EFFECT OF EXERCISE ON ACID-BASE STATUS AND VENTILATORY KINETICS

MRUNAL S. PHATAK*, GEETA A. KURHADE, SHOBA B. KAORE AND GOURI C. PRADHAN**

Department of Physiology, Gouv. Medical College, and **Department of Physiology, NKP Salve Institute of Medical Sciences, Nagpur - 440 003

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Abstract : The normal respiratory responses and changes in acid base status in twenty normal height, weight and age matched subjects were studied; using Auto Spiro AS 300 spirometer for ventilatory parameters and NOVA stat profile 3 analyser for gas analysis. Each subject performed a progressive incremental treadmill exercise by Bruce protocol to their symptom limited maximum. Minute ventilation (V̇e), tidal volume (V̇t) and frequency of respiration (f) increased significantly (P<0.001). Acidosis occurred following exercise as pH of arterialized venous blood declined significantly (P<0.05). Gas analysis of arterialized venous blood showed a rise in pO₂ (P<0.001) and a fall in pCO₂ (P<0.001). Recovery of acid base status as well as gaseous pressure in blood did not occur after 10 min. Expired gas pCO₂ declined significantly (P<0.05) and pO₂ increase significantly (P<0.05). These pressures returned to resting levels 10 min after exercise. Thus in normal young adults heavy exercise caused an increment in ventilatory kinetics producing hyperpnoea which recovers after a rest of 10 min. Acidosis stimulates the respiratory centre to cause hyperventilation which helps to meet the added metabolic demands of strenuous exercise.

Key words : exercise pO₂ pCO₂ minute ventilation tidal volume frequency of respiration acidosis arterialized venous blood expired air

INTRODUCTION

The ventilatory and cardiovascular systems are stressed during exercise and ability to respond adequately to this stress is a measure of their physiological health. How these organ systems respond to increased gas exchange required for exercise can only be assessed by exercise testing. Exercise tolerance tests provide an estimate of patient's over all functional status which has to be factored into clinical decision making process. The responses obtained are compared to previously set 'normal' values and based on variations from these norms an opinion regarding exercise limitation by cardiac, respiratory or other factors may be offered.

*Corresponding Author
The measurement of respiratory gas exchange data provide a great deal of physiologic information. The general pattern of expected changes in ventilatory parameters are well known. (1, 2, 3, 4) In the present study we have made a composite analysis of the acid base status along with the ventilatory parameters in healthy adolescent Indian subjects before and after severe exercise.

METHODS

Twenty male subjects, aged 17–20 years (18 ± 2.2 years) with height 160–172 cm (165.4 ± 5 cm) and weight 50–60 kg (53.6 ± 3.3 kg) were selected from 1st year M.B.B.S. students of Government Medical College, Nagpur. Cardiopulmonary dysfunction was ruled out by history, thorough clinical examination ECG and X-Ray chest. Subject undergoing any type of exercise training were not included. Tidal volume (VT); frequency of respiration (f) and expired minute ventilation (VE); was assessed using autospiro AS 300. Respiratory gas exchange data namely PH, PO₂ and PCO₂ of arterialized venous blood along with PO₂ of expired air was recorded by NOVA stat profile 3 analyser (NOVA Biomedical Waltham MA, USA). Subjects reported to the laboratory at 10 a.m. in post absorptive state. They were asked to rest for a period of 30 min before the test.

Arteralized venous blood samples were obtained 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 sample were withdrawn into heparinised syringes. During withdrawal of blood sample subject wore a loose fitting water proof glove and immersed hand in water bath maintained at 44°C. for a minimum of 10 min.

Each subjects performed exercise on motorised treadmill as per Bruce protocol (5) to their symptom limited maximum. All the parameters were measured before (REST) immediately after (PEAK) and 10 min after exercise (RECOVERY). The significance of variation was measured by applying Student's t test with the help of sterling computer SIVA PCAT 296.

RESULTS

It is observed that ventilation, tidal volume and frequency of respiration increase very highly significantly (P<0.001) at peak of exercise (Table I).

pH of arterialized venous blood declines significantly at the peak of exercise (P<0.05). PO₂ shows a very highly significant decrease (P<0.001) Table II.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Rest</th>
<th>Peak</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vₑ (L/M)</td>
<td>20.3 ± 6.74</td>
<td>77.9 ± 21.58***</td>
<td>24.76 ± 8.21</td>
</tr>
<tr>
<td>Vₑ (Lit)</td>
<td>0.9 ± 0.35</td>
<td>1.3 ± 0.36***</td>
<td>0.95 ± 0.42</td>
</tr>
<tr>
<td>f (per min)</td>
<td>23.15 ± 5.18</td>
<td>59.35 ± 5.82***</td>
<td>24.55 ± 5.46</td>
</tr>
</tbody>
</table>

***P<0.001 very highly significant.
Values are mean ± S.D. (n=20).
TABLE II: Effect of exercise on arterialised venous blood.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Rest</th>
<th>Peak</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35 ± 0.28</td>
<td>7.25 ± 0.3*</td>
<td>7.30 ± 0.32</td>
</tr>
<tr>
<td>pO₂ mmHg</td>
<td>41.5 ± 3.49</td>
<td>71.36 ± 3.67***</td>
<td>67.5 ± 2.06</td>
</tr>
<tr>
<td>pCO₂ mmHg</td>
<td>46.36 ± 6.34</td>
<td>42.69 ± 8.78***</td>
<td>43.22 ± 6.84</td>
</tr>
</tbody>
</table>

*P<0.05 significant.
***P<0.001 Very highly significant.
Values are mean ± S.D. (n=20).

TABLE III: Effect of exercise on expired air.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Rest</th>
<th>Peak</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>pO₂ mmHg</td>
<td>113.54 ± 8.48</td>
<td>106 ± 7.22**</td>
<td>117.47 ± 7.73</td>
</tr>
<tr>
<td>pCO₂ mmHg</td>
<td>25.47 ± 2.86</td>
<td>29.22 ± 4.85**</td>
<td>24.98 ± 3.16</td>
</tr>
</tbody>
</table>

**P<0.01 Highly significant.
Values are mean ± S.D. (n=20).

The pO₂ of expired air shows a decline and pCO₂ of expired air increases at the peak of exercise. Both these changes are statistically highly significant (P<0.01) Table III. All the above mentioned changes return approximately to resting levels in the recovery period.

DISCUSSION

As a person begins exercise the respiration increases in rate and depth in proportion to the concentration of CO₂ in blood. Depth of inspiration depends on number of motor units of inspiratory neurones firing and their frequency of discharge whereas respiratory rate depends on the length of time elapsing between firings. The afferent receptors which influence the medullary respiratory centre to match the rate and depth of breathing to the increasing metabolic demands of the body are centrally located medullary receptors and peripherally located receptors in respiratory muscles, arteries near heart and skeletal joints. Stimuli which activate the receptors are chemical i.e. CO₂, O₂ and [H+] and mechanical i.e. pressure in contracting muscles and moving joints. Thus at higher work loads the metabolism increases manyfold and ventilation increases proportionately. Severe exercise causes anaerobic metabolism which results in acidemia. (Table II) This stimulates the respiratory centre primarily medullary chemosensitive receptors (7).

The efficiency of ventilatory system is also enhanced by rise in fraction of minute volume that reaches the alveoli, while work of breathing continues to be minimized by appropriate matching of Vₜ and f (8). A combined rise of Vₜ and f is sufficient to keep carbondioxide at its resting level at low or moderate exercise levels but in severe exercise when blood lactic acid level increases, ventilation increases more than that caused by CO₂ production (Table I). So pCO₂ of blood drops as person expires more CO₂ than produced. Gas exchange ratio
increases so alveolar pCO₂ increases and pCO₂ of blood decreases. Alveolar ventilation rises in proportion to ventilated CO₂ level (VCO₂) so pO₂ of blood must increase (9). Increased oxygen utilisation by tissues during heavy exercise results in decreased pO₂ of expired gas (Table III) (6).

Exercise ventilation is highest at point of maximum exercise. Increasing ventilatory demand is met with declining ventilatory ability which is a positive feedback which result in exercise termination. When exercise stops ventilation decreases rapidly towards baseline. By 2-3 min after exercise it falls to approximately 1/3rd its highest value (10). The transient behaviour of partial pressure of O₂ and CO₂ of blood is determined by the relationships between the kinetics of the ventilatory and gas exchange responses. Hence the composite knowledge of these responses is crucial in exercise testing.

REFERENCES

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