Effect of Age on Substrate Oxidation During Total Parenteral Nutrition

Rima Al-Jaouni, Stephane M. Schneider, MD, Patrick Rampal, MD, and Xavier Hébuterne, MD, PhD

From the Department of Gastroenterology, Unit of Nutrition, Archet Hospital, Nice, France

OBJECTIVE: Parenteral nutrition is increasingly used in the elderly. Aging is accompanied by metabolic changes that can modify substrate use. We compared substrate oxidation during cyclic total parenteral nutrition (TPN) in elderly and middle-aged patients.

METHODS: Twelve elderly patients (eight women, four men; 72 ± 5 y) and 12 middle-aged patients (nine women, three men; 39 ± 13 y) who were on cyclic TPN for intestinal failure were investigated while in stable condition after at least 15 d of TPN. No patient was diabetic. Indirect calorimetry was performed during fasting and every 30 min during the 3 h of TPN infusion and 3 h after infusion, allowing the measurement of nutrient oxidation. Blood samples were obtained every hour for the measurement of glucose, insulin, triacylglycerols, and free fatty acids.

RESULTS: In the fasting state, resting energy expenditure was significantly higher in the elderly patients than in the middle-aged patients (39.3 ± 8.1 versus 31.9 ± 4.3 kcal/kg of fat-free mass per day, $P = 0.008$). During TPN, lipid oxidation was significantly higher in the elderly patients than in the middle-aged patients (1.09 ± 0.17 versus 0.84 ± 0.27 mg · kg^{-1} · min^{-1}, $P = 0.011$); glucose oxidation was significantly lower in the elderly patients than in the middle-aged patients (2.19 ± 0.93 versus 3.22 ± 1.54 mg · kg^{-1} · min^{-1}, $P = 0.038$). Areas under the curves of glycemia and free fatty acids were significantly higher in the elderly patients.

CONCLUSION: In the elderly, TPN was associated with significantly higher lipid oxidation and lower glucose oxidation than in younger patients. TPN formulas and flow rates should therefore be adapted in the elderly. *Nutrition* 2002;18:20–25. ©Elsevier Science Inc. 2002

KEY WORDS: total parenteral nutrition, elderly, body composition, indirect calorimetry, energy expenditure, substrate oxidation

INTRODUCTION

Elderly patients are more likely than younger adult patients to become nutritionally deficient, and deterioration of nutrition status during hospitalization has been described.[1–6] As a result, patients older than 65 y represent about 40% of the patients receiving total parenteral nutrition (TPN) in US hospitals, and 50% of all patients who receive tube feeding are older than 65 y.[7,8] Recent research conducted by our group suggests that malnutrition is more difficult to correct in elderly patients than in middle-aged patients.[9,10] Aging is accompanied by many changes that may explain the difficulty in gaining weight. Modifications in body composition and metabolism occur with aging. The basal metabolic rate, the thermogenic effect of food, and physical activity decrease with age[11,12]; the decline in the resting metabolic rate is between 1% and 2% per decade of age in men 20 to 75 y.[13] These changes are due mainly to the decrease of fat-free mass (FFM) that occurs with age.[14] Although few investigators have studied the effects of age on substrate oxidation, some studies have suggested the existence of modifications in lipid oxidation[15–18] and glucose tolerance[19–23] in elderly patients. To our knowledge, no reports have been published on substrate oxidation in elderly patients receiving TPN.

To test the hypothesis that metabolic changes due to aging might modify substrate oxidation and influence tolerance of TPN in the elderly, we compared substrate oxidation during cyclic TPN in stable and non-stressed elderly and middle-aged patients.

PATIENTS AND METHODS

Patients

Twelve elderly patients (eight women, four men; 72 ± 5 y) and 12 middle-aged patients (nine women, three men; 39 ± 13 y) who had been on cyclic parenteral nutrition for at least 2 wk were investigated while in stable condition. All patients had been admitted to the Nutritional Support Unit of our hospital for severe digestive failure requiring TPN (Table I). All patients were ambulatory (able to walk alone from the bed to the bathroom). Patients with active bacterial or viral infection; chronic renal, respiratory, liver, or cardiac failure; diabetes mellitus; or active cancer and patients treated with steroids, somatostatin, or antibiotics were excluded from the study. In all cases, dehydration was corrected before entry into the study. All patients gave their written informed consent, and this study was performed according to the ethical rules for human experimentation according to the Declaration of Helsinki. This protocol was approved by the Ethics Committee of the University of Nice Sophia Antipolis.

Methods

INDIRECT CALORIMETRY. The elderly and middle-aged patients were studied under similar conditions. Resting energy ex-

Correspondence to: Xavier Hébuterne, MD, PhD, Department of Gastroenterology, Unit of Nutrition, Archet Hospital, BP 3079, F-06202 Nice Cedex 3, France. E-mail: hebutern@unice.fr

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TABLE I.

DIAGNOSIS IN PATIENTS RECEIVING TOTAL PARENTERAL NUTRITION

Patients	Age (y)	Sex	Diagnosis	Clinical setting
Elderly				
1	67	M	Radiation enteritis	Short-bowel syndrome
2	76	F	Complication of surgery	Short-bowel syndrome
3	81	F	Mesenteric infarction	Short-bowel syndrome
4	70	F	Crohn's disease	Short-bowel syndrome
5	76	M	Crohn's disease	Short-bowel syndrome
6	65	M	Mesenteric infarction	Short-bowel syndrome
7	66	F	Radiation enteritis	Gut obstruction
8	65	F	Chronic pancreatitis	Fistula
9	76	F	Crohn's disease	Fistula
10	72	F	Radiation enteritis	Fistula
11	74	F	Crohn's disease	Short-bowel syndrome
12	72	M	Complication of surgery	Intractable diarrhea
Middle aged				
1	21	M	Crohn's disease	Gut obstruction
2	48	F	Crohn's disease	Fistula
3	58	M	Crohn's disease	Gut obstruction
4	59	F	Complication of surgery	Intestinal pseudoobstruction
5	27	F	Crohn's disease	Gut obstruction
6	38	F	Crohn's disease	Gut obstruction
7	51	F	Radiation enteritis	Short-bowel syndrome
8	38	F	Crohn's disease	Gut obstruction
9	40	F	Bullet wounds	Short-bowel syndrome
10	38	F	Crohn's disease	Gut obstruction
11	28	M	Idiopathic	Intestinal pseudoobstruction
12	22	F	Complication of surgery	Intestinal pseudoobstruction

F, female; M, male.

penditure (REE), diet-induced thermogenesis (DIT), and substrate oxidation were measured by indirect calorimetry (IC). IC was performed in the morning, after a 12-h overnight fast. Patients were studied while in a semirecumbent position, with a ventilated-hood, open-circuit indirect calorimeter (Deltatrac, Datex Instruments, Helsinki, Finland). After equilibrium was reached (10 min), respiratory exchanges were monitored continuously over 20 min; data were collected every minute and averaged over 20 min. The system was checked weekly by burning ethanol under standard conditions and calibrated daily with two standard gases.

BODY COMPOSITION. Body composition was measured by bipolar bioelectrical impedance analysis (BIA) with the use of an alternating electric current of 50 μA at two frequencies, 1 MHz and 5 kHz, as previously described and validated by Boulier et al.[24] A portable impedance analyzer (IMP BO 1, L'impulsion, Caen, France) equipped with a microprocessor and a computer was used to calculate impedance and body composition (including FFM, fat mass, body cell mass, total body water, intracellular water, and extracellular water). Measurements were taken after a 12-h overnight fast. The subjects had been supine for 30 min, with their arms relaxed at their sides without touching their bodies. Two stainless-steel needles were inserted subcutaneously: one on the anterointernal side of a foot, and the other in the first intermetacarpal space of the dorsal surface of the contralateral hand.

STUDY DESIGN. The study design is presented in Figure 1. The day before the study, parenteral nutrition was stopped in the morning, between 8 and 9 AM, and catheter permeability was obtained with a heparin lock (500 IU/5 mL). During the day, patients able to drink were allowed to have water, tea, or coffee. From 8 PM, patients were fasted. After the overnight fast, a catheter was placed into a forearm vein for blood sampling and kept patent with physiologic saline for biological analyses. At 8 AM, body composition was measured by BIA, and REE and fasting substrate oxidation were measured by IC. TPN was then initiated; patients were given a non-protein energy supply of 2.5 kcal $\cdot$ kg^{-1} $\cdot$ h^{-1} over 3 h as a ternary solution containing 34% non-protein energy as lipids (long-chain triacylglycerols) and 66% as glucose, with a nitrogen:energy ratio of 1:183. The infusion was administrated through the central venous catheter. IC was then performed every 30 min for 6 h, allowing measurement of DIT, carbohydrate oxidation (CHox), and lipid oxidation (Lox).

Venous blood samples were taken during fasting (B0), every hour during the 3 h of TPN (B1 to B3), and 3 h after the end of TPN (B4) for the measurement of glucose, triacylglycerols, free fatty acids (FFA), and plasma insulin. During the study period, urine samples were collected for measurement of nitrogen losses with the Kjeldahl method.[25]

DATA ANALYSIS. REE was calculated from oxygen consumption, carbon dioxide production, and urinary nitrogen output according to the method of Ben Porat et al.[26] Measured REE was expressed as a function of FFM (kcal/kg of FFM/24 h).

The cumulative increase in energy expenditure above the REE over the 6-h test period was defined as DIT. In the RESULTS section, DIT is expressed as a percentage of the metabolizable energy infused. The non-protein respiratory quotient and Lox were calcu-

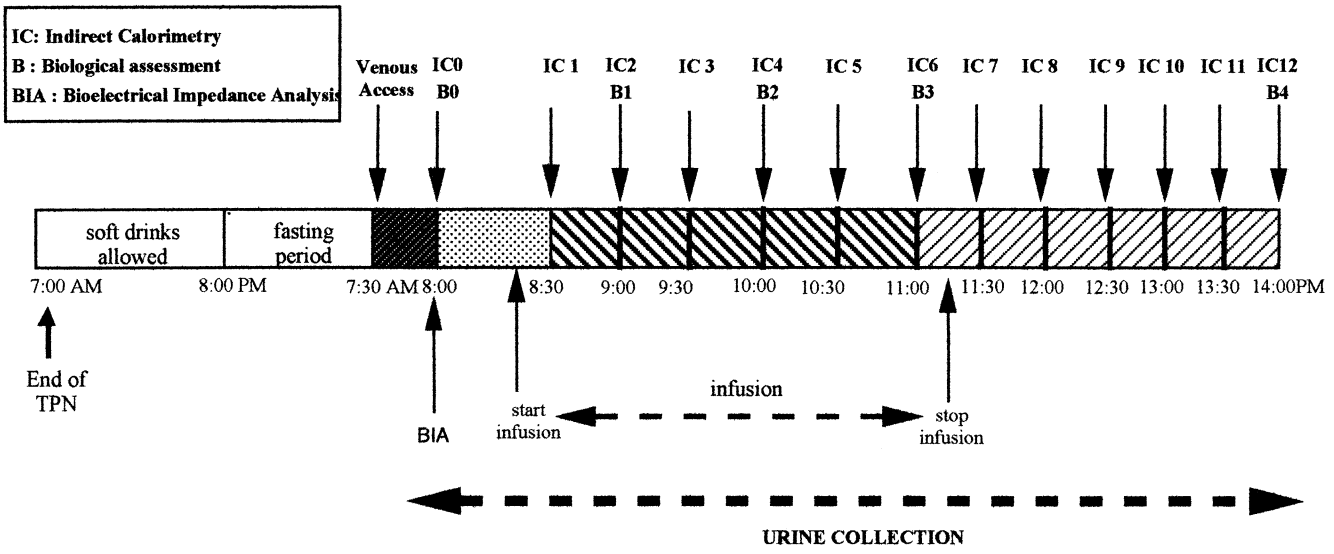

FIG. 1. Experimental protocol. TPN, total parenteral nutrition.

lated from the oxygen consumption rate, the carbon dioxide production rate, and nitrogen losses by using the equations of Ferranini[27] and Frayn.[28] Fasting and post-TPN Lox and CHox were expressed in milligrams per kilogram of body weight per minute ($mg \cdot kg^{-1} \cdot min^{-1}$).

ANALYTIC METHODS. Samples were centrifuged immediately (at 4°C for 15 min at 785*g*) and frozen at −20°C for later assay. The plasma glucose concentration was determined with the glucose-oxidase method on a Beckman Glucose Analyser II (Beckman Instruments, Fullenton, CA, USA). The plasma insulin concentration was measured by radioimmunoassay (ERIA Diagnostics Pasteur, Marnes la Coquette, France). Plasma triacylglycerols and FFA were determined by enzymatic methods (Wako, Neusss, Germany). C-reactive protein and α_1-glycoprotein acid levels were determined with standard in-house laboratory methods.

STATISTICAL ANALYSIS. Results were expressed as means ± standard deviation. Data for elderly and middle-aged patients were compared with the non-parametric Mann–Whitney test. Lipid and glucose oxidation during TPN versus baseline values and the mean increase of plasma serum above baseline value were assessed with analysis of variance. The *F* test for repeated measurements was used to compare the value obtained with the baseline values in each group. When significant *F* values emerged, data were analyzed with pairwise comparisons using Fischer's protected least significant difference test for repeated measurement analysis of variance. The area under the 0- to 180-min curve was calculated by the trapezoidal method.[29] Results were considered statistically significant at $P < 0.05$.

RESULTS

Twenty-four patients receiving TPN were studied. No significant differences were observed between the elderly and middle-aged patients concerning mean C-reactive protein level (7.8 ± 7.4 versus 6.5 ± 3.5 mg/L, respectively; $P = 0.609$) or α_1-glycoprotein acid level (1.6 ± 0.4 versus 1.3 ± 0.4 g/L, respectively; $P = 0.099$). The nutritional characteristics of the elderly and middle-aged patients are summarized in Table II. The body mass index (BMI) was similar in both groups, but, as expected, the elderly patients had significantly lower FFM and higher fat mass than the middle-aged patients.

In the fasting state, REE (kcal/24 h) did not differ between the elderly and middle-aged patients (1209 ± 142 versus 1352 ± 293 kcal/24 h, respectively; $P = 0.285$). When adjusted for FFM, REE was significantly higher in the elderly patients (39.3 ± 8.1 versus 31.9 ± 4.3 kcal/kg of FFM per day, respectively; $P = 0.008$). The non-protein respiratory quotient was similar in both groups (0.71 ± 0.06 versus 0.75 ± 0.06, respectively; $P = 0.182$). Consequently, fasting Lox and CHox did not differ between the elderly and middle-aged patients. However, the elderly patients had higher plasma levels of glucose and FFA and lower plasma levels of insulin (Table III) compared with the middle-aged patients.

During the 3-h TPN and the subsequent 3 h, DIT was similar in the elderly and middle-aged patients, whether considering global values (9.8 ± 21.2% versus 13.6 ± 15.0% of energy intake, respectively; $P = 0.862$) or comparing DIT on a time scale (Fig. 2). The mean non-protein respiratory quotient of the elderly pa-

TABLE II.

NUTRITIONAL CHARACTERISTICS OF ELDERLY AND MIDDLE-AGED PATIENTS

	Elderly ($n = 12$)	Middle aged ($n = 12$)
Body weight (kg)	49.3 ± 8.7	57.0 ± 15.7
Height (cm)	158.5 ± 10.8*	167.6 ± 8.2
Body mass index (kg/m^2)	19.6 ± 3.0	20.2 ± 5.0
Fat-free mass (kg)	32.2 ± 8.4*	43.1 ± 10.5
Fat mass (kg)	17.1 ± 3.7*	13.9 ± 9.9
Body cell mass (kg)	19.8 ± 6.4	25.0 ± 9.5
Total body water (kg)	25.8 ± 6.7	32.9 ± 8.0
Extracellular water (kg)	12.4 ± 3.4	15.2 ± 3.3
Intracellular water (kg)	13.4 ± 3.5	17.8 ± 4.9

* Significantly different from middle-aged patients ($P < 0.05$).

TABLE III.

BIOCHEMICAL CHARACTERISTICS IN FASTING (B0), DURING (B1–B3) AND AFTER (B4) INFUSION IN ELDERLY AND MIDDLE-AGED PATIENTS

	B0	B1 (1 h)	B2 (2 h)	B3 (3 h)	B4 (6 h)
Glucose (mmol/L)					
Elderly	4.20 ± 0.84*	8.02 ± 1.91†	11.81 ± 2.51*†	11.85 ± 2.78*†	3.81 ± 0.60
Middle aged	3.21 ± 1.19	6.68 ± 1.74†	9.63 ± 2.41†	9.61 ± 2.59†	3.26 ± 0.89
Insulin (mIU/L)					
Elderly	5.02 ± 0.12*	15.87 ± 8.49†	30.25 ± 14.51†	42.88 ± 21.14†	5.27 ± 0.58
Middle aged	6.28 ± 0.61	24.97 ± 16.46†	40.92 ± 29.80†	47.75 ± 30.84†	5.95 ± 2.47
Triacylglycerols (mmol/L)					
Elderly	1.79 ± 0.81	2.43 ± 1.13†	3.04 ± 1.33†	3.40 ± 1.95†	1.71 ± 0.88
Middle aged	1.15 ± 0.68	1.84 ± 0.76†	2.40 ± 1.00†	2.78 ± 1.14†	1.42 ± 0.97
Free fatty acids (mmol/L)					
Elderly	1.01 ± 0.45*	0.69 ± 0.16*†	0.51 ± 0.15*†	0.45 ± 0.14*†	0.81 ± 0.23*
Middle aged	0.65 ± 0.28	0.45 ± 0.21†	0.37 ± 0.18†	0.34 ± 0.14†	0.47 ± 0.21†

* Significantly different from middle-aged patients ($P < 0.05$).
† Significantly different from B0 ($P < 0.05$).

tients was significantly lower than that of the middle-aged patients (0.78 ± 0.03 versus 0.83 ± 0.05, respectively; $P = 0.030$). Lox was significantly higher in the elderly patients than in the middle-aged patients (1.09 ± 0.17 versus 0.84 ± 0.27 mg · kg^{-1} · min^{-1}, respectively; $P = 0.011$; Fig. 3A) and CHox was significantly lower in the elderly patients (2.19 ± 0.93 versus 3.22 ± 1.54 mg · kg^{-1} · min^{-1}, respectively; $P = 0.038$; Fig. 3B). Protein oxidation did not differ between the elderly and middle-aged patients (0.94 ± 0.50 versus 0.90 ± 0.37 mg · kg^{-1} · min^{-1}; $P = 0.9$), and a similar negative nitrogen balance was observed in the elderly and middle-aged patients during the 6 h of the study (−0.59 ± 1.34 versus −0.57 ± 1.40 g/6 h, respectively; $P = 0.908$).

Biological data are summarized in Table III. Glycemia was higher in the elderly patients than in the middle-aged patients after 2 and 3 h of TPN. Mean glycemia was significantly higher in the elderly than in the middle-aged patients (8.9 ± 1.7 versus 7.3 ± 1.6 mmol/L, respectively; $P = 0.046$), and the area under the glucose response curve during TPN infusion was significantly higher in the elderly patients than in the middle-aged patients (27.86 ± 5.62 versus 22.72 ± 5.43 mmol.hr/L, respectively; $P = 0.016$). Insulinemia did not differ between groups, and mean insulinemia was similar in both groups (23.6 ± 9.4 versus 29.9 ± 19.2 mIU/L, respectively; $P = 0.525$); the area under the insulin response curve during TPN infusion was not statistically different in the elderly patients versus the middle-aged patients (69.60 ± 27.74 versus 92.69 ± 61.04 mUI · h^{-1} · L^{-1}, respectively; $P = 0.123$). The area under the insulin/glucose curve was significantly lower ($P = 0.037$) in the elderly patients (2.54 ± 0.99) than in the middle-aged patients (4.35 ± 3.18) during TPN infusion. FFA were always significantly higher in the elderly patients than in the middle-aged patients; the mean plasma level of FFA was significantly higher ($P < 0.001$) in the elderly patients (0.62 ± 0.14 mmol/L) than in the middle-aged patients (0.41 ± 0.16 mmol/L); the area under the FFA response curve during TPN infusion was higher in the elderly patients than in the middle-aged patients (1.93 ± 0.85 versus 1.32 ± 0.56 mmol · h^{-1} · L^{-1}, respectively; $P = 0.025$). Triacylglyerol levels were similar in both groups.

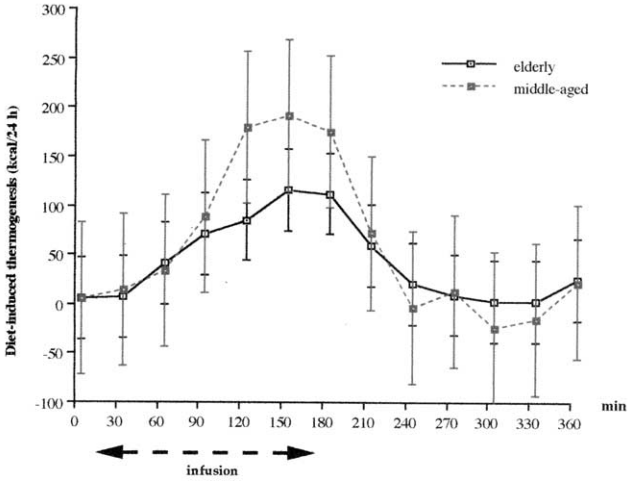

FIG. 2. Diet-induced thermogenesis in elderly and middle-aged patients.

DISCUSSION

This study conducted in patients managed by TPN for intestinal failure showed important metabolic differences between elderly and middle-aged patients: for similar energy supplies, elderly patients oxidized more fat and less glucose than middle-aged patients.

Our results must be interpreted in light of the methodology used. To avoid any bias due to their disease condition, only non-stressed patients without major organ failure, infection, or cancer who had been on cyclic TPN for at least 2 wk were studied. Moreover, no patient was diabetic, and all were ambulatory. Body composition was measured by BIA and a well-validated method.[24] For the determination of body composition, all methods are indirect and there is no gold standard in living subjects, but BIA is now considered a sufficiently accurate method for measurement of body composition in clinical investigations.[30] However, to validate the BIA in elderly malnourished patients, we compared the body compositions of similarly malnourished elderly patients as measured by BIA and anthropometric measurements. In 21 elderly (66 ± 10 y) malnourished (BMI = 16.8 ± 2.8 kg/m^2) patients, fat mass was determined by BIA and skinfold thicknesses at four sites, as proposed by Durnin and Womersley.[31] Bland–Altman analyses demonstrated a close correspondence between the two measurements. Therefore, we used BIA for the study. IC is a well-validated method for the measurement of energy expenditure and substrate

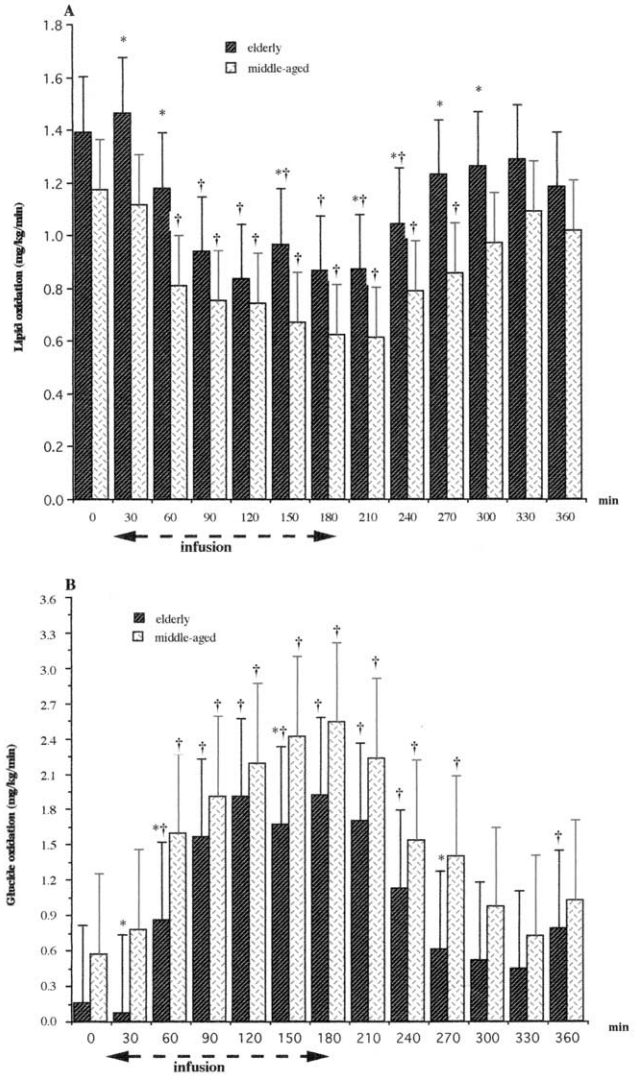

FIG. 3. (A) Lipid oxidation rates ($mg \cdot kg^{-1} \cdot min^{-1}$) during fasting, 3-h infusion, and the following 3 h in elderly and middle-aged patients. (B) Glucose oxidation rates ($mg \cdot kg^{-1} \cdot min^{-1}$) during fasting, 3-h infusion, and the following 3 h in elderly and middle-aged patients. *Significantly different from middle-aged patients, $P < 0.05$. †Significantly different from time 0 in the group considered, $P < 0.05$.

oxidation.[27,28] During cyclic TPN administered for intestinal failure, patients generally receive an energy supply of 30 to 35 kcal/kg over a 12- to 14-h overnight period, corresponding to a flow rate of 2.0 to 3.0 $kcal \cdot kg^{-1} \cdot h^{-1}$. To simulate cyclic TPN, patients were perfused at a flow rate of 2.5 $kcal \cdot kg^{-1} \cdot h^{-1}$ for 3 h, and IC was performed every 30 min for 6 h.

In our study population, REE adjusted for FFM was higher in the elderly patients than in the middle-aged patients. We assume that this difference is an effect of age per se and nutrition status because the underlying diagnoses and inflammatory status were similar in both patient groups (Table I). Moreover, at the time of the study, all patients were ambulatory and non-stressed.

It is well known that advancing age is accompanied by changes in body composition and energy metabolism. Owing to the decrease in FFM with age,[14] as in our study, older individuals generally have lower REE than young normal subjects.[12,13,32–34] When REE is adjusted for FFM, some investigators have reported lower REE in the elderly than in young subjects,[32,33,36] but others have not observed any difference between elderly and young individuals.[13,34] In one study comparing elderly and middle-aged women, the elderly subjects had a higher REE:FFM.[37] However, in contrast to our study, those investigations were done in healthy and non-malnourished subjects. In malnourished elderly subjects, Campillo et al.[38] found that the REE:FFM was higher in patients with BMI below 20 than in patients with BMIs above 20. The FFM accounts for 85% of the individual variations in REE.[39] As expected, the FFM of our elderly patients were significantly lower and their fat masses were higher than in the middle-aged patients. FFM decreases and fat mass increases with advancing age.[14] Sarcopenia occurs with aging, and the decrease in FFM is due essentially to a decrease in muscle mass. Cross-sectional and longitudinal body composition studies and autopsy dissection data confirm an overall decline in skeletal muscle mass, from 45% in young adults to less than 27% after the age of 70 y, with little change in non-muscle mass.[40] Malnourished elderly individuals with low FFM probably have lost a great quantity of muscle mass. Recent investigations on the relative contributions of different organs to REE have shown that, whereas muscle mass represents almost 40% of FFM, it represents only 20% of REE.[41] In contrast, organs such as the heart, brain, and kidney account for a very small percentage of FFM and a proportionally greater percentage of total REE. Extensive loss of muscle mass in malnourished elderly individuals may increase the contribution of other organs to FFM, thereby increasing REE when expressed as a ratio of FFM. Moreover, TPN may have contributed to the increase in REE.[42]

In the present study, we observed no effect of age on the thermogenic response to TPN. Previously published results concerning the effect of age on thermogenic response are contradictory. In some studies, glucose-induced thermogenesis was apparently decreased in elderly subjects compared with young subjects.[15,34] In other investigations, the thermogenic response after ingestion of a liquid mixed meal was decreased in elderly men[35,36] but not in elderly women.[36] Other investigators have reported that the thermogenic response to a protein load in elderly individuals does not differ from that in young individuals.[43] However, none of these studies concerned malnourished patients fed by TPN. We observed a thermogenic effect of TPN between 9.8% and 13.6% of the energy infused, a finding consistent with studies on young subjects in whom the thermogenic effect of TPN varies between 5% and 17%.[44–49]

The main findings of our study were increased fat oxidation and decreased glucose oxidation in elderly versus younger patients in response to TPN infusion of a mixed protein, lipid, and glucose diet. These observations are consistent with the results of Golay et al.[15] who observed a much higher glucose oxidation rate in young subjects than in elderly individuals after an oral glucose load of 100 g. Our observation is important because the reduction of glucose oxidation during TPN can affect treatment tolerance. Because the underlying diagnoses and inflammatory status were similar in both patient groups, our results suggest that the difference was the effect of age. This reduction in glucose oxidation was not due to decreased secretion of insulin because none of our patients were diabetic, and during infusion serum insulin levels were similar in both groups. The differences observed in the area under the curves of insulin and glucose suggest insulin resistance in the elderly. During the study, plasma FFA levels were always higher in the elderly than in the younger patients. Despite similar BMI in both groups, the body compositions of our elderly subjects were significantly altered (Table II). In particular, their fat mass accounted for 35% of their total body weight, whereas only 24% of the body weight of the younger subjects was due to fat. The higher baseline concentration of FFA in the elderly patients as opposed to the younger patients suggests increased mobilization of FFA from adipose tissue in older subjects.[18] Our results are in agreement with those of Bonadonna et al.[18] who, with the glucose clamp technique, showed an insulin resistance in a group of non-diabetic elderly subjects, reflected by increased FFA and lipid oxidation and decreased glucose oxidation.

The higher FFA levels associated with higher lipid oxidation and lower glucose oxidation in the elderly than in the middle-aged patients suggest that a substrate competition between FFA and glucose occurs according to the Randle cycle.[50] However, the methodology of the present study did not allow us to show this phenomenon, and Liu et al.[51] suggested that elderly obese patients do not use their fat stores as readily as young obese individuals. Although the metabolic changes in the aged probably have only minor consequences in non-stressed patients, parenteral formulas should be adapted for the elderly, and glucose tolerance must be checked, especially during cyclic TPN. These abnormalities may be enhanced in stressed patients, in whom aging can aggravate glucose intolerance.[21–23]

In conclusion, we showed that, in non-stressed and non-diabetic elderly patients, TPN is accompanied by lower glucose oxidation and higher fat oxidation than in younger patients, and that the glucose tolerance to TPN is decreased. We suggest that TPN formulas and/or flow rates should be adapted specifically for the elderly and that glucose tolerance of TPN should be checked carefully, particularly during cyclic TPN.

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