

(518 vs 350 kJ respectively,  $p < 0.05$ ). The DIT was significantly and negatively correlated with the percentage body fat ( $r = -0.51$ ,  $p < 0.005$ ). The DIT was highly correlated with the FFM and FM combined ( $r = 0.71$ ,  $p < 0.01$ ,  $\text{DIT (kJ)} = 17.5 \text{ FFM} - 11.3 \text{ FM} - 500.9$ ).

These results support 2 conclusions. First that the DIT was significantly higher for lean men than obese men of similar FFM. Second, that body composition is a significant determinant of the magnitude of the DIT.

**Deformability, membrane lipid composition and cytosolic cation contents of red blood cells in non-insulin dependent diabetic patients.** P Miossec<sup>1</sup>, M Garnier<sup>2</sup>, J Pariès<sup>1</sup>, F Zhiri<sup>2</sup>, M Dufilho<sup>3</sup>, MG Pernollet<sup>3</sup>, JR Attali<sup>1</sup>, P Valensi<sup>1</sup> (<sup>1</sup> *Diabetology-Nutrition*; <sup>2</sup> *Toxicology, Jean-Verdier Hospital, Bondy*; <sup>3</sup> *URA CNRS 1482, Necker Hospital, Paris, France*)

Many studies have clearly demonstrated hemorheological abnormalities in diabetic patients. The erythrocyte rigidity is higher, and is associated with changes in the membrane lipid composition. The aim of this work was to study, simultaneously, the variations in intra-erythrocyte cation concentrations and membrane lipids in 28 poorly controlled ( $\text{HbA}_{1\text{C}} = 8.8 \pm 3.6\%$ ,  $N < 6\%$ ) non-insulin-dependent diabetic patients (NIDDs) (age  $54.9 \pm 6.4$  years, BMI  $27.53 \pm 4.2$ ) compared with 26 control subjects (identical age and BMI). The red blood cell rigidity index (RI), measured on a Hanss' hemorheometer, was higher in the NIDDs than in controls ( $11.8 \pm 2$  vs  $10.4 \pm 2.2$ ,  $p < 0.03$ ), the RI correlated with post-prandial glycemia ( $r = 0.51$ ,  $p < 0.02$ ). Compared with the controls, in the NIDDs free cholesterol (FC) was lower ( $-16\%$ ,  $p < 0.01$ ), sphingomyelins (SP) were higher ( $+42\%$ ,  $p < 0.01$ ), free cholesterol/phospholipid ratio (FC/PL) was lower ( $-26\%$ ,  $p < 0.05$ ). In NIDD, intra-erythrocyte

sodium ( $\text{Na}_i$ ) was higher ( $29.4 \pm 10.3$  vs  $18 \pm 10.1$  mEq/l,  $p < 0.001$ ), while cytosolic potassium and magnesium were not significantly different. Intra-erythrocyte ionized calcium ( $\text{Ca}_i$ ) was higher ( $105 \pm 22.2$  vs  $69 \pm 8$  nmol/l,  $p < 0.001$ ) as well as the calcium influx estimated by incorporation of  $^{45}\text{Ca}^{2+}$  ( $V_i$   $^{45}\text{Ca}^{2+}$ ) ( $89 \pm 28.8$  vs  $47 \pm 20$   $\mu\text{mol/l/cell/h}$  ( $p < 0.001$ )). In the NIDDs,  $\text{Na}_i$  correlated with RI ( $r = 0.41$ ;  $p < 0.02$ ) and with fasting glycemia ( $r = 0.45$ ,  $p < 0.05$ );  $\text{Na}_i$  also correlated with membrane lipids: FC ( $r = -0.41$ ,  $p < 0.05$ ); phosphatidylethanolamine (PE) ( $r = -0.43$ ,  $p < 0.05$ ); phosphatidyl choline (PC) ( $r = -0.58$ ,  $p < 0.001$ ); FC/PL ( $r = 0.48$ ,  $p < 0.01$ ).  $V_i$   $^{45}\text{Ca}^{2+}$  correlated with  $\text{HbA}_{1\text{C}}$  ( $r = 0.51$ ,  $p < 0.02$ ), PC ( $r = 0.41$ ,  $p < 0.05$ ) and SP ( $r = 0.42$ ,  $p < 0.05$ ). In poorly controlled NIDDs the increase in  $\text{Na}_i$  and  $\text{Ca}_i$  may be in part responsible for the reduction in red cell deformability and may be the result of modifications in the membrane lipid constitution. The increase in  $\text{Ca}_i$  may result, at least in part, from the augmentation of  $V_i$   $^{45}\text{Ca}^{2+}$ . The changes in cation and lipid constitution of red blood cells correlate with glycemic control.

**Anthropometric characteristics of obese subjects with nocturnal oxygen desaturation.** D Cassuto<sup>1</sup>, G Panotopoulos<sup>1</sup>, E Orvoën-Frija<sup>2</sup>, B Saci<sup>2</sup>, A Basdevant<sup>1</sup>, B Guy-Grand<sup>1</sup> (<sup>1</sup> *Dept of Nutrition*; <sup>2</sup> *Dept of Pneumology, Hôtel-Dieu, 75181 Paris cedex, France*)

Nocturnal oxygen desaturation (NOD) can be observed in obese subjects (OB) with or without Obstructive Sleep Apnea Syndrome (OSAS). The aim of this prospective study was to clarify the anthropometric characteristics of OB with NOD with or without SAS.

Patients hospitalized in the Nutrition Unit ( $n = 66$ ; 45 women, body mass index (BMI)

45 ± 8 kg/m<sup>2</sup>; 21 males, BMI 44 ± 11 kg/m<sup>2</sup>) underwent nocturnal oxymetry recording, pulmonary functional testing (PFT) and diurnal arterial blood gas measurements. Their waist circumference (WC), waist/hip ratio (W/H) and neck circumference (NC) were measured. The NC was positively correlated with the WC ( $p < 0.001$ ), independently of BMI. Group 1 (15 F/17 M) consisted of OB with NOD > 15% of recorded sleep time spent with oxygen saturation (OS) < 4% of the diurnal OS. Group B (30 F/4 M) consisted of OB without NOD. As compared with group B, subjects in group A had significantly higher BMI ( $p < 0.003$ ) and W/H ( $p < 0.01$ ) but had no difference in NC. In women with NOD, W/H and WC were significantly higher than in women without NOD ( $p < 0.01$ ). Such a difference was not observed in men. In group A, the NC was positively correlated to the W/H ratio ( $p < 0.005$ ) and paCO<sub>2</sub> ( $p < 0.005$ ) and negatively to the residual volume (RV) ( $p < 0.05$ ), functional residual capacity (FRC) ( $p < 0.01$ ) and total lung capacity (TLC) ( $p < 0.05$ ) independently of BMI. The WC was positively correlated with FRC ( $p < 0.03$ ). The subjects in group A were classified according to the polysomnography recording. As compared with patients without OSAS ( $n = 8$ ), patients with OSAS ( $n = 21$ ) had significantly higher NC

( $p < 0.04$ ), similar WC and lower vital capacity (VC) ( $p < 0.05$ ) and TLC ( $p < 0.05$ ). In subjects with OSAS, NC was positively correlated to paCO<sub>2</sub> ( $p < 0.03$ ) and negatively to RV ( $p < 0.03$ ), FRC ( $p < 0.01$ ) and VC ( $p < 0.01$ ). It was concluded that: i) NOD is associated with abdominal obesity, especially in women; and ii) patients with SAS differ significantly from patients without SAS in terms of NC and PFT.

**Left ventricular function (LVEF) in patients with obstructive sleep apnea syndrome (OSAS) and massive obesity.**

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Obstructive apneas may induce acute left ventricular dysfunction mainly *via* an increase of after-load due to a negative intrathoracic pressure and a release of catecholamine. However, conflicting results have been reported concerning the chronic effects of OSA on LVEF. Previous studies did not take into account the cardiac diseases associated with obesity, and no data are available on the LVEF of massively obese patients with OSAS. In 59 consecu-

**Table I.** Data for patients with and without OSAS (Laaban *et al*).

	OSAS (n = 25)	No OSAS (n = 34)	p
LVEF (%)	57 (± 10)	60 (± 8)	NS
Apnea index (/h)	32 (± 23)	1 (± 0.5)	< 0.001
Hypertension (% pat)	37%	28%	NS
Diabetes (% pat)	37%	28%	NS
Myocardial ischemia (% pat)	27%	20%	NS
BMI (kg/m <sup>2</sup> )	51 (± 9)	50 (± 10)	NS
WHR female	0.95 ± 0.16	0.94 ± 0.11	NS
WHR male	1.04 ± 0.16	0.94 ± 0.3	NS