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Metabolic Response of Diabetic Patients with Obesity and No Obesity during Bruce Treadmill Test

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Abstract

Background: Physical exercise alters many biochemical parameters of human body. For that reason, it is recommended as a cure for many diseases of modern life like obesity and diabetics. This study investigates the biochemical and hormonal changes in diabetic, hypertensive and obese individuals after Bruce treadmill tests of different durations and intensities. **Method:** Our sample consisted of 59 individuals, divided into four groups: A: 12 normal body-weight diabetics, B: 11 overweight diabetics, C: 14 normal body-weight non-diabetics and D: 22 overweight non-diabetics. 66.1% of them were men. The comparisons were done between: 1) Diabetics versus non-diabetics; 2) Over-weight versus normal body-weight persons; 3) Over-weight diabetics versus normal weight diabetics; 4) Over-weight non-diabetics versus normal body-weight non-diabetics of 5) Hypertensives versus non-hypertensives. All individuals performed an intense, short duration treadmill exercise test, independently of the presence or not of DM2, hypertension and obesity. **Results:** The participants with DM2 (obese and non-obese) had decreased levels of glucose, triglycerides and TSH ($P < 0.01$) and increased levels of HDL-cholesterol, CK, CK-MB, LDH, renin, aldosterone ($P < 0.01$) after Bruce test. The non-diabetics participants (obese and non-obese) had decreased levels of glucose, urea, triglycerides, CK, CK-MB, LDH ($P < 0.01$) and decreased levels of hematocrit, HDL-cholesterol ($P < 0.01$) after Bruce test. All the other alterations have no statistical importance. **Conclusion:** Our results proved that the physical exercise, even if it has short duration like that of the Bruce test, has beneficial results in persons with DM2, obesity and arterial hypertension, as well as in normal persons.

Keywords

Diabetes Mellitus, Bruce Treadmill Test, Hypertension, Obesity

1. Introduction

In recent years, Western lifestyle diseases, such as diabetes mellitus, arterial hypertension, obesity, cardiovascular diseases, etc. have been made epidemic in our country like others. These diseases, to great extent, are due to food overconsumption, poor body training and unhealthy diets [1] [2] [3] [4]. Epidemiological and clinical studies in Greece (ATTICA study) [5] [6] [7] show that the frequency of the diseases, depending on the social and economic conditions, the age and the lifestyle of the population like similar studies in other countries.

In Europe, there are 48 million people with diabetes (7.8% of the population) while, in relative studies predicting that by the end of 2025, the percentage will increase to 20% and the disease will be offended increasingly young and obese individuals. These statistics concern Greece also, which holds one of the first places, in the frequency of obese children [6].

Today obesity is considered a "disease" and a health problem, because it is associated with serious diseases like diabetes and cardiovascular diseases, and is one of the risk factors of metabolic syndrome. Obesity is not combined only with the quantity and the poor quality of food, but more often is combined with a sedentary lifestyle and non-fitness. Genetic factors are responsible for the obesity and diabetes but these diseases would not have occurred if there was not a corresponding morbidly environment. It is known from international studies that even small changes in daily lifestyle (for instance improving the quality and reducing the amount of food and introducing physical activity in the daily program) contribute to the reduction of hyperglycemia, hypertension and BMI [1] [2] [3].

The latest WHO recommendations (2020) in all age groups of healthy subjects and individuals with chronic diseases (overweight, obesity, diabetes, hypertension, atherosclerotic, cardiovascular disease, cancer) report that the physical exercise must be at least 75 - 150 minutes of vigorous-intensity aerobic physical activity; or an equivalent combination of moderate- and vigorous intensity activity throughout the week for substantial health benefits. As part of their weekly physical activity, older adults with these chronic conditions should do varied multicomponent physical activity that emphasizes functional balance and strength training at moderate or greater intensity on 3 or more days a week, to enhance functional capacity and prevent falls [8]. Brisk walking seems to be the preferred aerobic exercise as improves the metabolic biomarkers (4). The same is happening with Yoga exercise where metabolic biomarkers and oxidative stress biomarkers are improved too [9].

Based on all mentioned before and because of diabetes, obesity and hyperten-

sion are major public health problems in our country, we studied the changes caused by physical exercise on biochemical and hormonal parameters in diabetic, obese and hypertensive patients. In spite of measuring metabolic biomarkers during certain training sport periods, we measured metabolic biomarkers at a given time and intensity exercise done during the stressing test by BRUCE [10].

2. Materials and Methods

The study took place in the Fatigue Laboratory of Cardiological Clinic of General Hospital of Zakynthos island. 59 patients participated, 39 men (66.1%) and 20 women (33.9%). Their age varied between 33 to 80 years old. We separated them in four groups according to their Body Mass Index (BMI) and if they suffered from type 2 diabetes mellitus (DM2) or not (Tables 1-3).

Among the 59 participants of our study 23 were diabetics with DM2 while the rest of them (36 persons) were non-diabetics. The average age of diabetics was 62 ± 9 years old, while the age of non-diabetics was 56 ± 12 years old. The two groups (DM2 vs non DM2) hadn't statistical important differences between their age (p = 0.060) and BMI (p = 0.293). On the contrary the participants with DM2

Table 1. The four patient's groups of our study.

	N	Glucose status	BMI value
GROUP A	12	Diabetic[1]	Non-obese
GROUP B	11	Diabetic	Obese[2]
GROUP C	14	Non-diabetic	Non-obese
GROUP D	22	Non-diabetic	Obese

[1]Persons with glucose levels > 115 mg/dL, [2]Persons with BMI > 27.

Table 2. The group's values of age and BMI.

	AGE				
	Mean	StDev	Median	Minimum	Maximum
GROUP A	62.08	9.26	63.5	44	76
GROUP B	62.09	8.58	63	50	74
GROUP C	58.93	9.63	61	41	80
GROUP D	54.41	12.92	53.5	33	76

Table 3. The group's values of age and BMI.

	BMI				
GROUP A	24.525	1.669	24.7	21.5	26.7
GROUP B	32.709	2.99	32.4	28	37.5
GROUP C	24.55	1.733	24.7	19.5	27
GROUP D	29.6	2.478	28.95	27.1	37.2

suffered more often from hypertension than of non DM2 participants with statistical significance (p = 0.002).

The BRUCE tests were done according to the international guidelines. Their duration (5 to 15 minutes) and their stress adjusted to each patient's age and physical condition. All blood determinations were done 30 minutes before the Bruce test and just after the end of it. The blood tests included glucose, urea, creatinine, potassium, sodium, cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, CRP, CK, CK-MB, LDH, CRP, TSH, FT_3, FT_4, T_3, T_4 C-peptide, insulin, growth hormone, cortisol and renin/aldosterone. All individuals had CRP values as negative.

The concentrations of the results are presented as: mean value ± standard deviation (Tables 4-6). We converted almost all units to SI (moles) in order our

Table 4. Comparisons of Diabetics according to BMI (Diabetic normal body weight persons [group A] vs Diabetic overweight persons [group B]). The table includes number of participants (N), average, standard deviation and the level of significance (P [Wilcoxon signed test]) of several laboratory tests before and after BRUCE test.

ANALYTES	DIABETICS with BMI < 27 kg/m² Group A				DIABETICS with BMI >27 kg/m² Group B			
	N	Before	After	P	N	Before	After	P
Hematocrit (%)	12	42.4 ± 2.9	42 ± 2.9	0.844	11	40.8 ± 2.7	40.8 ± 2.9	0.304
Glucose (mmol/L)	12	8.8 ± 2.72	8.0 ± 2.4	0.002	11	8.6 ± 2.23	7.9 ± 2.27	0.003
UREA (mmol/L)	12	6.65 ± 1.12	6.5 ± 2.55	0.058	11	7.05 ± 1.33	7.07 ± 1.28	0.718
Creatinine (μmol/L)	12	0.09 ± 0.01	0.09 ± 0.01	0.040	11	0.09 ± 0.01	0.09 ± 0.02	0.79
Potassium (mmol/L)	12	4.7 ± 0.5	4.8 ± 0.4	0.402	11	4.7 ± 0.48	4.7 ± 0.37	0.156
Sodium (mmol/L)	12	140.1 ± 2.4	139.9 ± 2.5	0.034	11	138.7 ± 2.15	139.5 ± 1.81	0.037
Cholesterol (mmol/L)	12	5.9 ± 1.42	5.9 ± 1.48	0.373	11	5.7 ± 0.98	5.3 ± 0.98	0.196
Triglycerides (mmol/L)	12	1.3 ± 0.69	1.2 ± 0.60	0.002	11	1.9 ± 0.96	1.7 ± 0.87	0.003
HDL-Cholesterol (mmol/L)	12	1.5 ± 0.39	1.5 ± 0.36	0.035	11	1.2 ± 0.25	1.3 ± 0.25	0.003
LDL-Cholesterol (mmol/L)	12	3.8 ± 1.14	3.8 ± 1.22	0.906	11	3.6 ± 0.90	3.5 ± 0.86	0.398
CK (U/L)	12	132 ± 128.9	140 ± 131.9	0.002	11	142 ± 120	153 ± 131	0.003
CK-MB (U/L)	12	17 ± 5.6	20 ± 5.4	0.002	8	15.9 ± 3.1	18.0 ± 3.5	0.011
LDH (U/L)	12	291 ± 89.5	302 ± 88.4	0.002	11	302 ± 40.6	322.5 ± 38.5	0.003
PT (sec)	12	12.6 ± 0.40	12.7 ± 0.3	0.624	11	12.5 ± 1.1	12.7 ± 1.2	0.151
T_3 (nmol/L)	12	1.2 ± 0.56	1.2 ± 0.49	0.455	11	1.3 ± 0.35	1.3 ± 0.32	0.789
T_4 (nmol/L)	12	115.4 ± 30.73	112.4 ± 33.66	0.388	11	105.6 ± 32.37	106.4 ± 31.33	0.304
TSH (μU/mL)	12	1.3 ± 0.95	1.0 ± 0.85	0.002	11	2.4 ± 1.63	1.9 ± 1.23	0.003
FT_3 (pmol/L)	12	4.07 ± 0.78	4.13 ± 0.89	0.480	11	5.05 ± 1.09	5.13 ± 0.97	0.327
FT_4 (pmol/L)	12	14.8 ± 3.86	15.9 ± 4.25	0.254	11	16.1 ± 2.96	16.7 ± 3.34	0.109
Microalbumin mg/dL	12	25 ± 46.05	30.7 ± 49.89	0.012	11	7.9 ± 6.67	9.9 ± 8.5	0.176
Growth hormone (μg/L)	11	1.3 ± 1.53	2.3 ± 2.37	0.021	9	0.8 ± 0.6	1.0 ± 0.9	0.766

Continued

Insulin (pmol/L)	10	33.3 ± 13.05	38.19 ± 14.51	0.168	8	82.2 ± 46.8	87.0 ± 45.3	0.674
C-peptide (nmol/L)	10	0.7 ± 0.16	0.5 ± 0.24	0.284	8	0.6 ± 0.43	0.7 ± 0.53	0.917
Cortisol (nmol/L)	10	525.5 ± 129.65	507.6 ± 177.65	0.508	8	475.0 ± 107.6	357.7 ± 162.8	0.017
Renin (pmol/L)	10	125.3 ± 156.20	209.5 ± 228.5	0.005	7	87.7 ± 41.39	112.8 ± 56.17	0.018
Aldosterone (nmol/L)	10	0.7 ± 0.21	1.0 ± 0.33	0.005	7	0.81 ± 0.27	1.1 ± 0.49	0.018

Table 5. Comparisons of Non-Diabetics according to BMI (Non-Diabetic normal body weight persons [group D] vs Non-Diabetic overweight persons [group C]). The table includes number of participants (N), average, standard deviation and the level of significance (P [Wilcoxon signed test]) of several laboratory tests before and after BRUCE test.

ANALYTES	NON-DIABETICS with BMI < 27 kg/m² Group C				NON-DIABETICS with BMI >27 kg/m² Group D			
	N	Before	After	p	N	Before	After	p
Hematocrit (%)	14	40.8 ± 5.2	41.0 ± 5.1	0.018	22	42.7 ± 3.90	42.8 ± 3.80	0.031
Glucose (mmol/L)	14	5.16 ± 0.27	4.82 ± 0.27	0.001	22	5.7 ± 0.44	5.1 ± 0.33	<0.001
UREA (mmol/L)	14	7.1 ± 2.72	6.9 ± 2.55	0.072	22	6.9 ± 2.58	6.5 ± 2.1	0.038
Creatinine (μmol/L)	14	0.08 ± 0.01	0.09 ± 0.01	0.086	22	0.1 ± 0.02	0.1 ± 0.02	0.321
Potassium (mmol/L)	14	4.4 ± 0.30	4.4 ± 0.30	1.000	22	4.5 ± 0.40	4.6 ± 0.40	0.202
Sodium (mmol/L)	14	140 ± 2.60	140 ± 2.60	0.623	22	139.7 ± 2.0	140.3 ± 2.0	0.051
Cholesterol (mmol/L)	14	5.6 ± 1.10	5.61 ± 1.08	0.722	22	6.0 ± 1.24	6.0 ± 1.32	0.974
Triglycerides (mmol/L)	14	1.17 ± 0.65	1.20 ± 0.63	0.002	22	1.6 ± 0.72	1.5 ± 0.66	<0.001
HDL-Cholesterol (mmol/L)	14	1.3 ± 0.26	1.3 ± 0.27	0.001	22	1.3 ± 0.28	1.3 ± 0.28	<0.001
LDL-Cholesterol (mmol/L)	14	3.8 ± 0.82	3.8 ± 0.82	0.087	22	4.0 ± 0.85	4.0 ± 0.93	0.426
CK (U/L)	14	104.0 ± 40.69	111.6 ± 40.12	0.001	22	147.2 ± 167.0	156.5 ± 172.0	<0.001
CK-MB (U/L)	14	19.0 ± 11.95	21.4 ± 12.31	0.001	22	17.0 ± 5.0	19.0 ± 5.0	0.003
LDH (U/L)	14	274.7 ± 51.45	284.2 ± 54.51	0.001	22	303.0 ± 60.0	320.0 ± 67.0	0.016
PT (sec)	14	13.0 ± 1.05	1.17 ± 0.23	0.120	22	12.7 ± 0.72	12.9 ± 0.65	0.304
T_3 (nmol/L)	14	1.2 ± 0.19	1.2 ± 0.020	0.441	22	1.1 ± 0.23	1.2 ± 0.22	0.149
T_4 (nmol/L)	14	105.6 ± 32.37	106.4 ± 31.33	0.414	22	93.8 ± 14.18	95.5 ± 16.51	0.986
TSH (μU/mL)	14	1.8 ± 1.39	1.5 ± 1.30	0.001	22	2.3 ± 2.84	1.9 ± 2.72	0.559
FT_3 (pmol/L)	14	4.5 ± 0.87	4.34 ± 0.95	0.087	22	4.6 ± 0.75	4.6 ± 0.86	0.559
FT_4 (pmol/L)	14	15.4 ± 3.6	15.4 ± 4.76	0.550	22	14.9 ± 4.24	15.1 ± 5.01	0.016
Microalbumin mg/dL	13	3.2 ± 3.76	8.9 ± 20.80	0.223	19	13.8 ± 19.91	39.5 ± 50.66	0.001
Growth hormone (μg/L)	12	1.8 ± 1.30	2.9 ± 2.60	0.026	20	0.61 ± 0.39	1.865 ± 1.96	0.099
Insulin (pmol/L)	12	32.8 ± 18.19	37.5 ± 19.16	0.593	20	63.9 ± 22.50	54.4 ± 24.72	0.084
C-peptide (nmol/L)	12	0.4 ± 0.22	0.4 ± 0.22	0.136	20	0.6 ± 0.41	0.5 ± 0.35	0.099
Cortisol (nmol/L)	12	471.0. ± 146.4	382.1 ± 81.93	0.099	20	545.6 ± 138.5	549.8. ± 178.2	0.073
Renin (pmol/L)	8	99.3 ± 138.2	187.2 ± 208.58	0.017	8	105.2 ± 66.60	146.0 ± 104.50	0.069
Aldosterone (nmol/L)	8	0.8 ± 0.35	0.6 ± 0.30	0.779	8	1.0 ± 0.22	1.3 ± 0.64	0.050

Table 6. Comparisons of hypertensive vs non-hypertensive persons. The table includes number of participants (N), average, standard deviation and the level of significance (P [Wilcoxon signed test]) of several laboratory tests before and after BRUCE test.

ANALYTES	HYPERTENSIVE (N = 19)				NON-HYPERTENSIVE (N = 40)			
	N	Before	After	P	N	Before	After	P
Hematocrit (%)	19	42.5 ± 3.67	42.5 ± 3.57	0.810	40	41.5 ± 3.99	41.7 ± 3.95	<0.001
Glucose (mmol/L)	19	7.9 ± 2.64	7.2 ± 2.43	<0.001	40	6.17 ± 1.75	5.65 ± 1.65	<0.001
UREA (mmol/L)	19	6.9 ± 1.22	6.8 ± 1.20	0.174	40	7.06 ± 2.48	6.78 ± 2.15	0.007
Creatinine (μmol/L)	19	0.09 ± 0.01	0.09 ± 0.01	0.445	40	0.09 ± 0.02	0.09 ± 0.02	0.041
Potassium (mmol/L)	19	4.6 ± 0.49	4.7 ± 0.41	0.166	40	4.6 ± 0.40	4.6 ± 0.37	0.174
Sodium (mmol/L)	19	139.7 ± 2.02	140.0 ± 2.5	0.327	40	139.6 ± 2.30	140.1 ± 2.34	0.010
Cholesterol (mmol/L)	19	5.8 ± 1.34	5.8 ± 1.37	0.256	40	5.9 ± 1.13	5.8 ± 1.17	0.867
Triglycerides (mmol/L)	19	1.60 ± 1.00	1.43 ± 0.89	<0.001	40	1.5 ± 0.65	1.3 ± 0.61	<0.001
HDL-Cholesterol (mmol/L)	19	1.34 ± 0.30	1.38 ± 0.29	0.001	40	1.3 ± 0.3	1.3 ± 0.31	<0.001
LDL-Cholesterol (mmol/L)	19	3.7 ± 1.02	3.7 ± 1.07	0.778	40	3.9 ± 0.86	3.9 ± 0.90	0.270
CK (U/L)	19	145.8 ± 130.08	156.7 ± 136.60	<0.001	40	126.9 ± 128.13	134.8 ± 132.17	<0.001
CK-MB (U/L)	19	17.1 ± 8.1	19.6 ± 8.05	<0.001	40	17.5 ± 6.7	19.5 ± 7.14	<0.001
LDH (U/L)	19	304.7 ± 71.08	322.2 ± 76.96	<0.001	40	288.0 ± 57.42	301.4 ± 58.85	<0.001
PT (sec)	19	12.1 ± 0.86	12.9 ± 0.85	0.057	40	12.7 ± 0.85	12.9 ± 0.81	0.030
T_3 (nmol/L)	19	1.2 ± 0.51	1.2 ± 0.46	0.968	40	1.2 ± 0.20	1.2 ± 0.21	0.033
T_4 (nmol/L)	19	110.6 ± 27.20	110.6 ± 28.48	0.968	40	99.1 ± 23.94	100.4 ± 23.81	0.375
TSH (μU/mL)	19	1.6 ± 0.88	1.3 ± 0.77	0.001	40	2.2 ± 2.39	1.8 ± 2.22	<0.001
FT_3 (pmol/L)	19	4.6 ± 0.77	4.5 ± 0.81	0.519	40	4.6 ± 0.76	4.54 ± 0.81	0.342
FT_4 (pmol/L)	19	15.4 ± 3.7	15.4 ± 3.9	0.131	40	15.4 ± 3.7	15.4 ± 3.9	0.393
Microalbumin mg/dL	15	13.2 ± 28.77	28.9 ± 47.05	0.009	38	12.7 ± 16.3	15.7 ± 19.02	0.001
Growth hormone (μg/L)	17	1.1 ± 1.01	2.0 ± 2.06	0.006	38	1.1 ± 1.27	2.2 ± 2.38	0.393
Insulin (pmol/L)	14	52.1 ± 41.81	54.1 ± 44.06	0.594	36	54.2 ± 26.87	51.4 ± 24.37	0.001
C-peptide (nmol/L)	14	0.5 ± 0.23	0.6 ± 0.40	0.826	36	0.5 ± 0.39	0.5 ± 0.33	0.255
Cortisol (nmol/L)	14	563.2 ± 151.65	493.9 ± 190.99	0.221	22	496.5 ± 124.96	411.0 ± 146.20	0.687
Renin (pmol/L)	10	124.4 ± 159.03	242.9 ± 232.37	0.005	22	82.9 ± 48.59	113.7 ± 71.34	0.001
Aldosterone (nmol/L)	11	0.7 ± 0.21	0.9 ± 0.26	0.003	22	0.88 ± 0.29	1.1 ± 0.55	0.019

results to be compared to other studies. All statistical tests were done by Wilcoxon signed test since all statistical distributions were no normal. The statistical package was SPSS v.28 (academic license).

3. Results

The results of the study were collected in three tables represented five different comparisons or our groups according to BMI, the presence DM2 and High Pressure.

Some of the participants suffered from arterial hypertension (50% of the diabetics and 15% of the non-diabetics). This finding led us to study if our test's results differed between hypertensive and non-hypertensive persons (**Table 6**).

The muscular work of Bruce treadmill had negative correlation with age and no correlation with other physical conditions of the participant like hypertension. dyslipidemia and coronary disease. The negative correlation of the muscular work with the age complies with previous studies which proved that the maximum aerobic capacity (VO_2max) decreases every new decade with an accelerated rate [10]. The participants with prolong endurance times in Bruce test ($t > 10$ min) were 33 - 54 years' old. These persons either they were working in agricultural industry or other high demanding muscular jobs either they were persons with daily exercise programs. All these had the highest capacity of aerobic work and the better performance in Bruce test.

4. Conclusions

Carbohydrates. The levels of glucose decreased in all participants' groups (statistical significance $p < 0.01$). The decrease is due to glucose overconsumption from the working muscles. It is known that the muscles' reliance on the glucose is parallel to the decrease of glycogen's reserves and to the intensity of the muscular effort [11].

Insulin/C-peptide. The levels of insulin and C-peptide are supposed to be increased as a feedback to the decrease of glucose. This happened in groups A, C and D (insulin) and in group B (C-peptide). Their differences, before and after Bruce test, were not statistical important.

Lipids. Lipids and LDL-cholesterol are supposed to be decreased after extensive exercise. Triglycerides and HDL had a statistical important reduction ($P < 0.05$), on the contrary, to the total cholesterol and LDL-cholesterol which change was not statistically important.

Thyroid hormones. Despite the physical condition of the participants (DM2, obesity, arterial hypertension) the level of TSH decreased after Bruce test. The overweight participants had a larger reduction of TSH than the normal weight persons. In order to find out the reasons for the TSH decrease, we studied the correlation between the TSH levels and age, TSH and BMI, TSH and duration of Bruce test and TSH of diabetics versus the TSH of non-diabetics. The correlation between BMI and TSH in diabetics before and after the Bruce test had statistical significance ($r = 0.518$, $p < 0.05$). The participants with DM2 increased the levels of renin and aldosterone after the completion of the muscular work. Both increases have statistical significance ($p < 0.05$). After the end of muscular activity, the levels of growth hormone increased too. These differences between diabetics and non-diabetics were statistically important ($p < 0.05$) only for the non-obese persons. The reason for TSH change is probably its feedback mechanism. Since the production of TRH hormone is decreasing during the exercise and the changes for thyroid metabolism during the stress [12] [13]. Furthermore, the

results of this study confirmed the theory about the involution of the thyroid gland in body exercise effects.

Cortisol. Cortisol is supposed to be increased because of the stress and the anxiety of the patients during the Bruce test [14] [15] [16]. This didn't happen in our experiment. Cortisol levels have a wide biological variation, a larger number of participants would probably provide more understanding results.

Growth hormone. Growth hormone increased in all groups (A, B, C and D) after Bruce tests. The differences were statistical important between non-obese persons. According to bibliography growth hormone increases during exercise since it stimulates fuel metabolism and decreases lipids [17]. Obesity blocks the normal growth hormone's stimulation during exercise [17] [18]. This proved in our study too.

Renin/aldosterone. Renin and aldosterone are supposed to be increased during the muscular work which activates the hormone mechanism of renin/angiotensin/aldosterone which tends to restore the blood pressure and increase the concentration of renin and aldosterone [19]. In our study, that proved true only between the diabetics. In diabetics, the increased levels of renin/aldosterone Diabetics increased values after Bruce tests had statistical importance ($p < 0.05$). Non-Diabetics without obesity had lower levels of aldosterone ($P < 0.05$) but their levels of renin increased like diabetics ($P < 0.05$).

Urine protein. The levels of urine microalbumin, before and after Bruce test, increased in all groups of our study. Among the diabetics with BMI < 27 and the non-diabetics with BMI > 27 the increase was statistically important ($p < 0.05$). The diabetics with BMI > 27 had already higher values of urine microalbumin than the non-diabetics, before the test, which become even higher after the test. Initially, these persons had small amounts of protein in urine strip, but these protein levels increased during the muscular work. We observed also that hypertensive persons had increased levels of microalbumin after the Bruce test. In one case, the increased values of microalbumin had a relation to chronic renal failure. Urinalysis revealed that some persons had "trace" levels of urine protein after the Bruce test. Persons with low values of urine microalbumin before the test and high values (>30 mg/L) after the test had also a hypertensive response to Bruce test [20] [21] [22].

5. Discussion

Nowadays, physical exercise is considered part of treatment protocol for diabetes mellitus, arterial hypertension and obesity. The triptych: consulting, diet (under specialists' supervision) and exercise, is capable to decrease the risk of DM2 in 60% of non-diabetics with impaired glucose tolerance [9] [11]. Additionally, the medium level physical exercise improves significantly the glycemic tests' results in persons with DM2 as diabetics who follow an aerobic exercise program of medium intensity have decreased death rates [23]. An obesity therapeutic program should include physical exercise besides the decrease of received calories.

This combination would have more possibilities of success compared to programs that ignore the physical exercise [24] [25].

Body exercise influences the levels of blood substances. This influence depends on exercise's intensity, duration, participation of particular muscles, kind of exercise (continuous or not), environmental conditions (temperature, moisture, etc.) and the dietary preparation of the person [26] [27]. This study attempted to prove that physical exercise, even with short duration like that of Bruce test, has beneficial results in persons with DM2, obesity and arterial hypertension, as well as in normal persons. These beneficial results can be summarized as follows: decrease of glucose, triglycerides and TSH, increase of HDL-cholesterol. These parameters are considered the target of every therapeutical intervention in patients with DM2, obesity and/or arterial hypertension.

Furthermore, persons with mild to medium primary hypertension, the body exercise decreases systolic and diastolic hypertension [28]. It is well known that in general population the ratio of hypertension in diabetics is twice more than in normal persons. This difference is due to the insulin resistance of the patients with DM2 [29]. Specifically, the percentage of hypertensive patients with DM2 patients varies between 30% - 50%, while the percentage of hypertensive patients with impaired glucose tolerance (IGT) varies between 20% - 40% [11] [29]. During Bruce test the produced muscular work is too high and the organism loses its ability to produce energy only from glucose's metabolism. So, the organism covers the needs of muscular cells' energy from the metabolism of carbohydrates and fats [30]. During the first stages of muscular work the muscle's glycogen and blood glucose are rapidly consumed and as the muscular exercise is continuing and the perfusion of the muscles is increasing, glycogen and glucose are replaced by other sources of energy like the free fatty acids, which is the basic source at this stage [31].

On the contrary to the reduction of glucose, there was no statistical significance in the reduction of cholesterol and LDL-cholesterol. Probably this is due to the short time interval of the muscles' work. It is proved that the prolonged muscles' work decreases more effectively the levels of total cholesterol and LDL-cholesterol [32].

The extremely high levels of urine microalbumin, after Bruce test, are due to the increase of arterial pressure because of the stress. The increase of urine microalbumin is related to diabetic nephropathy [20] and hypertension (systolic arterial pressure and 24h variation of systolic and diastolic arterial pressure) [21] [22]. Both are considered early signs of kidney disease.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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