However, after 48hours, the effect of exercise on RLPc was homogeneous. In control RLPc decreased in 6 out of 11 subjects by −30.05±50.25% and in T1DM it increased in 4 out of 10 subjects by 5.24±31.92 (p =0.091).
Conclusion: The effect of the exercise in RLPc is not observed immediately after the exercise, but after 48 hours it promotes a decrease in normal subjects, which is not observed in T1DM patients.

123 EFFECT OF STANDARD COMBINATION OF VALSARTAN WITH EITHER AMLODIPINE OR HYDROCHLOROTHIAZIDE ON LDL SUBFRACTION PROFILE IN PATIENTS WITH HYPERTENSION

L.G. Christogiannis[1], M.S. Kostapanos[1], H.J. Milionis[1], Z. Mitrogianni[1], E. Moutzouri[1], A.D. Tselepis[2], M.S. Elisaf[1]. *[1]Department of Internal Medicine, University of Ioannina School of Medicine, [2]Laboratory of Biochemistry, Department of Chemistry, University of Ioannina, Ioánnina, Greece*

To compare the effects of standard combination of valsartan with either amlodipine (VA) or hydrochlorothiazide (VH) on low-density-lipoprotein (LDL) subfraction profile in hypertensive patients.
Methods: Sixty drug-naïve patients with stage II or III hypertension were randomized to either VA (160/5 mg) or VH (160/12.5 mg) treatment [VA group (N = 30) and VH group (N = 30), respectively]. At baseline as well as 16 weeks post-treatment blood pressure (BP), serum lipid and apolipoprotein levels were determined. The analysis of the LDL subfraction profile was conducted by the LDL Lipoprint System.
Results: Both drug combinations effectively reduced BP levels; no difference between groups was observed. No effect of VA on serum lipid and apolipoprotein levels was noted. VH was associated with a significant increase in triglyceride and apolipoprotein-E levels [by 9.0% ($p < 0.05$) and 8.9% ($p < 0.05$), respectively] as well as with a significant decrease in high-density-lipoprotein (HDL)-cholesterol levels (by 5.0%, $p < 0.05$). A significant increase in the cholesterol concentration of small-dense LDL subfractions [6.0 (0.0–44.6) vs 10.5 (1.3–41.3) mg/dl, $p < 0.05$] was observed in the VH group, whereas this parameter did not change from baseline in the VA group ($p = 0.01$ for the comparison between groups). Consequently, mean LDL particle size decreased in the VH group (from 267±5 to 266±5 Å, $p < 0.05$), whereas remained unchanged in the VA group. No change in the cholesterol concentration of both large-buoyant and intermediate LDL subfractions was recorded in either group.
Conclusion: Despite similar reductions in BP, VH may adversely affect lipid and LDL subfraction profile as compared to VA.

124 APOLIPOPROTEINS A1 AND B AND THE RISK OF CARDIOVASCULAR RECURRENCES IN ISCHEMIC STROKE PATIENTS

S. Vidale, A. Sampietro, L. Tancredi, M. Arnaboldi. *Neurology Dept., Sant'Anna Hospital, Como, Italy*

Background and Purpose: Plasma apolipoproteins has been proposed as risk factors for cardiovascular disease. No studies analysed the contribution of those variables in the clinical follow up in stroke patients. Aim of this study was to evaluate the association between plasma apolipoproteins and recurrences of cardiovascular events (CVD) in patients with stroke.
Materials and Methods: We evaluated patients admitted in hospital for ischemic stroke between July and December 2004. Vascular risk factors, clinical features and blood variables (lipids and apolipoproteins) were registered during hospitalization. Follow up data were obtained by ambulatory visits and hospital registers. Statistical analysis was performed using chi-square and t-test.
Results: 129 patients were included (M/F: 1.4/1; mean age: 70 yrs). Hypertension was the most frequent vascular risk factor (55.8%). Mean values of apoA and apoB were 129 mg/dL and 98, respectively. Mean follow up time was 4.6 yrs. CVD recurrences occurred in 27 patients (20.9%). A positive association was observed between atrial fibrillation, apoB, apoB/A1 ratio and CVD recurrences ($p < 0.05$).
Conclusions: Apolipoproteins could contribute to increase the risk of cardiovascular accidents in ischemic stroke patients.

125 LDL SIZE DETERMINANTS DURING ANTIRETROVIRAL THERAPY INCLUDING PROTEASE INHIBITORS IN HIV-1 INFECTED PATIENTS

R. Bittar. *Unité Fonctionnelle de Biochimie des Maladies Métaboliques, Service de Biochimie Métabolique, Groupe Hospitalier Pitié-Salpétrière, Assistance Publique-Hôpitaux de Paris, Paris, France*

Background: Lipid disorders are frequent in HIV1-infected patients taking antiretroviral combinations that include protease inhibitors (PI). Hypertriglyceridemia in this population is frequently associated with increased small dense LDL. The LDL concentration and particle size might be an important predictive marker of cardiovascular disease in this setting.
Objective: To evaluate variables that influences the quantity and size of LDL in a substudy of the ANRS 126 trial.
Methods: We studied 81 HIV1-infected patients with dyslipidemia (LDL-cholesterol >4.1 mmol/l, triglycerides <8.8 mmol/l) who had been taking PI-including cART for at least two months, and who were not taking lipid-lowering drugs. Total cholesterol (TC), triglycerides (TG), LDL-cholesterol (LDL-c), and HDL-cholesterol (HDL-c) were assayed in serum. LDL diameter was assessed by gradient gel electrophoresis (GGE). Relationships between the LDL diameter, and demographic metabolic and HIV-related variables, were identified by using non parametric univariate tests and multiple linear regression models.
Results: In univariate analyses, LDL diameter was related to gender, ethnic origin, CDC stage, TG, HDL-c, duration of exposure to and numbers of NRTIs and PIs. In a multivariable linear regression model, LDL diameter was independently associated negatively with TG ($P < 0.0001$) and positively with HDL-c ($p < 0.0001$). Per 1 mmol/L increase in TG, the LDL diameter decreased by 0.281 nanometers. Conversely, per 1 mmol/L increase in HDL-c, the LDL diameter increased by 1.175 nanometers.
Conclusion: Based on the LDL size, an atherogenic phenotype was significantly related to TG and HDL-c concentrations. LDL diameter might help predict and monitor the cardiovascular risk (CVD) in HIV1infected patients.

126 CARDIOVASCULAR RISK IN PATIENTS WITH TYPE 2 DIABETES MELLITUS WITH OR WITHOUT METABOLIC SYNDROME

J. Millan[1], C. Garcia Calzado[2], C. Recarte[3]. *[1]Internal Medicine, Hospital GU Gregorio Marañón, Madrid, [2]Endocrinology, Universidad de Cadiz, Cadiz, [3]Internal Medicine, Universidad Complutense, Madrid, Spain*

Diabetes Mellitus is associated with a high cardiovascular morbi-mortality. This feature is related with macrovascular and microvascular complications. Metabolic syndrome, a clinical condition with high risk that is present frequently in diabetic patients may be a factor that increase the risk.
This study was made to detect differences in the estimated risk in diabetic patients depending of presence/absence of metabolic syndrome.
We have studied a group of patients with type 2 DM (n = 80) that were attended in primary care. Diagnosis of metabolic syndrome was made according with ATPIII criteria and IDF criteria. Cardiovascular risk was calculated according with Framingham scale.
In patients with tipo 2 DM metabolic syndrome was found in 47.5% (ATPIII) and 49% (IDF). Cardiovascular risk (Framingham) up to 20% was present in 28.8% of patients with and in 16.6% of patients without metabolic syndrome according with ATPIII.
Men with diabetes show metabolic syndrome in 41.9% of cases with Framingham >20%; but only 24.9% of diabetic men without metabolic syndrome show Framingham >20%. In women, 21% of diabetics with metabolic syndrome show Framingham >20%, and in diabetic women without metabolic syndrome we have not found any patients with Framingham >20%.
We can conclude that the Framingham score is available to show the increased risk attributable of metabolic syndrome in patients with diabetes mellitus.

127 CONSTITUTIVE INHIBITION OF PLASMA CHOLESTERYL ESTER TRANSFER PROTEIN (CETP) BY APOLIPOPROTEIN C1 IN NORMOLIPIDEMIC, BUT NOT IN HYPERLIPIDEMIC PATIENTS WITH CORONARY ARTERY DISEASE. CONSEQUENCES ON PLASMA CHOLESTEROL DISTRIBU

X. Pillois[1], T. Gautier[2], J.-P. Pais de Barros[2], A. Jeannin[2], J. Bonnet[1,3], L. Lagrost[2,4]. *[1]University Victor Segalen of Bordeaux II − INSERM Research Center UMR828, Bordeaux, [2]University of Burgundy − INSERM Research Center UMR866, Dijon, [3]University Hospital of Bordeaux, Bordeaux, [4]University Hospital of Dijon, Dijon, France*

Objectives: Plasma cholesteryl ester transfer protein (CETP) promotes the cholesterol enrichment of triglyceride-rich lipoproteins at the expense of HDL. In earlier studies, HDL was found to inhibit CETP activity in a concentration-dependent manner. We were able to show that, among all the apolipoprotein components of HDL, apolipoprotein (apo) C1 is a potent inhibitor of CETP *in vitro. In vivo* studies in apoC1-knocked out/human CETP transgenic and human apoC1 transgenic/human CETP transgenic mice came in direct support of the inhibitory effect of human apoC1 that was initially observed *in vitro*. Accordingly, we found a negative correlation between plasma concentrations of apoC1 and levels of CETP activity in healthy, normolipidemic subjects. Our goal was to establish whether modulation of cholesteryl ester transfer protein (CETP) activity by apolipoprotein C1 (apoC1) can modify plasma cholesterol transport in humans and whether it is influenced by hyperlipidemia in high-risk patients.
Methods: Plasma CETP activity, apoC1 concentration, and lipoprotein profile were determined in 240 patients with documented Coronary Heart Disease.
Results: ApoC1 levels correlated negatively with CETP activity in the total population studied. However, this negative relationship was observed in normolipidemic patients only, but not in those with hyperlipidemia (LDL cholesterol >2.6 mmol/l and/or triglycerides >1.7 mmol/l). As a consequence of CETP inhibition, and in the former group only, a high apoC1 level was accompanied with higher HDL to LDL cholesterol ratio.