OPEN

European Journal of Clinical Nutrition (2013) 67, S5–S8

www.nature.com/ejcn

REVIEW

Assessment of growth and nutrition in children with cerebral palsy

L Samson-Fang[1] and KL Bell[2,3]

This manuscript provides an update on the assessment of growth and nutrition in children with cerebral palsy and children with similar neurodevelopmental disabilities. Topics include the assessment of linear growth using segmental measures, avoidance of commonly used tools to assess nutritional status in typically developing children that are not valid in this population of children and how to use other nutritional assessment tools that have been developed specific to this population of children.

European Journal of Clinical Nutrition (2013) **67,** S5–S8; doi:10.1038/ejcn.2013.223

Keywords: neurodevelopmental disability; nutrition; growth; cerebral palsy; malnutrition

INTRODUCTION

Monitoring linear growth and nutritional status is critical in the health care of all children. In neurotypical children, assessment is performed using growth charts published by the World Health Organization and others. Although certain challenges exist such as variation across ethnicities, assessment is generally straightforward.

For children with cerebral palsy (CP) and similar neurodevelopmental disabilities, assessing growth and nutritional status is challenging. However, with careful consideration, the use of multiple clinical tools and repeated assessments over time, a clinician can approach nutritional care with confidence.

NUTRITION AND FEEDING HISTORY

The first step in growth and nutritional assessment is to review the patient and families perspective. Feeding is a critical social component of a child's life, and a child's growth may be viewed by families to be a reflection of their ability to nurture. Clinicians should review these issues in the supportive context of family-centered collaborative care. Important questions include the following:

1. Have the parents been concerned about their child's growth, weight, eating patterns or health?
2. Is the child able to eat and drink efficiently and safely? How long does it take for the child to eat meals? Is the feeding experience pleasant for the child and family?
3. Do the parents have to prepare the child's food in special ways to accommodate food intolerances, texture aversions, swallowing or chewing challenges?
4. What are the child's preferred foods and what does this tell about caloric density of foods and desired textures? Does the child's diet have enough variety to provide iron, calcium, other micronutrients and fibre?
5. Is fluid intake adequate and is urine output good?
6. Who feeds the child at school and when parents are not available?
7. How is the child positioned for feeds (for example, in a wheel chair, on a parents lap and in bed)?
8. How frequently does the child eat (for example, how many meals and snacks each day)?
9. How does illness have an impact on the child's feeding? Has the child experienced episodes of weight loss or dehydration?
10. Does the child take any caloric, protein, vitamin or herbal supplements?
11. If there are concerns, what interventions have been tried, how have these been tolerated and what has their impact been upon the child's nutritional status and health and upon the family?

The 3-day prospective diet diary is often used. In some research settings, diaries have performed well, whereas in others they have performed poorly with recorded caloric intake significantly in excess of the child's true intake.[1,2] Extensive parent training and provision of a food scale is required to obtain a reliable diary. In the clinical setting, with less rigorous parent training, diet diaries should be utilized with caution and interpreted primarily qualitatively (for example, what types and variety of foods does an individual eat) with less emphasis on quantitative interpretation.

MEDICAL HISTORY

Medical history should be reviewed with special attention to the underlying condition, level of functioning, degree of high tone, involuntary movements, activity level, respiratory (for example, signs of aspiration) and gastrointestinal issues (for example, vomiting, retching, constipation and diarrhea), medications that might have an impact on nutrition (for example, seizure medications) and clinical issues that might reflect nutritional compromise (for example, episodes of skin break down, poor wound healing, recurrent rashes, abnormal hair or nails and fractures).

ANTHROPOMETRIC MEASUREMENT AND INTERPRETATION

Anthropometric assessment is the cornerstone of evaluation. Quality control in measurement is paramount. Weights and

[1]University of Utah School of Medicine, Salt Lake City, UT, USA; [2]Queensland Cerebral Palsy and Rehabilitation Research Centre, Children's Nutrition Research Centre, Queensland Children's Medical Research Institute, The University of Queensland, Brisbane, Queensland, Australia and [3]Department of Paediatric Rehabilitation, Children's Health Queensland Hospital and Health Service, Herston, Brisbane, Queensland, Australia. Correspondence: Professor L Samson-Fang, University of Utah School of Medicine, PO Box 581289, Salt Lake City, UT 84158, USA.
E-mail: Lisa.Samson-Fang@HSC.Utah.EDU
This supplement was derived from a meeting with experts facilitated by Josephine Garvey and Annemiek Goedhart, held in Orlando in 2012.

heights obtained in certain settings (for example, outpatient settings not focused on nutritional issues, inpatient bed scales) are often not accurate. Weight should be obtained on a digital scale. For children unable to stand, a wheel chair scale can be utilized. It is critical that infants are naked when weighed. For older children, a thin set of clothes will suffice. Disposable undergarments should be dry. Recumbent length should be obtained in children under 2 years or older children who are unable to stand independently. Ideally, a recumbent measuring board should be used instead of a tape measure laid on an examining table. Standing height should be obtained using a stadiometer in those over 2 years who are able to stand. Recumbent length and height are unreliable if a child has contracture, high tone, scoliosis or poor cooperation interfering with the optimal positioning. A clinician might be tempted to use a flexible tape measure along the patient in a lying position ('crown rump length') or rely on a recumbent length in a suboptimal position, but these are not valid or reproducible measures. Using calipers, ulnar (forearm) length can be measured in the majority of children and published growth curves can be used to monitor linear growth.[3] Equations are available to calculate height from ulnar length, knee height and tibial length in some age groups (see Table 1).[3–6] The clinician must have the needed equipment, understand the landmarks and techniques of measure, and recognize that each equation is validated in a specific age, gender and diagnostic group. For example, it would not be appropriate to use an equation validated in a 12-year old boy with CP to calculate the height of a 15-year old boy with CP or a 12-year old boy with CP in combination with a genetic syndrome that has an impact on skeletal proportions. The derived equations to calculate height include a degree of error, which affects the ability to use calculated heights in certain ways. For example, calculated heights should not be used to assess body mass index, as the error is magnified when squared. Assessing growth velocity may also be unreliable when using calculated heights.[7]

In the child with CP or similar neurodevelopmental disability, interpreting anthropometric data has significant caveats. If a reliable height or a calculated height can be obtained, the clinician can plot it on a World Health Organization or, if over 2 years old, locally used growth chart. Debate exists with regard to the degree of short stature, which can be attributed to the underlying impacts of neurological function on growth. Regardless, a lack of growth or deviation from the growth curve through time should be considered abnormal. Similarly, although weight for age may be reduced (for example, because of short stature and/or reduced muscle mass), longitudinally, weight should track along an established percentile.

When faced with such challenges, it is tempting for a clinician to search for a population-derived growth chart. Although growth charts specific to children with CP have been published, the use of these charts to evaluate the growth of a specific child with CP is not necessarily the optimal solution and the use of such population-derived growth charts is not recommended by the US Centers For Disease Control (and Prevention). These population-specific growth charts in general describe 'what is' and not necessarily ideal growth. The use of population-based growth curves may give the clinician some perspective on the broad range of growth seen in CP, but growth along a lower percentile of such a chart should raise alarm for the clinician as opposed to reassure the clinician that the child is 'growing as expected'.

In neurotypical children, clinicians generally use weight for height, percentage ideal body weight and/or body mass index in comparison with published age/gender normative data to determine whether a child's fat stores are low, normal or excessive. The validity of these tools is highly suspected in children with CP or similar neurodevelopmental disabilities. They should be used with caution if at all. Differences in body composition including altered muscle and bone mass makes these assessment tools unreliable indicators of nutritional stores.[8–10] Using them in a traditional manner in children with reduced muscle mass will inadvertently result in a child whose nutritional status 'on paper' looks healthy, but who in reality has excessive fat stores. Measurement of skinfolds is another tool the clinician can use to assess fat stores. Although a more direct measure of fat storage, skinfold interpretation can also be a challenge in that children with CP appear to store fat in unique distribution (more centrally such as in the abdominal cavity).[10,11] Skinfolds may underestimate the child's fat stores. In the setting that the child's fat stores appear fairly robust based upon skinfold measures in comparison with

Table 1. Formulas for estimating stature from segmental measures

Equations for estimation of stature from segmental measures		
Segmental measure	*Equation to estimate stature (S) (cm)*	*SE of estimate (cm)*
Children with CP (age: birth –12 years)[4]		
Upper arm length, UAL	$S = (4.35 \times \text{UAL}) + 21.8$	1.7
Tibial length, TL	$S = (3.26 \times \text{TL}) + 30.8$	1.4
Knee height, KH	$S = (2.69 \times \text{KH}) + 24.2$	1.1
Estimation of stature (S) from knee height (KH) in children aged 6–18 years		
Race and gender	*Equation to estimate stature (cm)*	*SE of estimate (cm)*
Derived in typically developing children, validity demonstrated in a small group of children with CP[5,6]		
White males	$S = (2.22 \times \text{KH}) + 40.54$	4.21
Black males	$S = (2.18 \times \text{KH}) + 39.6$	4.58
White females	$S = (2.15 \times \text{KH}) + 43.21$	3.90
Black females	$S = (2.02 \times \text{KH}) + 46.59$	4.39
Estimation of height (H) from ulna length (U)		
Gender	*Equation to estimate height (cm)*	*RMSE*
Derived in typically developing children aged 5–19 years. Not validated in children with CP. Performed better in typically developing children than prior ulnar equations, which had shown validity in CP (construct validity)[3]		
Males	$H = 4.605\text{U} + 1.308\,\text{A} + 28.003$ (A = age)	3.896
Females	$H = 4.459\text{U} + 1.315\,\text{A} + 31.485$ (A = age)	3.785

Abbreviation: RMSE, root mean-square error.

normative data (for example, multiple skinfolds greater than 10th percentile for age), the clinician may feel reassured that the child's lower weight for height is likely to be 'non-nutritional' in nature (that is, reflects reduced muscle or bone mass). However, given a tendency to store fat centrally, reduced peripheral skinfolds may not necessarily mean low fat stores. In children with CP, equations to calculate percentage body fat from multiple skinfolds have been developed and their validity is being evaluated in ongoing studies.[11] Using such equations will allow the clinician to better assess total body fat storage from peripheral skinfold measures.

If a center has the availability of dual X-ray absorptiometry (DEXA) using a machine that has pediatric normative data, DEXA will allow for assessment of body composition including assessment of lean and non-lean (fat) stores. DEXA is not reliable in some children because of the presence of hardware or the positioning difficulties.[12] Interpretation requires careful consideration of clinical factors such as the presence of severe stunting, and delayed/precocious pubertal development as these have an impact on normative expectations. Regardless, if DEXA can be obtained and interpreted with confidence, it may be one of the best ways to ensure that a child's fat stores are neither deplete nor excessive.

For assessment of fat stores in children with CP, bioelectrical impedance analysis holds promise.[13,14] It is low cost, widely available, portable, quick and generally easy to perform. However, in this population, performing bioelectrical impedance analysis presents challenges (for example, impacts of hardware, movement artifact and hydration concerns) and equations derived to convert impedance to lean body mass in children with CP have not yet demonstrated the validity desired for broad clinical use.

LABORATORY ANALYSIS

- Inadequate intake is the most common reason for growth and nutritional issues in children with CP but clinicians should be vigilant to identify contributing medical problems. If history suggests a medical concern (for example, celiac disease, cystic fibrosis or inflammatory condition), lab tests should be obtained to evaluate for the condition.
- If a child's linear growth is the primary concern, screening for hormonal abnormalities (for example, levels of thyroid-stimulating hormone and serum markers of growth hormone activity (insulin-like growth factor 1 and insulin-like growth factor 1-binding protein 3) are indicated.
- It is common to measure albumin and pre-albumin. Given the primarily caloric nutritional compromise this population experiences, albumin and pre-albumin are generally normal.[15,16] Their normality should not be interpreted as evidence of adequate nutritional status. Low values may be nutritional (chronic low-protein intake) or non-nutritional (the fluid shifts of an acutely ill patient, protein losses in urine or stool or liver disease).
- Serum electrolytes do not necessarily reflect intake. For example, chronic-deficient intake of calcium will not have an impact on serum calcium as calcium will be mobilized from bone stores to maintain serum equilibrium.
- Zinc and carnitine levels are often obtained and carnitine levels are particularly important in the child taking valproic acid. If low, supplements should be given but the extent to which serum levels reflect body stores is debated.
- Anecdotally, vitamin D (measured with a 25 hydroxy vitamin D level) and iron stores (measured with a serum ferritin along with C-reactive protein to exclude artificial elevation by concurrent inflammation) may yield low values in a significant percentage of patients. Normalizing vitamin D values is important, given the multiple risks to bone health in children with CP. If ferritin is low, iron stores should be repleted but consideration should be given to the possibility that deficiency may reflect chronic unrecognized oesophagitis/gastritis.
- Many children will have alterations in their red blood cell indices because of the impact of medications (for example, elevated mean corpuscular volume from phenobarbital) and it is important for the clinician to ensure that nutritional contributions (for example, low vitamin B12 or folate) have been excluded.

IMAGING

DEXA was discussed above for assessment of fat storage. Most children with CP and significant nutritional compromise will be found to have a low bone density on DEXA, although the extent to which its measurement has an impact on therapeutic approach is debatable.[17] Other imaging modalities have a limited role in evaluating nutritional status but are important in evaluating feeding or gastrointestinal concerns complicating the nutritional picture. If unclear from history, further investigations might be indicated to clarify the presence of constipation. An upper gastrointestinal series may be helpful in providing information about anatomic issues causing feeding intolerance (for example, superior mesenteric artery syndrome), poor gastric emptying, and severe gastroesophageal reflux. A modified barium swallow might be obtained to better understand the child's mechanical eating abilities including optimal textures and positioning. If a feeding tube and/or fundoplication is being considered, other evaluations may be important in planning the surgery (for example, pH probe).

DETERMINING NEED FOR INTERVENTION

Nutritional assessment is challenging in children with CP. The keys to success are collaboration with families, use of multiple methodologies (for example, feeding history, anthropometry, multiple skinfolds, DEXA (when available) and serum nutritional markers) and longitudinal repeated assessments. Given the complexity and the clinical variability among patients, there are no strict criteria to define 'malnutrition'. From a big picture perspective, the child and family should enjoy a safe eating experience, diet should be varied enough to provide micronutrients and fibre and the child should not experience dehydration. Children should gain weight and grow. Plateaus in growth or 'deviation from established pattern' is worrisome. Fat stores should be in the broad range of normal. Micronutrient intake (assessed by qualitative diet history supplemented by select serum levels) should be adequate. In many cases, the clinician will be able to reassure a family that their child with CP is thriving. If a child's nutritional situation is at risk (for example, not gaining weight over time, linear growth deviating from an established curve, fat stores appear low, periods of dehydration and poor intake of micronutrients), a collaborative family-centered, longitudinal, 'big picture' approach lays the foundation for successful intervention.

This supplement is provided as a professional service by the Paediatric Division of Nutricia Advanced Medical Nutrition.

CONFLICT OF INTEREST

KLB and LSF have received lecture fees from Danone. Spouse of LSF is investor and Chief Scientific officer of Veritract (specialized feeding tube). KLB has received grant support from the national Health and Medical Research Council (NHMRC 569605) Australia.

REFERENCES

1 Stallings VA, Zemel BS, Davies JC, Cronk CE, Charney EB. Energy expenditure of children and adolescents with severe disabilities: a cerebral palsy model. *Am J Clin Nutr* 1996; **64**: 627–634.

2 Walker JL, Bell KL, Boyd RN, Davies PS. Validation of a modified three-day weighed food record for measuring energy intake in preschool-aged children with cerebral palsy. *Clin Nutr* 2013; **32**: 426–431.
3 Gauld LM, Kappers J, Carlin JB, Robertson CF. Height prediction from ulna length. *Dev Med Child Neurol* 2004; **46**: 475–480.
4 Stevenson RD. Use of segmental measures to estimate stature in children with cerebral palsy. *Arch Pediatr Adolesc Med* 1995; **149**: 658–662.
5 Chumlea WC, Guo SS, Steinbaugh ML. 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* 1994; **94**: 1385–1388, 91; quiz 9–90.
6 Hogan SE. Knee height as a predictor of recumbent length for individuals with mobility-impaired cerebral palsy. *J Am Coll Nutr* 1999; **18**: 201–205.
7 Samson-Fang L, Stevenson RD. Linear growth velocity in children with cerebral palsy. *Dev Med Child Neurol* 1998; **40**: 689–692.
8 Samson-Fang LJ, Stevenson RD. Identification of malnutrition in children with cerebral palsy: poor performance of weight-for-height centiles. *Dev Med Child Neurol* 2000; **42**: 162–168.
9 Spender QW, Cronk CE, Stallings VA, Hediger ML. Fat distribution in children with cerebral palsy. *Ann Hum Biol* 1988; **15**: 191–196.
10 Kuperminc MN, Gurka MJ, Bennis JA, Busby MG, Grossberg RI, Henderson RC *et al.* Anthropometric measures: poor predictors of body fat in children with moderate to severe cerebral palsy. *Dev Med Child Neurol* 2010; **52**: 824–830.
11 Gurka MJ, Kuperminc MN, Busby MG, Bennis JA, Grossberg RI, Houlihan CM *et al.* Assessment and correction of skinfold thickness equations in estimating body fat in children with cerebral palsy. *Dev Med Child Neurol* 2010; **52**: e35–e41.
12 Henderson RC, Lark RK, Renner JB, Fung EB, Stallings VA, Conaway M *et al.* Dual X-ray absorptiometry assessment of body composition in children with altered body posture. *J Clin Densitom* 2001; **4**: 325–335.
13 Bell KL, Boyd RN, Walker JL, Stevenson RD, Davies PS. The use of bioelectrical impedance analysis to estimate total body water in young children with cerebral palsy. *Clin Nutr* 2013; **32**: 579–584.
14 Veugelers R, Penning C, van Gulik ME, Tibboel D, Evenhuis HM. Feasibility of bioelectrical impedance analysis in children with a severe generalized cerebral palsy. *Nutrition* 2006; **22**: 16–22.
15 Lark RK, Williams CL, Stadler D, Simpson SL, Henderson RC, Samson-Fang L *et al.* Serum prealbumin and albumin concentrations do not reflect nutritional state in children with cerebral palsy. *J Pediatr* 2005; **147**: 695–697.
16 Ohwada H, Nakayama T. The distributions and correlates of serum albumin levels in institutionalised individuals with intellectual and/or motor disabilities. *Br J Nutr* 2008; **100**: 1291–1296.
17 Henderson RC, Berglund LM, May R, Zemel BS, Grossberg RI, Johnson J *et al.* The relationship between fractures and DXA measures of BMD in the distal femur of children and adolescents with cerebral palsy or muscular dystrophy. *J Bone Miner Res* 2010; **25**: 520–526.