PULMONARY CAPILLARY BLOOD VOLUME IN HIGH-ALTITUDE PULMONARY OEDEMA

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Summary: Pulmonary capillary blood volume (Vc) and membrane diffusing capacity (D_M) 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 D_M 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 (V_c) 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 V_c might be expected to decrease. Measurements of D_M and V_c in HAPE have not been reported so far. The present study was undertaken with the hope that changes in V_c 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 th patients were studied within the first three days of the onset of the symptoms; all the 4 be ween 4-10 days and 2 patients after more than 10 days of the episode. Two patients we studied thrice and the other two only twice.

All the studies were done at an altitude of 3,658m with the patient sitting comfortably o a chair. Pulmonary diffusing capacity was determined by the modified steady state techniqu utilizing a Rahn and Otis end-tidal sampling device for obtaining alveolar air (2). Diffusio measurements were made at two levels of oxygenation; one utilizing an air-CO mixture and th other with an O_2-CO mixture. Alveolar air oxygen tension was determined by direct samplin Pulmonary capillary CO was determined by a breath holding method. The portable circu utilized for these measurements has been described in detail earlier (5). Membrane diffusin capacity and pulmonary capillary blood volume were calculated by the method of Roughto and Forster (11).

RESULTS

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

TABLE I : DM and Vc in high altitude pulmonary oedema

Subject	*Age (yrs)*	*Height (cm)*	*Weight (kg)*	*B.S.A. (M^2)*	*Days after onset of HAPE*	*DLCO, STPD (ml/min/ mm Hg)*	*DM, STPD (ml/min/ mm Hg)*	*Vc (ml*
1. HS	28	170.0	67.0	1.78	5 days	15.27	60.8	22.
					13 days	22.91	46.8	51.
					23 days	23.14	212.5	27.
2. JSP	27	162.5	59.0	1.63	1 day	15.29	32.4	32.
					8 days	25.23	118.8	35.
					18 days	19.26	32.4	52.
3. DB	29	162.5	50.0	1.52	3 days	8.28	13.3	23.
					6 days	15.63	21.7	58.
4. PVM	36	165.0	61.0	1.67	1 day	9.13	10.6	67.
					5 days	18.87	25.3	76.
Mean		Initial study			2.5 days	11.99	29.3	36.
		Subsequent studies			12.1 days	20.84	76.3	50.
Mean values for 9 acute inductees						22.55	42.9	61.

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_c 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_c 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_c to be increased considerably above normal. Decreased V_c 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 changes in pulmonary blood volume and V_c in HAPE might be because the latter represents only a fraction of the former. Thus even a 50% decrease in V_c 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_c on pulmonary blood volume.

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