EDITORIAL CORRESPONDENCE

Measuring resting energy expenditure in pediatrics

To the Editor:

We read with interest the study by Kaplan et al.[1] and particularly the editorial by Pencharz and Azcue[2] on the measurement of resting energy expenditure (REE) in clinical practice.[1, 2] We agree that REE measurements in children are frequently necessary to provide a good clinical "energy diagnosis" and treatment in patients with failure to thrive, protein-energy malnutrition, or obesity. Although REE is 65% to 75% of total daily energy expenditure, the assessment of this compartment is invaluable in these cases because disease causes an increase in the energy expenditure variability.[3]

The editorial suggests that REE does not change significantly with the time of day that the evaluation is performed. However, several investigators have shown that REE has a high intraday variability that can be reduced if the measurement conditions are standarized, as occur with other biologic variables as blood pressure. We currently measure REE at the same time of day, after adequate time for nutrient absorption, and after 30 minutes of rest. After performing more than 1000 calorimetric measurements in our laboratory, we have observed that the measurement of REE is nearly impossible in healthy children who weigh less than 8 kg, because we cannot expect them to remain at rest or in fasting conditions before and during the test. In those cases, we should measure energy expenditure for a period long enough to obtain a representative value of the total daily energy expenditure (3 hours or more for a newborn infant), or we must use the published equations to predict energy expenditure.

Because REE is closely related to body cell mass, it is logical that Kaplan et al.[1] observe that it is better to predict REE from height and weight than from weight only. In effect, as previously published,[4] 89%, 91%, or 96% of the variance in REE can be explained by weight, weight and height, or lean body mass, respectively, in a very large hospitalized pediatric population. In our study, we developed REE prediction equations with the measured lean body mass because this is the variable that accounts for the greater variability of REE.

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REFERENCES

1. Kaplan AS, Zemel BS, Neiswender KM, Stallings VA. Resting energy expenditure in clinical pediatrics: measured versus predicted equations. J PEDIATR 1995;127:200-5.
2. Penchard PB, Azcue MP. Measuring resting energy expenditure in clinical practice [Editorial]. J PEDIATR 1995;127:269-71.
3. Abdulrazzaq YM, Brooke OG. Respiratory metabolism in preterm infants: the measurement of oxygen consumption during prolonged periods. Pediatr Res 1984;18:928-31.
4. Salas JS, Moukarzel E, Dozio E, Goulet OJ, Putet G, Ricour C. Estimating resting energy expenditure by simple lean-body-mass indicators in children on total parenteral nutrition. Am J Clin Nutr 1990;51:958-62.

Effect of weight reduction on serum transaminase activities in children with simple obesity

To the Editor:

We read with interest the article by Vajro et al.,[1] who described a correlation between weight reduction and serum transaminase activities in obese children. Abnormal results of liver tests are commonly observed in both adults and children with obesity.[1-3] Although most are suspected of having fatty liver associated with simple obesity, there is no easy way to reliably exclude primary liver disease. Weight reduction may be the best method to solve such a dilemma. Normalization of hepatic test results required a 10% weight reduction; the duration recommended was not reported.[1, 2] Hence we conducted a 3-month observation study to examine the fact reported by Vajro et al.

Our study included 73 obese children (69 boys and 4 girls) ranging from 6 to 14 years of age, who were referred to our outpatient clinics because of persistent elevation of serum aspartate aminotransferase/alanine aminotransferase (AST/ALT) of unknown origin. Genetic causes of obesity were clinically excluded, and primary liver diseases were ruled out by a battery of pertinent examinations (serum antigen and antibody tests for hepatitis A, B, and C viruses, cytomegalovirus, Epstein-Barr virus and herpesvirus, antinuclear antibodies, ceruloplasmin, α_1-antitrypsin, sweat chloride test, amino acids, urinary reducing sugar, fasting blood sugar, ammonia, and ultrasound tomography). All patients received no medication. We recommended both dietary and exercise measures for weight reduction. The outpatients were retrospectively divided into four groups, according to the percentage changes in weight after 3 months; group A, weight loss >5%; group B, weight loss of ≤5%; group C, an increase of <5%; group D, an increase >5%. There were no significant differences in terms of body height, weight, excess weight, and age among the four groups before weight reduction recommendations. To evaluate the effect of weight reduction on serum AST/ALT activity, the absolute change in AST/ALT and the percentage with return to normal levels of AST/ALT were examined. The changing rates of serum AST/ALT significantly correlated with the grades of weight reduction, respectively ($r = 0.56$, p 0.01; $r = 0.60$, p <0.01). The frequency of normalization or improvement in serum transaminase activities also correlated with the grades of weight reduction. In group A (n = 10), all 10 patients had improvement with normalization of both AST and ALT activity in 7 patients. In group B (n = 26), 88% showed