expenditure (DEE) of elderly people in free-living conditions: the doubly labelled water (DLW) technique [Ritz and Coward, (1995), *Diabete & Metab*], the factorial method (MFact) [Visser et al (1995), *Metab*] and the heart rate recording method (MHR) [Ceesay et al (1989), *Br J Nutr*]. For the individual calibration of MFact and MHR, energy costs of the various typical activities and individual relationships between HR and energy expenditure (EE) were determined from continuous measurements of HR and EE during 3 consecutive days in two opencircuit whole-body calorimeters in 12 healthy subjects (six males and six females; 70.1 ± 2.7 years; mean ± SD). In free-living conditions, DEE was determined by DLW during 17 days and by MFact and MHR from recordings of activities and HR during 14 and 4 days, respectively. Mean free-living DEE estimated using DLW, MFact and MHR was 12.8 ± 3.1, 12.7 ± 2.2 and 13.5 ± 2.7 $MJ.day^{-1}$ in men and 9.6 ± 0.8, 8.8 ± 1.2 and 10.2 ± 1.5 $MJ.day^{-1}$ in women, respectively. No significant differences were found between the three methods for both genders, using the *Bland & Altman* test which is based on a paired *t* test (*Lancet*, 1986).

It was concluded that MFact and MHR are satisfactory alternatives to DLW when considering the mean DEE of groups of subjects in free-living conditions, while MFact seems more suitable than MHR to estimate DEE of individuals.

Circadian variation of the energetic and hormonal response to a meal. M Romon [1], JL Edmé [2], C Le Fur [1], B Hecquet [3] (*[1] Service de nutrition, CHU Lille; [2] Cereste, 5, avenue Oscar-Lambret, Lille cedex; [3] Centre Oscar-Lambret, 59000 Lille, France*).

The aim of this work was to study the effect of meal time on the energetic and hormonal response. It was realised among 12 healthy men (BMI: 22.2 ± 1.7 kg/m^2, age 25.2 ± 5.3 years). They were given in a random order a standard meal; either they remained fasting at night (01h00) or during the day (13h00). The meal contained 40% of the estimated daily energy expenditure. During the 6 h following meal time, energy expenditure (EE) was measured by indirect calorimetry and blood samples were drawn at base line and every 20 min for assay of glucose, insulin, C Peptide, cortisol, glucagon and GH. The diet induced thermogenesis (DIT) was calculated as the areas under the curve of the differences between the post meal and the corresponding fasting energy expenditure. Comparisons between the sessions were made by a repeated two-way variance analysis (day/night and hour). During fed sessions, there was an interaction between the two factors ($P = 0.002$), post-meal increase of energy expenditure was blunted during the night, but there was no difference in DIT. All blood parameters were increased post-meal; this increase was significantly higher during the night for glycemia ($P = 0.01$) and insulinemia ($P = 0.04$); for the others parameters there was an interaction between the two factors: post prandial increase is delayed during the night. Post-meal energy expenditure was correlated with C Peptide and 2 h cortisol during the day. During the night, no correlation was found. Multiple regression analysis were performed with energy expenditure as dependent variable and day/night, cortisol, C Peptide and glycemia as independent variables. It resulted in the identification of C Peptide as significant variable ($\beta = 2.68$, $P = 0.01$). This result confirms that the metabolic response to a meal is modulated by meal time.

Precision of energy expenditure (EE) measurement in pre-term infants: contribution of natural isotopic abundance (NIA) variations. JC Picaud [1], S Normand [2], C Pacchiaudi [2], JP Riou [2], BL Salle [1] (*[1] Neonatal Unit Hospital Edouard-Her-*

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Association of EE measurement and nutrient balance study in very low birth weight (VLBW) infants allows precise evaluation of different feeding regimen (nutrient utilization, weight gain composition). EE measurement can be performed with doubly labelled water (DLW) method, but little is known about NIA in oxygen 18 (O18) and deuterium (D2) and influence of its variations on precision of EE measurement with DLW method.

During nutritional balance studies, urine samples were collected each day during 5 days in 13 VLBW infants (post-natal age: 28.0 ± 4.4 days, gestational age: 32.5 ± 1.1 SA, body weight: 1 509 ± 124 g) fed human milk (n = 5) or pre-term formula (n = 8). NIA was measured (n = 54) with isotope ratio mass spectrometer (Optima®, Fisons). Then daily values of O18 and D2 were used for calculation of EE in the 13 subjects.

NIA was –3.13 ± 0.83 ∂‰ for O18 and –24.56 ± 3.63 ∂‰ for D2, which is close to the adults values [Ritz et al (1996), *Am J Physiol* 270, E164-E169]. The mean coefficient of variation of EE measurement in relation with NIA variations was 1.98 ± 0.78% (0.62 to 2.98%).

In conclusion, during nutrient-balance study in VLBW infants, the variations in NLA did not significantly influence precision of EE measurement by DLW method.

Metabolic effects of oral glucose in CAPD patients versus healthy subjects. J Delarue [1], C Maingourd [2], C Couet [1], Ph Bagros [2], F Lamisse [1] *([1] Clinique médicale A et laboratoire de nutrition; [2] Association régionale d'aide aux urémiques du Centre Ouest, service de néphrologie, hôpital Bretonneau, 37044 Tours, France).*

Patients with end stage renal failure display an insulin-resistant state which is not reversed by continuous ambulatory peritoneal dialysis (CAPD). The insulin-resistance associated to chronic peritoneal glucose administration can induce chronic hyperinsulinaemia. In addition, these patients gain adiposity over the course of CAPD. The mechanism(s) sustaining this alteration of body composition is(are) not elucidated. Metabolic and oxidative responses to oral glucose were compared in six CAPD patients (68 ± 5 years) and six healthy subjects (HS) (24 ± 1 years) retrospectively selected after having ingested a similar oral glucose load per kg of fat free mass (FFM) (1.20 ± 0.3 g.kg FFM^{-1} vs 1.20 ± 0.6 g.kg FFM^{-1}; CAPD vs HS). Body composition was determined from anthropometric measurements. Substrates oxidation was obtained over 6 h from indirect calorimetry. CAPD patients had similar BMI (21.4 ± 1.3 vs 22.9 ± 1.1 $kg.m^{-2}$), a higher fat mass (25.8 ± 3.7 vs 16 ± 2.2%; $P < 0.05$) and a lower FFM (42.2 ± 2.2 vs 56.5 ± 2.6%; $P < 0.01$) than HS. CAPD patients displayed a higher glycemic and insulinaemic response to glucose than HS ($P < 0.05$). After adjustment for FFM, glucose oxidation was not different between the two groups ($P = 0.5$) while fat oxidation tended to be lower in CAPD patients than in HS ($P = 0.06$). Fat oxidation was related to fat mass in CAPD patients ($r^2 = 0.77$, $P < 0.05$) but not in HS ($r^2 = 0.05$). Basal plasma FFA were similar (405 ± 87 vs 483 ± 50 μM; CAPD vs HS). Following glucose, FFA concentrations were more inhibited in CAPD patients than in healthy subjects from 240 to 360 min; $P < 0.01$).

In conclusion, the marked adiposity of CAPD patients could result from an increased inhibition of fat oxidation secondary to hyperinsulinaemia which results from insulin-resistance. The increased adiposity was not sufficient to overwhelm the inhibiting effect of hyperinsulinaemia on fat oxidation.