Soltani R. E. Energy metabolism and tissue respiration in patients with obstructive sleep apnoea hypopnoea syndrome. Journal of Education, Health and Sport. 2020;10(10):252-258. eISSN 2391-8306. DOI http://dx.doi.org/10.12775/JEHS.2020.10.10.024
https://apcz.umk.pl/czasopisma/index.php/JEHS/article/view/JEHS.2020.10.10.024
https://zenodo.org/record/4412188

The journal has had 5 points in Ministry of Science and Higher Education parametric evaluation. § 8. 2) and § 12. 1. 2) 22.02.2019.

Received: 12.10.2020. Revised: 16.10.2020. Accepted: 30.10.2020.

ENERGY METABOLISM AND TISSUE RESPIRATION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNOEA HYPOPNOEA SYNDROME

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Abstract

A comparative study of the features of energy metabolism and tissue respiration in patients with obstructive sleep apnoea hypopnoea syndrome during the day and at night was performed. It has been established that during the day in the patients with OSAHS energy metabolism is higher than normal basal metabolism; in this case the cardiovascular system transports more oxygen and its tissues extract more than in norm. At night, due to cyclic attacks of apnoea hypopnoea, the release of sympathetic nervous system mediators is stimulated, as a result the intensity of tissue respiration also increases significantly and oxygen delivery is activated mainly by cardiovascular system. Such mechanisms are crucial for development of hypertension in the patients with OSAHS.

Key words: obstructive sleep apnoea hypopnoea syndrome; energy metabolism; tissue respiration.

Introduction

Obstructive sleep apnoea hypopnoea syndrome is a pathological condition of pulmonary ventilation break for more than 10 seconds; during a 7-hour sleep apnoea hypopnoea is evidenced at least 30 times [1]. The prevalence of obstructive sleep apnoea hypopnoea syndrome (OSAHS) in highly developed countries is 5-7% among the population

of over 30 years of age. At the same time, about a third of these patients suffer from moderate or severe stage of this disease and appropriate correction is needed [2].

Mortality from a separate senile asthenia syndrome is 6.0-8.0% (death in sleep).

The problem of sleep apnoea syndrome is urgent because OSAHS is a risk factor for such life-threatening conditions as stroke, myocardial infarction, heart rhythm disorders [3].

Each episode of apnoea is in actual fact asphyxia and leads to hypoxia, hypercapnia and metabolic acidosis that stimulate activation of the sympathetic nervous system and adversely affect the function of various organs and systems of the body [4].

The aim of the study was to investigate energy metabolism, tissue respiration and its features in the patients with OSAHS during the day and at night.

Material and Methods

Anthropometric data (age, sex, body weight, height, body area) were determined for patients, as well as hemodynamic parameters (heart rate, blood pressure), arterial blood saturation (SpO_2a) and cardiac output (echosonography) were measured.

The intensity of tissue respiration of the body was assessed by:

1. Evaluation of the patient's normal basal metabolism (NBM), using the Harris-Benedict formulas (taking into account sex, body weight, height and age of each patient, kcal.) [5]:

NBM (male) =66+13.7· body weight (kg) + 5 x height (cm) – 6.8 x age (years) (1)

NBM (female) =665+9.6· body weight (kg) x 1.8 · height (cm) – 4.7 x age (years) (2)

2. The percentage of deviations (PD) of the basic metabolism from the norm according to the Reed formula (taking into account the level of blood pressure and heart rate, %) [5]:

PD = 0.75· (HR + pulse BP x 0.74) – 72 (3)

PD - percentage of deviations of the basic metabolism from the norm, %

HR – heart rate, beats per minute,

pulse BP– pulse blood pressure, mm Hg

3. Since the level of energy metabolism (EM) consists of the sum of the basic metabolism and any deviation from the norm (+ or – sign), it is determined as:

EM = NBM + PD (kcal) (4)

4. The amount of oxygen that the body consumes in one minute (MO_2C) at the time of the study is

$$MO_2C = \frac{EM}{7.07} \quad (5)$$

MO_2C – minute oxygen consumption ($ml \cdot min^{-1}$),

EM – energy metabolism (kcal),

7.07 is a constant value and is the product of the average caloric value of 1 litre of oxygen per number of minutes per day (1440), divided by 1000

5) The volume of oxygen consumed by the body per one minute for the body area (BA) of every 1 m^2 (oxygen consumption index (O_2CI)) [6]:

$$O_2CI = MO_2C : BA \quad (6)$$

O_2CI - oxygen consumption index ($ml \cdot min^{-1} \cdot m^{-2}$)

6) The oxygen content in the arterial blood (O_2Ca) was calculated by:

$O_2Ca = SpO_2a$ x aBOC (actual blood oxygen capacity ($ml \cdot l^{-1}$) (7)

7) The amount of oxygen transported to the tissues was measured by oxygen content and cardiac output CO (real tissue respiration index (r ITO2))

$$r\ ITO_2 = O_2Ca \cdot CO \quad (ml \cdot mon^{-1} \cdot m^{-2}) \quad (8)$$

Results

In the control group of our study, the average value of the normal basal metabolism (NBM), calculated by the Harris-Benedict tables, was (2347.0±17.3) kcal (Table 1). In the patients with OSAHS, the level of NBM was almost the same. At such energy consumption, the normal minute oxygen consumption (NMO_2C) was (329-338) ml x min^{-1}.

Taking into account the parameters of hemodynamics the percentage of deviations (PD) of energy metabolism from the norm was calculated by the Reed formula. During the day, in the patients of all groups it was with a + sign and ranged from 12.5% (in the control group) to 20.1% (in the most severe patients).

Therefore, during the period of activity at the daytime energy metabolism in the studied patients of all groups was higher than the normal basal metabolism by 14.5% in the control group, by 14.8% in mild pathology, by 19.1% in moderate pathology and by 31.1% in the patients with severe pathology. Accordingly, during the period of activity at the daytime the same dynamics (in the same amount) of increase of the oxygen consumed by body tissues per minute (minute oxygen consumption, MO_2C) was evidenced in all individuals.

Table 1. Energy metabolism and tissue respiration in patients with OSAHS during the day (M±m)

Parameter	Control group	OSAHS degree:		
		mild	moderate	severe
NBM, kcal	2347.0±17.3	2332.1±22.4	2367.8±19.4	2392.6±24.5*
Heart rate, beats /min^{-1}	76.0±2.0	79.9±1.6	84.1±1.8	86.2±1.2*
AT syst., kPa	18.4±0.3	19.3±0.3	19.9±0.3	21.2±0.3*
BP diast, kPa	11.3±0.2	13.0±0.3	13.6±0.2	15.5±0.2*
PD, %	12.5±0.3	14.1±0.6**	17,3±0.5**	20.1±0.6**
NMO_2C, $ml \cdot min^{-1}$	331.9±12.1	329.9±14.2	334.9±10.9	338.4±12.0
EM, kcal	2687.1±15.9	2679.5±21.6	2819.1±20.1**	3136.1±22.4**
MO_2C, $ml \cdot min^{-1}$	380.3±9.2	379.0±8.6	398.7±8.8*	443.6±9.1**
O_2CI, $ml \cdot min^{-1} \cdot m^{-2}$	181.1±5.4	180.5±3,9	189.9±4.9	211.2±5.5**
a-vDO_2, $ml \cdot min^{-1} \cdot m^{-2}$	317.2±4.2	320.2±4.8	364.1±4.4**	416.0±6.1**
$rITO_2$, $ml \cdot min^{-1} \cdot m^{-2}$	498.3±17.0	500.7±19.8	554.0±22,1*	627.2±24.7**
ITR, %	114.6±0.9	114.9±1.7	119.0±1.3*	131.0±1.5**
CUO_2, %	36.3±1.4	36.0±2.2	34.3±2.0	33.7±1.9*
Notes * - $p<0.05$; ** - $p<0.01$ compare to the parameters of the control group				

The oxygen consumption index (O_2CI) determined the amount of oxygen that the body cells took in one minute to meet their metabolic needs per 1 m^2 of body area. In the studied patients, this indicator increased slightly in the first and second groups, and significantly only in the most severe patients, $p<0.01$.

Comparing the results of measurements and calculations of the volume of oxygen delivery to tissues ($rITO_2$) and its consumption by the body (O_2CI), we obtained the parameter of the arterio-venous difference in oxygen indicator (a-vDO_2). It was found out that in the patients with mild OSAHS a-vDO_2 rate did not differ from that in the control group, (317-320) ml x min^{-1} x m^{-2}; in the patients of the 2nd group it was higher by 40-45 ml, and in the patients of the 3rd group – by 90-100 ml compare to the control group ($p<0.01$).

The body consumed more oxygen during the day than it needed to maintain its basic metabolism; the intensity of tissue respiration (ITR) in all patients was over 100%, and in the mild patients it was slightly higher than the control; in moderate patients it increased by 4.4%, and in the severe patients – by 16.4% compare to the control group (the results are statistically significant, $p<0.01$).

The coefficient of oxygen utilization (CUO_2) determined how much oxygen (%) the body consumed in relation to the amount of its delivery. It was established that during the

period of activity at the daytime this coefficient was the highest in the control group (36.3±1.4) %. In the patients with OSAHS it was slightly lower, ranging from 36.0% to 33.7% of the amount of oxygen delivered by the cardiovascular system to the tissues to provide their respiration.

Energy metabolism and tissue respiration in the studied patients at night was different. During the night in the patients with OSAHS basal blood arterialization significantly decreased in apnoea attacks. Oxygen delivery to tissues decreased. At the same time during patients sleep in the absence of exercise energy metabolism should have decreased.

We relied on the concept that the level of normal basal metabolism and minute oxygen consumption for metabolic reactions of the body in basal metabolism were constant values.

Their absolute values are presented in the Table 2.

Table 2. Energy metabolism and tissue respiration (basal) in the patients with OSAHS at night (M±m)

Parameter	Control group	OSAHS degree:		
		mild	moderate	severe
NBM, kcal	2347.0±47.3	2332.1±42.4	2367.8±37.4	2392.6±44.5
NMO_2C, $ml \cdot min^{-1}$	331.9±40.3	329.9±39.4	334.9±41.1	338.4±36.7
PD, %	-5.2±0.5	0.7±0.2**	1.5±0.2**	11.2±0.9**
EM basal, kcal	2225.4±37.8	2348.4±39.0**	2403.3±42.4**	2660.5±49.8**
MO_2C, $ml \cdot min^{-1}$	314.7±7.4	332.2±8.0*	340.0±9.6**	376.1±10.1**
O_2CI, $ml \cdot min^{-1} \cdot m^{-2}$	149.9 ±3.8	158.2±3.9*	161.9±4.0**	179.10±4.3**
$rITO_2$, $ml \cdot min^{-1} \cdot m^{-2}$	413.6±16.8	418.6±29.4	453.5±22.2*	496.5±27.7**
$a\text{-}vDO_2$, $ml \cdot min^{-1} \cdot m^{-2}$	263.7±8.0	260.4±10.2	291.6±11.9**	317.4±14.0**
ITR, %	94.8±2.0	100.7±1.7*	101.5±1.2**	111.1±2.1**
CUO_2, %	36.2±1.4	37.8±2.2	35.7±2.3	36.0±1.9

Notes
* - $p<0.05$;
** - $p<0.01$ compare to the parameters of the control group

At night, in the control group a percentage of deviations from normal energy metabolism was (-5.2±0.5) %. The results are expected, because at night during sleep all energy processes reduce, especially muscle tone, and energy metabolism is lower than the basal metabolism. However, in the patients with OSAHS the percentage of energy metabolism deviations increased at night compare to the norm, reaching results with a + sign. At the same time, a clear proportional dependence of these indicators on the pathology severity was observed, from (0.7±0.2) % to (11.2±0.9) %.

Energy metabolism (basal) in the patients increased from (2348.4±39.0) to (2660.5±49.8) kcal. These results are attributable to the systematic cyclic activation of the sympathetic division of the autonomic nervous system in respiratory arrest, the frequency of which correlates with the severity of the pathology.

In apnoea hypopnoea attacks in the patients a change in the phase of sleep by micro-awakening took place; at this period muscle tone increased, heart rate and cardiac output increased and tissue respiration activated. Therefore, an increase in minute oxygen consumption as well as oxygen consumption index was observed in the patients compare to the controls. However, the patients' MO_2C parameters at night were still significantly lower compare to those during daytime activity.

The intensity of tissue respiration was lower than 100% only in the control group; in the patients this rate increased from (100.7±1.7) % to (111.1±2.1) %. However, these parameters were lower than those of the same patients at the daytime.

The oxygen utilization coefficient at night was significantly higher than normal that evidenced intensification of tissue respiration. At the same time, it proved a tendency (unreliable) to decrease simultaneously with the pathology severity, although it was still higher by 5-8% compare to day rates.

Conclusion

At the daytime, energy metabolism in the patients with OSAHS is higher than normal basal metabolism; in this case the cardiovascular system transports more oxygen and its tissues extract more than normal. To ensure increased tissue respiration, the body initiates compensatory mechanisms: hyperactivity of the cardiovascular system that is manifested by increased cardiac output and blood pressure.

At night, in cyclic attacks of apnoea hypopnoea the release of mediators of the sympathetic nervous system stimulates, as a result the intensity of tissue respiration increases significantly and oxygen delivery is activated mainly by the cardiovascular system. Such mechanisms are crucial for development of hypertension in the patients with OSAHS.

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