information and education should be given to communities on ensuring that water sources, food sources and general sanitation do not put the lives of paediatrics at risk of contracting illness. Hand-washing practices using soap have been proven to reduce microorganism levels on hands to close to zero and reduce the transmission of faecal-oral microorganisms on hands.\textsuperscript{34}

**The management and treatment of infectious diarrhoea in children**

Guidelines from the WHO and SA on the treatment of diarrhoea both highlight hydration as being the most important aspect when treating paediatrics. Dehydration is the most common cause of death in children under the age of five years who have been diagnosed with diarrhoea.\textsuperscript{3} The level of dehydration must be identified immediately and hydration intervention given according to the severity thereof, with the first-line therapy being ORS.\textsuperscript{35} Table III describes the different classifications, description and treatment of paediatric diarrhoea. This is in accordance with the principles stated by the South African standard treatment guidelines.\textsuperscript{35}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{diarrhoea_diagram.png}
\caption{The microorganisms depicted in this figure are the most common causes of diarrhoea in children. The leading cause is rotavirus, followed by adenovirus. The other common causes are food-borne, with the most common being \textit{Staphylococcus aureus}.\textsuperscript{27}}
\end{figure}
As depicted in Figure 2, Vitamin A supplemented with magnesium, iron, copper and zinc should be given to a child over the age of one year with persistent diarrhoea: Vitamin A is one of the essential vitamins to be administered in paediatrics with diarrhoea; infants 6–11 months: 100 000IU and children 12 months to five years: 200 000IU.5

Table III. Treatment steps for dehydration in paediatrics presenting with diarrhoea35

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>No visible dehydration Plan A</td>
<td>Only one or no other sign of dehydration.</td>
<td>Educate caregiver on how to give ORS with a spoon for children &lt; two years old in frequent small sips, or in frequent small sips from a cup in a child that is able to hold a cup. ORS should be given 10 ml/kg after each loose stool until diarrhoea stops. 50–100 ml should be given in children ≤ two years of age. 100–200 ml should be given in children &gt; two years of age. Caregivers should be encouraged to continue to feed the child especially children still being breastfed. Educate caregivers on how to make ORS/SSS at home in order to continue treatment.</td>
</tr>
<tr>
<td>Some dehydration Plan B</td>
<td>Two of the dehydration signs: Irritability or restlessness Sunken eyes Thirsty and drinks eagerly Moderate decrease in skin turgor by slow pinch, returning in ≥ two seconds</td>
<td>Give ORS 80 ml/kg over four hours or 5 ml/kg every 15 minutes. Give more if child wants more. Educate caregiver on how to give ORS with a spoon for children &lt; two years old in frequent small sips, or in frequent small sips from a cup in a child able to hold a cup. If vomiting occurs wait 10 minutes then continue slowly. Caregivers should be encouraged to continue to feed the child especially children still on breastfeeding. If, after four hours: There are no signs of visible dehydration treat as “no visible dehydration” Still some dehydration signs, continue as usual Signs of severe dehydration, treat as “severe dehydration”</td>
</tr>
<tr>
<td>Severe dehydration Plan C</td>
<td>Two of the dehydration signs: Lethargic or unconscious Sunken eyes Drinks poorly or unable to drink Severe decrease in skin turgor, skin pinching returning ≥ two seconds</td>
<td>Rapid administration of NaCl 0.9% IV 20 ml/kg. Give bolus of 10 ml/kg over 10 minutes if there are signs of acute severe malnutrition. If radial pulse is weak or undetectable, administration should be repeated twice. Continue with 20 ml/kg every hour for the next five hours. Refer urgently for further treatment of 20 ml/kg for another five hours, unless child is classified as “some dehydration”. Reassess every two hours while awaiting transfer. Give IV fluids more rapidly if hydration status does not improve. Give ORS oral 5 ml/kg/hr as soon as the child can drink, usually 3–4 hours for infants and 1–2 hours for children. If IV administration is not possible, insert a nasogastric tube while waiting or during urgent transfer giving ORS nasogastrically 20 ml/kg/hr over the next six hours. If oral administration is not possible, or condition improving transfer child urgently, and while waiting and during transfer give ORS oral 20 ml/kg/hr. Reassess and reclasify the child every four hours and if improvement is evident, reclasify child as “some dehydration”.</td>
</tr>
</tbody>
</table>

(Adapted from the Essential Drugs List, 2018)

Persistent diarrhoea35

Rehydration is the first point of care for the treatment and prevention of dehydration. It is important to assess if the patient should be treated according to Plan B or C with ORS and zinc.

The HIV status of the patient should be determined and caregiver(s) should be educated on the importance of adequate feeding:

- Breastfed infants should be fed more frequently and for longer periods.
- Instances where replacement feeding is used, normal milk should be substituted with breast milk where possible or with fermented milk products e.g. maas or yoghurt.
- Solid foods should be given in small quantities and more frequently, up to six meals a day.

As depicted in Figure 2, Vitamin A supplemented with magnesium, iron, copper and zinc should be given to a child over the age of one year with persistent diarrhoea: Vitamin A is one of the essential vitamins to be administered in paediatrics with diarrhoea; infants 6–11 months: 100 000IU and children 12 months to five years: 200 000IU.5

Figure 2. This depiction shows which vitamins and minerals should be administered to a child with persistent diarrhoea. They include vitamin A, magnesium, iron, copper and zinc.1
**Acute inflammatory diarrhoea/dysentery**

Rehydration remains the most important aspect in treating a patient with diarrhoea. The patient is assessed and treated according to Plan A, B or C with the initiation of zinc therapy. Nutritional support is essential for acute and persistent diarrhoea. Loperamide administration to slow down gastric motility is a total contraindication as it may cause toxic megacolon.

One of the most important factors is to determine the causative agent of the patient’s condition. Stool sampling should be taken in order to identify the causative microorganism and sensitivity to antimicrobials. The most likely strain causing dysentery in paediatrics is *Shigella*. The treatment is as follows: ciprofloxacin 15 mg/kg twice a day for five days and ceftriaxone 50–80 mg/kg/day in cases of severe infection.

**Cholera**

Due to very watery stools being present, severe dehydration can occur. The first step is to aggressively address the hydration status of the child and treat according to Plan A, B or C. Zinc therapy should be initiated. Antimicrobial therapy is as follows: ciprofloxacin 20 mg/kg as a single dose should be given orally.

**Cryptosporidiosis diarrhoea**

Hydration remains the most important aspect in treating a patient with diarrhoea. The patient should be assessed and treated according to Plan A, B or C and zinc therapy should be initiated. Nutritional support is essential as for acute and persistent diarrhoea.

Being an AIDS-defining disease, there is no specific antimicrobial therapy, but it responds well to antiretroviral therapy (ART). Treatment is as follows: loperamide 4 mg orally immediately followed by 2 mg up to four times in one day.

**Antibiotic-associated diarrhoea**

The first step is to discontinue the antibiotic causing the diarrhoea. Hydration remains the most important aspect in treating a patient with diarrhoea. The patient should be assessed and treated according to Plan A, B or C and zinc therapy should be initiated. Nutritional support is essential as for acute and persistent diarrhoea. The use of loperamide is contraindicated due to the possibility of causing toxic megacolon due to decreased gastric motility. Antimicrobial therapy: metronidazole 400 mg three times a day orally only when withdrawal of the offending antimicrobial does not stop the diarrhoea. Stool that contains traces of pseudomembranous colitis should be treated with vancomycin orally 125 mg every six hours; if there is no response add metronidazole after five days with the consultation of a specialist.

**Campylobacter species**

Antimicrobial therapy with erythromycin started more than four days after onset of symptoms appears to produce no benefit for the patient.

**Clostridium difficile**

The discontinuing of potential causative antibiotics is the first point of therapy. When discontinuation of causative antimicrobials is ineffective, oral metronidazole should be administered.

**Escherichia coli**

Treatment with trimethoprim/sulphamethoxazole (TMP-SMX) if diarrhoea is moderate or severe; for systemic complications, a parenteral second-generation or third-generation cephalosporin with rehydration remains first-line therapy for uncomplicated diarrhoea.

**Salmonella species**

Uncomplicated diarrhoea should be treated with rehydration solution, but antimicrobial therapy may be considered for infants younger than three months and for high-risk patients (e.g. those who are immunocompromised or have sickle cell disease); for drug-sensitive strains, ampicillin or alternatively, TMP-SMX fluoroquinolones, or third-generation cephalosporins may be considered.

**Shigella species**

Rehydration is essential in cases of non-complicated diarrhoea. No antimicrobial treatment is necessary for most mild infections, but for moderate-to-severe cases, ampicillin for drug-sensitive strains and TMP-SMX for ampicillin-resistant strains or in cases of penicillin allergy may be used; fluoroquinolones may be considered in patients with highly resistant organisms.

Treating persistent diarrhoea with antimicrobials has no effect and can be viewed as a contraindication, unless a non-intestinal infection such as pneumonia, sepsis or urinary tract infection is evident along with the diarrhoea, which would justify effective antimicrobial therapy. When an intestinal infection is evident, the appropriate antimicrobial therapy should be administered.

**Conclusion**

Gastrointestinal diseases in children are common and prevalent in significant percentages. The diagnosis and treatment may present a challenging predicament for clinicians. The ability to differentiate between functional, organic and infectious diseases is significant in determining the appropriate treatment for the patient. Most patients with functional diseases do not require pharmacological treatment. There is minimal evidence to support the initiation of pharmacological therapy in FGIDs. This is also observed in infections that are self-limiting and only require supportive therapy. Biopsychosocial management options have been used in FGIDs by clinicians and have shown to be beneficial in certain patients. It is therefore important for clinicians to always keep abreast with the development of the classifications of GI conditions, and the updates on the diagnosis and treatment of these conditions in order to improve the quality of life of children.