The Correlation of Lung Vital Capacity, VO_2Max, and Heart Rate Recovery With Changes in Blood Lactate Levels in Young Male: Cross Sectional Study in Provoked By Repeated Sprint Sessional-3

Mustofa[1], Susiana Candrawati[1], Khusnul Muflikhah[1], Tiara Dwivantari[2], Rahardita Alidris[2] and Dessy Dwi Zahrina[2]

[1]*Department of Physiology, Faculty of Medicine Jenderal Soedirman University, Jl. Dr. Gumberg, Purwokerto, Indonesia*
[2]*Faculty of Medicine JenderalSoedirman University, Purwokerto, Indonesia*
mustofa.dr@gmail.com

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Abstract: Muscle fatigue is lactate accumulation effect from chronic response to strong muscle contraction in high intensity of activity. The elevated of lactate can be caused by the inability of the cardiorespiratory system to meet oxygen requirement. Lung vital capacity (LVC) determines the availability of oxygen in the body.Maximum oxygen consumption(VO_2max) and Heart-rate recovery (HRR) aretwo of cardio-respiration health indicator that able to assess the ability of body metabolism to come up muscle fatigue. This study was aimed to investigate the influence ofLVC, VO_2max and HRR on the lactate changes in young men provoked by Repeated Sprint Sessional3.The cross-sectional study with 23 young male (18-22 y.o)subjects which were selected used consecutive sampling method. Lung Vital Capacity was measured using Chestgraph HI-101. VO_2max was measured using Queen College step Test Method. HRR was measured by calculated the difference between the heart rate maximum after queen's college step test and heart rate after 1 minute recovery. Blood lactate level was measured by finger prick test method which was measured in pre- and post- RSS 3 intervention. This research protocol had been ethically agreed by Ethical Commission of JenderalSoedirman University. Statistical analysis used paired t test, Pearson and Spearman correlation test.The result showed significantly correlation between HRR with the changes of blood lactate($p<0, 05$; $r= - 0,469$).The correlation between LVC and VO_2max with the changes of blood lactate were not significant ($p>0, 05$). Heart-rate recoverywas proven correlate with blood lactate changes during activity.

1 INTRODUCTION

Muscle fatigue occurs after strong muscle contraction with high intensity (Hall and Guyton, 2011) and / or lasts longer. Muscle activity requires ATP to be met through aerobic and anaerobic metabolism. High-intensity muscle activity requires the availability of large and immediate ATPs. This causes a shift in muscle metabolism from aerobic to anaerobic, resulting in increased blood lactate and muscle fatigue(Giriwijoyo and Sidik, 2012).

Metabolic shifts are affected by aerobic capacity that includes the capacity of muscle oxidative enzymes, blood and muscle oxygen content, substrate content of blood and muscle, circulation in muscle tissue and others.These changes differ for each individual depending on gender, age, nutritional status, nutrition, and fitness level.The level of fitness can be assessed based on the value of the maximal oxygen volume (VO2max) influenced by many factors, including vital pulmonary capacity(Moxnes and Sandbakk, 2012), and cardiovascular condition as reflected in the heart rate recovery (HRR)(ACSM, 2014).

Previous studies have shown an association between HRR and blood lactate. Higher HRR is associated with lower lactate accumulation in the blood(Buchheit *et al.*, 2010). In Their article(Daanen *et al.*, 2012)showed that HRR correlates with blood lactate accumulation. In their study, (Zebrowska *et al.*, 2012; Green *et al.*, 2014)showed a correlation of VO_2max to blood lactate acid levels.

The aims of this study was to determine the correlation of lung vital capacity, VO_2max and HRR with changes in blood lactate levels in young men after repeated Sprint Sessional 3 (RSS3).Changes in

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blood lactate can be provoked using the RSS3 method. The study of (Gharbi *et al.*, 2014), suggesting that RSS3 may lead to elevated blood lactate levels (> 10 mmol / l).

2 METHODS

This research use cross sectional study design. Twenty-three sedentary young male (18-23 years old) volunteered to participate in this study.Inclusion criteria were Body Mass Index (BMI) in the 19-25 kg / m2 range. Exclusion criteria are having bone or joint problems and have cardiovascular disease.The researchprotocol and possible risks were communicated verbally and in writing to all study participants, who then gave their informed written consent. The subjects were asked not to perform vigorous exercise twenty four hour before and not to take food, caffeine or smoke two hour before testing. The study was approved by the Ethics Committee of medical faculty-Jenderal Soedirman university.

Lung Vital capacity was measured using Chestgraph HI-101 (Chest M.I, Japan) while sitting. The study measured forced vital capacity (FVC) using manufacture guidance book.Maximum oxygen consumption was measured by submaximal exercise testing using Queen's College Step Test, a reliable method to indirectly estimate VO_2max.The equipment consisted of a sturdy bench16.25 inch high, a stop watch, and a metronome set at 96 beats/min.The subjects were then asked to step up and down in rhythm with the metronome for three minutes. After completion of the exercise, the subject was asked to remain standing and the radial pulse rate was was counted five seconds after stopping, for a period of 15 seconds of the recovery periodand multiplied by four to get the pulse rate/min. This recovery pulse rate was used in the following equation to determine the subject's VO2max and read on an age-adjusted rating scale(Hingorjo *et al.*, 2017). Heart-Rate Recovery is determined by calculating the pulse rate difference of the peak pulse during exercise with pulse pulse after 1 minute of rest. The peak pulse is provoked by queen's college step test (QCST). Changes in lactate levels were obtained from the difference in blood lactate levels before and 1 min after RSS3 were measured using the Accutrend Plus System-Cobas lactate test analyzer with finger tip sampling.

Lung vital capacity, VO_2max, HRR measured on day I, while changes in lactic acid levels were measured at day II. RSS3 is a sprint on a 30 meter (2x15 meter) straight trajectory with three repetitions. Rest time between repetitions were 30 seconds. Changes in blood lactate levels after provocation of RSS3 were tested using paired t test. The correlation of pulmonary vital capacity and HRR with changes in blood lactate levels were tested using Pearson test. The VO2max correlation with changes in blood lactate levels was tested using the Spearman test because the VO2max data was not normally distributed. The significance level set is $p < 0.05$.

3 RESULTS

This study was conducted at Faculty of Medicine, University of General Soedirman from November, 17th to December, 7th, 2016. Research subjects meeting the inclusion criteria and exclusion criteria were 23 people. The data were normally distributed ($p > 0,05$) with Saphiro-Wilk test except VO2max, as in table 1. Table 2 shows the characteristics of research subjects.

Paired t-test is used to find significant differences of blood lactate levels before and after RSS 3. The result of bivariate analysis using paired t-test showed significant value ($p < 0.001$)(table 2). Changes in blood lactate levels before and after RSS 3 are shown with values from 2.0 ± 1.0 mmol / L to 9 , 6 ± 3.1 mmol / L. This means RSS 3 is able to significantly increase blood lactate levels so that the effectiveness of RSS 3 is considered good in provoking an increase in blood lactate levels.

Table 1: Data distribution of research subject.

Variable	Shapiro-Wilk Test
Heart Rate Recovery (HRR)	0, 241
Lung Vital Capacity (LVC)	0,769
VO_2 max	0,014
Lactate levels : Before RSS3 After RSS3	0,694 0,346
Changes in blood lactate levels (ΔL)	0,695

Table 2: Characteristics of research subject.

Variable	*Mean ± SD* *Median (min – max)*
HRR, beat/menit	34,26± 12,06
LVC, liter	3,58 ± 0,54
VO_2max, ml/kg/min	67,65 (57,57-72,69)
Lactate levels : Before RSS3 After RSS3	2,0 ± 1,0 9,6 ± 3,1
Changes in blood lactate levels (ΔL), mmol/L	(7,59 ± 3,14)***

***p<.001

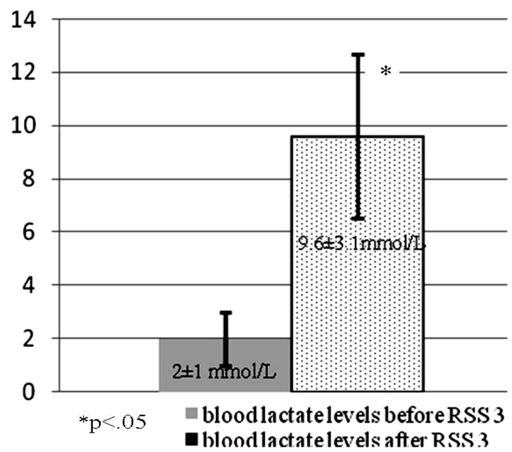

FIgure 1: RSS 3 provoked enhancement of blood lactate levels.

Table 3. shows the significant correlation between of HRR and changes in blood lactate levels ($p = 0.024$; $r = -0.469$). The higher the HRR, the lower the lactate level changes. However, this study showed no significant correlation between VC and changes in blood lactate levels ($p = 0.950$) and between VO2max and changes in blood lactate levels ($p = 0.805$).

Table 3: Correlation test of HRR, VC, VO_2max with changes in blood lactate levels.

Variable	*Mean ± SD* *Median (min–max)*	Correlation tests
HRR ΔL	34,26±12,06 7,59 ± 3,14	p = 0,024* r = - 0,469
LVC ΔL	3,58 ± 0,54 7,59 ± 3,14	p = 0,950 r = 0,014
VO_2max ΔL	67,65(57,57–72,69) 7,59 ± 3,14	p = 0,805 r = - 0,054

*p<.05

4 DISCUSSION

The prevous study (Buchheit *et al.*, 2010) showed a correlation between HRR1 minute and blood lactate levels after the 3rd, 5th, 7th, 9th and 10th sprints with strongly negative correlation ($p < 0.001$, $r = -0.67$). The higher HRR1 minute value is associated with lower blood lactate levels.(Thomson *et al.*, 2016)also supports this study. The results suggest low HRR is associated with fatigue or lactate buildup. If HRR is affected by the sympathetic nervous under fatigue conditions, there is a decrease in the sensitivity of the adrenergic receptor and the sympathetic nervous system will decrease after maximal exercise through the modulation of the larger parasympathetic nervous system. As a result high HRR value and pulse will return to normal. (Fernando *et al.*, 2015)said this HRR-1 minute has a correlation with aerobic type physical fitness. Aerobic physical fitness is a physical fitness in oxygen fulfillment and the body is able to perform physiological adaptations of the cardiovascular system in regulating acute stressor responses such as blood lactate levels and speeding up the reduction process.

Heart-rate recovery is physiologically a combination of several body responses such as feedback effects of cardiac stimulation of the cerebral cortex, stimulation of induced reflexes from stress system metabolites, chemoreceptors, blood lactate from muscle contraction results, and cardiac output(Leunkeu *et al.*, 2014). High physical exercise intensity increased heart work in meeting the needs of cell metabolism and muscle fatigue. Muscle fatigue after exercises results in reduced muscle ability to withstand load and strength(Finsterer, 2012).

The next results of this study showed no association between LVC and VO_2max on changes in blood lactate levels. This is assumed because the data collection technique of 2 variables is more subjective than HRR. Measurements of VO_2max in the non-athlete population are often affected by the lack of internal motivation of the study subjects. While LVC data retrieval techniques often become invalid because of the inability of research subjects to follow the instructions given.

Increased blood lactate results from compensated cardiorespiratory system that has not been maximal in meeting the needs of muscle oxygen. The major changes of the heart and blood vessel system are the increased pulse rate given exercise load. Increased excessive heart work due to lactate accumulation results in increased sympathetic nerve induction and decreased parasympathetic nerve activity characterized by low HRR values(Daanen *et al.*, 2012).

Subjectsin this study were taken based on consecutive techniques without the classification based on the level of physical exercise habits undertaken subjects. Data for the HRR distribution range in this study reached 18.0 - 61.0 bpm and for changes in blood lactate levels of this study in the range 2.5 - 13.9 mmol / L. These data indicated a wide range of data variations because they are influenced by both acute and chronic physical activity habits that affect both main variables.

Individuals who regularly perform physical exercise with a proper dose and regularly able to minimize fatigue and improve maximum performance. Appropriate exercise can increase lactate threshold levels, in untrained people having a 65% threshold level of maximal heart rate, while athletes who follow continuous exercise can achieve 90% of maximal heart rate(Ayabe *et al.*, 2015) (Ayabe *et al*., 2015).

5 CONCLUSIONS

There is a correlation of HRR levels with changes in lactic acid blood levels of medical students with moderate correlation strength and negative correlation direction. The higher the HRR level, the lower the lactate blood levels changes.

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