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LETTER TO THE EDITOR

EXERCISE INDUCED CHANGES IN PLASMA POTASSIUM AND VENTILATION IN HEALTHY ADULTS

Sir,

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In order to exercise without experiencing hypoxaemia or hypercarbia a subject has to increase alveolar ventilation to meet the higher metabolic demands (1). There is elevation of serum potassium level along with hyperpnoea during maximal exercise in normal young adults. There has been a search for a "work fator" that could explain hyperpnoea following maximum exercise. There are recent persuasive proposals that elevation of serum potassium level (K^+) is one of the important influences leading to exercise hyperpnoea. There are very few studies exploring this interesting possibility (2, 3, 4). Also there are no studies of this type in central India. Hence the present work was undertaken to explore the correlation between serum potassium level and ventilation during exercise.

Twenty Ist MBBS male students from Government Medical College, Nagpur mean age 18 ± 2.2 years, mean height 164.4 ± 0.5 cm, mean weight 60.5 ± 4 kg were selected for the study. Thorough clinical examination, X-ray chest and ECG were done to exclude cardiopulmonary abnormalities. Exercise trained subjects were not included in the study. They were asked to abstain from tobacco and caffeine containing drinks on the day of tests. Each subject reported to the laboratory at 10 am and was asked to rest for 30 min. Exercise protocol was explained to the subjects. Arterialized venous blood sampling was used by inserting a teflon cannula (18 gauge) into antecubital vein and an extension catheter was attached. Patency of the cannula was maintained by intermittent flushing with heparinised saline solution. Prior to sampling the dead space was flushed by withdrawing 2 ml of blood. Blood samples were withdrawn into heparinised syringes.

Progressive incremental exercise on treadmill as per Bruce protocol (5) to symptom limited maximum was performed by the subjects. Serum pottasium levels were measeured by flame photometry (Ellico-Hyderabad) before (REST) immediately after (PEAK) and 10 min after exercise (RECOVERY). Ventilation was measured by autospiro AS 300 spirometer. The results were analysed by sterling computer SIVA PCAT 296 by applying Student's 't' test to obtain significance.

The Table shows mean serum potassium level in mmol/L and ventilation in L/min at rest, peak and recovery of exercise.

The increase in serum potassium level was very highly significant ($P<0.001$). It declined after 10 min of exercise though it did not come to resting level ($P<0.05$).

Ventilation was highest at the point of maximum exercise (peak) ($P<0.001$) and it

returned towards normal within 10 min of termination of exercise (recovery) (P<0.001).

TABLE I : Effect of exercise on serum potassium level and ventilation.

	Rest	*Peak*	*Recovery*
Serum potassium level (mmol/L)	3.89±0.21	4.22±0.25***	4.08±0.26
Ventilation (L/min)	20.30±6.74	77.90±21.59***	24.76±8.21

Values represent mean ± SD. (n = 20)
***Very highly significant (P < .001) than resting values.

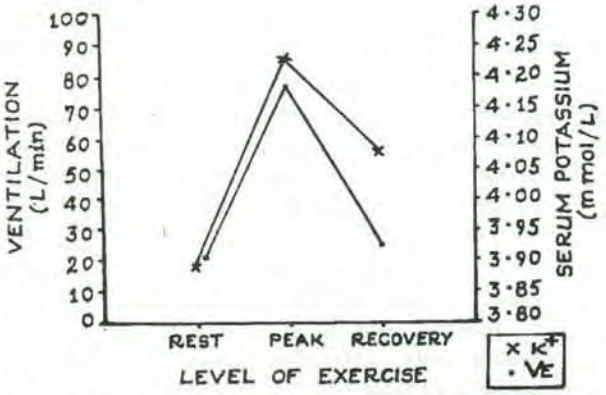

Fig. 1 : Correlation of minute ventilation and serum potassium level at rest, peak and recovery of exercise.

Exercise in man increases both ventilation and sensitivity of the ventilatory response to acute hypoxia. But how these occur is not clear. As arterial $P^{H}PO_2$ and PCO_2 remain unchanged; recently role of potassium as a factor that could be involved in the control of breathing during exercise has drawn attention.

Acute physical exercise increases plasma potassium as the efflux from working muscles and its level is not being adequately counteracted by uptake by the muscle (6). There is effusion of potassium from active muscle cells particularly in the falling phase of every action potential. Certain fraction of elevated plasma potassium is due to loss of plasma water and when plasma volume is restored after exercise, the plasma K^+ and total K^+ content return to pre-exercise level (7). Also at high work rates, there is accumulation of ADP and phosphate ions which inhibit ATPases. As a consequence of this, the rise in plasma potassium is enhanced. The combined effect of both raised noradrenaline and potassium may contribute to the enhanced sensitivity of the arterial chemoreflex in exercise (8). Elevation of arterial plasma K^+ similar to that seen in exercising man (9) stimulates the peripheral chemoreceptors (10) and may act as a drive to ventilation during exercise (11). It has been reported that during incremental exercise testing the patterns of change in ventilation and arterial potassium are similar; suggesting that changing arterial K^+ may lead to the phenomenon of the ventilatory threshold through its action on the peripheral chemoreceptors (12). K^+ channels are generally believed to be involved in type I carotid body cell chemotransduction (13). Hence this study supports the hypothesis of serum K^+ as a possible "Work Factor" in exercise induced increment in ventilation.

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