



## EVALUATING MAXIMUM OXYGEN UPTAKE OF MALE SOCCER PLAYERS WITH BRUCE PROTOCOL

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### Abstract

**Purpose.** Maximal oxygen consumption (VO max) is defined as the ability to transfer and consume oxygen during exhausted work and is associated with cardiorespiratory fitness. The main purpose of this research is to measure and analyze the VO<sub>2</sub>max of eleven soccer players who compete in regional soccer league in Los Angeles. Additionally, secondary purpose of this study is to determine the relationship between VO<sub>2</sub>max values and heart rate, ventilation, RER, and V<sub>O</sub>2, and CO<sub>2</sub>.

**Methods.** Study participants included 11 male soccer players who competed in Los Angeles recreational regional league. All subjects performed a maximal exercise test using a treadmill with Bruce protocol. The tests starting at speed 2,74km/hr and grade of 10% and, gradually more increased at every 3 minutes until exhaustion. The following parameters were recorded from the cardiorespiratory exercise test: VCO<sub>2</sub> /VO values, the duration of the test, the maximal pulmonary ventilation (VE), the maximal heart rate (HR), and the respiratory exchange ratio (RER). Descriptive statistics included calculation of mean values for subjects VO<sub>2</sub> max, VCO<sub>2</sub> /VO<sub>2</sub> values, the duration of the test, the maximal pulmonary ventilation (VE), the maximal heart rate (HR), and the respiratory exchange ratio (RER). Correlation coefficients were also calculated to evaluate the relationships for each value.

**Results.** The subjects reached VO<sub>2</sub>max because they meet at least 3 criteria's for reaching VO<sub>2</sub>max and VO<sub>2</sub> strongly correlated with all other variables.

**Conclusion.** As expected VO<sub>2</sub> values of eleven soccer players was closely related with heart rate, ventilation, RER, and V<sub>O</sub>2, and CO<sub>2</sub> of the subjects.

**Key words:** Bruce protocol, maximum oxygen consumption, RER.

### Introduction

Maximal oxygen consumption (VO max) is defined as the ability to transfer and consume oxygen during exhausted work and is associated with cardiorespiratory fitness. (American College of Sports Medicine 2006). There are several exercise treadmill protocols for the prediction of VO max (Astorino, 2011). The most common maximal GXT for the treadmill is the Bruce protocol that provides outstanding correctness and a consistent testing procedure for all participants. It requires all participants to progress from one stage to the next at the same speed and grade making it comparable between participants based on the same exercise intensity requirements. Also, total exercise time can be used to precisely categorize participants according to cardiorespiratory fitness or cardiovascular risk (Glass, Gregory, 2007. George, Paul, 2009). The presentation of ventilatory expired gas analysis into traditional stress test procedures led to the direct

measurement of VO<sub>2</sub>. There are not many studies using the Bruce protocol in respect to relationship of VO<sub>2</sub> with its other parameters in a specific population such as soccer. Thus, the main purpose of this research is to measure and analyze the VO<sub>2</sub>max of eleven soccer players who compete in regional soccer league in Los Angeles. Additionally, secondary purpose of this study is to determine the relationship between VO<sub>2</sub>max values and heart rate, ventilation, RER, and V<sub>O</sub>2, and CO<sub>2</sub> values. We predict that VO<sub>2</sub>max values will be positively related to heart rate, ventilation, RER, and V<sub>O</sub>2, and CO<sub>2</sub> values.

### Methods

**Subjects.** Study participants included 11 male soccer players who competed in Los Angeles recreational regional league. The physical and anthropometric characteristics of the subjects are listed in Table I. Before the study, subjects provided informed consent for their participation.

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**Table 1: Subject Characteristics**

Number Of Subjects	Mean Age (Y)	Mean Weight (kg)	Years of playing soccer(Mean)
11	24.94 ± 5.05	73.84 ± 22.16	9

**Testing Procedures**

All subjects underwent health screening before exercise testing to make sure they are free of any cardiovascular or any kind of risks. All subjects performed a maximal exercise test using a treadmill. The treadmill was calibrated in order to make sure the accuracy of grade and speed. Expiratory gas analyzer Oxycon Pro of Jaeger (Viasys) were used to analyses gases. The tests starting at speed 2,74km/hr and grade of 10% and, gradually more increased at every 3 minutes until exhaustion (R.A. Bruce, 1949). Additionally, heart rates of the subjects were measured by Polar Vantage NV heart rate monitors (Polar Electro Oy, Kempele, Finland). The players were informed not to take part in any vigorous training 48 hours prior to the measurements to get out of any possible side effects. Also, the following parameters were recorded from the cardiorespiratory exercise test: VCO /VO values, the

duration of the test, the maximal pulmonary ventilation (VE), the maximal heart rate (HR), and the respiratory exchange ratio (RER).The following exercise test criteria were used for the attainment of VO max:

1. Respiratory exchange ratio (VCO /VO) superior than 1.10 (Virtual Exercise Physiology Laboratory 2004).
2. Leveling off (plateau) of oxygen uptake with an increase of work rate (N. Koutlianos, et al. 2004).
3. Attainment of 90% of the age-adjusted estimation of maximal heart rate (T.M. Gibson, et al. 1979).

Data Analysis.Descriptive statistics included calculation of mean values for subjects VO2 max, VCO /VO values, the duration of the test, the maximal pulmonary ventilation (VE), the maximal heart rate (HR), and the respiratory exchange ratio (RER). Correlation coefficients were also calculated to evaluate the relationships for each value.

**Results.**The subjects reached VO2max because they meet at least three out of the five criteria's for reaching VO2 max.

At the end of the subjects' heart rate reached to estimated MaxHR. At the end of the subjects had an RER greater than 1.10 and they had a plateau in VO2.

**Table 2: VO2 and related parameters**

Stage	VO2(Mean)(ml/kg/min)	Heart Rate (Mean) (bpm)	RER (Mean)	VCO2 (Mean) (ml/kg/min)	VO2 (Mean) (L/min)	Ve (Mean) (L/min)
1	17	122	0.75	12.75	1.36	35.9
2	21.9	141	0.94	20.5	1.75	52.58
3	38.9	171	0.96	37.3	3.11	85.17
4	48.7	186	1.09	53.25	3.9	124.18
5	52.3	197	1.17	61.3	4.19	154.37

All parameters are increased as VO2 increases.

**Table 3: Correlation table**

R(Correlations)	Heart Rate (Mean) (bpm)	RER (Mean)	VCO2 (Mean) (ml/kg/min)	Ve (Mean) (L/min)
VO2(Mean)(ml/0.	0.99	0.92	0.99	0.97

All parameters are correlated with VO2.(p<0.05)

## Discussion

As expected VO values of eleven soccer players was closely related with heart rate, ventilation,

RER, and  $\dot{V}O_2$ , and  $\dot{V}CO_2$  of the subjects

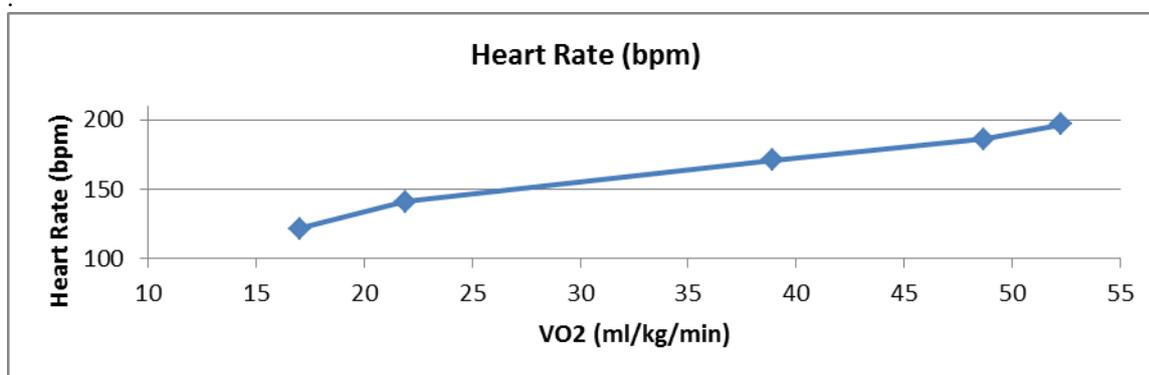


Fig. 1: Heart rate vs. VO<sub>2</sub>

This graph shows a linear increase in heart rate as intensity increases. As intensity and duration increases heart rate needs to increase to meet the increase demand of oxygen to do the working skeletal muscles. The observed heart rate response indicates that with an increase in intensity, there is an increase need for blood flow. The increase in heart rate is caused mainly by two mechanisms. One mechanism is the withdrawal from the vagus nerve, meaning a decrease in the neurotransmitter acetylcholine. A decrease in this neurotransmitter from the decrease in innervations from the vagus nerve results in an increase in heart rate. However, this is only the means to increase heart rate up to 100 beats per minute, therefore this was only the physiological basis for the raise in heart rate between stage one and stage 2. After heart rate reaches 100 beats per minute, input of plasma epinephrine is responsible for the increase in heart rate. This means, as intensity increases, the plasma levels of epinephrine increase and uptake by the Beta 1 cells, which causes an increase in heart rate as needed, per exercise intensity (American College of Sports Medicine 2006).

Muscle blood during exercise will have a greater A-vo<sub>2</sub> to V-vo<sub>2</sub> difference as exercise intensity increases. What this means is oxygen uptake by the muscle cells from the blood is increased with exercise intensity. Therefore the venous muscle blood will have less O<sub>2</sub> in it during exercise than at rest. The muscle cells themselves will receive greater blood flow. This is due to a decrease in splanchnic blood flow, due to vasoconstriction caused by increased plasma epinephrine levels to alpha-1 receptors. The other contributing factor is vasodilatation of the blood vessels at the exercising muscle due to increased

plasma epinephrine levels and up take at the Beta 2 receptors (American College of Sports Medicine 2006). As exercise intensity increases, plasma epinephrine levels increase. When these come across alpha-receptors of the splanchnic region, it causes vasoconstriction. This vasoconstriction decreases blood flow to the splanchnic region (American College of Sports Medicine 2006., American College of Sports Medicine, 2010). The autonomic nervous system aids in blood flow redistribution by release of the neurotransmitters norepinephrine and epinephrine. As exercise intensity increases, the ratio of plasma epinephrine to norepinephrine becomes greater than 1. The effect this has on blood flow is that these neurotransmitters in these proportions are what is needed to stimulate the aforementioned changes in blood flow when received by both beta and alpha receptors of the appropriate tissue (American College of Sports Medicine 2006., American College of Sports Medicine 2010., Virtual Exercise Physiology Laboratory, 2004). NO (Nitrous Oxide) is a powerful vasodilator. It is also a waste product of protein metabolism during exercise. Carbon dioxide is a waste product of aerobic metabolism, as well as glycolysis. An increase in carbon dioxide yields an increase in Hydrogen ion concentration in the blood, through the carbonic acid buffering system. This decrease in blood pH may overpower the effects of catecholamine's, due to inhibition of metabolic enzymes. Furthermore, circulating nitrous oxide may cause vasodilatation in areas the catecholamine's would have signaled vasoconstriction (American College of Sports Medicine, 2006).

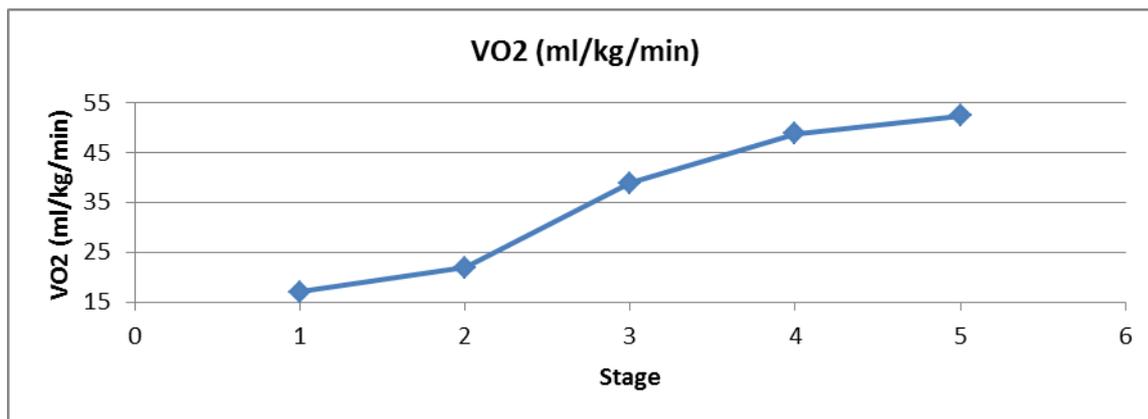


Fig. 2: Stage vs. VO2.

This graph shows a linear increase in Oxygen consumption as intensity and duration of the test increases. This increase in O<sub>2</sub> consumption is caused by the increase need of oxygen to working muscles. Based on VO<sub>2</sub>max values, the subjects are in excellent cardiovascular shape because he has a VO<sub>2</sub> greater than 51. As exercise intensity increases, oxygen consumption increases. This is because the more ATP needs to be produced to meet the new exercise intensity. Since more ATP is needed, aerobic ATP production becomes the prime bioenergetics pathway.

This is because the amount of ATP yielded during oxidative phosphorylation is greater than the amount of ATP being used during anaerobic ATP production. Furthermore, since more ATP is needed with greater intensity, more ATP is being produced, thus more oxygen is needed to help produce it by aerobic means. For this reason oxygen consumption increases with an increase in exercise intensity (American College of Sports Medicine, 2006., American College of Sports Medicine, 2010)

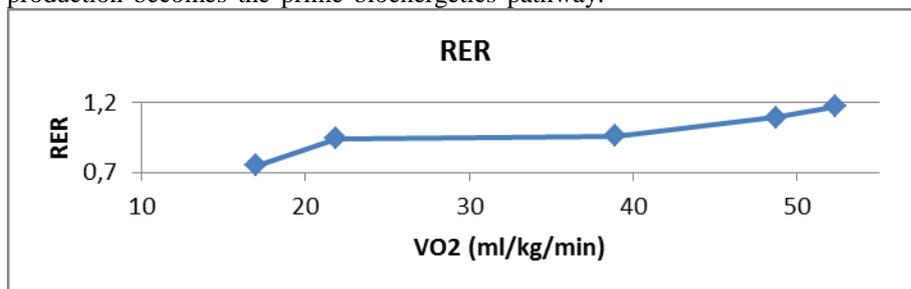


Fig. 3: RER vs. VO2.

This graph shows a curvilinear increase in RER. At a Vo<sub>2</sub> intensity of 21.9 ml/kg/min RER plateau until reaching a Vo<sub>2</sub> intensity of 38.9 ml/kg/min. As the respiratory exchange ratio increases, it means glucose (carbohydrates) rather than fat is starting to be used as a fuel source. By the time the RER reaches 1, it means glucose (carbohydrates) are the only fuel source, not fat. The reason RER would increase is that as exercise intensity increases, the cells start to need more energy, and this energy comes in the form of ATP. The quickest way to get energy aerobically is by breaking down glucose as a fuel source as opposed to fat. For this reason, if the intensity is high enough, only glucose will be used as a fuel source. What the RER of the subjects show here on the graph is that through stage 4, fat was being used as a fuel source, however it was being used less with each stage. This means at each level of intensity, less fat and more glucose was being used for fuel. After stage 4, the RER reached 1.00. This means only glucose was being used as fuel for the aerobic means of ATP production after stage

4(American College of Sports Medicine 2006., American College of Sports Medicine 2010).

As exercise intensity increases, the demand to produce Adenosine Tri Phosphate (ATP) increases. Exercise intensity can increase to a point where all fuel being consumed is glucose for the purpose of producing ATP aerobically. This would be at RER 1.00. At this point, if exercise intensity is increased even further, since ATP is already being produced as quickly as it can be aerobically, the only way to produce enough ATP to provide energy for the new, higher intensity exercise is to also start using anaerobic bioenergetics pathways along with aerobic energetic pathways to produce ATP. The main anaerobic bioenergetics pathway is glycolysis. Glycolysis produces a waste product of carbon dioxide. When glucose is being used as the only fuel source for aerobic ATP production, the waste product carbon dioxide is produced in a 1:1 ratio of the oxygen consumed to produce it. However, at exercise intensity high enough that glucose metabolism must be coupled

with glycolysis to produce enough ATP for the needed exercise, the carbon dioxide produced from glycolysis must be added to the carbon dioxide produced from aerobic metabolism. Aerobically produced carbon dioxide plus anaerobically produced carbon dioxide yields greater carbon dioxide than the amount of oxygen brought in for aerobic metabolism (American College of Sports Medicine, 2006). For this reason, there is now more carbon dioxide being produced, and going out, than there is oxygen being brought in. Therefore, the ratio of  $VCO_2/VO_2$  will be greater than 1. As pyruvate is produced, lactate as a result will be produced. This lactate, which circulates in the blood is normally taken up by the liver and converted back into glucose in what is known as the Cori cycle. However, as exercise intensity increases, the demand for pyruvate increases, due to the increase in ATP production. The increase in pyruvate will also yield an increase in lactate. Eventually this increase in lactate occurs to a point where lactate is being produced faster than the liver can remove it. Therefore, the lactate starts to accumulate in blood. As exercise intensity increases,

ATP production increases, which means lactate increases. At the exercise intensity where the lactate production in blood exceeds the liver's ability to uptake all of it a point is reached known as lactate threshold. Lactate threshold is the point where blood lactate begins to rise exponentially with exercise intensity. Therefore, the amount of lactate in the blood can be used to determine the intensity of the exercise. During graded exercise then, at each stage blood lactate should be increasing in an exponential fashion for each stage (American College of Sports Medicine 2006, Virtual Exercise Physiology Laboratory, 2004). As exercise intensity increases, plasma epinephrine increases. This increase in plasma epinephrine causes an increase in metabolism. An increase in metabolism means an increase in lactate, which is a waste product of metabolism. Therefore, as epinephrine increases, blood lactate should increase linearly until the point of lactate threshold. At lactate threshold, the increase will no longer be linear, but exponential for an increase in blood lactate to blood epinephrine.

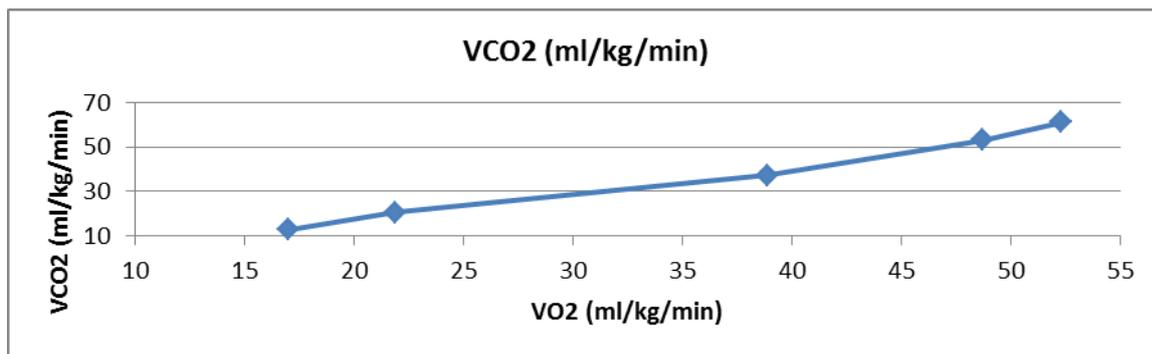


Fig. 4: VCO<sub>2</sub> vs. VO<sub>2</sub>.

This graph shows a linear increase in VCO<sub>2</sub> with an increase in intensity. The VCO<sub>2</sub> breakpoint is seen at a Vo<sub>2</sub> of 38.9ml/kg/min. The increase in VCO<sub>2</sub> during this stage is due to the body's increase reliance on glycolysis. As exercise intensity increases, VCO<sub>2</sub> will increase. This is because more carbon dioxide is produced when glucose is metabolized than when fat is metabolized, and the shift to glucose metabolism increases as exercise intensity increases. Furthermore, if the intensity is high enough, glycolysis may contribute to energy production, which yields carbon dioxide (American College of Sports Medicine 2006., American College of Sports Medicine 2010., Virtual Exercise Physiology Laboratory. 2004). This was demonstrated in the graph. As the exercise intensity increased, the subjects VCO<sub>2</sub> increased in a near linear fashion, or 1:1 ratio.

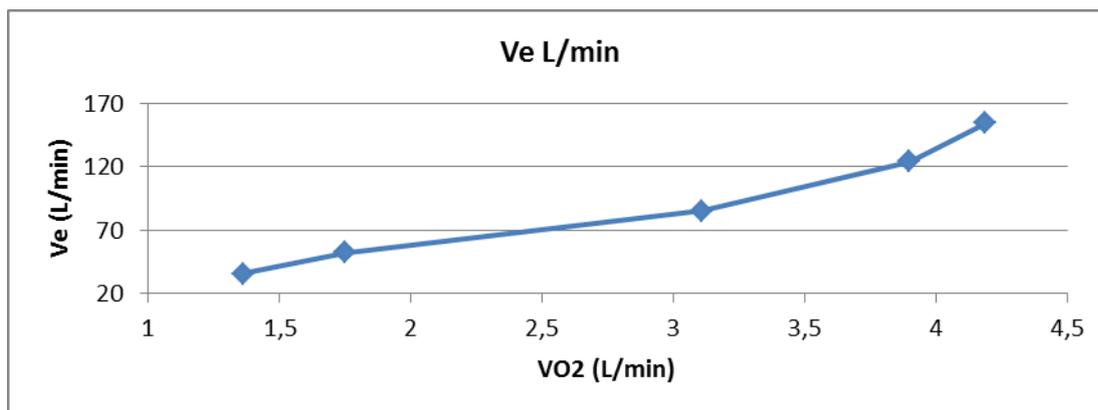


Fig. 5: VE vs. VO2.

This graph shows a linear increase in Ventilation with an increase in intensity. Ventilation breakpoint was seen at a VO<sub>2</sub> of 3.11 l/min. The ventilation breakpoint was caused by the body's increase need to sent O<sub>2</sub> to the working muscles. As exercise intensity increases, ventilation rate increases. This is because there is both a greater demand for oxygen, as well as an increased production of carbon dioxide, as exercise intensity increases (American College of Sports Medicine, 2006).

**Conclusions.** To conclude, there is strong relationship between VO<sub>2</sub> and its other parameters (VCO<sub>2</sub> /VO values, the duration of the test, the maximal pulmonary ventilation (VE), the maximal heart rate (HR), and the respiratory exchange ratio) of male soccer players who competed in regional soccer league.

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