

## Ventilation, Potassium and Lactate During Incremental Exercise in Men Athletes

<sup>1</sup>S. Darabi, <sup>2</sup>M.H. Dehghan, <sup>2</sup>S. Refahi and <sup>3</sup>E. Kiani

<sup>1</sup>Qom University of Medical Sciences, Qom, Iran

<sup>2</sup>Department of Biochemistry, Qazvin University of Medical Sciences, Qazvin, Iran

<sup>3</sup>Islamic Azad University, Burujerd Branch, Iran

**Abstract:** The present investigation was undertaken to examine the changes in Ventilation (VE), plasma Potassium ( $k^+$ ) and Lactate (La) concentration during incremental exercise. Fifteen normal athletes men performed an incremental cycle ergometer exercise to extreme fatigue. Antecubital venous blood samples were taken at the end of each step and exercise onset. The VE and plasma  $k^+$  and La concentration were measured from blood samples in every step. We investigated linear regression analysis of the slopes of the ventilation, plasma  $k^+$  and La levels vs workload during incremental exercise. Our findings showed that VE and plasma  $k^+$  and La concentration proportionally increase during incremental exercise, but an inflection see in the curves. This study demonstrated that inflection on the plasma La plot occurs earlier than break points on VE and plasma potassium plots during incremental exercise.

**Key words:** Ventilation, potassium, lactate, men athletes, incremental, exercise

### INTRODUCTION

It is generally accepted that the control of breathing during exercise is regulated by a combination of both neural and humoral drives (Paterson *et al.*, 1990). The relationship between plasma  $k^+$  and ventilation has not been extensively studied during exercise. Recently, attention has focused on the role of potassium and lactate as a factor that could be involved in the control of breathing during exercise (Qayyum *et al.*, 1994). We have examined the changes in ventilation (VE), plasma potassium and lactate concentrations during incremental exercise in 15 normal athletes' men.

### MATERIALS AND METHODS

We studied 15 men athletes with no history of respiratory and cardiovascular disease (mean $\pm$ SD: age 22.6 $\pm$ 1.4 years, weight 78 $\pm$ 9 kg, height 182.3 $\pm$ 8.6 cm). All subjects performed incremental cycle ergometer exercise to extreme fatigue. The exercise started at a workload of 50 w, followed by gradual increases of 30 w every 3 min. On the day of the experiment, the subjects arrived at the laboratory in the morning after an overnight fast. Antecubital venous blood samples were taken at the end of each step and exercise onset. Samples analyzed for plasma potassium concentration by Flam photometry (Corning 480) and for plasma lactate concentration by

enzymatic method (measurement kit from Sigma). Ventilation (VE) during incremental exercise was also measured. Ventilation was obtained breath-by-breath by dividing expired volume by breath duration. We investigated linear regression analysis of the slopes of the ventilation, plasma potassium and lactate levels vs workload during incremental exercise. All subjects signed written informed consent. The investigation was approved by the Central Research Ethics Committee at the Tabriz University of Medical Sciences.

### RESULTS

Examples of VE, Mean values plasma potassium and lactate are plotted against workload at the each step. Figure 1 shows VE plotted against workload. It has been proposed that linear regression model be fitted to the VE-Workload relationship to estimate the location of the Respiratory Compensation Point (RCP). The RCP shown by the downward arrow is seen to mark the most evident angle in the curve and would be easily detected.

Figure 2 is a plot of plasma potassium vs workload, showing a break point marked by the intersection of the regression lines that represented the 2 linear segments above and below this point. This point of increased slope marks the start of respiratory compensation like, which is shown in Fig. 1.

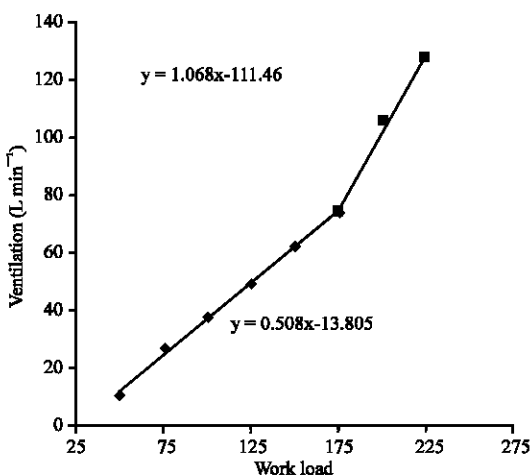


Fig. 1: Ventilation response vs workload during incremental exercise

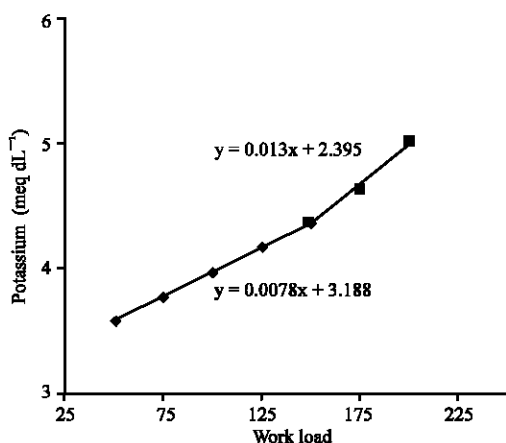


Fig. 2: Plasma potassium response vs workload during incremental exercise

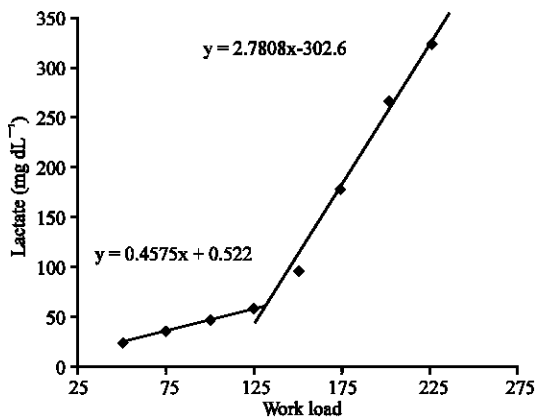


Fig. 3: Plasma lactate response vs workload during incremental exercise

At the start of the incremental workload, plasma lactate rises approximately linearly with workload up to the Anaerobic Threshold (AT). Above the AT, plasma lactate increases more steeply relative to workload, as is shown in Fig. 3, which is a plot of plasma lactate vs workload. The 2 regression lines represent the 2 linear portions of the curve that join at the point where lactate clearly begins to increase more rapidly.

## DISCUSSION

The Respiratory Compensation Point (RCP) marks the onset of hyperventilation during incremental exercise. Its physiological meaning has not yet been definitely determined, but the most common explanation is a failure of the body's buffering mechanisms which leads to metabolic (lactic) acidosis. The RCP was determined from the ventilation-workload plot. Researchers determined RCP independently by visually estimating the point of departure from linearity. Several studies have demonstrated the presence of a second rise in ventilation during incremental exercise, RCP, which is clearly distinguishable from the Ventilatory Threshold (VT) and must, hence, be based on different physiological mechanisms (Meyer *et al.*, 2004). Our study demonstrates that onset of hyperventilation during incremental exercise is at workload by 175 w, that is, the loss of linearity in a plot between VE and workload. Acidosis due to failure of lactate buffering seems to be a major determinant for exercise hyperventilation. For the 1st time it was directly demonstrated that exercise induced lactic acidosis is causally involved in the hyperventilation which starts at RCP. However, it does not represent the only additional stimulus of ventilation during intense exercise (Meyer *et al.*, 2004). Findings of this study show a break point in lactate level vs. workload curve at 125 w. It has been recognized since the 1920's by Hill *et al.* (1924) that lactate increases in the blood during heavy exercise. As exercise increases above a certain workload threshold, an anaerobic component of metabolism causes lactate to increase significantly (Beaver *et al.*, 1986). The increased accumulation of lactate in the skeletal muscle with exercise has been reported by Barlow (Barlow *et al.*, 1998).

Our results are in accordance with results from Schneider and Berwick (Schneider and Berwick., 1998). Currently there is no evidence in humans that shows a direct stimulatory effect of potassium on VE during exercise (Antonutto and Prampero, 1995). Previous demonstrations of plasma potassium and lactate levels during incremental exercise in human are rare. Potassium

is released by the skeletal muscle during exercise, mainly during repolarization of the muscle membrane (Barlow *et al.*, 1998). McCoy suggested that  $k^+$  may make an important contribution to the regulation of ventilation during incremental exercise (McCoy and Hargreaves., 1992). The break points appear in both curves of  $k^+$  and VE vs. workload at 175 w. This is similar to the finding of Patterson that showed close relationship between VE,  $k^+$  has been reported during exercise (Paterson *et al.*, 1990).

There are several possible means by which  $k^+$  release from the recruited muscles could regulate exercise ventilation. As previously stated, we observed a non-linear regression with a break point between VE and  $k^+$  during incremental exercise which is in agreement with previous experimental observations in those it was observed that  $k^+$  and VE increased in a coordinated manner (Yaspelkis *et al.*, 1994). In accordance with our hypothesis that VE is influenced by the  $k^+$  during incremental exercise, relationship between these parameters was consistent with the observations of the Antonutto and Prampero (1995) and Busse *et al.* (1992).

### CONCLUSION

VE and plasma potassium and lactate concentration proportionally increase during incremental exercise, but an inflection see in the curves, inflection on the plasma lactate plot occurs earlier than break points on VE and plasma potassium plots during incremental exercise.

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