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Digestive tract neural control and gastrointestinal disorders in cerebral palsy

Liubiana A. Araújo,[1] Luciana R. Silva,[2] Fabiana A. A. Mendes[3]

Abstract

Objectives: To examine the neural control of digestive tract and describe the main gastrointestinal disorders in cerebral palsy (CP), with attention to the importance of early diagnosis to an efficient interdisciplinary treatment.

Sources: Systematic review of literature from 1997 to 2012 from Medline, Lilacs, Scielo, and Cochrane Library databases. The study included 70 papers, such as relevant reviews, observational studies, controlled trials, and prevalence studies. Qualitative studies were excluded. The keywords used were: cerebral palsy, dysphagia, gastroesophageal reflux disease, constipation, recurrent respiratory infections, and gastrostomy.

Summary of the findings: The appropriate control of the digestive system depends on the healthy functioning and integrity of the neural system. Since CP patients have structural abnormalities of the central and peripheral nervous system, they are more likely to develop eating disorders. These range from neurological immaturity to interference in the mood and capacity of caregivers. The disease has, therefore, a multifactorial etiology. The most prevalent digestive tract disorders are dysphagia, gastroesophageal reflux disease, and constipation, with consequent recurrent respiratory infections and deleterious impact on nutritional status.

Conclusions: Patients with CP can have neurological abnormalities of digestive system control; therefore, digestive problems are common. The issues raised in the present study are essential for professionals within the interdisciplinary teams that treat patients with CP, concerning the importance of comprehensive anamnesis and clinical examination, such as detailed investigation of gastrointestinal disorders. Early detection of these digestive problems may lead to more efficient rehabilitation measures in order to improve patients' quality of life.

J Pediatr (Rio J). 2012;88(6):455-64: Cerebral palsy, dysphagia, gastroesophageal reflux disease, constipation.

Introduction

Cerebral palsy (CP) is any disorder characterized by motor impairment secondary to non-progressive neuropathological abnormalities in the developing brain. It is a broad term, used for a variety of non-progressive motor signs, arising from an injury that prevents the full development of the central nervous system in utero, during birth or in the first years of life.[1]

CP is one of the three leading causes of neurodevelopment disability, the other two being autism spectrum disorder and mental retardation.[2] Motor manifestations may be

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associated with sensory symptoms, intellectual deficit, epilepsy, behavioral disorders and diseases of the digestive tract and other systems.[3]

CP is the most frequent cause of disability in childhood, affecting 2-2.8/1,000 live births.[4] CP has important effects on the functionality and quality of life of individuals and their families. Current data on the prevalence of CP in Brazil are unknown, but it is estimated that the incidence is high due to poor care provided to pregnant women and their newborns in many regions.[5] Estimates suggest that in Brazil there are approximately 30,000 to 40,000 new cases per year.[6]

Despite growing technology in perinatal intensive care, statistics until the year 2000 showed that the incidence and prevalence of CP were not decreasing, because, in parallel, there was an increase in the survival of very low birth weight preterm infants, in whom neurological disorders are more frequent.[7,8] Recent studies from developed countries showed that since 2000, the prevalence has remained stable, except for extreme preterm newborns.[9-12]

The term CP implies movement disorders, but the presence of other disorders should be investigated; among these stand out: cognitive, visual, hearing, language, sensory cortex, attention, alertness, and behavioral abnormalities, epilepsy, hormonal disorders, orthopedic conditions, gastrointestinal disorders, and growth retardation.

Children with CP may have gastrointestinal disorders and feeding difficulties that directly affect their growth and quality of life. Virtually all individuals with CP have some degree of digestive and nutritional status disorders at some point in their lives, since the neurological connection with the digestive tract is of paramount importance.[13]

The incidence and prevalence of feeding difficulties in these subjects are described in several studies in the literature. In children with CP, feeding difficulties are described in about 30 to 40%, and these are changed according to disease severity.[4,14]

Among the most frequent difficulties are dysphagia for solids and liquids, regurgitation and vomiting, prolonged time to offer a meal, and intestinal constipation. Sullivan et al. described that in 271 children with CP, 89% presented dysphagia, 28% prolonged time for feeding, 8% were gastrotomized, 22% presented frequent vomiting, and 26%, intestinal constipation.[15]

Abnormalities of the high gastrointestinal tract can trigger recurrent respiratory conditions, inadequate nutritional intake, nutritional deficit on the growth curve, greater number of hospitalizations and need for gastrostomy.[16,17]

These gastrointestinal disorders impact on the quality of life of children and their caregivers, also because of the stressor factor in the families. Based on these aspects, this review was designed with the aim to examine the physiopathology of neural control of the gastrointestinal tract and the main digestive manifestations in CP.

In recent years, evidence shows that the life expectancy of people with CP is increasing and, partly, it is due to improved nutritional care.[18,19] There is a clear need for an interdisciplinary team that is aware of these changes and that is qualified to perform the appropriate treatment of these conditions, preventing effects in nutritional status and health of these individuals.

Neural control of the digestive system

Adequate control of the digestive tract depends on the functioning and integrity of the central and peripheral nervous system.

Swallowing represents a neuromuscular synergistic action that involves food preparation, the formation of the bolus, oral transit and bolus propulsion through the pharynx with airway protection. It consists of sequentially and harmonically interrelated stages (Figure 1).

Swallowing is a complex process involving a total of 31 pairs of striated muscles, which are innervated by the following cranial nerves: trigeminal, facial, glossopharyngeal, vagus, accessory and hypoglossal.

The preparatory phase is controlled by the cerebral cortex and there is reflex participation from afferents departing from mechanical proprioceptors, which are sensitive to changes in pressure and deformation of the mucosa of the lips, cheeks, and tongue dorsum.[20,21] During this preparatory phase, trigeminal (V), facial (VII), glossopharyngeal (IX), and hypoglossal (XII) nerves are involved. The facial nerve participates in motor control of lips and cheeks, the muscle tone of the face and mouth, and the muscles that perform the opening and lateralization of the jaw (platysma and lateral pterygoid). Furthermore, it controls taste on the anterior 2/3 of the tongue. The trigeminal nerve acts in the muscles that close the jaw and grind foods (temporalis, masseter and medial pterygoid). The hypoglossal nerve controls the rotation and lateralization of the tongue. Concomitantly, the glossopharyngeal nerve that innervates the soft palate must keep it at rest to increase nasal airway and narrow the oropharyngeal passage, reducing the possibility of premature entry of food into the pharynx, in addition to controlling the palate in the posterior 1/3 of the tongue.[1,22,23]

In the oral phase, occur elevation and propulsion of food bolus, specially controlled by the genioglossus and styloglossus tongue muscles, which are innervated by cranial pair XII. The trigeminal performs occlusion of the anterior part of the oral cavity by contraction of the mylohyoid, generating a negative pressure that facilitates the propulsion of food.

In the transition between oral and pharyngeal phases, the swallowing reflex is triggered with interruption of breathing.

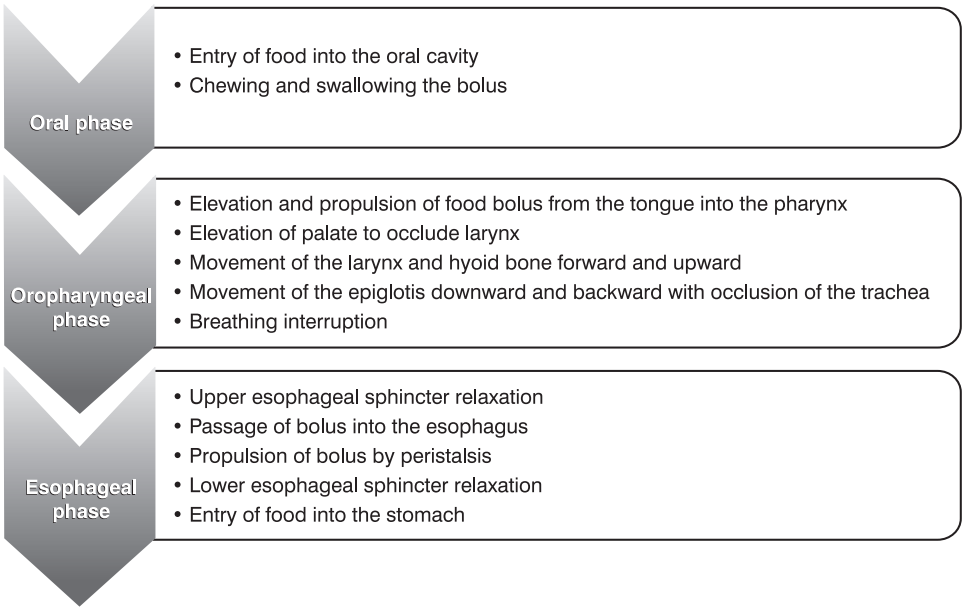


Figure 1 - Deglutition phases

The cough reflex that occurs to prevent aspiration of food is also controlled by the nervous system.

The pharyngeal phase is controlled by the trigeminal nerve (V), glossopharyngeal (IX), vagus (X), accessory (XI), and hypoglossal (XII), in addition to spinal segment C_1-C_3.

In esophageal phase, sensory and motor controls of peristalsis occur under the influence of the X nerve.

The swallowing coordination center is located in the lenticular nucleus, bulbar region.[24,25] The pyramidal system acts in the voluntary motor control, through the fibers originated in the cerebral cortex (corticospinal and corticobulbar tract). The extrapyramidal system participates in the coordination and harmonization of the automatic movements, providing the respective postural muscle adaptation.

The motor cortex is essential for oral motor skills, but it also participates in the mechanisms of swallowing. The basal ganglia integrate all cortical afferents and participate in the planning and initiation of movements and organization of associated postural adjustments and motor learning.[26] The cerebellum also participates in motor learning, since it compares motor orders generated with the muscle performance and corrects errors in advance through the mechanism of feedback.[27]

The distension of the stomach walls triggers a vagal reflex that results in contraction of the lower esophageal sphincter, preventing stomach contents from flowing back into the esophagus. There must be an adequate pressure of the lower esophageal sphincter and control of transient sphincter relaxation, in order to also avoid the pathological reflux. The control of esophageal peristalsis helps to regulate proper time of esophageal clearance, a measure that also has an important role to avoid irritation of the esophagus nerve endings when there is reflux of gastric acid. The control of the lower esophageal sphincter and esophageal peristalsis involves the solitary tract nucleus, which is also the swallowing center.[28]

Gastric emptying and motility of the foregut are also regulated by the vagus nerve.

The distension of the stomach and the duodenum by the entry of food is followed by gastrocolic and duodenocolic reflexes, which trigger colonic contractions of high amplitude intended to transport the fecal material into the colon.[29-31] Peristalsis of the descending colon, sigmoid, and rectum propel the feces. In the colon, there are segmentation

movements, whose purpose is to mix the colonic content, facilitating the absorption of water and nutrients; and propulsion, peristalsis mass movement, pushing the feces into the rectum for defecation.

In the intestine, the importance of the enteric nervous system stands out, which suffers the action of the autonomic nervous system, the presence of food, the action of gastrointestinal hormones, determining motility, digestion, absorption, and defecation.

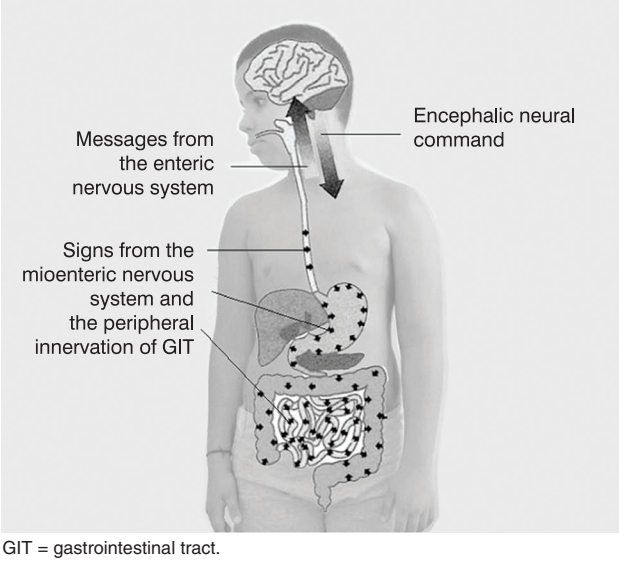
Defecation is initiated by an intrinsic and a parasympathetic reflex. The distension of the large intestine walls by feces stimulates nerve endings, and from there, nervous stimuli that propagate through the myenteric plexus are sent. This triggers increased peristalsis of the descending colon, sigmoid and rectum. As the peristaltic waves approach the anus, the inhibitory pulses of the myenteric plexus cause relaxation of the internal anal sphincter. When there is parallel relaxation of the external anal sphincter, the feces are expelled. This is the intrinsic reflex.

The parasympathetic reflex involves sending parasympathetic impulses by sacral segments of the spinal cord, providing more effective peristaltic waves to trigger defecation and a more efficient relaxation of the internal anal sphincter. The internal sphincter is formed by three circular muscles and its innervation occurs by the pelvic nerves, which are involuntary. The external sphincter is composed of a striated muscle, whose innervation is controlled voluntarily, except in the early years of life.

For the act of defecation, there is the involvement of the diaphragm, the muscles of the abdominal wall, the musculature adjacent to the perineum, and the muscles of the rectum and anus. After the entry of feces in the rectum, there is an increase in intraluminal pressure, and when it exceeds about 50 mmHg, occurs the relaxation of the internal anal sphincter, and the need to evacuate becomes conscious. When the conscious relaxation of the external anal sphincter occurs, peristalsis and contraction waveform propagate toward the rectosigmoid junction. There is contraction of the levator ani and the rectococcygeus muscles, and shortening of the rectal ampulla. In parallel, occurs the contraction of the muscles of the abdominal wall and chest wall, with flattening of the diaphragm. The intra-abdominal pressure rises from 100 to 200 mmHg, and the expulsion of fecal material occurs. Messages are sent to the brain for the voluntary control of defecation. Fecal continence and evacuations depend on the perfect functioning of the pelvic muscles.[31,32]

The center of defecation is present in the bulb, which performs its control and coordination. It is associated with a chemoreceptor trigger zone that is located on the floor of the fourth ventricle.

For these steps to occur throughout digestion, the integrity of all these routes is essential. As in individuals with CP lesions often compromise the cortical and subcortical regions responsible for the harmonious functioning of the digestive system (Figure 2), there are frequent changes at various times throughout those routes. There are other factors associated to these neurological changes that, together, make digestive manifestations frequent and multifactorial in these individuals. Next, the factors that are interrelated and are responsible for this etiology will be described.



GIT = gastrointestinal tract.

Figure 2 - Neural control of the digestive tract

Feeding difficulties

The difficulties that limit food intake in children with CP range from neurological immaturity to the interference with mood and preparation of their caregivers. Among the known factors, stand out[20,33]:

- positioning: inappropriate trunk stability, inappropriate cervical stability;
- eating disorder: hypotonia, suction disorder, absent tongue lateralization, persistent tongue thrust, poor lip closure, inefficient esophageal clearance, mastication disorder;
- abnormal postures: persistence of primitive reflexes (Moro, asymmetric tonic-neck), dystonic movements and choreoathetosis, opisthotonos, orofacial dyskinesia;
- neurological maturation: swallowing incoordination, "tonic bite", exacerbated gag reflex, gastroesophageal reflux, aspiration, sensory deficit;
- associated conditions: seizures, irritability, poor dental preservation, chronic pain, gastrointestinal discomfort by reflux/constipation, mental retardation, visual impairment;
- language delay: inability to express thirst, hunger, or food preferences;
- decreased activity: decreased appetite;
- motor impairment: inability to serve and to access food;
- use of medications: side effects, such as hypersecretion in airways, dry mouth, constipation, and decreased appetite;
- change in the consistency of food: offering liquid and/or pasty foods with possible loss of nutrients and calories during dilution, detailing of the food inventory;
- behavioral problems: food refusal, food selectivity, early satiety;
- interference of caregivers: stress, depression and tiredness, lack of guidance;
- financial difficulties in the family: no appropriate armchair/chair, lack of adequate tools, cost of food, cost of food thickener;
- lack of training of health professionals who deal with these children.

For the evaluation of a child with CP, anamnesis and a complete assessment covering all this information are essential.

Dysphagia

Dysphagia is a swallowing disorder that occurs due to the difficulty of transporting the bolus from the mouth into the stomach and that can be caused by neurological damage that interferes with swallowing dynamics. Neurogenic dysphagia is found in 58-86% of individuals with CP.[14,34,35] It is generally related to losses in growth curves, eating pleasure, associated respiratory infections, and hospitalizations, worsening psychomotor development and quality of life.[35-38]

Dysphagia in CP can be, among other causes, secondary to neurological changes that generate inadequate control of the oral, pharyngeal, or esophageal phases of swallowing. In addition to the changes in innervation and motor control already described previously, there are also changes in oral sensitivity and esophageal dysmotility.[36,39]

Feeding difficulties may case aspiration of food and liquids into the airways, which is a major cause of morbidity and mortality in CP.[15,40] The prevalence of aspiration in children with CP is high, of around 68-70%.[17]

Aspiration occurs when there is interference with the synchronism of the swallowing/breathing process. It is defined when there is passage of food inside the vestibule of the larynx, passing under the vocal cords and into the trachea and lungs.[41]

Dysphagia can be assessed by clinical and complementary methods. In clinical evaluation, weight, height, and anthropometric measures of the individual should be obtained. Spontaneous swallowing of saliva and intake of different food consistencies should be carefully observed, from thin and thickened, pasty liquids, to solids, in different volumes, with different instruments.

In this evaluation, dysphagia presents the following signs[42-44]:

- cough or hypoxemia during or after feeding;
- nasal regurgitation;
- extra-oral escape;
- poor oral motor skills;
- delay in pharyngeal response to trigger pharyngeal swallow reflex;
- multiple swallows;
- increased secretion in the airways;
- fatigue during or after feeding;
- stridor caused by fluid in the upper airways during or after feeding;
- apnea or dyspnea during feeding;
- changes in the respiratory rhythm;
- tearing eyes, throat clearing, grimacing;
- prolonged time for swallowing and eating the meal (45-60 min);
- residues in the oral cavity;
- tongue thrust;
- head tilt, opisthotonos, diplopia, halitosis.

On clinical examination there should be cervical auscultation with a stethoscope located in the larynx during and after swallowing as an auxiliary method for noninvasive detection of aspiration, besides saturimetry, where a drop of more than 5 % may be indicative of aspiration.[44]

There are scales to assess dysphagia that are used for individuals with mental or physical disability in general, such as the Dysphagia Severity Scale, but in Brazil there is no validated scale targeted for the evaluation of children with CP.[36,45]

Clinical examination may be supplemented by videofluoroscopy. This is a complementary method that visualizes the function of anatomical structures, their operation and the direction of the bolus during mastication and swallowing of different consistencies with contrast.[46,47] One of the main indications of this exam is the suspicion of silent aspirations.[48,49]

The treatment of dysphagia when oral and pharyngeal phases are mainly compromised, aims to establish the positioning and types of appliances, adjustment of food consistencies, indication of the food thickener and reduction of the fractional volume that is offered in each food supply. In those individuals where dysphagia occurs in all phases or determines aspirations, malnutrition, and recurrent respiratory infections, gastrostomy is indicated.[50]

Gastroesophageal reflux

Gastroesophageal reflux occurs when there is involuntary retrograde flow of gastric contents into the esophagus, which can de defined as infant regurgitation or gastroesophageal reflux disease (GERD).[51] GERD can be defined by regurgitation with symptoms and effects such as retrosternal pain, heartburn, post prandial irritability and during sleep; with negative consequences in nutritional assessment and growth, and with recurrent respiratory infections represented by symptoms of upper and low respiratory tract (ear infections, wheezing, cough, hoarseness, bronchitis, pneumonia). GERD may be primary, when associated with abnormal esophageal functioning. When associated with neurologic diseases and others, it is considered secondary, but also related to esophageal malformations, cystic fibrosis, postoperative upper gastrointestinal tract, and obesity.[52] GERD is described in about 20-90% of children with neurologic impairment.[53]

There are several reasons that cause GERD in CP: dysfunction of the neural control of esophageal peristalsis; abnormalities on the innervation of the lower esophageal sphincter, with consequent episodes of transient relaxation; prolongation of gastric emptying; prolonged supine position; increased intra-abdominal pressure secondary to scoliosis, spasticity, or constipation; seizures; medications; obesity and alteration of diet consistency, such as predominantly liquid or pasty.[54,55]

Children may present the following symptoms related to GERD[56-58]:

- regurgitations, with elimination of gastric content without effort;
- vomiting, with eliminations of higher amount of gastric content associated with the contraction of the abdominal muscles and effort;
- retrosternal pain, heartburn, crying, irritability and sleep disorders due to the presence of the acid content in the esophageal mucosa, with irritation of the nerve endings and esophageal mucosal injury;
- aspirations and respiratory infections, chronic night coughing, recurrent pneumonia, non-allergic bronchial hyperreactivity, asthma;
- upper gastrointestinal bleeding;
- chronic esophagitis and even esophageal stricture ulcer, Barrett's esophagus (replacement of esophageal squamous epithelium by intestinal columnar epithelium) and Sandifer's syndrome (which may be associated with severe esophagitis, rotational movements of the head and neck and anemia);
- food aversion;
- dental problems;
- worsening of dystonic movements and spasticity;
- growth deficit, anemia, and hypoproteinemia, due to associated losses and food aversion.

GERD may be diagnosed clinically, and some additional tests may be performed, such as upper digestive endoscopy with biopsy, esophageal pH monitoring, radiological examination of the esophagus, stomach, and duodenum, and impedance analysis, depending on the predominant symptoms of the individual. However, there is no gold standard test, and in CP professionals must consider the risk associated with sedation for gastrointestinal endoscopy, for instance.[51,59]

If there are clinical criteria suggestive of GERD, treatment should be instituted. Posture and diet measures related with volume and consistency are fundamental. Drug prescription of proton pump inhibitors or anti-H2 drugs may be indicated, and if there is clinical improvement, it reinforces diagnosis. Lately, the use of prokinetics is being questioned, and the evaluation of the gastroenterologist is indicated in these situations.[60]

In individuals with CP it has been observed that baclofen, a GABAergic medication prescribed as muscle relaxant to assist in dystonia and spasticity, has shown effect in reducing the frequency of episodes of regurgitation.[61] However, there is insufficient data on the use of this drug in order to treat GERD.

Another treatment option would be fundoplication in some cases, such as: severe esophagitis, individuals resistant

to medical treatment, patients with Barrett's esophagus, repeated respiratory aspiration, and cyanosis. In subjects with CP subjected to gastrostomy, fundoplication may also be recommended.[61]

Constipation

Constipation can be defined according to the Roma III criteria, and the diagnosis is established by the presence of two or more of the following symptoms: fewer than three evacuations per week; at least one episode of fecal incontinence weekly if the child already presents control of the sphincter; history of retentive behavior or excessive intentional fecal retention, history of painful defecations, painful or difficult bowel movements; presence of fecal mass in the rectum; history of large diameter stools that may obstruct the toilet.[62] There may be more effort than usual during the act of defecation, change in caliber and consistency, with elimination of parched or hard stools and pain, strain or bleeding during evacuation and presence of fecal soiling. The liquid content of the stool can be reduced to less than 70% of its weight in constipated individuals.[63]

Intestinal motility disorders associated with genetic, environmental, and psychological factors are responsible for functional constipation, in addition to dietary mistakes with low-fiber diet, low water intake and lack of physical activity. In CP, intestinal motility is altered throughout the entire colon due to neurological abnormalities. The following motility disorders can be described in CP[64]:

- abnormally high sphincter pressure;
- failure of internal anal sphincter relaxation after rectal distention;
- altered rectal sensibility;
- relaxation impediment or paradoxical contraction of the internal anal sphincter or puborectalis muscle;
- reduced colonic propulsion.

In individuals with CP, intestinal constipation is common. Its etiology depends on several factors that may be present and interrelated to each other. Some of the factors are related to[65,66]:

- lifestyle: children with CP are usually sedentary, with physical inactivity, there is a deficient intake of fiber and fluids due to dysphagia and difficulties related to the supply of the diet;
- change in neurological control: brain lesions involving cortical and subcortical regions, bulb, and others that are responsible for the harmonious functioning of the digestive system; decrease in the sensation of rectal filling and need for greater volumes of inflation for triggering the recto-anal reflex; lack of synchrony between central nervous system, autonomic nervous system and enteric nervous system;
- use of medications: anticonvulsants, combined antacids, inadequate laxatives;
- abnormalities of the structures of the intestine, rectum and anus: fecalomas with dilatation of the colonic wall and megacolon, anal fissure;
- emotional factors: stress, pain and suffering to evacuate, soiling;
- associated disorders: bone deformities, seizures that are difficult to control, mental retardation, malnutrition, hidroelectrolyte disturbances.

The longer the feces remain detained, the more parched and bulky they become, and they may form fecalomas. There may be distension of the bowel walls and, in the long term, decreased peristalsis, causing a vicious circle and further accentuating constipation. Consequences in children with CP determine various symptoms that interfere negatively with their quality of life. They can present a condition of chronic pain, bloating, crying and irritability, restless sleep, hemorrhoids, anal fissure, fecal soiling, discomfort, change in appetite and early satiety, mood shift, urinary tract infection, and aggravation of gastroesophageal reflux.[67,68]

The treatment of constipation in individuals with CP follows the guidelines of the treatment in general: disimpaction followed by dietary modifications, positioning, and, in some cases, use of medications. Dietary guidelines are performed with increasing intake of fiber and adequate fluid intake, but as the pathophysiology involves neurological changes, there may be little or no response with these measures. The use of rectal or oral laxatives may also be prescribed, as well as osmotic laxatives.[69,70]

Conclusion

Digestive manifestations are, thus, common in individuals with CP and are interconnected with each other (Figure 3). The most serious consequences are mainly nutritional deficits, respiratory infections, and recurrent hospitalizations, interfering with global health and neuropsychomotor development of these individuals.

The issues examined in this review are essential to emphasize proposals for interdisciplinary teams treating individuals with CP about the importance of a comprehensive anamnesis and detailed clinical examination including the investigation of associated gastrointestinal disorders.

The team that treats the child with CP should be interdisciplinary, due to the multiple interrelated needs of these individuals.

Anamnesis should include questions about all data described in this review that may suggest digestive diseases, besides dietary history (with detailed assessment on the intake of nutrients, fiber and water), degree of participation

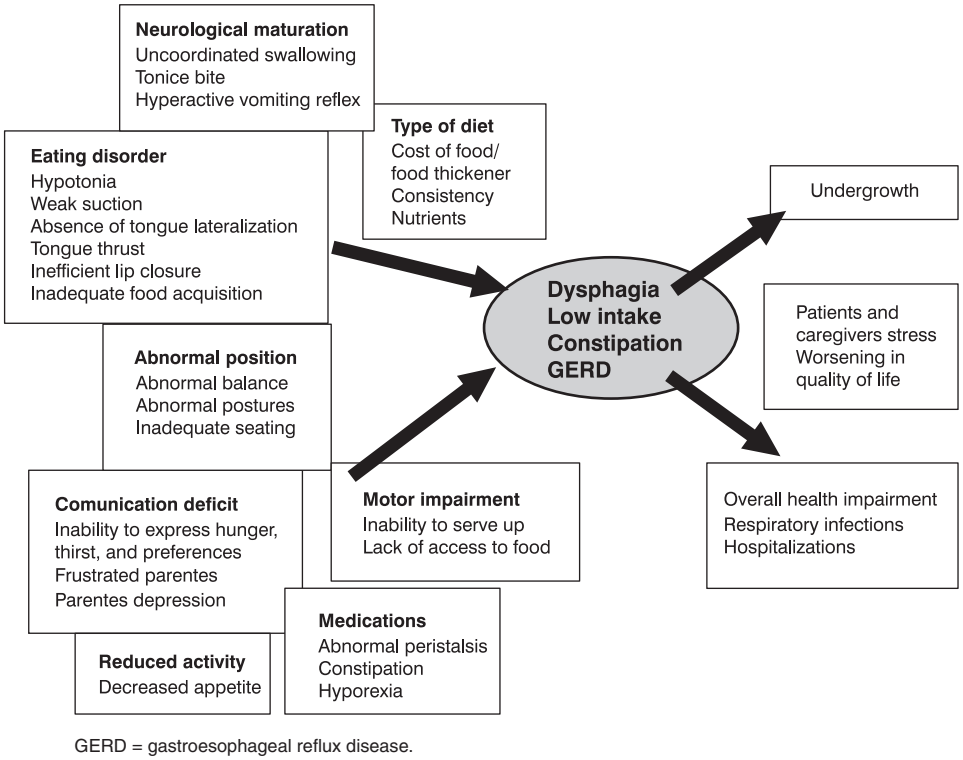


Figure 3 - Factors involved in the digestive abnormalities in cerebral palsy and its consequences

on activities of daily living, positioning during these activities, growth curves, use of medications, previous treatments of digestive abnormalities, and results, in addition to data about the caregiver (who is the main caregiver, which are the strategies used to manage these digestive abnormalities, what is their position concerning all those abnormalities and difficulties).

The general physical examination must consider all the peculiarities described and emphasize also adequate anthropometric data for the individual, suggestive signs of nutritional deficits, posture, deformities associated, and the type of abnormality in posture and movement.

Treatment should be directed and individualized, introduced as early as possible, taking into consideration the diagnosis and the overall socioeconomic, familiar, and cultural context of each individual. Training and emotional support should be provided for caregivers, in order to make these measures more effective.[68]

The sharing of knowledge and experience between each team that works with patients affected by CP may help in early detection of these digestive changes, in order to support early and effective rehabilitation measures and interdisciplinary treatment to improve the quality of life of these individuals and their families.

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