PULMONARY CAPILLARY BLOOD VOLUME IN HIGH-ALTITUDE PULMONARY OEDEMA

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Summary: Pulmonary capillary blood volume (Vc) and membrane diffusing capacity (DM) were measured on 10 occasions in four patients with acute high altitude pulmonary oedema. In the acute phase both the parameters were found to be significantly decreased as compared to the normal values at that altitude. Recovery from pulmonary oedema was accompanied by an increase in DM as well as Vc. It has been hypothesized that a significantly reduced Vc in presence of pulmonary oedema suggests exudation of fluid from the lung capillaries at normal pressure, presumably because of capillary endothelial damage.

Key Words: pulmonary capillary blood volume high-altitude pulmonary oedema

While the clinical and pathological features of acute high-altitude pulmonary oedema (HAPE) have been reported by several investigators (1,6,8-10,13), scanty information is available regarding the circulatory and respiratory changes in this condition. Fred et al. (4) and Hultgren et al. (7) found normal left atrial or pulmonary arterial wedge pressures in all the five patients studied. Recently Roy et al. (12) reported normal left atrial pressures in six patients with HAPE. Wedged pulmonary arterial pressure was measured in three of them and found to be normal. The pulmonary blood volume was increased in two of the six patients. The pathogenesis of HAPE is obscure. If it were produced as a result of increase in hydrostatic pressure in the pulmonary capillaries, the volume of blood contained therein (Vc) would be increased. On the other hand, if it was the result of exudation of fluid from the capillaries because of hypoxic damage to their endothelium, the Vc might be expected to decrease. Measurements of DM and Vc in HAPE have not been reported so far. The present study was undertaken with the hope that changes in Vc in the acute stage of HAPE might give a clue to its pathogenesis.

MATERIALS AND METHODS

Ten studies were performed on 4 subjects who developed HAPE at an altitude of 3,658m. All the patients were males with age ranging from 27-36 yrs. Two of them were smokers. One of the patients (DB) was a high altitude native who developed HAPE on ascending to 4,200m. The remaining three were lowlanders who had been freshly inducted to high altitude. Pulmonary oedema was diagnosed on the basis of typical clinical presentation and radiological findings in the absence of any underlying heart disease (13). All the patients had already received con-
ventional therapy with morphine, frusemide and oxygen before the initial study. Three of the patients were studied within the first three days of the onset of the symptoms; all the others between 4-10 days and 2 patients after more than 10 days of the episode. Two patients were studied thrice and the other two only twice.

All the studies were done at an altitude of 3,658m with the patient sitting comfortably in a chair. Pulmonary diffusing capacity was determined by the modified steady state technique utilizing a Rahn and Otis end-tidal sampling device for obtaining alveolar air (2). Diffusion measurements were made at two levels of oxygenation; one utilizing an air-CO mixture and the other with an O₂-CO mixture. Alveolar air oxygen tension was determined by direct sampling. Pulmonary capillary CO was determined by a breath holding method. The portable circuit utilized for these measurements has been described in detail earlier (5). Membrane diffusing capacity and pulmonary capillary blood volume were calculated by the method of Rought and Forster (11).

RESULTS

The results of DLco, DM and Vc estimations have been given in Table I. The mean values for these parameters in a group of 9 lowlanders studied within the first week of arrival at high altitude have been included in the Table for the sake of comparison (5). The DLco was reduced in the acute stage and it increased with recovery from pulmonary oedema. DM was significantly decreased in 2 of the 4 patients at the time of the initial study and it showed a tendency to increase on subsequent studies. There was a considerable variation, however, in the results obtained on different occasions.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>B.S.A. (M²)</th>
<th>Days after onset of HAPE</th>
<th>DLco, STPD (ml/min/mm Hg)</th>
<th>DM, STPD (ml/min/mm Hg)</th>
<th>Vc (ml)</th>
</tr>
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<tbody>
<tr>
<td>HS</td>
<td>28</td>
<td>170.0</td>
<td>67.0</td>
<td>1.78</td>
<td>5 days</td>
<td>15.27</td>
<td>60.8</td>
<td>21</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>13 days</td>
<td>22.91</td>
<td>46.8</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>23 days</td>
<td>23.14</td>
<td>212.5</td>
<td>27</td>
</tr>
<tr>
<td>JSP</td>
<td>27</td>
<td>162.5</td>
<td>59.0</td>
<td>1.63</td>
<td>1 day</td>
<td>15.29</td>
<td>32.4</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8 days</td>
<td>25.53</td>
<td>118.8</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>18 days</td>
<td>19.26</td>
<td>32.4</td>
<td>52</td>
</tr>
<tr>
<td>DB</td>
<td>29</td>
<td>162.5</td>
<td>50.0</td>
<td>1.52</td>
<td>3 days</td>
<td>8.28</td>
<td>13.3</td>
<td>27</td>
</tr>
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<td></td>
<td>6 days</td>
<td>15.63</td>
<td>21.7</td>
<td>56</td>
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<td>PVM</td>
<td>36</td>
<td>165.0</td>
<td>61.0</td>
<td>1.67</td>
<td>1 day</td>
<td>9.13</td>
<td>10.6</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5 days</td>
<td>18.87</td>
<td>25.3</td>
<td>76</td>
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<td>Mean</td>
<td>Initial study</td>
<td></td>
<td></td>
<td></td>
<td>2.5 days</td>
<td>11.99</td>
<td>29.3</td>
<td>36.5</td>
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<td></td>
<td>Subsequent studies</td>
<td></td>
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<td></td>
<td>12.1 days</td>
<td>20.84</td>
<td>76.3</td>
<td>50.2</td>
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<td>Mean values for 9 acute inductees</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>22.55</td>
<td>42.9</td>
<td>61</td>
</tr>
</tbody>
</table>

Pulmonary capillary blood volume showed a consistent increase on recovery. It was the minimum value obtained at the initial value was within the non-pulmonary oedema.

If the cause of high-altitude pulmonary oedema is high-altitude pressure the same should be done. It is difficult technically. The findings of Roy et al. (12) in HAPE exonerates the cause of high-altitude pulmonary oedema but it does not exclude its role. The reported normal measurements, however, may not indicate the pressure at a point.

Roy et al. (12) observed that the Vc of patients with HAPE. It was about 1.5 ml per min per kg. Reduced Vc may have been normal because the time the studies were performed with pulmonary oedema. DM was increased in 2 of the 4 patients at the time of the initial study and it showed a tendency to increase on subsequent studies. There was a considerable variation, however, in the results obtained on different occasions.

The dissimilarity of our findings is significant arterial unsation because the latter represents the effect of decreased Vc on the volume of blood in the pulmonary capillary oligemia.

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Pulmonary capillary blood volume was decreased in the acute phase and showed a consistent increase on recovery. In three of the 4 patients the initial $V_e$ was <34 ml which was the minimum value obtained in 9 healthy acute inductees (5). In one patient (PVM) the initial value was within the normal range but it showed further increase on recovery from pulmonary oedema.

**DISCUSSION**

If the cause of high-altitude pulmonary oedema lies in elevated pulmonary capillary pressure the same should be demonstrable on direct pressure measurements. Unfortunately it is difficult technically. The finding of normal left atrial pressure by Fred *et al.* (4) and Roy *et al.* (12) in HAPE exonerates the failure of left ventricle as being the cause of pulmonary oedema but it does not exclude pulmonary capillary hypertension. Hultgren *et al.* (7) and Roy *et al.* (12) reported normal pulmonary arterial wedge pressures in their patients. These measurements, however, may not truly reflect the pulmonary capillary pressure but may rather indicate the pressure at a point distal to the capillaries.

Roy *et al.* (12) observed the pulmonary blood volume to be increased in 2 of the 6 patients with HAPE. It was argued that the pulmonary blood volume in the remaining patients may have been normal because the fluid had already exuded into the alveolar spaces by the time the studies were performed. The findings of the present study do not support this contention. If the exudation of the fluid occurred because of pulmonary blood hypervolemia, the $V_e$ should at least be normal if not elevated. In a single patient of acute pulmonary oedema due to aortic valvular disease, Finlayson *et al.* (3) found the $V_e$ to be increased considerably above normal. Decreased $V_e$ in HAPE suggests exudation of fluid from the pulmonary capillaries at a normal pressure, presumably because of the hypoxic endothelial injury, resulting in pulmonary capillary oligaemia.

The dissimilarity of change in pulmonary blood volume and $V_e$ in HAPE might be because the latter represents only a fraction of the former. Thus even a 50% decrease in $V_e$ would appear as only a slight decrease in pulmonary blood volume. Moreover, in HAPE there is a significant arterial unsaturation with consequent hypoxic vasoconstriction resulting in moderate degree of pulmonary arterial hypertension (12). This would produce an increase in the volume of blood in the precapillary pulmonary vessels which might more than overshadow the effect of decreased $V_e$ on pulmonary blood volume.

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REFERENCES


Summary: The swallowing reflex is well established by making series of swallowing movements. It is controlled in the dorsal motor reticular formation, in the medulla oblongata. The swallowing pathway involves cranial nerve X (12), which is composed of both sensory and motor components. The sensory component is activated by stimulation of the palate, pharynx, and larynx, while the motor component is activated by the pharyngeal and laryngeal muscles. The swallowing reflex is initiated by the activation of the swallowing center in the medulla oblongata, which then sends signals via cranial nerve X to the pharynx and larynx, causing the muscles to contract and the food to be propelled into the esophagus. The swallowing reflex is essential for the proper functioning of the digestive system, as it allows for the efficient and safe passage of food from the mouth to the stomach.