35
FEEDING AND NUTRITION

Valentina Intagliata and Richard Stevenson

Introduction

Cerebral palsy (CP) is the most common form of neurodevelopmental disability, with prevalence estimates of about 2.1 to 3.6 per 1000 children (Yeargin-Alssopp et al. 2008). CP is a clinical diagnosis based on the presence of impairment in movement and posture resulting from injury to the developing brain. However, various associated impairments commonly exist simultaneously. Among these, intellectual disability, seizures, and sensory impairment are important. Feeding disorder due to oral–motor dysfunction is one other associated impairment, affecting approximately 30% to 40% of children with CP at least to some significant degree (Sullivan et al. 2000, Fung et al. 2002). For some children, the consequence of oral–motor impairment may require gastrostomy tube feedings to maintain safe and effective nourishment; others may only have difficulty with certain textures of food, which can be overcome through dietary adaptation. The functional effect of these associated impairments can be so profound that the impact on daily activity and participation can be even greater than from the motor impairment of CP, depending on its severity.

In children with CP affected by feeding disorder, it is essential first to recognize that the problem exists. Next, it is important to perform a comprehensive assessment to identify contributing factors. These steps are crucial for informing an appropriate – and successful – management plan. Furthermore, a range of secondary conditions are frequently comorbid in these children, such as malnutrition, growth problems, osteopenia/osteoporosis, gastrointestinal reflux disease, constipation, chronic aspiration, and drooling. Not only are these conditions commonly present, but more importantly it is thought that feeding disorder may influence their development. Some of these secondary conditions may also further exacerbate the feeding disorder.

The relationship between feeding disorder, malnutrition, and poor growth is exceedingly complex. It is vital to pursue a better understanding of the associations between these issues. Feeding disorder clearly can lead to malnutrition, which can cause poor growth (Stallings et al. 1993a, b, Stevenson et al. 1994, Fung et al. 2002, Sullivan et al. 2002). Children with CP are generally shorter, smaller, and lighter than matched peers with typical development, and this growth disparity consists of differences in stature as well as body composition (Stallings et al. 1993a, Stevenson et al. 1994, 2006, Krick et al. 1996, Samson-Fang and Stevenson 1998, Fung et al. 2002, Henderson et al. 2002, Day et al. 2007). For typically developing children, growth and nutritional status are basic and central components of health and well-being. It is usually perceived that a child who grows normally is healthy, whereas a child who grows abnormally is not. However, for children with CP the causes of poor growth are usually multifactorial, including nutritional deficits, neuroendocrine abnormalities, psychosocial influences, and growth-promoting activity effects (Henderson et al. 2005, 2007, Stevenson 2007, Kuperminc et al. 2009). Thus, it can be difficult to identify whether poor growth indicates malnutrition, or whether it is a marker for the underlying CP and associated gross motor and activity deficits. It may be that growth is not the most important outcome, and that other measures of health and well-being are more revealing of the effects of adequate nourishment. For example, life expectancy is increasing in people with CP and data suggest that this may be in part related to improved nutritional care in recent years (Strauss et al. 2007).

This chapter provides an overview of feeding disorder in children with CP, and discusses the malnutrition and poor growth experienced by many of these children.

The first section begins with a description of normal feeding, and abnormal feeding in the context of neurological impairment. Next follows a summary of the issues related to malnutrition and poor growth in children with CP. The chapter culminates with details about proper assessment and management of feeding disorder, nutritional status, and growth.

Feeding in children with cerebral palsy

Anatomy and physiology of normal feeding

When infants with typical development and young children are learning to feed, it may appear simple and effortless. It is important to note that eating and feeding are different skills. The process of eating and swallowing is physiological and requires a series of complicated steps that must be well coordinated (Stevenson and Allaire 1996, Delaney and Arvedson 2008). The development of feeding skills occurs as a result of a complex interaction between the child's biology and behaviors, as well as the caregiver's responses to the child. This developmental feeding dynamic occurs within a larger sociocultural background. Thus, learning to feed is an intricate process involving the child's innate physiological skills and experiential

Mark

'Will he have gained weight this month? I do hope so.' The anxiety induced by regular attendances at the multi-disciplinary feeding clinic is a major source of stress for Mark's parents and for his maternal grandmother, who contributes significantly to his care.

Mark is now aged 3. He has bilateral dystonic cerebral palsy (CP) and is in Gross Motor Function Classification System (GMFCS) level V and Eating and Drinking Ability Classification System level IV. Oral feeding has always been prolonged and difficult. He has gastro-oesophageal reflux with vomiting. His length and weight are below the third centiles using charts for children with typical development. Videofluoroscopy has shown precarious swallowing with some aspiration of unthickened fluids. Yet Mark is healthy, alert, and socially responsive. His caregivers perceive that he enjoys being fed. At the clinic they anticipate again being advised to consider allowing Mark to undergo percutaneous gastrostomy insertion so that his oral intake can be supplemented.

They have met parents whose children have gastrostomies and who have benefitted. They have met other parents who have elected to continue feeding orally.

They have looked recently at growth charts for children with CP that have been published (Brooks et al. 2011) and have noted that children with CP who are in GMFCS level V are, historically, significantly smaller and lighter than either their uninjured peers or those with less severe motor disabilities than Mark. They are also aware from the literature that low weight and failure to thrive are adverse factors for long-term survival.

At the clinic, Mark's paediatrician has put time aside to discuss these issues with the parents.

learning, which arises from child–caregiver–environment interactions (Stevenson and Allaire 1996, Delaney and Arvedson 2008).

It is most useful to begin with a review of the anatomy involved in eating and swallowing because several notable differences exist between the adult and infant oral–pharyngeal structures (Figs 35.1 and 35.2) (Stevenson and Allaire 1996). In the infant, the tongue lies exclusively within the oral cavity, the larynx is high and anterior, and the tip of the epiglottis reaches to the soft palate (Kramer and Eicher 1993). Importantly, the position of the epiglottis serves to direct liquid away from the larynx and thus protect against aspiration. In addition, infant cheeks comprise dense fatty tissue to aid in sucking, but which also lead to crowding of the oral cavity (Bosma 1985). The resultant anatomical relationship is one in which the infant's tongue, soft palate, and arytenoid mass (arytenoid cartilage, false vocal cords, and true vocal cords) are large relative to the surrounding anatomical structures. However, this is ideal for the typically developing infant who feeds by sucking from the breast or bottle in a recumbent position (Bosma 1985, Kramer 1989).

As the infant grows, the oral cavity expands owing to physical enlargement of the head/neck, posterior descent of the larynx, and disappearance of the fatty cheek pads (Kramer and Eicher 1993, Stevenson and Allaire 1996). Generally, at about 6 months of age teeth erupt, which provide both mechanical and sensory functions (Delaney and Arvedson 2008). The appearance of dentition coincides with the child's increasing caloric needs, requiring the ability to bite and chew foods that are more energy dense. Although children's feeding skills are clearly influenced to some extent by the anatomical changes that arise as they grow to 3 to 4 years of age, in large part these feeding skills develop earlier in the first 2 years of life and occur as a result of central nervous system (CNS) maturation combined with experiential learning (Stevenson and Allaire 1996).

The physiology of eating and swallowing is complex, and differs between the newborn infant and the older mature feeder. These distinctions occur mostly due to the significant brain development that occurs during the first 2 years of life (Stevenson and Allaire 1996). Nevertheless, for feeding to occur safely and effectively, the individual anatomical structures must each work independently, yet in concert with each other in a rhythmical, successive, coordinated movement pattern directed by the CNS (Strudwick 2009). The feeding of a newborn infant who has an immature brain is automatic and based on primitive feeding reflexes. Consequently, infants use a single feeding pattern of suck, swallow, breathe (Stevenson and Allaire 1996, Qureshi et al. 2002). An older child with mature feeding skills, in contrast, has the capacity to choose among several different feeding strategies (e.g. chew the food, eat off a spoon) and to manipulate the food bolus in the oral cavity through volitional oral movements. The rate and rhythm of delivery of the food bolus into the pharynx of an infant is also different compared with a mature feeder (Stevenson and Allaire 1996).

Infants are born with several different reflexes that enable them to feed at the breast and bottle, such as the suck–swallow and rooting reflexes (Table 35.1) (Stevenson and Allaire 1996). The rooting reflex serves as a response to locate food. It is easily

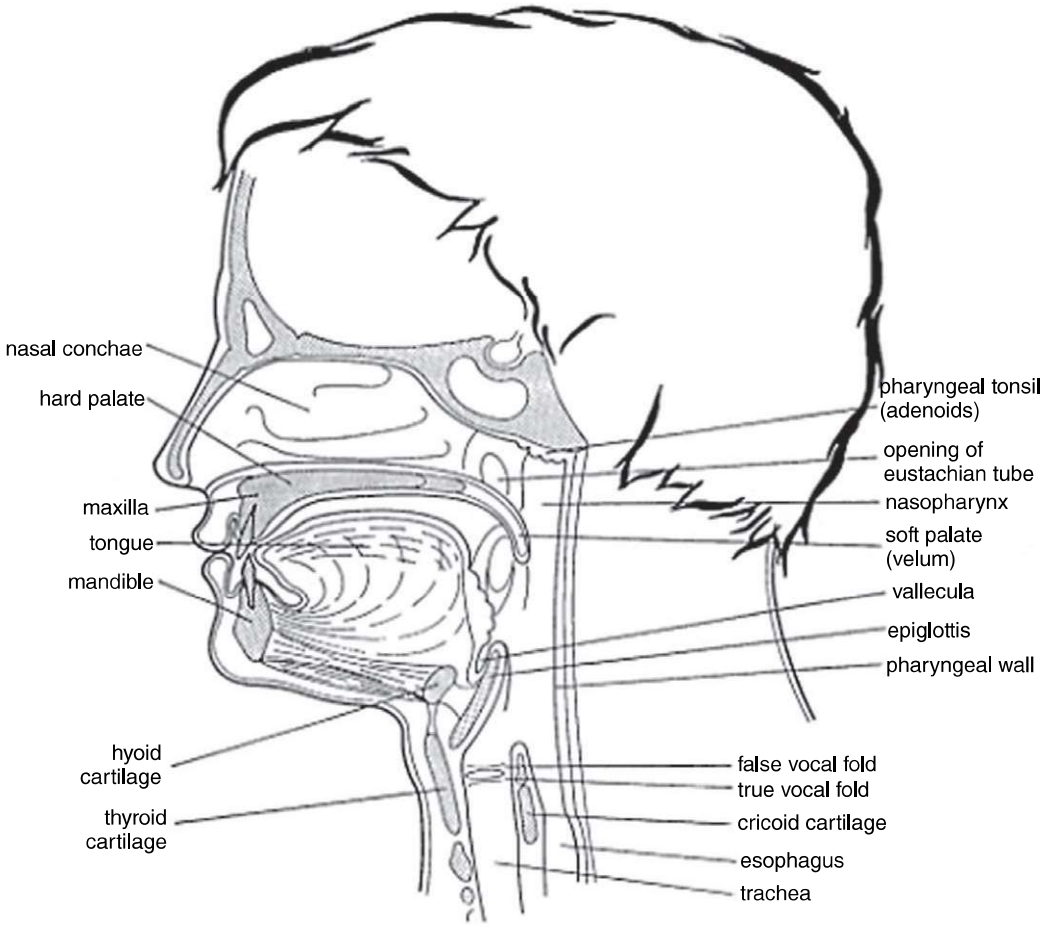

Fig. 35.1. The mouth and pharynx of the adult. (Reprinted with the permission of the author from Morris [1982] and http://www.new-vis.com.)

provoked by a soft stroke near the edge of the mouth (e.g. the nipple), causing the head to turn towards the source of food. These reflexes may be stronger or weaker depending on various factors such as hunger state, level of alertness, and neurological intactness. Generally, these primitive reflexes disappear by 6 months as the CNS matures, and more mature feeding strategies dominate.

The suck, swallow, breathe pattern is the sole feeding strategy for the newborn infant (Qureshi et al. 2002). During sucking/suckling, the tongue contracts in a peristaltic fashion, which causes nipple compression and thus milk expression (Kramer and Eicher 1993, Stevenson and Allaire 1996). Owing to anatomical crowding of the infant oral cavity, the tongue initially at birth moves in a pattern of extension and retraction, termed suckling (Delaney and Arvedson 2008). A true suck, on the other hand, involves an up-and-down movement of the tongue in unison with the mandible. This technique allows the lips to close more tightly around the nipple, and thus to generate more negative pressure to pull in fluid. By 6 months of age, infants primarily engage in true sucking as a result of developmental advancements and because it is more efficient. Once the fluid enters the oral cavity with suckling/sucking, the action of swallowing is immediately initiated, when in one continuous motion the tongue moves the liquid bolus to the posterior oral cavity (Kramer and Eicher 1993, Rogers and Senn 2008). When an adequate amount of fluid reaches the epiglottic vallecula, the liquid bolus is thrust into the pharynx by a combination of tongue contraction and changing pressure gradients (Bosma 1992).

During feeding, the breathing pattern and rate change because of the requisite apnoea that occurs with swallowing (Stevenson and Allaire 1996, Qureshi et al. 2002). With continuous feeding, the infant's overall respiratory rate decreases while the expiratory phase lengthens and the inspiratory phase shortens. The breathing pattern of a newborn infant consists of two or more sucks before pausing to swallow and breathe. As the infant nears 6 months of age, longer periods of sucking can occur before the need to breathe and swallow. Infants are also obligate nose-breathers while feeding, which occurs as a consequence of the small oropharynx concurrent with occlusion of the oral cavity owing to nipple placement. Coordinated breathing is crucial for successful feeding, and a child suffering any type of respiratory compromise (e.g. nasal congestion, choanal atresia) does not feed as well (Stevenson and Allaire 1996).

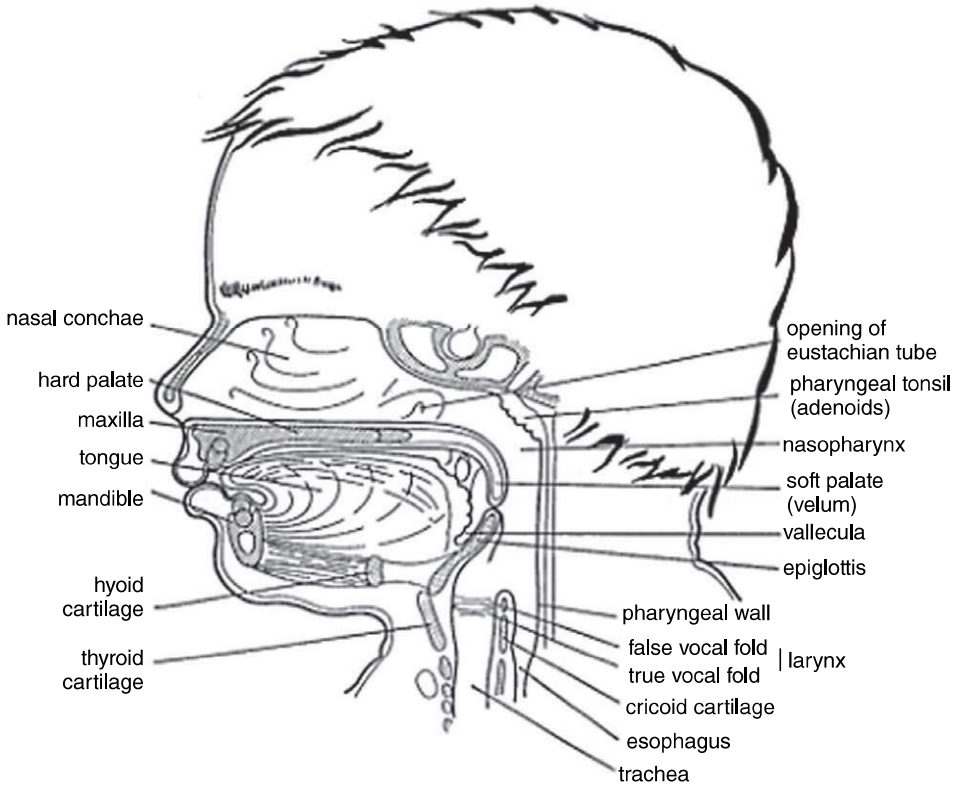

Fig. 35.2. The mouth and pharynx of the newborn infant. (Reprinted with the permission of the author from Morris [1982] and http://www.new-vis.com.)

TABLE 35.1
Feeding reflexes present in the newborn infant

Reflex	*Stimulus*	*Response*	*Time of disappearance (mo)*
Suck–swallow	Stroke anterior third of tongue or center of lips	Suckle/suck, swallow	
Rooting	Stroke around mouth	Head moves toward source of stimulus, mouth latches onto nipple	3–4 (later if breast-fed)
Biting	Stroke the gum	Rhythmic up and down biting motions of jaw	6
Gagging	Touch mid-third portion of tongue	Gag, eye-widening	Changes to posterior third of tongue at 7 (remains)
Babkin reflex	Stroke palms of both hands	Mouth opens, head flexes and rotates to midline	3–4

Adapted from Stevenson RD, Allaire JH (1996) The development of eating skills in infants and young children. In: Sullivan PB, Rosenbloom, L, editors. *Feeding the Disabled Child.* London: Mac Keith Press, pp. 11–22.

In mature eating and swallowing, the physiological process consists of four phases: oral preparatory, oral, pharyngeal, and esophageal phases (Rogers and Senn 2008). The food preparation phase allows for the breakdown of food through mastication and mixing with saliva. The tongue plays an important role in this stage by moving food laterally towards the teeth for grinding and crushing. A child with typical development will develop tongue movements first in the horizontal direction, then vertically, and finally accomplish lateral movements at about 9 months of age (Delaney and Arvedson 2008). Full coordination of mastication matures between 3 and 6 years old (Kramer and Eicher 1993).

In the brief oral phase, the bolus rapidly moves into the posterior oropharynx, which through sensory signaling triggers the swallow reflex (Dodds, Stewart, and Logemann 1990, Rogers and Senn 2008). This begins the phase of swallowing that is completely involuntary. During this pharyngeal phase, respiration stops briefly as the soft palate elevates to close off the nasopharynx, and the pharyngeal musculature propels the bolus into the relaxed upper esophagus (Kramer and Eicher 1993). Concurrently, the larynx closes at three different levels (epiglottis/aryepiglottic folds, false vocal cords, and true vocal cords), each forming a separate safeguard against food penetration into the airway. The cricopharyngeal muscle at the top of the esophagus functions to protect from aspiration and usually remains continuously contracted, relaxing only briefly to allow food to pass during swallowing. As food enters the esophagus, this marks the onset of the esophageal phase, and transfer of

the food bolus into the stomach by continued peristalsis (Rogers and Senn 2008).

Neurophysiological control of eating and swallowing is important in understanding why a child with CP and brain injury may have difficulties with feeding. In older children, the voluntary components of mature feeding involve motor and sensory signaling by cranial nerves, as well as higher-order CNS control (prefrontal cortex) (Dodds, Stewart, and Logemann 1990). On the other hand, oral feeding in young infants is purely based on primitive reflexes, which are brainstem-mediated and do not necessitate supra-bulbar control. It is through experiential learning and 'encephalization' (maturation of the brain allowing for higher-order CNS control of feeding) that ultimately enables typically developing infants to use different voluntary feeding strategies. This also explains why some children with CP may feed reasonably well initially because of reflexive feeding, but then do not progress to mature eating and swallowing. Swallowing in the mature feeder entails a blend of voluntary and involuntary actions, with sensory stimuli playing an essential role for determining which motor response occurs. The swallowing center in the medullary brainstem is fundamental and works to integrate and process various sensory afferent information, as well as to coordinate efferent signaling for the appropriate motor action (Dodds, Stewart, and Logemann 1990).

Pathophysiology of abnormal feeding

Feeding dysfunction in children with CP is generally multifaceted and complex. It is essential to consider the constellation of contributing influences for each child, and to understand the relationship between them. These contributory factors may be composed of the various manifestations secondary to the inciting brain injury responsible for the CP, such as oral–motor dysfunction, as well as components of gross and fine motor dysfunction that specifically affect feeding ability. Other comorbid conditions including gastro-esophageal reflux (GER) and constipation commonly affect children with CP and interfere with feeding (Clayden 1996, Sullivan 2009b). Lastly, environmental factors including medications, family dynamics, and economic resources also play an important role (Strudwick 2009).

One of the most important etiological aspects of feeding dysfunction to consider in children with CP is oral–motor impairment and swallowing dysfunction caused by injury to the brain in the areas of the motor–sensory cortex and the brainstem. Feeding is a complex task necessitating the concurrent use of motor and sensory pathways, as well as neuromuscular synchronization (Dodds, Stewart, and Logemann 1990). As a result, young infants with oral–motor impairment due to brain injury may experience a weak suck, uncoordinated swallow, poor lip closure, and inefficient food acquisition (Strudwick 2009). Other issues in older children related to abnormal neurological maturation include poor food manipulation and chewing, tonic bite, and persistent tongue thrust (Strudwick 2009). Commonly, children with CP who have abnormal oral–motor function also have oral–sensory impairment. This can adversely affect feeding when there is a hyperactive gag response, or hypersensitivity to particular textures, temperatures, and tastes (Strudwick 2009). More than likely, a child with CP has more than one of these contributing to their feeding problem. Furthermore, the presence of one or more of these issues often leads to inefficient and slow feeding, which if severe enough results in inadequate caloric/nutrient intake, dehydration, and prolonged feeding times (Sullivan 2009a). Children with CP are a heterogeneous group, but even those with relatively mild eating and drinking dysfunction may be at risk for poor nutritional status (Fung et al. 2002).

Some children with oral–motor impairment have such significant swallowing dysfunction that they are unable to protect their airway, and consequently are at increased risk for aspiration (Shaw 2007, Strudwick 2009). Aspiration is the passage of foreign material (either food, fluid, or refluxed gastric contents) into the larynx and below the vocal cords into the trachea and lungs. The prevalence of aspiration in children with severe neurodevelopmental disability is as high as 70% (Griggs et al. 1989). Many variables can contribute to aspiration, such as poor general coordination of oral–motor movements or absent/delayed swallowing reflex (Shaw 2007, Strudwick 2009). Weak respiratory musculature, scoliosis with chest wall deformity, and coexisting bronchopulmonary dysplasia of prematurity are other potential factors to consider. Fatigue that occurs during the course of a feeding session can increase aspiration risk because the child ultimately cannot keep up with trying to manage the food safely. Because aspiration can lead to pneumonia, recurrent wheezing and cough, and chronic lung damage, it is a major cause of morbidity and mortality for these children (Shaw 2007). Acute aspiration does not necessarily have to be associated with respiratory symptoms, as it may be silent and still cause harm (Arvedson et al. 1994). Furthermore, the frequency and severity of chronic aspiration does not uniformly correlate with development of lung injury (Cass et al. 2005).

As a side note, sialorrhea – or drooling – is a problem that may affect individuals with neurological impairment manifesting as oral–motor impairment (Blasco 1996). In particular, those with CP who have feeding disorders are at high risk for drooling. Drooling generally occurs from poor lip closure and inefficient swallowing, rather than excessive saliva production (Erasmus et al. 2009). Problematic drooling spans a continuum of severity, with about 33% to 58% of children with CP estimated to have drooling problems significant enough to hamper daily social and functional activities (Tahmassebi and Curzon 2003). This may include drooling that is so profuse that clothing, papers, equipment, and other possessions get soaked. For older children drooling can be a significant source of embarrassment. Pooling of saliva in the hypopharynx may lead

to congested breathing, coughing and gagging, vomiting, and possible aspiration. Malodor may occur as well due to mixing of pooled saliva with food. As such, chronic drooling not only affects the general well-being of the individual, but it may also pose problems for caregivers as well (Blasco and Allaire 1992).

The degree of gross and fine motor impairment in children with CP is associated with problems related to eating and drinking (Sullivan et al. 2000). Several different factors related to motor impairment can hamper safe and effective feeding. Significant interference in feeding for many children with CP derives from persistence of primitive reflexes. For instance, the asymmetric tonic neck reflex causes a child's head to turn away from the flexed arm used for self-feeding (Strudwick 2009). A persistent tongue thrust can also make it difficult to eat from a spoon or drink with a cup. Furthermore, the child may not have the hand/arm strength and dexterity to be able to manipulate food and drink for feeding. Of course, sensory impairment also plays a role here owing to decreased proprioception.

Another common problem is poor head and trunk control due to hypotonia. In fact, the most influential postural factor affecting eating and drinking is head control (Strudwick 2009). Head position in relation to the rest of the body is critically important for safety, such that if the head is not at midline it is more difficult for the child to swallow and maintain a safe airway. If the head is tilted backwards the airway is open but unprotected; conversely, if the head is tilted forwards, the airway can obstruct as well as make it difficult to swallow (Strudwick 2009). Those who cannot lift their heads and who depend on a feeding tube are at highest risk for death in early childhood (Strauss et al. 1997). Children with CP are often limited in their activity level as a result of their motor impairment, and this may also affect feeding by causing decreased appetite (Sullivan 2009a). In summary, a child with CP who has oral–motor impairment and swallowing dysfunction may have their feeding disorder further aggravated by impaired motor control and posture.

Other factors contributing to abnormal feeding

Other comorbid medical conditions often have a large impact on feeding through effects on appetite, oral–motor function, and energy needs. These may include GER, retching, constipation, dental caries/gingivitis, chronic lung disease, seizures, and chronic infections. Associated impairments of cognition, behavior, and sensation also affect feeding and contribute to the overall difficulty in providing sufficient nutrients to the child (Stevenson 2007).

GER is a comorbid condition frequently present in children with CP, which can have a significant influence on feeding in these children (Sullivan 2009b). GER, constipation, delayed gastric emptying, and retching are all possible manifestations of the underlying CNS dysfunction in children with CP. The brain has 1% of its neurons (as many as one billion neurons) allocated to modulation of the enteric nervous system; thus it is no wonder that these children suffer gastro-intestinal dysfunction. Theory implicates failure of the lower esophageal sphincter and gastro-esophageal dysmotility as the main culprits in the etiology of GER in children with neurological impairment. Other reasons include hiatal hernia, chronic supine positioning, increased abdominal pressure secondary to spasticity, scoliosis, and seizures. If abdominal contents reflux high enough into the pharynx, pulmonary aspiration occurs. Chronic GER can result in esophageal mucosal ulceration and stricture formation, (Sullivan 2009b) which may lead to lack of appetite, food refusal, abdominal pain, nausea, and aspiration (Strudwick 2009). However, these symptoms are often incorrectly attributed to oral–motor difficulties, tone abnormalities (e.g. extensor spasms), and/or aversive feeding behaviors, thus leaving GER undiagnosed.

In children with disabilities, the diagnosis and management of constipation often comes second to other conditions seen in children with neurological disability, such as airway difficulties, seizures, poor nutrition, and recurrent infections (Clayden 1996). This may be due to the documented importance to health and survival of these other issues, whereas the degree to which bowel issues contribute to health, survival, and quality of life are less well understood. Constipation, defined as delayed defecation or passage of hard stools, can lead to abdominal pain and fullness, as well as anal discomfort. In addition, constipation can augment other problems as well. Some children lose their already modest appetites, whereas others exhibit deterioration in behavior. Evidence also supports the association of urinary tract infections with constipation, theoretically because of bladder compression from a full rectum (Giramonti et al. 2005). Constipation may arise from a constellation of factors, including poor fiber in the diet, chronic dehydration, intestinal dysmotility, poor rectal sensation, and decreased ambulation (Clayden 1996). The situation can turn into a vicious cycle in which these factors lead to constipation, which serves to exacerbate the feeding disorder further by decreasing appetite and worsening behaviors.

Poor oral hygiene leading to mouth ulcers, oral thrush, dental caries, and gingivitis causes discomfort and worsens feeding disorder (Strudwick 2009). In particular, children who mouth-breath and drool tend to have oral hygiene problems (Strudwick 2009). In children with CP, adequate oral hygiene is often difficult to accomplish for various reasons. For instance, it may be difficult for caregivers, and even dentists inexperienced in children with neurological disability, to provide adequate oral care. GER may also contribute to dental problems because chronic acid leads to enamel erosion (Sullivan 2009b).

In a child who already struggles with consuming sufficient calories, nutrients, and fluids because of oral–motor impairment, any additional stress can interrupt a tenuous energy balance. Chronic lung disease, uncontrolled seizures, and recurrent illness are common comorbid conditions in children with CP that add to the energy demands.

Communication ability and cognitive level of the child are highly important factors to consider in relation to the nutrition of children with CP. Oral–motor impairment is often associated with dysarthria, which makes it difficult for these children to express themselves verbally when hungry or thirsty, or with food preferences (Strudwick 2009). They may also have limited communicative gestures due to motor impairment. Children with severe intellectual disability probably also have difficulty understanding their caregiver's language and non-verbal cues, as well as the mealtime experience in general. Visual impairment further complicates the feeding process because the child cannot visualize other non-verbal cues, nor anticipate food or drink. Additionally, children with CP are frequently on medications to help control disorders (e.g. seizures, hypertonia, drooling), some of which impair alertness, decrease appetite, and worsen constipation (Strudwick 2009).

The child's difficulty with eating and drinking as a result can lead to aversive feeding behaviors related to past feeding experiences associated with discomfort, pain, fear, or other distress (e.g. aspiration, nasogastric feeds or suctioning). Furthermore, caregivers may inadvertently ignore these behavioral responses because of their own emotional stress and anxiety about feeding a child enough nutrients and doing it safely (Strudwick 2009).

Prevalence and natural history of feeding disorder

About 30% to 40% of children with CP are affected by feeding disorder, as documented in recent epidemiological studies (Sullivan et al. 2000, Fung et al. 2002). It is also evident that those with more severe motor deficit have increased feeding and nutritional problems, shown both in the North American Growth in CP Project and the Oxford Feeding Study (Sullivan et al. 2000, Fung et al. 2002). For instance, in the Oxford Feeding Study, 47% and 32% of children with severe and moderate CP had feeding difficulty, respectively, whereas only 10% of those with a mild form were affected (Sullivan et al. 2000). Furthermore, an overwhelming majority (almost 90%) of children with CP, regardless of severity, required at least some assistance with feeding. Other common feeding issues included frequent choking and vomiting, as well as prolonged feeding times of greater than 3 hours per day.

The Oxford Feeding Study also revealed that feeding and nutrition was not part of the care plan for many children with CP, at least at the time of the study (Sullivan et al. 2000). More than half of the caregivers for these children reported that their child had never had a feeding and nutritional status evaluation, and only about one-fifth of children had had contact with a dietician in the previous 12 months. Fortunately, more recent practice supports increased awareness of feeding and nutrition issues in children with CP, and emphasizes a multidisciplinary team for the care and management of children with CP to prevent adverse outcomes related to feeding dysfunction and malnutrition (Marchand et al. 2006).

TABLE 35.2
Risk assessment of aspiration during eating and drinking

- Inability to handle own secretions
- Coughing or choking during/after feeds
- Delayed swallow
- Multiple swallows to clear single bolus
- Limited endurance for feeding
- Noisy/wet upper airway sounds during feeding
- Wet voice quality during/after feeding
- Apnea or increased congestion during feeds
- Change in breathing rhythm(faster or slower or both) during feeds
- Change in color of oral mucosa (cyanosis) during feeds
- Blinking/eyes widening/eye watering during feeds
- Throat clearing during feeds
- Grimacing during feeds
- Head tilting during feeds
- Arching during feeds

Adapted from Strudwick W (2009) Oral motor impairment and swallowing dysfunction: assessment and management. In: Sullivan PB, editor. *Feeding and Nutrition in Children with Neurodevelopmental Disabilities.* London: Mac Keith Press, chapter 3.

The North American Growth in CP Project was a large, multicenter study designed to characterize feeding dysfunction as perceived by parents of children with CP and its relationship to health and nutritional status (Fung et al. 2002). An important conclusion of the study was that feeding dysfunction was not only commonplace for children with moderate to severe CP, but that it was associated with poor health (more days in ill in bed, hospitalization, missed school) and poor nutritional status. Moreover, even children with mild CP who have feeding problems, for example those who require chopped or mashed foods, were found to be at risk for poor nutrition. The study also reported a lack of support for the caregivers of these children with feeding difficulties, which tends to produce significant stress on the family (Fung et al. 2002). Other studies have also corroborated this finding of increased psychological stress and decreased caretaker quality of life as a result of having a child with neurological impairment and related feeding difficulty (Sullivan et al. 2004).

Poor growth and malnutrition

Feeding disorder secondary to oral motor impairment is clearly associated with poor growth and malnutrition (Stallings et al. 1993a, b, Stevenson et al. 1994, Fung et al. 2002, Sullivan et al. 2002). In fact, children with CP have poor growth that correlates positively with increasing severity of motor impairment including ability for feeding (Stevenson et al. 2006, Day et al. 2007). For children with neurodevelopmental disabilities, poor growth and malnutrition usually evolve gradually over months to years (Stevenson 2007). A disabled child who is a slow and inefficient eater can only take in a limited amount of food over a period of time. Various other factors may also contribute to poor oral intake, but inefficient and dysfunctional

oral skills are largely culpable. The caretaker may attempt to compensate by lengthening oral feeding times and through other physical adaptations, but as the child grows energy needs outpace these strategies (Sullivan 2009a). Further influence on this chronic process of under-nutrition occurs from acute episodes of illness combined with failure to recover acute weight loss (Stevenson 2007). Moreover, malnutrition weakens the immune system, thus predisposing the child to more frequent infections (Katona and Katona-Apte 2008). If no intervention occurs to interrupt this process of under-nutrition, the potential outcomes for the child with CP include short stature, low fat stores, reduced muscle mass, poor bone density, and micronutrient deficiency (Krick et al. 1996, Henderson et al. 2002, Stevenson et al. 2006, Day et al. 2007, Sullivan 2009a, Schoendorfer et al. 2010).

Differences in growth and body composition

It is well-documented that the growth of children with CP is distinct compared with age-matched children with typical development (Krick et al. 1996, Samson-Fang and Stevenson 1998, Henderson et al. 2002, Stevenson et al. 2006, Day et al. 2007, Sullivan 2009a). In general, these children tend to be shorter and lighter, with the differences that are more pronounced with increasing severity of motor impairment and feeding abilities (Day et al. 2007). These differences were first documented by comparing the growth curves of children with CP with the standards from the National Center for Health Statistics (Krick et al. 1996). For both females and males with bilateral CP (quadriplegia) up to age 10 years, the 50th centile was shown to be below the 10th centile National Center for Health Statistics standards for weight- and length-for-age, and approximately equal to the 10th centile for weight-for-length. These findings of decreased stature and weight have been corroborated by other more recent and larger studies as well, even in children with bilateral CP (diplegia) and unilateral CP (hemiplegia) (Stallings et al. 1993a, b, Stevenson et al. 1994, Day et al. 2007).

Body composition also differs in children with CP (Bandini et al. 1991, Henderson et al. 1995, 2005, Stallings et al. 1995). Decreased weight in a child with CP does not imply the specific etiology, for example decreased fat stores, muscle mass, or bone density. Research has focused on these individual components, and through technology such as dual-energy X-ray absorptiometry (DXA) scans children with CP have been shown not only to have decreased fat mass, but generally also reduced lean muscle and bone density (Stallings et al. 1995, Henderson et al. 2005). Severity of neurological impairment, difficulty in feeding the child, and use of antiepileptic drugs contributed to low bone mineral density (BMD). Bone density has also been shown to correlate with poor nutritional status and low calcium intake (Henderson et al. 1995). Altered skeletal maturation also occurs commonly (>50%) in children with CP, and correlates with diminished bone growth, reduced bone density, and decreased body fat (Henderson et al. 2005).

Children with CP also experience distinctive growth patterns (Krick et al. 1996, Samson-Fang and Stevenson 1998, Day et al. 2007). For instance, growth velocity is decreased in children with CP. Documentation of poor growth over a period of time is important as it may mean growth failure, which is notably different from constitutional short stature. Interestingly, children with more severe CP do not have the typical pubertal growth spurt of typically developing peers (Worley et al. 2002). A typical population of children tends to have a logistic S-shaped curve whereas children with CP show a straight-line increase in weight during puberty. Children with moderate to severe CP also tend to begin puberty earlier, which terminates later compared with children with typical development.

Growth in relation to nutrition and health

In the past, it was believed that poor nutritional status accounted for most of the growth impairment in children with CP. However, recent research supports the idea that the etiology of poor growth is probably multifactorial and includes neuroendocrine abnormalities, psychosocial influences, and growth-promoting activity effects (Henderson et al. 2005, 2007, Stevenson 2007, Kuperminc et al. 2009). Despite this, the role of nutrition continues to be important and documented as a contributor to impaired growth in children with CP. One study demonstrated that children with CP at nutritional risk (triceps skinfold thickness <5th centile) grew more poorly over time than those with adequate triceps skinfold thickness ($p = 0.009$) matched for age and sex (Samson-Fang and Stevenson 1998). In children with bilateral spastic CP (quadriplegia), nutritional status may contribute up to 10% to 15% of the variability in linear growth (Stallings et al. 1993a). Even children with unilateral CP (hemiplegia) and bilateral CP (diplegia) demonstrate poor linear growth due at least in part to malnutrition (Stallings et al. 1993b). In a cross-sectional study of children with CP, almost 40% were below the third centile for height and weight compared with standard growth curves, and 30% were below the third centile for triceps skinfold. Multiple linear regression analysis revealed that nutrition contributed significantly to the variance in growth (Stevenson et al. 1994).

Poor growth is only one outcome in children with CP related to malnutrition. Indeed, it may be that growth impairment is not the most important outcome, especially because it may merely be a marker of the underlying disease severity rather than nutritional status (Stevenson 2007). Further research is necessary to better explain growth in children with CP and the contributing factors. It is also essential to clarify how growth and nutritional status are associated with general health and overall quality of life (Samson-Fang and Stevenson 2000). For example, it has been shown that malnutrition is one of the most common predictors of early death among people with severe disabilities (Strauss et al. 1997, 2007).

Preliminary insight into the relationship between nutritional status, growth, and general health and well-being in children with CP does exist (Samson-Fang et al. 2002). In this study, it was shown that triceps skinfold thickness z-score correlated significantly with child general health, child participation, and family activities. Importantly, this finding occurred independently of Gross Motor Function Classification System level, age, and sex. Each standard deviation increase in triceps skinfold thickness was associated with a 20% reduction in doctor visits, a 22% reduction in days missed from school, and a 33% reduction in missed activities for the family for the preceding 1 to 2 months. Other nutritional markers (arm muscle area, arm fat area, and subscapular skinfold thickness) had a similar relationship to these outcomes that were statistically significant. Reference growth curves using this same data were later developed for children with CP and demonstrated that overall growth was consistently associated with health and participation (Stevenson et al. 2006). In other words, larger children had less use of healthcare and more social participation than smaller children.

Other consequences of malnutrition

Other consequences of malnutrition have been studied, mostly in children without disabilities. Malnutrition has been ascribed to abnormalities in neurological and developmental outcomes, which is particularly important when considering children with CP who already experience problems in these areas. School-age children who suffer from early childhood malnutrition generally have poorer IQ levels, cognitive function, school achievement, and greater behavioral problems than matched peers (Grantham-McGregor 1995). Some evidence suggests that undernourished children have differences in temperament as well (Baker-Henningham et al. 2009). Supplementation of under-nourished children has been shown to improve psychomotor development compared with non-supplemented children (Grantham-McGregor et al. 1991). Relative to these issues scarce data exist for children with CP, but it is possible that nutritional deficits exacerbate the effects of primary brain injury in young children with CP owing to the rapid brain development that occurs the first 2 years of life. Proper nutrition and treatment of GERD may also improve motor function in children with CP, as shown in one study (Campanozzi et al. 2007).

Clinical assessment of feeding, growth, and nutrition

A multidisciplinary team approach is recommended for a comprehensive assessment of feeding disorder and malnutrition in children with CP. This strategy encourages team members to contribute their specific expertise to the overall understanding of the child's situation, so that together the team can develop a cohesive and thorough management plan that addresses each potential etiology. It is not appropriate to assess only the adequacy of nutritional intake and the safety of oral feeding in children with CP. Feeding dysfunction with associated malnutrition and poor growth can be extremely distressing for the child and family involved. Thus quality of life should be given significant consideration as well (Strudwick 2009). Accordingly, a multidisciplinary team is essential for fully appreciating the effect of the child's eating and drinking in the context of family dynamics (Andrew, Parr, and Sullivan 2011).

As for gross motor function and hand function and communication, eating and drinking skills can be classified using the Eating Drinking Assessment Classification System (EDACS; Sellers et al. 2014; see also Appendix 4). The scale classifies eating and drinking ability in terms of efficiency and safety, with levels of assistance applied to EDACS levels I to III, and is currently valid for children with CP from the age of 3 years (see also Chapter 25).

Oral–motor skills and swallowing

One of the main responsibilities for speech and language pathologists is to assess the child's oral–motor skills and swallowing, and interpret adequacy for nutritional requirements as well as feeding safety. The speech and language pathologist can also help promote the child's eating potential, reduce anxiety around feeding, and assist with decision-making when oral intake in not sufficient or possible (Strudwick 2009). The child should be evaluated at rest and while feeding, ideally in different environments, such as in the school and at home. Different textures should be attempted to assess safety with chewing and swallowing. Unfortunately, a standardized tool for assessment of oral–motor skills validated for children with neurological impairment is not available currently (Strudwick 2009).

Anatomical and functional examination of the jaw, lips, cheeks, and tongue during feeding is required for an appropriate assessment (Strudwick 2009). It is important to examine the position of the jaw in children with CP because they often have over-bites, which can exacerbate biting and chewing dysfunction. Tonic biting can be a significant issue as well, in which the child bites down firmly with their jaw and has difficulty releasing. Children with CP may suffer from inability to open/close the jaw appropriately, resulting in ineffective manipulation of food and fluid in the mouth, especially with bolus formation. In assessing the function of the lips for feeding, it is necessary to observe lip tone, presence of athetosis or asymmetry, and coordination/timing of movements. In particular, the lips are important for removing food off a spoon, preventing spillage of food outside the oral cavity, and creating sufficient negative pressure for an effective swallow. The speech and language pathologist must also examine the tongue of a child with CP because abnormal tone and movements of the tongue can cause slow or ineffective bolus formation, poor oral transit backwards in preparation for the swallow, and inability to manipulate the food for chewing. Tongue thrusting is common in children with CP, and may cause difficulty with spoon or cup placement, or food loss. The cheeks support the lip and

tongue to contain food in the oral cavity and move the bolus inside the mouth. Hypotonic cheeks can contribute to food collecting on the lateral sulci, which causes difficulty in forming and swallowing a food bolus (Strudwick 2009). In summary, each of these components must be assessed individually, in addition to how they interact with each other.

Aspiration

Screening for aspiration can be performed quite well through clinical assessment, especially aspiration of fluids, but it cannot be used alone and confirmatory testing with videofluoroscopy is required (Strudwick 2009). Aspiration of fluids occurs most commonly, accounting for 40% among aspiration events. Furthermore, fluid aspiration is easier to detect clinically (about 92% of the time versus 33% for solids) (Dematteo et al. 2005). Clinical signs of aspiration during and after feeds include coughing or choking, noisy/wet upper airway sounds, wet voice quality, throat clearing or grimacing, change in breathing rhythm, blinking and watery eyes (Strudwick 2009, Andrew, Parr, and Sullivan 2011) (Table 35.2). It is critical to be aware of silent aspiration, which can occur with no obvious signs at all, and actually accounted for most aspiration events in one study (Arvedson et al. 1994). Other findings often associated with aspiration are inability to handle secretions, delayed swallow reflex or multiple swallows to clear a single bolus, very poor oral–motor coordination, and a history of recurrent pneumonia (Strudwick 2009). Essentially, children with more severe impairment in oral–motor function and swallowing are more likely to experience aspiration.

Cervical auscultation is an additional strategy for assessing children at risk for aspiration. This technique is increasingly integrated into the clinical assessment because of its easy accessibility and non-invasiveness, but it does rely on the experience of the clinician (Strudwick 2009). Of course, confirmatory detection of aspiration must be made through videofluoroscopy, because this is the definitive way of demonstrating how the anatomical structures are functioning independently and together during eating and drinking. Videofluoroscopy is a crucial tool for determining whether feeding is safe, but can also serve to inform management strategies about ideal feeding position, appropriate textures, and feeding pace (Strudwick 2009). In addition, it can be quite helpful for having families understand through visualization that their child is indeed aspirating with feeding.

Gross and fine motor skills related to feeding

The speech and language pathologist, jointly with the physical therapist and occupational therapist, should provide an evaluation of fine and gross motor skills with particular emphasis as to how they relate to feeding skills. This includes most importantly an assessment of arm/hand use, postural control, and seating. Ideally, the therapists should have experience and expertise in working with children with CP. These therapists serve to help recognize the particular aspects of the child's movements and positioning that are hampering safe and effective feeding.

It has been documented that improved posture in children with neurodevelopmental disabilities leads to better safety and ability to eat and drink (Redstone and West 2004). Children with CP should be evaluated for their seating position during feeding, because this may be different when engaging in other activities and can require certain equipment (Strudwick 2009). Seating is important because even slight changes in body posture can impede a child's functional feeding skills. As one speech and language pathologist describes, 'The aim is to achieve stability and symmetry. The child ideally should be positioned with their hips and knees at right angles to their body. Their head should be in the midline, with a slight chin tuck, and with no extension in the neck. Their shoulders should be slightly flexed' (Strudwick 2009).

It is also important to consider the positioning of the caregiver while feeding the disabled child, which can positively or negatively impact the positioning and oral–motor skills (Strudwick 2009). Lastly, despite best assessment and education efforts, resources and location may restrict the options for ideal seating during feeding. For instance, the child may have customized seating for school, but unfortunately may lack a suitable chair at home. Growing children also have needs and an environment that are constantly changing, thus necessitating routine evaluations to preserve appropriate seating strategies (Strudwick 2009).

Growth and nutritional status

Growth and nutritional status are fundamental aspects of health and well-being in children (Stevenson 2007). A child who grows inappropriately or appears malnourished on assessment is indicative of an underlying pathology or environmental stress. The combination of assessment tools, including the physical examination, growth curves, anthropometric measures, nutritional quantification, laboratory studies, and DXA are essential components in the evaluation of child's growth and nutritional status. Evaluation is important for determining whether there is a growth or nutritional issue, but also informs management strategies for intervention.

At first glance, a general impression can often alert a clinician that a child is malnourished owing to diffuse wasting and growth stunting. However, any child with CP is at risk for these issues and necessitates a thorough assessment even if cursory physical examination is unrevealing. Other specific findings on physical examination can provide clues to whether a child is lacking calories or nutrients. Generalized edema is characteristic of protein inadequacy, and can be a sign of heart failure. Pallor, or a heart murmur, often indicates anemia due to iron, folate, or cobalamin deficiency. The lungs may exhibit wheezing from chronic aspiration. The skin, nails, and oral mucosa should also be examined for abnormalities suggestive of vitamin B

deficiencies. Evidence of poor wound healing is often observed with caloric insufficiency and possibly deficiencies in vitamin C, vitamin E, and zinc (Langermo et al. 2006). In severe vitamin D deficiency, beading of the ribs, widening of the wrists, and bowing of the legs are classic features on musculoskeletal examination. The eye examination can be revealing as well for vitamin A deficiency (corneal clouding, xerosis) or thiamine deficiency (nystagmus, ophthalmoplegia). More generalized findings include altered mental status or irritability, which may indicate extreme dehydration or thiamine deficiency. Delayed puberty may also reflect malnutrition (Worley et al. 2002).

The use of growth curves has become standard practice for health professionals monitoring the nutrition and health of children (Stevenson 2007). Growth reference curves based on population averages of children with typical development allow for assessment of growth by comparison of an individual with the norm. Significant deviation from the predicted average pattern of growth may indicate a biological or environmental problem. In general, plotting children with CP on standard growth curves reveals substantial divergence. Thus, it is not practical or highly informative to use typical pediatric growth charts for children with CP. Instead, reference charts specific to children with CP provide the opportunity for a uniform assessment of growth and nutritional status. Such growth charts have been developed (Krick et al. 1996, Stevenson et al. 2006, Day et al. 2007, Brooks et al. 2011). However, even these CP-specific growth charts are not ideal because their clinical utility has not been established (Stevenson 2007). Furthermore, these growth curves are descriptive in nature. In other words, they portray how a group of children have grown and do not indicate how an individual child should grow. In clinical practice it would be more useful to have prescriptive growth curves that correlate statistically and clinically significant anthropometric measurements with health and participation outcomes (Stevenson 2007) The most recently developed CP-specific growth chart (Brooks et al., 2011) may prove useful as it reports weight below a threshold as associated with an increase risk of mortality in subsequent years. Further study is needed to corroborate this report and determine the clinical utility of the growth charts.

It is often challenging to obtain reliable anthropometric measures in children with CP using traditional strategies such as weight and height. This is due to limitations inherent to the diagnosis of CP, including motor impairment, involuntary muscle spasm, fixed joint contractures, and poor cooperation secondary to intellectual disability (Stevenson 2007). An alternative method for determining anthropometric information is the use of segmental measures, which have been shown to be reliable and valid measures in children with CP (Spender et al. 1989, Stevenson 1995). Specialized growth charts using segmental measurements of the upper arm and lower leg length exist and are useful for assessing the growth of children with CP compared with children with typical development. Others have developed formulas to estimate the stature of children with CP based on segmental measures such as upper arm length, tibial length, and knee height (Chumlea et al. 1994, Stevenson 1995).

Obtaining a weight measurement is often more straightforward than height, especially for infants and small children (Stevenson 2007). It can be more difficult, however, for larger children with disabilities. A bed scale or chair scale is a good substitute if the child is too large for the infant scale, or cannot stand independently. However, these specialized scales may not be available in the typical primary care office. If this is the case, an inexpensive and reliable strategy is to have the caregiver and child weigh themselves together, and then subtract the caregiver's weight. At times even this method is not possible if the child is too large. In this case, unfortunately, the child must be taken to a local hospital with a bed scale.

Normally, assessment of nutritional status relies heavily on measurement of weight. For children with CP, particularly those who have severe motor impairment (non-ambulatory), this strategy frequently is inadequate owing to the differences in body composition and poor availability of less-than-ideal reference standards (Stevenson 2007). Instead, skinfold thickness and arm circumference measurements can be used as a proxy for adiposity, and have standard techniques established for children with disabilities (Cameron 1986, Lohman et al. 1988). Specifically, triceps skinfold thickness is the single best screening tool for the assessment of nutritional status in children with CP (Samson-Fang and Stevenson 2000). Some data suggest that skinfold thickness should be used in clinical practice regularly based on evidence linking it to overall health and participation (Samson-Fang et al. 2002).

Determination of actual caloric and micronutrient intake relative to ideal physiological need is an important piece of the assessment. For example, a clue to the etiology of malnutrition arises from knowing whether the child is already taking in sufficient calories or not. For estimating a child's caloric requirements, various methods are available (Stevenson 2007). The preferred equations for prediction of basal metabolic rate are from the World Health Organization. These are simple to use, and in general accurately estimate resting energy expenditure in children with CP (Bandini et al. 1991, Stallings et al. 1996). Children with CP who exhibit low fat stores are an exception to this, however, and the World Health Organization equations tend to underestimate their caloric needs. Other formulas exist but are either more complicated or require further investigation (Stevenson 2007). A dietician as part of the multidisciplinary team can assist with these calculations.

Laboratory studies can be quite useful, and even necessary for the severely malnourished individual. Generally, preliminary investigation includes a complete blood count, iron studies, and vitamin D 25-OH level. Depending on the presence and type of anemia, further evaluation may warrant folate and cobalamin levels. Routine evaluation of vitamin D 25-OH,

and possibly alkaline phosphatase, calcium, and phosphorus, are indicated in children with CP who are non-ambulatory and/or have seizures treated with antiepileptic medications (Fehlings et al. 2012). For any child with severe malnourishment and/or dehydration, a basic metabolic panel is necessary because electrolyte disarray and renal failure may be present. Low pre-albumin, retinol-binding protein, and transferrin are useful for supporting a concern for protein inadequacy, though albumin is a better indicator of chronic malnutrition because of its longer half-life. Based on other findings on the physical examination and in the history, other laboratory investigations are required to eliminate other causes of poor growth, such as hypothyroidism or hyperthyroidism (free thyroxine, thyroid-stimulating hormone), growth hormone deficiency (insulin-like growth factor binding protein-3 and insulin-like growth factor-1), delayed puberty (follicle-stimulating hormone, leutinizing hormone, estradiol, testosterone), inflammatory disease (erythrocyte sedimentation rate, C-reactive protein), celiac disease (tissue trans-glutaminase, immunoglobulin A), and chronic urinary tract infection (urinalysis, urine culture), among others.

DXA is the most common and preferred means of evaluating bone health (Fehlings et al. 2012). In simple terms, the technology relies on measurement of energy transmission based on the degree to which it attenuates as it passes through bone or soft tissue before it passes out of the body. This allows measurement of BMD, fat mass and lean mass. In children with CP, the distal femur regional scan is favored for measurement of BMD because this is the area most frequently fractured and because of ease of access. Current guidelines, based on expert opinion only, recommend obtaining a DXA scan only after a fragility fracture. Certainly a DXA scan at baseline and after intervention are useful to assess changes in BMD. Bone density results should be compared with pediatric reference standards and reported as z-scores adjusted for the child's age, sex, and height (Aronson and Stevenson 2012).

In summary, limited scientific guidelines exist to support clinicians in assessing adequate growth and nutritional status in children with CP (Stevenson and Conaway 2007). Instead, clinicians must assess and manage patients in large part based on clinical impression in combination with available anthropometric data (Stevenson 2007). As a result, much variability can occur among practitioners in terms of assessment and management of growth and nutrition in children with CP.

Management strategies

When managing children with neurodevelopmental disability, a 'biopsychosocial' approach is recommended and supported by the literature to develop and implement a successful healthcare plan (Craig et al. 2003, Sleigh 2005). This approach requires acknowledgment and respect for the caregivers' perspectives, transparent and interactive conversation with the family about the child's medical status and recommended treatments, and a collaborative multidisciplinary team approach. For example, in a situation where a child with CP has nutritional wasting and gastrostomy tube placement is recommended by the physician, a mother might feel guilt about the poor growth of her disabled child, blaming herself that she is not able to feed him (Sullivan 2009a). In her mind, the recommendation for a feeding tube might validate the idea of failure as a caregiver. A mother might be quite opposed to the suggestion for supplemental tube feedings, and even after placement might choose to continue feeding orally. This case illustrates the need for ongoing conversation addressing the mother's concerns and hesitations, while using a multidisciplinary approach to explain the benefits of tube feeds. Otherwise, the disconnect between caregiver and therapeutic team will interfere with any intervention. It is also necessary to consider feeding as an issue tightly associated with quality of life for the child and family, and not only one of function. Using this 'biopsychosocial' approach, the feeding and nutrition goals can be uniform among an integrated team of physicians, therapists, and caregivers. Together, the team should collaborate with the family to outline the various aspects (e.g. positioning, diet modification) important for safe and effective feeding to prevent malnutrition, dehydration, and aspiration, and to encourage growth, health, and well-being.

Child positioning, seating, and oral control

Positioning and seating of the child is of utmost importance for maximizing feeding ability (Strudwick 2009). The ideal situation is to position the child's head with a slight chin tuck and elongated neck so that the airway remains open. Appropriate seating of the child may require special equipment, and should be customized to maximize stability and head control, while minimizing abnormal tone and reflexes. In some cases, the child feeds best in a standing frame. Caregivers must be instructed on their positioning during feeding as well, so that they are facing the child in order to observe non-verbal cues and monitor for aspiration. Correct caregiver positioning is key so that the child does not have to move their head or trunk towards them, which may impair feeding or increase the risk of aspiration. The individual feeding the child can also assist with oral control through hands-on positioning to aid lip closure, stabilize the jaw, reduce abnormal movements, and facilitate useful movements. Importantly, to help children with desensitization and acceptance of having another's hands on their faces, oral control techniques should be initiated when the child is not feeding (Strudwick 2009).

Food delivery and texture

The caregiver and others responsible for feeding the child with CP must be informed on the proper technique for providing food and drink. Components of correct technique include pacing of bites/sips, choosing the food amount, and responding to the child's cues (Strudwick 2009). Presenting the next bite

too quickly, or placing too much food on the spoon, can lead to over-filling the mouth and overwhelming the child, thereby increasing the risk for aspiration. The key to determining the rate and quantity of food/drink involves observation and responsiveness by the individual feeding the child. Consistency of presentation also improves the feeding process, as demonstrated by a study using a robotic aid to eating (Pinnington and Hegarty 2000). Importantly, a positive reciprocal interaction between the child and caregiver can help ameliorate the inconsistencies inherent to human nature. In addition, the type of utensils and cups can influence delivery of food to the child (Strudwick 2009). It is important to consider spoon shape, size, and material, noting that most children with feeding difficulties do best with plastic cutlery. A variety of cups exist as well, and a flexible plastic cup with space for the nose is often recommended for optimal delivery of fluid and observation of drinking.

Selecting appropriate food and drink textures for a child with a feeding disorder is critical for making mealtimes safe and efficient, and should be aided by an experienced speech and language pathologist (Strudwick 2009). If the child cannot chew well, then puréed or mashed foods are often recommended. Foods that contain a mix of textures are generally troublesome and are often avoided in children with oral–motor impairment. More challenging textures can increase feeding fatigue, and potentially reduce feeding safety. Fluids may require thickening to a consistency that minimizes risk for aspiration, although some children do better with thin liquids. Simultaneously, it is important to consider texture and fluid thickness in the context of oral transit time. A careful balance should be maintained between maximizing efficiency and nutritional value while promoting safety as a priority. For instance, thickening food and fluids to reduce nutritional losses due to spillage from the oral cavity may make it too difficult for a child to manipulate the bolus safely for swallowing. Furthermore, seeking the advice of the dietician for densely caloric foods may also help to maximize feeding efficiency and safety, by reducing feeding time and effort (Strudwick 2009).

Sensorimotor therapy for feeding disorder

The use of oral sensorimotor therapy can be extremely useful in the management of feeding dysfunction, and is used frequently by speech and language pathologists (Gisel 2008, Strudwick 2009). Oral stimulation before feeding can help prepare the child for eating and drinking. Specifically designed chew toys can foster chewing skills through sensorimotor practice. Sometimes these children can suffer from oral hypersensitivity, and implementation of a desensitization program can be quite useful for decreasing oral defensiveness and improving feeding. For children who do not eat orally, sensorimotor therapy may improve oral hygiene and reduce drooling. Sensorimotor therapy can also help with abnormal tone that impacts on feeding ability. Alerting therapy for hypotonia may consist of fast tapping or brushing, whereas calming therapy for hypertonia involves massage and vibration. Oral sensorimotor therapy together with positioning and seating, and food delivery and texture, can benefit the child with CP and a feeding disorder; however, it should be performed frequently and consistently to be effective (Strudwick 2009).

Surgical treatment and other medical options

Children with CP and associated malnutrition and poor growth due to feeding disorder often require a gastrostomy feeding tube to supplement or replace oral feeds (Samson-Fang et al. 2003, Andrew, Parr, and Sullivan 2011). These children may also require surgical fundoplication because of aspiration risk. Although the medical team recommending a gastrostomy views it as a beneficial and often necessary treatment, families are commonly resistant to the idea initially. Some of their reservation originates from feelings of 'caregiver failure' and 'relinquishing normality' (Sullivan et al. 2004). Other concerns relate to the surgical risks of gastrostomy feeding tube placement. These include peritonitis, gastro-colic fistula, bowel perforation, hemorrhage, and anesthetic complications (Samson-Fang et al. 2003, Andrew, Parr, and Sullivan 2011). Later complications include stoma leakage, cellulitis, granulation tissue at the gastrostomy site, and displacement. Principal methods for placement are percutaneous endoscopic gastrostomy and surgical gastrostomy, with the risk of complications possibly varying depending on the exact technique. In children with CP, factors such as abdominal spasticity, diaphragmatic flaccidity, and scoliosis may further increase the complications associated with the procedure (Mollitt et al. 1985).

Clinical observation suggests that gastrostomy placement in children with CP seems to reduce feeding times, provide better nutrition, facilitate drug administration, and overall improve the quality of life for children and their families (Stevenson 2007). Although generally perceived by clinicians as a valuable option for treatment in these children, some controversy remains as to whether gastrostomy is overall beneficial because of surgical complications, costs, and emotional stress (Samson-Fang et al. 2003, Sleigh, Sullivan, and Thomas 2010). A Cochrane Review in 2010 revealed that no randomized controlled trials exist addressing these issues. A recent well-performed study supports the benefits of gastrostomy feeding for children and their caregivers (Sullivan et al. 2005). This was a prospective, longitudinal, multicenter trial involving children with severe CP who were followed for 6 and 12 months after gastrostomy placement. Results established significant improvements in weight and subcutaneous fat deposition after gastrostomy placement using reliable anthropometric measures. Almost all parents reported a significant improvement in their child's health and decreased time spent feeding after the intervention. A separate data analysis of the same cohort demonstrated significantly improved quality of life for

caregivers, and the measures of quality of life were no different from reference standards 1 year after gastrostomy placement (Sullivan et al. 2004).

Other non-invasive medical treatments for malnutrition and growth impairment in children with CP have not been well studied. In general, treatment of hypertonia with medications such as baclofen, diazepam, or trihexyphenidyl can be helpful when spasticity or dystonia interfere with feeding. Recently, a systematic review has provided clinical practice guidelines for the use of bisphosphonates, vitamin D, and calcium in children with CP who have low BMD and are at risk for fragility fractures (Fehlings et al. 2012). Overall, evidence suggests that bisphosphonates, when given with calcium and vitamin D to children with CP, are probably effective in improving BMD, and are possibly effective in reducing fragility fractures in children who have a history of fractures. However, when to use bisphosphonates in children with CP remains a matter of clinical judgment and the literature does not provide guidance for decision-making.

Supplemental vitamin D and calcium are also likely to be effective in improving BMD, but data were insufficient to make recommendations on their effectiveness to prevent fragility fractures (Frehlings et al. 2012). Anecdotally, routine use of supplemental calcium and vitamin D seem to minimize risk of fragility fractures. Another pharmacological option for consideration currently is growth hormone. Growth hormone has been reported to improve lean muscle mass, bone density, and possibly physical functioning. Based on a case report supporting these potential effects, and reported growth hormone deficiency in some children with CP, further research is indicated (Coniglio and Stevenson 1995, Shim et al. 2004).

Management algorithm

If a child is considered to be malnourished, an algorithm (Stevenson 2007) is available to facilitate decision-making for the management plan (Stevenson 2007). To begin, it is critical to use information from the history, physical examination, diet history, and anthropometry and apply it in the context of the child's nutritional status. This will enable detection of any factors that might be contributing to malnourishment, including feeding, medical, and environmental issues, some of which may be amenable to intervention. Incorporating a multidisciplinary approach is highly beneficial for obtaining this background information. Secondly, a target nutritional status should be identified, and may consist of either a specific weight or skinfold measurement (e.g. weight at which skinfold measurements are adequate). The interventions that follow will aim to work towards this nutritional goal. Initial strategy should include maximization of oral intake through increasing oral supplementation, adjusting textures, optimizing seating, and treating medical problems (e.g. constipation, GER). Lastly, supplemental nasogastric or gastrostomy feeds become necessary when the child's nutritional status remains inadequate despite ideal oral intake and medical management. Tube feedings, whether short-term or long-term, have become standard practice for children with disabilities.

Tube feedings may be administered by continuous drip, bolus, or a combination of both, and can be supplemental to oral feedings as well (Stevenson 2007). Whether the tube feedings provide 100% of caloric needs or less depends on the extent of malnourishment. The choice of formula or blended food recipe should be decided with the help of a nutritionist, and feeding volume should begin small and advance slowly to diminish vomiting risk. Tolerance and family schedule inform the timing and specific mode of delivery. Close monitoring of nutritional status is necessary to ensure that the child is indeed growing better, but also to avoid over-nutrition once children reach a state of adequate nourishment. It is important to note that as calories are reduced to control weight gain, nutrients are also reduced (protein, calcium, vitamin D, etc.) and thus require replacement through supplementation (Stevenson 2007).

Conclusion

In addition to motor dysfunction, other associated impairments and secondary conditions often afflict children with CP and contribute significantly to overall function and participation. Feeding disorder is a common associated impairment, particularly among the most individuals with severe motor impairment, affecting about 30% to 40% of children with CP (Sullivan et al. 2000, Fung et al. 2002). Malnutrition and poor growth are secondary conditions frequently resulting from feeding disorder. Other reasons for the differences in stature, weight, and body composition are responsible as well. The important relationship between nutrition, growth, health status, and quality of life is an area of research now requiring further attention.

In the assessment and management of children with CP, a multidisciplinary and 'biopsychosocial' strategy is needed to achieve safe and effective feeding (Craig et al. 2003, Sleigh 2005). In some cases, oral feeding will not be safe or it will not meet the child's hydration and nutrition needs. The options for intervention, including gastrostomy, should be discussed sensitively with parents, including them as part of the healthcare team. This general approach of inclusiveness and candor is essential in all aspects of care for children with CP and their families.

Currently, few reliable and valid assessment tools and formal management guidelines exist for the CP population. Consequently, clinicians must frequently rely on clinical judgment to establish a plan for the patient. Advancement in this field will facilitate more effective treatment guidelines or 'care maps' with creation of improved evaluation tools and treatment interventions. Although our knowledge of feeding disorder and malnutrition in patients with CP has advanced, further research is needed to better our understanding of the relationship with growth and other health outcomes.

REFERENCES

Andrew MJ, Parr JR, Sullivan PB (2011) Feeding difficulties in children with cerebral palsy. *Arch Dis Child Pract Ed*: 1–9.

Aronson E, Stevenson SB (2012) Bone health in children with cerebral palsy and epilepsy. *J Pediatr Health Care* 26: 193–9.

Arvedson J, Roger B, Buck G, et al. (1994) Silent aspiration prominent in children with dysphagia. *Int J Pediatr Otorhinolaryngol* 28: 73–81.

Baker-Henningham H, Hamadani JD, Huda SN, et al. (2009) Undernourished children have different temperaments than better-nourished children in rural Bangladesh. *J Nutr* 139: 1765–71.

Bandini LG, Schoeller DA, Fukagawa NK, Wykes LJ, Dietz WH (1991) Body composition and energy expenditure in adolescents with cerebral palsy or myelodysplasia. *Pediatr Res* 29: 70–7.

Blasco PA (1996) Drooling. In: Sullivan PB, Rosenbloom, L, editors. *Feeding the Disabled Child. Clinics in Developmental Medicine No. 140*. London: Mac Keith Press, pp. 92–105.

Blasco PA, Allaire JH (1992) Drooling in the developmentally disabled: management practices and recommendations. Consortium on Drooling. *Dev Med Child Neurol* 34: 849–62.

Bosma JF (1985) Postnatal ontogeny of performances of the pharynx, larynx, and mouth. *Am Rev Respir Dis* 131: S10–5.

Bosma JF (1992) Pharyngeal swallow: basic mechanisms, development, and impairment. *Head Neck Surg* 6: 225–75.

Brooks J, Day S, Shavelle R, Strauss D (2011) Low weight, morbidity, and mortality in children with cerebral palsy: new clinical growth charts. *Pediatrics* 128: 299–307.

Cameron, N (1986) The methods of auxological anthropology. In: Falkner F, Tanner JM, editors. *Human Growth: A Comprehensive Treatise*, 2nd edition, volume 3. New York: Plenum Press, pp. 3–46.

Campanozzi A, Capano G, Miele E, et al. (2007) Impact of malnutrition on gastrointestinal disorders and gross motor abilities in children with cerebral palsy. *Brain Dev* 29: 25–9.

Cass H, Wallis C, Ryan M, et al. (2005) Assessing pulmonary consequences of dysphagia in children with neurological disabilities: when to intervene? *Dev Med Child Neurol* 47: 347–52.

Chumlea WC, Guo SS, Steinbaugh ML (1994) Prediction of stature from knee height for black and white adults and children with application to mobility-impaired or handicapped persons. *J Am Diet Assoc* 94: 1385–8, 1391.

Clayden, G (1996) Constipation in disabled children. In: Sullivan PB, Rosenbloom, L, editors. *Feeding the Disabled Child. Clinics in Developmental Medicine No. 140*. London: Mac Keith Press, pp. 106–16.

Coniglio SJ, Stevenson RD (1995) Growth hormone deficiency in two children with cerebral palsy. *Dev Med Child Neurol* 37: 1013–5.

Craig GM, Scambler G, Spitz, L (2003) Why parents of children with neurodevelopmental disabilities requiring gastrostomy feeding need more support. *Dev Med Child Neurol* 45: 183–8.

Day SM, Strauss DJ, Vachon PJ, Rosenbloom L, Shavelle RM, Wu YW (2007) Growth patterns in a population of children and adolescents with cerebral palsy. *Dev Med Child Neurol* 49: 167–71.

Delaney AL, Arvedson JC (2008) Development of swallowing and feeding: prenatal through first year of life. *Dev Disabil Res Rev* 14: 105–17.

Dematteo C, Matovich D, Hjartarson, A, et al. (2005) Comparison of clinical and videofluoroscopic evaluation of children with feeding and swallowing difficulties. *Dev Med Child Neurol* 47: 149–157.

Dodds WJ, Stewart ET, Logemann JA (1990) Physiology and radiology of the normal oral and pharyngeal phases of swallowing. *AJR Am J Roentgenol* 154: 953–63.

Erasmus CE, Van Hulst K, Rotteveel LJ, et al. (2009) Drooling in cerebral palsy: hypersalivation or dysfunctional oral motor control? *Dev Med Child Neurol* 51: 454–9.

Fehlings D, Switzer L, Agarwal P, et al. (2012) Informing evidence-based clinical practice guidelines for children with cerebral palsy at risk of osteoporosis: a systematic review. *Dev Med Child Neurol* 54: 106–16.

Fung EB, Samson-Fang L, Stallings VA, et al. (2002) Feeding dysfunction is associated with poor growth and health status in children with cerebral palsy. *J Am Diet Assoc* 102: 361–73.

Giramonti KM, Kogan BA, Agboola OO, Ribons L, Dangman, B (2005) The association of constipation with childhood urinary tract infections. *J Pediatr Urol* 1: 273–8.

Gisel, E (2008) Interventions and outcomes for children with dysphagia. *Dev Disabil* 14: 165–73.

Grantham-McGregor, S (1995) A review of the studies of the effect of severe malnutrition on mental development. *J Nutr* 125: 2233S–8S.

Grantham-McGregor SM, Powell CA, Walker SP, Himes JH (1991) Nutritional supplementation, psychosocial stimulation, and mental development of stunted children: the Jamaican Study. *Lancet* 338: 1–5.

Griggs CA, Jones PM, Lee RE (1989) Videofluoroscopic investigation of feeding disorders of children with multiple handicap. *Dev Med Child Neurol* 31: 303–8.

Henderson RC, Gilbert SR, Clement ME, Abbas A, Worley G, Stevenson RD (2005) Altered skeletal maturation in moderate to severe cerebral palsy. *Dev Med Child Neurol* 47: 229–36.

Henderson RC, Grossberg RI, Matuszewski J, et al. (2007) Growth and nutritional status in residential center versus home-living children and adolescents with quadriplegic cerebral palsy. *J Pediatr* 151: 161–6.

Henderson RC, Lark RK, Gurka MJ, et al. (2002) Bone density and metabolism in children and adolescents with moderate to severe cerebral palsy. *Pediatrics* 110: e5.

Henderson RC, Lin PP, Greene WB (1995) Bone-mineral density in children and adolescents who have spastic cerebral palsy. *J Bone Joint Surg Am* 77: 1671–81.

Katona P, Katona-Apte J (2008) The interaction between nutrition and infection. *Clin Infect Dis* 46: 1582–8.

Kramer SS (1989) Radiologic examination of the swallowing impaired child. *Dysphagia* 3: 117–25.

Kramer SS, Eicher PM (1993) The evaluation of pediatric feeding abnormalities. *Dysphagia* 8: 215–24.

Krick J, Murphy-Miller P, Zeger S, Wright, E (1996) Pattern of growth in children with cerebral palsy. *J Am Diet Assoc* 96: 680–5.

Kuperminc MN, Gurka MJ, Houlihan CM, et al. (2009) Puberty, statural growth, and growth hormone release in children with cerebral palsy. *J Pediatr Rehabil Med* 2: 131–41.

Langermo D, Anderson J, Hanson D, Hunter S, Thompson P, Posthauer ME (2006) Nutritional considerations in wound care. *Adv Skin Wound Care* 19: 297–300.

Lohman TG, Roche AF, Martorell R (editors) (1988) *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books.

Marchand V, Motil KJ, NASPGHAN Committee on Nutrition (2006) Nutrition support for neurologically impaired children: a clinical report of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition. *J Pediatr Gastroenterol Nutr* 43: 123–35.

Mollitt DL, Golladay ES, Seibert JJ (1985) Symptomatic gastroesophageal reflux following gastrostomy in neurologically impaired patients. *Pediatrics* 75: 1124–6.

Morris SE (1982) *The Normal Acquisition of Oral Feeding Skills: Implications for Assessment and Treatment*. New York, NY: Therapeutic Media.

Pinnington L, Hegarty, J (2000) Effects of consistent food presentation on oral–motor skill acquisition in children with severe neurological impairment. *Dysphagia* 15: 213–23.

Qureshi MA, Vice FL, Taciak VL, Bosma JF, Gewolb IH (2002) Changes in rhythmic suckle feeding patterns in term infants in the first month of life. *Dev Med Child Neurol* 44: 34–9.

Redstone F, West JF (2004) The importance of postural control for feeding. *Pediatr Nurs* 30: 97–100.

Rogers BT, Senn KM (2008) Pediatric dysphagia. In: Accardo PJ, editor. *Capute and Accardo's Neurodevelopmental Disabilities in Infancy and Childhood*, 3rd edition. Baltimore, MD: Paul H Brookes, chapter 10.

Samson-Fang L, Butler C, O'Donnell M, AACPDM (2003) Effects of gastrostomy feeding in children with cerebral palsy: an AACPDM evidence report. *Dev Med Child Neurol* 45: 415–26.

Samson-Fang L, Fung E, Stallings VA, et al. (2002) Relationship of nutritional status to health and societal participation in children with cerebral palsy. *J Pediatr* 141: 637–43.

Samson-Fang L, Stevenson RD (1998) Linear growth velocity in children with cerebral palsy. *Dev Med Child Neurol* 40: 689–92.

Samson-Fang LJ, Stevenson RD (2000) Identification of malnutrition in children with cerebral palsy: poor performance of weight-for-height centiles. *Dev Med Child Neurol* 42: 162–8.

Schoendorfer N, Boyd R, Davies PS, et al. (2010) Micronutrient adequacy and morbidity: paucity of information in children with cerebral palsy. *Nutr Rev* 68: 739–48.

Sellers D, Mandy A, Pennington L, Hankins M, Morris C (2014) Development and reliability of a system to classify the eating and drinking ability of people with cerebral palsy. *Dev Med Child Neurol* 56: 245–51.

Shaw BNJ (2007) The respiratory consequences of neurological deficit. In: Sullivan PB, Rosenbaum L, editors. *Feeding the Disabled Child. Clinics in Developmental Medicine No. 140*. London: Mac Keith Press, chapter 5.

Shim ML, Moshang Jr T, Oppenheim WL, Cohen, P (2004) Is treatment with growth hormone effective in children with cerebral palsy? *Dev Med Child Neurol* 46: 569–71.

Sleigh, G (2005) Mothers' voice: a qualitative study on feeding children with cerebral palsy. *Child Care Health Dev* 31: 373–83.

Sleigh G, Sullivan PB, Thomas AG (2010) Gastrostomy feeding versus oral feeding alone for children with cerebral palsy. *Cochrane Database Syst Rev* 2: CD003943.

Spender QW, Cronk CE, Charney EB, Stallings VA (1989) Assessment of linear growth of children with cerebral palsy: use of alternative measures to height or length. *Dev Med Child Neurol* 31: 206–14.

Stallings VA, Charney EB, Davies JC, Cronk CE (1993a) Nutrition-related growth failure of children with quadriplegic cerebral palsy. *Dev Med Child Neurol* 35: 126–38.

Stallings VA, Charney EB, Davies JC, Cronk CE (1993b) Nutritional status and growth of children with diplegic or hemiplegic cerebral palsy. *Dev Med Child Neurol* 35: 997–1006.

Stallings VA, Cronk CE, Zemel BS, Charney EB (1995) Body composition in children with spastic quadriplegic cerebral palsy. *J Pediatr* 126: 833–9.

Stallings VA, Zemel BS, Davies JC, Cronk CE, Charney EB (1996) Energy expenditure of children and adolescents with severe disabilities: a cerebral palsy model. *Am J Clin Nutr* 64: 627–34.

Stevenson RD, Allaire JH (1996) The development of eating skills in infants and young children. In: Sullivan PB, Rosenbloom, L, editors. *Feeding the Disabled Child*. London: Mac Keith Press, pp. 11–22.

Stevenson RD (1995) Use of segmental measures to estimate stature in children with cerebral palsy. *Arch Pediatr Adolesc Med* 149: 658–62.

Stevenson RD (2007) Nutrition and growth. In: Accardo PJ, editor. *Capute and Accardo's Neurodevelopmental Disabilities in Infancy and Childhood,* 3rd edition, volume II: *The Spectrum of Neurodevelopmental Disabilities*. Baltimore, MD: Paul H Brookes, chapter 9.

Stevenson RD, Conaway M (2007) Growth assessment of children with cerebral palsy: the clinician's conundrum. *Dev Med Child Neurol* 49: 164.

Stevenson RD, Conaway M, Chumlea WC, et al. North American Growth in Cerebral Palsy, Study (2006) Growth and health in children with moderate-to-severe cerebral palsy. *Pediatrics* 118: 1010–8.

Stevenson RD, Hayes RP, Cater LV, Blackman JA (1994) Clinical correlates of linear growth in children with cerebral palsy. *Dev Med Child Neurol* 36: 135–42.

Strauss D, Ashwal S, Shavelle R, Eyman RK (1997) Prognosis for survival and improvement in function in children with severe developmental disabilities. *J Pediatr* 131: 712–7.

Strauss D, Shavelle R, Reynolds R, Rosenbloom L, Day S (2007) Survival in cerebral palsy in the last 20 years: signs of improvement? *Dev Med Child Neurol* 49: 86–92.

Strudwick W (2009) Oral motor impairment and swallowing dysfunction: assessment and management. In: Sullivan PB, editor. *Feeding and Nutrition in Children with Neurodevelopmental Disabilities*. London: Mac Keith Press, chapter 3.

Sullivan PB (2009a) Feeding and nutrition in neurodevelopmental disability: an overview. In: Sullivan, P, editor. *Feeding and Nutrition in Children with Neurodevelopmental Disabilities*. London: Mac Keith Press, chapter 1.

Sullivan PB (2009b) Gastrointestinal disorders: assessment and management. In: Sullivan PB, editor. *Feeding and Nutrition in Children with Neurodevelopmental Disabilities*. London: Mac Keith Press, chapter 6.

Sullivan PB, Juszczak E, Bachlet AM, et al. (2004) Impact of gastrostomy tube feeding on the quality of life of carers of children with cerebral palsy. *Dev Med Child Neurol* 46: 796–800.

Sullivan PB, Juszczak E, Bachlet AM, et al. (2005) Gastrostomy tube feeding in children with cerebral palsy: a prospective, longitudinal study. *Dev Med Child Neurol* 47: 77–85.

Sullivan PB, Juszczak E, Lambert BR, Rose M, Ford-Adams ME, Johnson, A (2002) Impact of feeding problems on nutritional intake and growth: Oxford Feeding Study II. *Dev Med Child Neurol* 44: 461–7.

Sullivan PB, Lambert B, Rose M, Ford-Adams M, Johnson A, Griffiths P (2000) Prevalence and severity of feeding and nutritional problems in children with neurological impairment: Oxford Feeding Study. *Dev Med Child Neurol* 42: 674–80.

Tahmassebi JF, Curzon ME (2003) Prevalence of drooling in children with cerebral palsy attending special schools. *Dev Med Child Neurol* 45: 613–7.

Worley G, Houlihan CM, Herman-Giddens ME, et al. (2002) Secondary sexual characteristics in children with cerebral palsy and moderate to severe motor impairment: a cross-sectional survey. *Pediatrics* 110: 897–902.

Yeargin-Alssopp M, Van Naarden Braun K, Doernberg NS, et al. (2008) Prevalence of cerebral palsy in 8-year-old children in three areas of the United States in 2002: a multisite collaboration. *Pediatrics* 121: 547–54.