Rheumatology 2002;41:1172–1177

Paediatric Rheumatology/Series Editor: P. Woo

Measuring the nutritional status of children with juvenile idiopathic arthritis using the bioelectrical impedance method

C. M. Lofthouse†, F. Azad, E. M. Baildam and A. K. Akobeng

Departments of Gastroenterology and Rheumatology, Booth Hall Children's Hospital, Charlestown Road, Blackley M9 7AA, UK

Abstract

Objective. To assess the nutritional status of children with juvenile idiopathic arthritis (JIA) using anthropometric measurements and bioelectrical impedance.

Methods. Twenty-two consecutive JIA patients (seven pauciarticular, 15 polyarticular) attending the rheumatology clinic at Booth Hall Children's Hospital were compared with 22 age- and sex-matched controls attending the accident and emergency department of the same hospital. There were no patients with systemic-onset JIA in the cohort. Height, weight, head circumference and skinfold thickness at four sites (biceps, triceps, subscapular and suprailiac) were measured. Regression equations were used to calculate body fat as a percentage of weight, and arm muscle circumference. In addition, bioelectrical impedance measurements were made using a Holtain body composition analyser. These measurements were then used to calculate the total body water, which could be used as an indirect estimate of the lean body mass.

Results. Of the JIA patients, 22.7% were below the third centile for height, 18.1% had a weight less than the third centile. Mid-arm circumference was below the fifth centile in 36.4% of the patients. Patients with polyarticular disease showed significantly more signs of malnutrition than patients with pauciarticular disease. In the polyarticular group, comparison with controls revealed significant P values for reduction in height (0.047), weight (0.045), mid-arm circumference (0.002), arm muscle circumference (0.012), percentage body fat (0.008) and total body water (0.031).

Conclusions. In view of the findings of lower total body water, indicating lower lean mass, in more nutritionally deprived JIA patients (as deduced by the other physical parameters measured), we conclude that bioelectrical impedance is a useful adjunct to anthropometric measures in assessing nutritional status in JIA.

KEY WORDS: Bioelectrical impedance, Juvenile idiopathic arthritis, Nutritional assessment, Total body water.

There is evidence that children with juvenile idiopathic arthritis (JIA) may have impaired growth [1–6]. Factors such as anorexia, cytokine-mediated depletion of protein stores, anaemia, gastrointestinal side-effects of anti-rheumatic medications and unprescribed diet modifications may all contribute to the nutritional disturbances observed in these patients [7]. Several studies have demonstrated that protein–energy malnutrition is very prevalent amongst these patients, with rates that vary between 20 and 40% [8–13].

Assessment of the state of nutrition is made difficult by the number of factors that contribute to nutritional well-being (protein stores, fat stores, trace elements, etc.).

Many of the nutritional parameters are often more difficult to interpret in the paediatric population with rheumatic diseases than in malnourished patients with non-inflammatory disease. Hip or knee contractures can make it difficult to obtain an accurate height. Measures of somatic protein stores (arm circumference, arm muscle circumference) may also be altered by muscle atrophy secondary to disuse in cases of upper extremity arthritis.

Visceral protein stores, such as albumin, prealbumin [14] and retinol binding protein, are also negatively influenced by active inflammation that drives protein synthesis towards the manufacturing of acute-phase reactants [15]. The Seltzer index [16] (relying on the

Submitted 3 August 2001; revised version accepted 29 April 2002.

Correspondence to: E. Baildam.

†Dr Lofthouse is deceased.

albumin concentration and lymphocyte count) and the nutritional assessment of Blackburn [17] are more useful, yet there is no scoring system which can adequately combine multiple measurements into a more accurate 'nutritional index' that can be calculated. The aim of this study was to determine the nutritional status of children with JIA using anthropometric parameters and bioelectrical impedance analysis.

Subjects and methods

Subjects

Twenty-two consecutive patients attending the rheumatology clinic at Booth Hall Children's Hospital were studied from May to July of 1998. There were five males and 17 females with JIA, ranging in age from 3 to 14 yr. Twenty-two age- and sex-matched children attending the accident and emergency department were used as a control group. Children who had previous surgery, those with foci of active infection or inflammation and those with any other pathological condition or chromosomal abnormality were excluded from the control group. There were no patients with systemic-onset JIA included in this study as none were seen in the consecutive patients attending during the study period. Informed consent was obtained from the parents of the children.

Bioelectrical impedance analysis

Since 1940, changes in hydration have been known to cause changes in total body resistance and capacitive reactance. The relationship between total body water and electrical impedance was delineated by Hoffer *et al.* in 1969 [18]. Reactance is a measure of cell membrane capacitance and an indirect measure of the intracellular volume or body cell mass. Body fat, total body water and extracellular water offer resistance to electrical current; only cell membranes offer capacitive reactance. Because fat tissue cells are not surrounded by cell membranes, reactance is not affected by the quantity of body fat.

Single-frequency, dual-frequency and multi-frequency methods for the analysis of bioelectrical impedance have been developed and tested in various health and disease populations.

As shown below, body density is derived from skinfold thickness according to the formulae of Durnin and Rahaman [19]. Body fat as a percentage of weight is calculated by the formula of Siri [20]. Total body water is calculated by the formula of Hoffer *et al.* [18]. Total body water is then used to estimate lean mass. The body composition can thus be separated into estimates of lean and fat mass.

Methods

Anthropometric measurements were performed in clinic using the same equipment and the same observer. Height was measured with a stadiometer (Holtain, Crymych, UK) to the nearest 0.01 cm and weight to the nearest 0.01 kg [Weylux; Autoweigh scale (UK), Halifax, UK]. Head circumference was measured with a non-stretchable tape to the nearest 0.01 cm and mid-arm circumference was measured. Skinfold thickness was determined to the nearest 0.2 cm at the triceps and biceps, in the suprailiac area and just below the angle of the scapula on the left side using a skinfold caliper (Holtain). The Holtain skinfold caliper was used throughout and the measurement recorded on each occasion was the average of three readings.

Body density was calculated from the regression equations of Durnin and Rahaman [19]. For boys, density $= 1.1533 - 0.0643 \times \log$ sum of the skinfold thicknesses at four sites. For prepubertal boys, density $= 1.1690 - 0.0788 \times \log$ sum of the skinfold thicknesses at four sites. For girls, density $= 1.1369 - 0.0598 \times \log$ sum of the skinfold thicknesses at four sites. For prepubertal girls, density $= 1.2063 - 0.0999 \times \log$ sum of the skinfold thicknesses at four sites. Calculation of body fat as a percentage of weight was based on the equation given by Siri [20]: $((4.95/\text{body density}) - 4.5) \times 100$. Arm muscle circumference (AMC) was then calculated using the formula: AMC = mid-arm circumference (MAC) $-$ ($0.314 \times$ triceps skinfold) [21–23].

Bioelectrical impedance measurements were made using a Holtain body composition analyser. Electrodes were applied to the dorsum of the right wrist and to the flexor surface of the right ankle according to the manufacturer's instructions. An 800 μA, 50 kHz alternating current was passed, allowing the impedance to be measured [24, 25]. Total body water was then calculated using the regression equation of Hoffer *et al.* [18]: total body water $= 0.79 + 0.55$ (height^2/impedance).

Statistical analysis

The one-sample Kolmogorov–Smirnov goodness-of-fit test showed the nutritional indices that were studied to be normally distributed. Therefore, Student's *t*-test was used to calculate any statistical significance between data in the different patient groups.

Results

The 22 JIA patients comprised five males and 17 females; seven patients had pauciarticular JIA and 15 polyarticular JIA. Eight of the 22 patients were being treated with steroids.

Anthropometric measurements showed a trend towards small body size in the variables studied. Of the children with JIA, 22.7% were below the third centile for height *vs* 0% of controls and 45.4% were below the 10th centile *vs* 9% of controls; 18.1% had a weight less than the third centile *vs* 0% of controls and 50% were below the 10th centile *vs* 9% of controls. Although these results strongly suggest that JIA patients tend to be shorter and weigh less than the healthy population, the observed differences are not significant ($P = 0.096$ and 0.075 respectively).

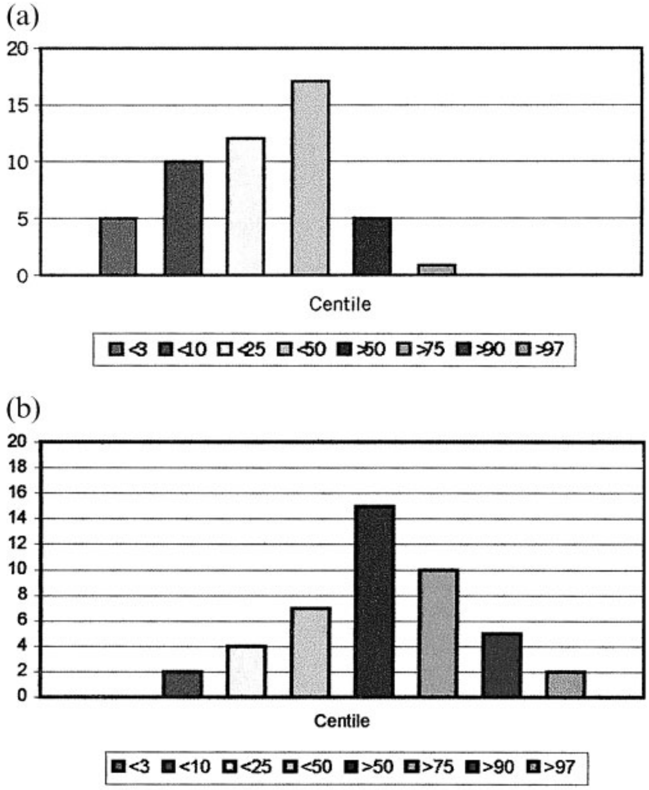

FIG. 1. Centile distribution of height: (a) JIA patients, (b) control group. <3, <10, <25, <50, >50, >75, >90, >97.

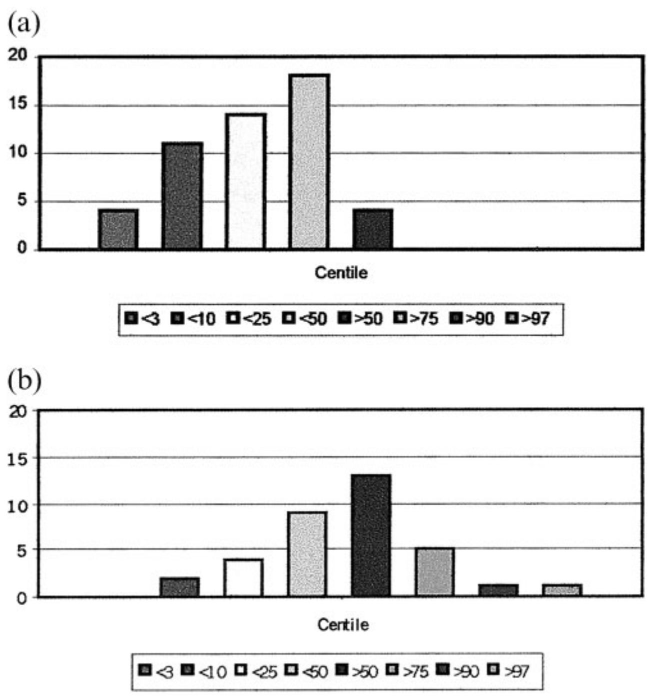

FIG. 2. Centile distribution of weight: (a) JIA patients, (b) control group. <3, <10, <25, <50, >50, >75, >90, >97.

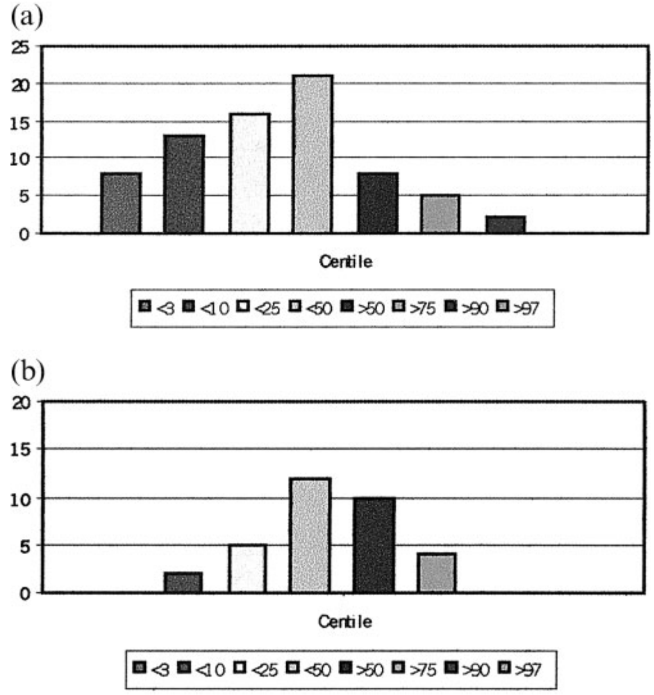

FIG. 3. Centile distribution of MAC: (a) JIA patients, (b) control group. <3, <10, <25, <50, >50, >75, >90, >97.

Centile distribution of height

Of the 22.7% of JIA patients who were below the third centile for height, 20% were on steroids and 80% had polyarticular disease. Similarly, of the 18.1% of patients that fell below the third centile for weight, 50% were on steroids and 75% had polyarticular disease (Fig. 1a and b).

Centile distribution of weight

There was no significant difference in weight between the JIA patients and controls (Fig. 2a and b).

Centile distribution of MAC

Results are shown in Fig. 3a and b. The percentage of patients who were below the fifth centile for MAC was 36.4. Out of this number, 37.5% were on steroids and 87.5% had polyarticular disease; 62.5% had upper extremity arthritis and therefore disuse atrophy could be a contributory factor to the low MAC observed. There were no children in the control group who had an MAC less than the fifth centile. Further statistical analysis revealed that there was a significant difference between the JIA patients and controls for MAC [$P < 0.01$ ($P = 0.002$)] and AMC [$P < 0.01$ ($P = 0.009$)]. AMC can be affected by muscle atrophy in JIA, but we felt it was necessary to measure it in the control and JIA populations to try to compare muscle mass directly at one site between the two groups.

The remaining variables also showed statistically significant differences between JIA and control subjects. These

TABLE 1. Nutritional indices of all JIA patients and controls

	JIA patients (n = 22)		Controls (n = 22)		
	Mean	S.D.	Mean	S.D.	P
Height (cm)	129.6	18.5	140.2	22.3	0.096
Weight (kg)	28.0	9.4	34.6	14.2	0.075
Head circumference (cm)	52.4	1.6	53.1	2.1	0.205
MAC (cm)	18.1	2.4	21.0	3.2	0.002*
AMC (cm)	21.2	4.3	26.1	5.0	0.001*
Body fat (%)	15.0	2.3	17.1	2.8	0.009*
Total body water (kg)	14.3	5.5	18.6	7.3	0.033*

*Significant.

TABLE 2. Nutritional indices of polyarticular patients and controls

	Polyarticular ($n = 15$)		Controls ($n = 15$)		
	Mean	S.D.	Mean	S.D.	P
Height (cm)	136.1	16.9	147.5	19.6	0.047*
Weight (kg)	30.5	8.9	38.9	13.4	0.045*
Head circumference (cm)	52.6	1.6	53.7	2.1	0.132
MAC (cm)	18.7	2.5	22.0	3.7	0.002*
AMC (cm)	15.5	2.3	17.9	2.6	0.012*
Body fat (%)	22.9	3.3	27.3	4.9	0.008*
Total body water (kg)	15.2	6.1	20.6	7.1	0.031*

*Significant.

TABLE 3. Nutritional indices of pauciarticular patients and controls

	Pauciarticular (n = 7)		Controls (n = 7)		
	Mean	S.D.	Mean	S.D.	P
Height (cm)	115.9	14.5	124.4	20.5	0.389
Weight (kg)	22.6	8.7	25.6	12.3	0.605
Head circumference (cm)	51.8	1.5	51.8	1.8	0.987
MAC (cm)	16.9	2.0	19.0	3.3	0.175
AMC (cm)	14.0	2.1	15.5	2.8	0.275
Body fat (%)	17.7	4.1	23.5	4.6	0.027*
Total body water (kg)	12.5	3.6	14.2	5.8	0.514

*Significant.

TABLE 4. Nutritional indices of patients on steroids and controls

	Patients on steroids ($n = 8$)		Controls ($n = 8$)		
	Mean	S.D.	Mean	S.D.	P
Height (cm)	125.8	18.2	137.7	20.2	0.163
Weight (kg)	26.5	8.4	33.0	13.9	0.201
Head circumference (cm)	52.6	1.4	53.2	1.9	0.407
MAC (cm)	17.6	2.1	20.7	3.2	0.018*
AMC (cm)	14.8	1.8	16.9	2.8	0.046*
Body fat (%)	20.1	4.7	25.8	6.4	0.028*
Total body water (kg)	13.3	5.0	18.0	6.9	0.082

*Significant.

TABLE 5. Nutritional indices of patients not on steroids and controls

	Patients not on steroids (n = 14)		Controls (n = 14)		
	Mean	S.D.	Mean	S.D.	P
Height (cm)	133.4	18.9	142.7	25.0	0.341
Weight (kg)	29.4	10.5	36.2	15.0	0.235
Head circumference (cm)	52.1	1.8	53.0	2.5	0.361
MAC (cm)	18.6	2.7	21.4	3.3	0.044*
AMC (cm)	15.3	2.7	17.3	3.0	0.103
Body fat (%)	22.4	3.6	26.4	3.5	0.015*
Total body water (kg)	15.3	5.9	19.2	4.8	0.048*

*Significant.

were a lower body fat percentage ($P < 0.01$) and lower total body water ($P < 0.05$) in JIA patients *vs* controls.

Nutritional indices

Results are shown in Tables 1–5.

Comparison of polyarticular and pauciarticular subgroups with controls. When polyarticular JIA patients ($n = 15$) were compared with age- and sex-matched controls, it was found that, with the exception of head circumference ($P = 0.132$), significantly lower values of all measurements were found in the polyarticular subgroup ($P < 0.05$). Similarly, pauciarticular patients ($n = 7$) were compared with age- and sex-matched controls. In this subgroup, only the body fat percentage was significantly lower than in controls ($P = 0.027$).

Patients on steroids vs those not on steroids. JIA patients on steroids (eight patients) showed statistically significantly lower values for MAC ($P = 0.018$), AMC ($P = 0.046$) and body fat percentage ($P = 0.028$) compared with patients not receiving steroids. The remaining variables showed no significant differences ($P > 0.05$).

Patients not on steroids (14 patients) showed significantly lower measurements than the controls for MAC ($P = 0.044$), body fat percentage ($P = 0.015$) and total body water ($P = 0.048$). Unexpectedly, AMC did not differ significantly between these two groups ($P = 0.103$).

Comparisons between male ($n = 5$) and female ($n = 17$) patients and between pauciarticular ($n = 7$) and polyarticular ($n = 15$) presentations were not performed, as the subgroup sizes were too small.

Discussion

We have shown that an impaired nutritional state may be present in some patients with JIA, especially those with polyarticular disease. In 1999, Knops *et al.* [7] showed that nutritional status in patients with systemic JIA was diminished compared with controls for height ($P < 0.01$) and fat-free mass (FFM) ($P < 0.03$). Oligo- and polyarticular patients with JIA had normal height and FFM. After correction for body weight and FFM, the resting energy expenditure (REE) in systemic JIA patients was 18% higher per kg body weight (196 *vs* 162 kJ/kg/day, $P < 0.01$). Oligo- and polyarticular JIA patients had 8% higher values for REE per kg body weight of FFM, but this was not statistically significant. The data suggest that assessment of individual energy requirements should include correction for FFM in the treatment of patients with JIA. By contrast, the present study has shown significant differences between polyarticular patients and controls. Systemic-onset JIA patients were not included in our study.

In 1986, Johansson *et al.* [10] found that impaired growth occurred only in patients with systemic or polyarticular onset of disease. He looked at the nutritional status of 26 girls with JIA and of healthy controls, with emphasis on nutrients involved in inflammatory processes. Children with JIA had decreased plasma selenium compared with controls. This is in accordance with previous reports on adult arthritic patients [26]. Glutathione peroxidase activity in blood was slightly depressed in JIA. The decrease in blood glutathione peroxidase was more pronounced in patients with high to medium disease activity. Our finding that height and weight were lowest in children with polyarticular JIA is also consistent with the study of Bacon *et al.* [27], who also demonstrated that systemic JIA patients had both mean caloric and mean vitamin E intakes below the recommended daily allowance.

Although differences in mean weight and height between the JIA patients and controls were not statistically significant, there was a trend for all measurements, apart from occipitofrontal circumference, to be lower in JIA than in controls. Lack of significance could be due to the small number studied; a much larger study is required to assess nutrition fully in pauciarticular patients. Bernstein *et al.* [28] and Henderson and Lovell [12] also found height and weight measurements to be lower in JIA children.

The total body water, AMC and MAC were lower in the arthritic patients, indicating lower muscle mass and reduced somatic protein stores. This is also compatible with the findings of Henderson and Lovell [12], who showed that 70% of JIA patients had protein–energy malnutrition. These measures of protein stores may be affected by muscle disuse atrophy secondary to upper extremity arthritis. Johansson *et al.* [10] published on nutritional status in girls with chronic arthritis, finding that 20–50% of children with JIA have protein–energy malnutrition. The body fat percentage was also significantly lower in JIA patients than in controls, showing that they have reduced fat mass.

The present study tended to show that there was no real difference in growth retardation between patients on steroids and those not on them. It is unlikely that the growth retardation is due solely to steroid use, as 53.3% of our patients with polyarticular disease had not been treated with steroids. Furthermore, decreased heights have been reported in JIA patients not receiving steroids who have growth factor abnormalities [29, 30].

The maintenance of nutritional homeostasis is fundamental to normal health. In children, the added demands of growth makes this goal more difficult to achieve, but in those with JIA it is not surprising that their state of nutrition is lower than that of the normal population.

Decreased intake of calories and selected nutrients in children may result from a number of factors [31]. Inflammatory mediators might cause appetite suppression, abnormal absorption or decreased utilization of nutrients. Fever could increase nutrient requirements. Children experiencing depression or severe pain because of their disease may become anorexic.

The measurement of skinfold thickness as a means of assessing body fat percentage may be unreliable because the distribution of fat within the body may vary with sex, age and race [32]. The technique requires practice to reduce the error to the minimum possible with a single observer. It has been shown that it is not possible in infants to predict total body fatness from the measurement of skinfold thickness to an appropriate level of accuracy [33], and the equations derived for use in childhood and adolescence are often extremely population-specific. In contrast with anthropometric methods, bioelectrical impedance analysis is more accurate and much less population-specific. However, as the technique theoretically predicts total body water, care should be taken in assuming that the same degree of precision can be applied to derived estimates of FFM. The conversion of body water to measures of FFM assumes a constant level of hydration in lean tissue.

Current care of children with JIA requires a paediatric rheumatology team consisting of a paediatric rheumatologist, a physiotherapist, an occupational therapist and a social worker. Our preliminary findings suggest the need to include a nutritionist in the team.

In summary, this study shows that children with JIA differ in several aspects of nutritional status. Further investigation is necessary to elucidate whether dietary changes or supplements are helpful in normalizing nutritional status or affecting the course of the disease. We advocate the simple but accurate method of measuring bioelectrical impedance to estimate lean body mass in JIA.

References

1. Still GF. On a form of chronic joint disease in children. Trans R Med Chir Soc 1897;62:47–50.
2. Chaffard A, Raymond F. Adenopathies in chronic infectious rheumatism. Rev Med 1897;16:345–59.
3. Kuhns JG, Swaim LT. Disturbances of growth in chronic arthritis in children. Am J Dis Child 1932;43:1118–3.
4. Ansell BM, Bywaters EG. Growth in Still's disease. Ann Rheum Dis 1956;15:295–318.
5. Laaksonen AL. A prognostic study of JRA: analysis of 544 cases. Acta Paediatr Scand 1966;166(Suppl.):49–55.
6. Lovell DJ, White PH. Growth and nutrition in JRA. In: Woo P, White P, Ansell B, eds. Paediatric rheumatology update. Oxford: Oxford University Press, 1990:47–56.

7. Knops N, Wulffraat N, Lodder S, Houwen R, de Meer K. Resting energy expenditure and nutritional status in children with juvenile rheumatoid arthritis. J Rheumatol 1999;26:2039–43.
8. Koerper MA, Stempel DA, Dallman PR. Anaemia in patients with JRA. J Paediatr 1978;92:930–3.
9. Ostrov BE. Nutrition and pediatric rheumatic diseases. Hypothesis: cytokines modulate nutritional abnormalities in rheumatic diseases. J Rheumatol 1992;33(Suppl.):49–53.
10. Johansson U, Portinsson S, Akesson A, Swantesson H. Nutritional status in girls with JCA. Hum Nutr Clin Nutr 1986;40:57–67.
11. Helliwell M, Coombes EJ, Moody BJ, Batsone GF, Robertson JC. Nutritional status in patients with rheumatoid arthritis. Ann Rheum Dis 1984;43:386–90.
12. Henderson CJ, Lovell DJ. Assessment of PEM in children and adolescents with JRA. Arthritis Care Res 1989; 2:108–13.
13. Miller ML, Chacko JA, Young EA. Dietary deficiencies in children with JRA. Arthritis Care Res 1989;2:22–4.
14. Richter M, Coughlan R, Trull A, Panayi GS. Prealbumin as a sensitive marker of disease activity. Ann Rheum Dis 1983;42:220–1.
15. Billingham ME, Gordon AH. The role of the acute phase reaction in inflammation. Agents Actions 1976;6:195–200.
16. Seltzer MH, Bastidas JA, Cooper DM, Engler P, Slocum B, Fletcher HS. Instant nutritional assessment. J Parenteral Enteral Nutr 1979;3:157–9.
17. Blackburn GL. Nutritional assessment and support during infection. Am J Clin Nutr 1977;30:1493–7.
18. Hoffer EC, Meador CK, Simpson D. Correlation of whole body impedance with total body impedance with total body water volume. J Appl Physiol 1969;27:531–4.
19. Durnin JV, Rahaman MM. The assessment of the amount of fat in the human body from measurements of skinfold thickness. Br J Nutr 1967;21:681–9.
20. Siri WE. Body composition from fluid spaces and density: analysis of methods. In: Brozek J, Henschel A, eds. Techniques for measuring body composition. Washington DC: National Academy of Science, 1961:223–44.
21. Smith DE, Booth IW. Nutritional assessment of children: guidelines to collecting and interpreting anthropometric data. J Hum Nutr Diet 1989;2:217–24.
22. Tanner JM, Whitehouse RH. Revised standards for triceps and subscapular skin folds in British children. Arch Dis Child 1975;50:142–5.
23. Warady BD, McCammon SP, Lindley CB. Anthropometric assessment of patients with JRA. Top Clin Nutr 1989;4:7–14.
24. Gregory JW, Greene SA, Scrimgeour CM, Rennie MJ. Body water measurement in growth disorders: a comparison of bioelectrical impedance and skinfold thickness techniques with isotope dilution. Arch Dis Child 1991; 66:220–2.
25. Pencharz PB, Azcue M. Use of bioelectrical impedance analysis measurements in clinical management of malnutrition. Am J Clin Nutr 1996;64:4855–85.
26. Aaseth J, Munthe E, Forre O, Steinnes E. Trace elements in serum and urine of patients with rheumatoid arthritis. Scand J Rheumatol 1978;7:237–40.
27. Bacon MC, Raiten DJ, Saini S, White PH. Nutritional status and its relationship to growth among children with rheumatoid arthritis. Arthritis Care Res 1989;2:S14.
28. Bernstein BH, Stobie D, Singsen BH, Koster-King K, Hanson V. Growth retardation in JRA. Arthritis Rheum 1977;20:212–6.
29. Bennett AE, Silverman ED, Miller JJ, Hints RL. Insulin-like growth factors 1 and 2 in children with systemic onset juvenile arthritis. J Rheumatol 1988;15:655–8.
30. Allen R, Jiminez M, Cowell C. Physiologic growth hormone secretion and somatomedin levels in JRA. Arthritis Rheum 1988;31:S118.
31. Balogh Z, El-Ghobarey AF, Fell GS, Brown DH, Dunlop J, Dick WC. Plasma zinc and its relationship to clinical symptoms and drug treatment in RA. Ann Rheum Dis 1980;39:329–32.
32. Garrow JS. JS body fat distribution changed by dieting? Acta Med Scand 1988;723(Suppl.):199–203.
33. Davies PS, Lucas A. The prediction of total body fatness in early infancy. Early Hum Dev 1990;21:193–8.