Physical Activity Review vol. 4, 2016

Original Articles

DOI: http://dx.doi.org/10.16926/par.2016.04.03

Obesity: reversible biological adaptation or disease?

Authors' Contribution:

A – Study Design
B – Data Collection
C – Statistical Analysis
D – Manuscript Preparation
E – Funds Collection

Karol Pilis[1], Anna Pilis[1], Krzysztof Stec[1], Cezary Michalski[1], Michał Zych[1], Jacek Buchta[1], Wiesław Pilis[2]

[1] Institute of Physical Education, Tourism, and Physiotherapy, University of Czestochowa, Poland

[2] Institute of Physiotherapy, Public Medical Higher Vocational School, Opole, Poland

Abstract

Obesity is an unfavorable state of health as a result of which come in the initial adaptation, that could gradually transform into specific disease condition. The aim of the study was to assess the degree of middle-aged men adaptation to obesity in terms of somatic changes, exercise capacity and cardiopulmonary fitness. The study involved 12 obese middle-aged men (OG) - BMI = 34.32 ± 4.11 kg/m^2. The control group (CG) consisted of 12 non-obese middle-aged men- BMI = 23.72 ± 1.83 kg/m^2, with similar body height (BH) to OG. After recording somatic and physiological data at rest, participants were subjected to the bicycle ergometer test (BT) gradually increasing intensity. During the BT aerobic power (AP) was recorded and analyzed along with parameters characterizing the efficiency of the circulatory and respiratory systems at anaerobic threshold (AT) and the maximum load (ML). This study has shown that obese men have a higher body mass (BM), a higher content of fat (BF), an increased lean body mass (FFM) and a higher content of water (TBW) than CG ($p < 0.001$). Absolute and relatively expressed in relation to the FFM respondents' values of AP and VO_2 were similar in both groups and after taking under the consideration the body weight, they were significantly lower in OG than in the CG. Furthermore, adverse effects have been observed that emerged on some cardiovascular and respiratory variables at rest in case of OG; these effects did not occur during physical exertion. Beyond negative changes occurring obesity in the first stage led to favorable somatic adaptation that entails an increase of FFM in OG, which gives a positive impact on the capacity of physical work of obese men and produces a beneficial effect on the resulting compensation impairment in the respiratory and circulatory systems of these subjects.

Keywords: obesity, exercise, body weight, exercise capacity, cardio-respiratory fitness.

INTRODUCTION

Obesity is one of the factors affecting inter alia on the quality and duration of life [1], and causing the premature death [2-3]. It has been noted that obesity reduces vital capacity and its components [4-5]. By modifying the metabolic processes of the lungs and the mechanics of breathing obesity alters the body's exercise tolerance [6]. Also in the circulation system obesity leads to adverse changes, which are manifested amongst other by: high blood pressure, elevated levels of low-density lipoprotein cholesterol, low levels of high-density lipoprotein cholesterol [7], increased left ventricular heart mass [8], renal hyperfiltration [9]. These changes can cause low cardiovascular fitness and reduced body's adaptation to physical exertion. Since obesity is often accompanied by hypertension hence these two ailments jointly affect deterioration in the structure and function of the cardiovascular system. It is known that the hallmark of hypertension is: an increased total peripheral resistance, a contracted intravascular volume, maintaining a normal cardiac output and increased left ventricular stroke volume due to an increased afterload, as well as concentric hypertrophy of left ventricular. In contrast, in case of obese patients the following enhanced values were recorded: intravascular volume, left ventricular filling pressure and cardiac output, but decrease total peripheral and renal vascular resistance. Left ventricular changes consist of eccentric hypertrophy [10].
In case of somatic conditions it was observed that obese people have an increased FFM as an expression of adaptation to the continuous body workload with excess fat and exercise capacity of obese people do not have to be lowered, on the contrary, it may be increased [11].

Given the above divergent facts we have decided in this study to check whether in middle-aged men obesity affects somatic, exercise capacity, and the formation of metabolic changes as well as the efficiency of the circulatory and respiratory systems.

MATERIALS AND METHODS

The study involved 12 obese men (OG) and 12 lean men (CG). Twenty four hours before testing it was recommended to participants to properly hydrate by drinking 2.5 liters of still mineral water and to avoid coffee and alcohol consumption at least 12 hours before the testing. Respondents described their health as good, have not used any medication and 6 of them cultivated recreational physical activity. In the first stage of the study respondents' age and body height (BH) were recorded. Their body weight (BW), fat content (BF), lean body mass (FFM), and the total water content of the body (TBW) were measured using body composition analyzer (Tanita BS 418 – MA). Then after 10 minutes of resting (R) in a sitting position systolic (SBP) and diastolic blood pressure (DBP) as well as heart rate (HR – using a device Polar) were measured. In the next stage the following values were calculated: mean arterial pressure (MAP), pulse pressure (PP) and the rate pressure product of systolic pressure x heart rate (RPP = SBP * HR * 1000^{-1}). The subjects with breathing mask on their face were attached to a rapid gas analyzer (Ergo Card), which recorded the following variables: minute ventilation (V_E), minute oxygen uptake (VO_2), minute carbon dioxide excretion (VCO_2), respiratory quotient (RER), and cardiovascular variables such as cardiac output (CO), and stroke volume (SV). Cardiac output (CO) was calculated using the Fick's equation based on a noninvasive method of oxygen uptake by the body [12].

Based on this data were calculated the following physiological variables: oxygen pulse (VO_2/HR), the ratio SV/PP, the ventilatory equivalent for oxygen (EQO_2- V_E/VO_2), the

ventilatory equivalent for carbon dioxide ($EQCO_2$- V_E/VCO_2), total peripheral vascular resistance (TPR = MAP/CO), and the minute amount of energy expenditure (EE) by the modified Weir equation [13].

$$EE = (3.94 \times VO_2) + (1.1 \times VCO_2)$$

where, EE - is the energy expenditure (kcal/min), VO_2 - is the volume oxygen uptake (l/min), VCO_2 - is the volume of carbon dioxide excretion (l/min).

In a further step the subjects performed a bicycle ergometer test (BT) with increasing intensity, where workload was started at the power equal to 60W, then every 3 minutes the load was increased by 30W until the individual's maximum potential exertion, which was considered to be the maximum aerobic power (APmax). All the above-mentioned physiological variables were measured and the similar calculations were done for all loads. The dynamic stress changes V_E, VCO_2 and RER were used to calculate with the method proposed by Beaver et al. [14] anaerobic threshold (AT), which was expressed in watts [W]. After the BT has completed, the amount of oxygen debt (OD) was measured for five minutes.

The data was presented as means and standard deviations. The significance of intergroup differences between the means was calculated with t-test and for the simultaneous determination of differences inter- and intra-values obtained in the R, at AT, and at ML was used a two-way analysis of variance for repeated measures with Bonferoni post hoc test (SPSS software, version Statistics 20). The level of significance was set at 0.05.

RESULTS

The tested people were of similar age and had similar body height (Table 1). On the other hand OG were characterized by higher body weight, BF and BMI, and this group had a higher absolute as well as lower relative value of FFM, and TBW than CG ($p<0.001$).
Power developed in BT expressed in absolute terms and in relation to FFM was similar in both groups, at AT and at ML, while expressed in relations to the body mass was higher at AT ($p=0.011$) and at ML ($p = 0.01$) in CG than OG. Two other variables describing exercise capacity, ie. AT (% VO_2max) and the OD did not differ between the two groups (Table 2).

The analysis of variance showed that the main effects of determining the difference between the two groups occurred only for SV ($F=14.127$, $p=0.003$), but post hoc analysis revealed that the resting variable and attained at the AT for OG were adequately higher ($p<0.01$ and $p<0.05$) than in the CG. TPR achieved lower values ($F=4.967$, $p=0.048$) at rest ($p<0.01$) in the group OG than in CG. In terms of other intergroup variables analyzed at R and during the BT there were not recorded statistically significant main effects but post hoc calculations have shown higher resting values of: SBP ($p<0,05$), CO ($p<0,001$), MAP ($p<0,05$) and RPP ($p<0,01$) in OG compared to CG (Table 3). In addition, despite the absence of the main effects of differences, respiratory variables showed higher post hoc values of V_E, VO_2 and VCO_2 at rest ($p<0.001$) and lower resting values for SaO_2, EQO_2 and $EQCO_2$ ($p<0.05$) as well as lower relative VO_2max ($p<0$ 05) achieved at ML in the OG as compared to CG. Also, analysis of variance had shown lack of main statistical effect in relation to EE but in post hoc calculation it was observed that OG spent at rest more energy ($p<0,001$) than CG (Table 4). All recorded and calculated variables in R at AT and at ML during BT beyond the DBP changed significantly under the influence of

applied effort (p<0.001). With respect to $V_{E'}$ PP, EQO_2, $EQCO_2$ and TPR there were significant interactions group- exercise intensity (Table 3 and 4).

Table 1. Somatic characteristics of investigated subjects.

Variables	Groups	x	±SD	t	p<
Age [years]	CG	51,50	11,28	0,733	0,471
	OG	48,23	11,00		
BW [kg]	CG	76,62	6,98	-7,975	**0,001**
	OG	109,94	12,81		
BH [cm]	CG	179,67	4,48	0,283	0,780
	OG	179,08	5,79		
BF [%]	CG	18,98	4,02	-7,960	**0,001**
	OG	31,55	3,87		
BF [kg]	CG	14,75	4,13	-8,097	**0,001**
	OG	34,88	7,63		
FFM [%]	CG	81,00	4,02	7,932	**0,001**
	OG	68,45	3,89		
FFM [kg]	CG	61,87	4,01	-5,220	**0,001**
	OG	75,06	7,85		
TBW [%]	CG	59,28	2,95	7,926	**0,001**
	OG	50,10	2,84		
TBW [kg]	CG	45,28	2,94	-5,231	**0,001**
	OG	54,95	5,73		
BMI [kg/m^2]	CG	23,72	1,83	-8,201	**0,001**
	OG	34,32	4,11		

Table 2. Exercise capacity and physical fitness of investigated subjects

Variables	Groups	x	±SD	t	p<
APAT [W]	CG	150,00	65,23	0,654	0,519
	OG	133,85	58,24		
APML [W]	CG	195,00	56,49	-0,767	0,451
	OG	210,00	40,62		
APAT/BW [W/kg]	CG	1,94	0,75	2,761	**0,011**
	OG	1,23	0,54		
APML/BW [W/kg]	CG	2,54	0,63	2,823	**0,010**
	OG	1,93	0,43		
APAT/FFM [W/kg]	CG	2,40	0,96	1,822	0,081
	OG	1,78	0,75		
APML/FFM [W/kg]	CG	3,13	0,80	1,172	0,253
	OG	2,81	0,56		
AT[%VO2max]	CG	72,54	11,19	0,698	0,493
	OG	69,19	12,31		
OD [l/5min]	CG	2,98	1,60	-0,514	0,612
	OG	3,24	0,71		
OD [ml/kg/5min]	CG	38,18	17,49	1,649	0,12
	OG	29,31	6,42		

Table 3. Cardiovascular variables of investigated subjects

Variables	Groups	Exercise						Group		Exercise		Group vs. Exercise intensity (Interaction)	
		R		AT		ML							
		x	SD	x	SD	x	SD	F	p<	F	p<	F	p<
HR [ud/min]	CG	74,50	14,68	147,92	23,06	170,08	22,85	0,972	0,345	221,29	**0,001**	2,795	0,109
	OG	78,75	9,25	133,00	23,14	164,00	19,40						
SBP [mm/Hg]	CG	124,58*	11,17	196,25	35,04	209,17	30,96	2,651	0,132	118,950	**0,001**	0,853	0,455
	OG	139,58	12,69	208,33	27,50	227,50	19,48						
DBP [mm/Hg]	CG	83,75	6,78	87,50	10,55	86,67	11,74	1,875	0,198	0,932	0,425	0,399	0,681
	OG	92,50	11,18	92,50	12,70	91,25	13,67						
CO [l/min]	CG	4,50***	0,90	16,83	4,61	19,33	4,29	1,959	0,189	334,560	**0,001**	0,615	0,560
	OG	6,83	1,27	17,67	3,08	20,92	2,23						
SV [ml]	CG	61,78**	14,17	113,66*	21,56	113,66	19,79	14,127	**0,003**	110,111	**0,001**	0,772	0,488
	OG	82,57	20,43	134,27	18,93	129,10	17,88						
MAP [mmHg]	CG	97,42*	7,55	123,83	17,11	127,50	16,26	3,877	0,075	59,820	**0,001**	0,129	0,880
	OG	108,25	10,91	131,08	10,08	136,67	9,97						
RPP [mmHg/bpm]	CG	9,30*	2,19	29,19	7,18	35,70	7,17	0,26	0,730	169,514	**0,001**	1,691	0,233
	OG	11,00	1,1,71	28,15	8,09	37,41	6,34						
TPR [mmHg/l/min]	CG	22,36**	4,65	7,93	2,80	6,99	2,31	4,967	**0,048**	106,679	**0,001**	6,97	**0,013**
	OG	16,38	3,65	7,60	1,23	6,63	1,08						
VS/PP [ml/mmHg]	CG	1,58	0,47	1,11	0,33	0,98	0,32	0,818	0,385	23,605	**0,001**	1,895	0,201
	OG	1,78	0,49	1,27	0,46	0,98	0,25						

*- statistical difference between CG and OG; *-p<0,05; **-p<0,01; ***-p<0,00

Table 4. Respiratory variables and energy expenditure of investigated subjects

Variables	Groups	Exercise						Group		Exercise		Group vs. Exercise intensity (Interaction)	
		R		AT		ML							
		x	SD	x	SD	x	SD	F	p<	F	p<	F	p<
V_E [l/min]	GK	10,20***	1,59	60,10	30,87	99,67	41,82	0,853	0,376	82,299	**0,001**	4,103	**0,050**
	GO	14,57	2,88	59,87	14,43	116,24	17,99						
VO_2max [l/min]	GK	0,30***	0,07	2,20	1,02	2,94	0,95	0,395	0,542	297,419	**0,001**	1,859	0,206
	GO	0,49	0,12	2,23	0,61	3,20	0,55						
VO_2max [ml/min/kg]	GK	3,92	1,00	28,33	11,32	37,83*	10,19	4,225	0,064	210,819	**0,001**	3,198	0,084
	GO	4,50	1,24	20,42	5,77	29,83	5,25						
VCO_2 [l/min]	GK	0,25***	0,05	2,21	1,01	3,32	0,85	0,703	0,420	222,875	**0,001**	2,835	0,106
	GO	0,42	0,11	2,23	0,60	3,64	0,35						
RER	GK	0,85	0,05	1,00	0,02	1,15	0,11	0,34	0,857	306,071	**0,001**	0,193	0,827
	GO	0,86	0,04	1,00	0,02	1,13	0,11						
SaO_2 [%]	GK	96,17*	1,27	93,83	1,47	93,25	1,60	0,001	1,000	20,504	**0,001**	2,815	0,107
	GO	95,17	1,27	94,17	1,11	93,92	1,50						
VO_2/HR [ml/bpm]	GK	4,13**	1,21	14,76	5,26	17,47	4,44	2,233	0,163	314,488	**0,001**	0,376	0,696
	GO	6,27	1,76	16,53	3,05	19,82	3,80						
PP [mmHg]	GK	40,83	8,21	108,75	29,40	122,50	25,72	0,875	0,370	156,108	**0,001**	6,054	**0,019**
	GO	47,08	8,65	115,83	34,17	136,25	26,12						
EQO_2	GK	35,11*	6,01	27,01	2,41	33,71	4,65	0,044	0,838	22,520	**0,001**	5,713	**0,022**
	GO	30,49	4,44	27,21	2,38	37,30	8,58						
$EQCO_2$	GK	41,00*	6,27	26,89	2,43	33,71	4,65	0,223	0,646	47,022	**0,001**	8,093	**0,008**
	GO	35,23	5,15	27,12	2,09	37,31	8,58						
EE [kcal/min]	GK	1,46***	0,34	11,10	5,13	15,23	4,68	0,444	0,519	299,562	**0,001**	2,233	0,158
	GO	2,39	0,60	11,23	3,08	16,62	2,39						

*- statistical difference between CG and OG; *-p<0,05; **-p<0,01; ***-p<0,001

DISCUSSION

The main achievement of this work is that obese men compared to lean individuals, who are the same age and have similar body height, have reached a higher FFM and TBW. Higher values FFM are the expression of a specific body's adaptation to continuous load excess with body mass in obese individuals [15-16]. Our studies of the people led us to the fact that FFM in obese men were significantly higher than in CG ($p<0.001$) and this increase was due to "static continuous training" of these persons consisting in the carrying of excess body weight. This increase in FFM in obese individuals is a paradox in which the pathophysiologic factor undermining health may also become an advantage, improving the functioning of the body [17]. Occurring in sports increase of FFM is stimulated in a different way, because the physiological factors, which is training loads affecting only sessionally the body and as a result is a significant increase in this variable by the strength training [18] and endurance training [19], or specific loads used during sports team games [19]. Appearing at the same time increased FFM and higher TBW in OG is an indirect proof of the increased muscle mass in this group compared to CG, since among the FFM muscles are the largest tissue containing about 65-80% water.

In Aiello et al. [11] studies increase of FFM in obese people has led to an increase in the absolute exercise capacity. In our study, aerobic power reached at AT and ML in both groups was similar, but when the values of these two powers were divided by the weight of the subjects, the computed indicators were significantly higher for CG than OG. After calculating aerobic power in relation to FFM it was found that the two so calculated relations between the investigated groups did not differ statistically, and this proves that exercise capacity of both groups was similar. Thus, occurring obesity has not diminished exercise capacity in OG but thanks to the kind of training, consisting in constant carrying of increased body mass there was no reduction in muscle mass, despite commonly less occurring physical activity in obese individuals [20]. Other indirect indicators assessing physical performance subjects such as VO_2max or OD did not differ between the two groups, which points to their similar physical performance. The relatively high values of AT ($\%VO_2max$) oscillating around 70% did not differ between the two groups and indicate good adaptation of the respondents to exercise [21-22]. It is difficult to point to factors that could cause so high VO_2max at AT workload, although in an interview with the tested men it was found that in the each group (OG and CG) were three persons, who in the past practiced competitive sports and currently continued with a recreational training. While in the case of OG one could postulate that the relatively high value VO_2 achieved at the AT results in a longer training as an effect of carrying excess body weight, in the case of CG is difficult to name the factor for the increase in exercise capacity.

Observation of resting and exercise variables depicting the pulmonary function suggests a slightly severe adverse changes occurring in obese people. They were manifested in the range of larger values of V_E, VO_2 and VCO_2 measured at rest ($p<0.001$) and lower rest value of SaO_2, EQO_2 and $EQCO_2$ and lower relative VO_2max ($p<0.05$) in OG as compared to CG. It should be noted that there were no statistically significant effects in comparisons of the mean values of the two groups in terms of the variables described above. With some certainty it could be concluded that these unfavorable changes occurring in the OG had not yet reached an advanced stage and the emergence of such a strong stressor, which was the applied physical effort led to their temporary leveling off. Larger rest values of V_E in obese people may partially result from changes in the relations of end-expiratory lung volume (EELV) to the inspiratory capacity (IC =

IRV + V_T), which may be accompanied by adverse effects of excessive body fat distribution affecting the thickening of the chest wall and in this way could occur disorder in respiratory mechanics of these people [15,23]. Some slight impairment of respiratory mechanism in OG in relation to CG is also indicated by reduced resting of SaO_2, EQO_2 and $EQCO_2$ values [6,11]. Larger rest V_E value may also be due to greater body mass and/or FFM, as it was also shown by the higher resting values of VO_2 and VCO_2 of obese men in relation to CG. The negative impact of excess body mass in OG ultimately lead to lower relative VO_2max for this group, which contributed to achieving smaller relative values (to body mass) of the AP, at the AT and ML than in CG. However, both the absolute values of the AP, and VO_2 achieved at AT and ML were similar, hence the physical capacity of the two groups were not considered statistically differed.

Similar negative effects as in the respiratory system were observed in certain cardiovascular variables. So there were main effects between groups in terms of SV and TPR. Moreover, post hoc calculations indicated that in OG was observed the higher resting values of SBP, MAP, RPP, CO than in CG, where there was observed the absence of the main effects of these variables. Elevated resting values of SBP, MAP, RPP in OG should be considered as adverse effects occurring in the circulatory system [10,24,25]. However increased FFM occurring in OG caused a particular compensation effect through the function helping to pump blood through increased skeletal muscle mass, exemplified by the increased value of the SV and CO. This compensated circulatory benefit function was also manifested by a higher resting value VO_2/HR in OG as compared with CG; it was also stated in previous studies [11]. Significantly lower resting TPR observed in OG in comparison to CG is an interesting achievement of this paper, but Lavie and Messerli (1986) [10] have considered reduced TPR in a group of obese people as an negative circulatory change.

Chaput et al. [26] defined obesity as a chronic excess of energy accumulated in the body then increased value of the rest EE OG in relation to CG is a results of higher body mass or natural defensive response connected with energy excretion.

Significant changes of all respiratory and cardiovascular variables during R at AT and at ML during BT beyond the DBP under the influence of applied effort are well known and they are not being commented in this paper.

Portrayal of adverse changes in respiratory and circulatory systems only under resting conditions the above-mentioned variables (with no main effect analysis of variance in the range of intergroup comparison of these parameters) as well as presence of the main effects on the SV and TPR, and the appearance of significant interactions (group-exercise intensity) involving V_E, SV, EQO_2, $EQCO_2$, and TPR indicates a deterioration of cardiopulmonary conditions in obese people. These interim adverse effects occurring at the stage of obesity I^0 in OG are mostly compensated during the exercise test performed by both groups at similar load, which was positively impacted by higher FFM occurring in the obese group.

CONCLUSIONS

1. Obese men characterized by a greater FFM and TBW than CG, which suggests that the first group had more muscle mass.
2. Increased FFM in obese men lay at the root of relative exercise capacity in the researched groups, due to that fact it was similar in OG and CG.
3. Obesity I^o occurring in OG leads to adverse changes in the respiratory and circulatory

systems observed only at rest and regressing under workloads.

4. Reasons for the lack of differences in exercise adaptation of the respiratory and circulatory systems in the research groups should be seen in their similar exercise capacity, adjusted in OG by a greater FFM in respect to CG.
5. Expanded research on people with obesity II^o and III^o should complement the controversial results of the present study.

REFERENCES

1. Adams KF, Schatzkin A, Harris TB, Kipnis V, Mouw T, Ballard-Barbash R, Hollenbeck A, Leitzmann MF: Overweight, obesity, and mortality in a 4 large prospective cohort of persons 50 to 71 years old. N Engl J Med, 2006; 355: 763-778.
2. Stamler J, Stamler R, Neaton JD: Blood pressure and diastolic and cardiovascular risks: U.S. population data. Arch Intern Med, 1993; 153: 598–615.
3. Calle EE, Thun MJ, Petrelli JM, Rodriguez C: Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med, 1999; 341: 1097–1105.
4. Ora J, Laveneziana P, Ofir D, Deesomchok A, Webb KA, O'Donnell DE: Combined effects of obesity and chronic obstructive pulmonary disease on dyspnea and exercise tolerance. Am J Respir Crit Care Med, 2009; 180: 964–971.
5. Ora J, Laveneziana P, Wadell K, Preston M, Webb KA, O'Donnell DE: Effect of obesity on respiratory mechanics during rest and exercise in COPD. J Appl Physiol, 2011; 111: 10–19.
6. O'Donnell DE, O'Donnell CD, Webb KA, Guenette JA: Respiratory consequences of mild-to-moderate obesity: impact on exercise performance in health and in chronic obstructive pulmonary disease. Pulm Med, 2012; 2012:818925. doi: 10.1155/2012/818923.
7. Nicholas SB: Lipid disorders in obesity. Curr Hypertens Rep, 1999; 1: 131-136.
8. de Simone G, Devereux RB, Roman MJ: Relation of obesity and gender on left ventricular hypertrophy in normotensive and hypertensive adults. Hypertension, 1994; 23: 600-606.
9. Ribstein J, Callar G, Mimran A: Combinet renal effects of overweight and hypertension. Hypertension, 1995; 26: 610-615.
10. Lavie CJ, Messerli FH: Cardiovascular adaptation to obesity and hypertension. Chest, 1986; 90(2): 275-279.
11. Aiello M , Teopompi E, Tzani P, Ramponi S, Gidia MR, Marangio E, Hetta A: Maximal exercise in obese patients with COPD: the role of fat free mass . BMC Pulm Med, 2014; 14:96. doi: 10.1186/1471-2466-14-96.
12. Stringer WW, Hansen JE, Wasserman K: Cardiac output estimated noninvasively from oxygen uptake during exercise. J Appl Physiol, 1997; 82(3): 908-912.
13. Fujii TK, Phillips BJ. Quick Review: The Metabolic Cart. Int J Internal Med., 2003; 3(2).
14. Beaver WL, Wassermann K, Whipp BJ: A new method for detecting the anaerobic threshold by gas exchange. J Appl Physiol, 1986; 60(6): 2020-2027.
15. Babb TG, Wyrick BL, DeLorey DS, Chase PJ, Feng MY: Fat distribution and end-expiratory lung volume in lean and obese men and women. Chest, 2008; 134:704–711.
16. Wood RE, Hills AP, Hunter GR, King NA, Byrne NM: VO_2max in overweight and obese adults:

Address for correspondence:

Wiesław Pilis - Institute of Physiotherapy, Public Medical Higher Vocational School, Opole, Poland, E-mail: pilwies@wp.pl

Recevied: 09.12.2015; Accepted: 18.02.2016; Published online: 04.03.2016