19

The Pulmonary Circulation of High Altitude Natives

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La Paz, the capital of Bolivia, a city with about 700,000 inhabitants, extends itself from an altitude of 4000 m above sea level down to 3200 m. The altitude of the Bolivian Institute of Altitude Biology (I.B.B.A.) is 3600 m or 12,200 ft. At this altitude the atmospheric pressure averages about 499 mmHg, which provides an inspired oxygen tension (PI_{O_2}) of 95 mmHg.

Since Rotta et al. (8) first published their data, it has been known that the pulmonary circulation behaves quite differently at high altitude than at sea level with respect to hemodynamics. In particular, a state of pulmonary hypertension exists due to the low alveolar oxygen tension ($Pa_{O_2} = 60$ mmHg in La Paz) which increases pulmonary vascular resistance (12). Rotta et al., who made the first high altitude studies with right heart catheterization at Morococha, Peru (4540 m or 14,900 ft.), found an increase in mean pulmonary artery pressure to 24 mmHg in natives of that altitude and to 18 mmHg in newcomers. Peñaloza et al. (7), who studied larger series also at Morococha, indicated that the mean pulmonary artery pressure of such people is in fact higher. In a study of 38 native residents of Morococha they found mean pulmonary artery pressure to be 28 mmHg. Blount and Vogel (3) recorded an average value of 24 mmHg in newcomers to Leadville, Colorado (altitude 10,150 ft).

The differences in magnitude of the mean pulmonary artery pressure in natives and newcomers have been attributed to an increased bulk of medial muscle in the peripheral pulmonary arteries of those permanently living at high altitude (1) and to pulmonary vasoconstriction stemming from the critical reduction in alveolar oxygen tension in newcomers (11). Above the altitude of 10,000 ft the mean pulmonary artery pressure can be predicted from an empiric relationship, which states that the mean pulmonary artery pressure varies directly with the inspired oxygen tension (PI_{O_2}) and inversely with the product of the existing barometric pressure multiplied by the alveolar oxygen tension. It is possible to set up a linear regression equation relating the mean pulmonary artery pressure at altitude to the alveolar oxygen tension (6). In this way the mean pulmonary artery pressure can be estimated and predicted; for instance, for the altitude of La Paz (12,200 ft), the predicted pulmonary artery pressure was approximately 23 mmHg.

The first cardiac catheterization data derived from nine male and two female vol-

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Table 19-1. *Right Heart and Pulmonary Vascular Pressures of 11 Normal High Altitude Natives at La Paz (3600 m).*

	RAP (mmHg)			RVP (mmHg)			MPAP (mmHg)			PCP (mmHg)		
	S	*D*	$\bar{X}$	*S*	*D*	$\bar{X}$	*S*	*D*	$\bar{X}$	*S*	*D*	$\bar{X}$
$\bar{X}$	6.2	0.90	3.81	36.7	0	18.9	38.0	14.5	22.9	10.1	4.2	6.8
SE	0.73	0.54	0.32	2.01	0	2.05	2.47	0.76	1.34	1.07	0.46	0.59

RAP, right atrial pressure: RVP, right ventricular pressure; MPAP, main stem pulmonary artery pressure; PCP, pulmonary wedge pressure; S = systolic; D = diastolic; $\bar{X}$ = mean.

unteers at La Paz, whose average age was 22.4 years, showed a mean pulmonary artery pressure of 22.9 mmHg, which is very close to that predicted from the regression equation (9). On exercise this pressure rose to a mean of 49 mmHg which constitutes an increase of 109%. Pulmonary wedge or capillary pressure in these subjects was 6.8 mmHg and increased only slightly during exercise, i.e., to 9.8 mmHg. The results of this first study are shown in Tables 19-1–19-3. In these normal individuals, oxygen breathing at that altitude for 4 min resulted in a mean pulmonary artery pressure drop from 22.9 to 16 mmHg, which is about 30%. No change was demonstrated with regard to the mean capillary pressure, and cardiac output was not affected. An infusion of acetylcholine caused mean pulmonary artery pressure to fall, but not as much as that noticed with oxygen. The mean fall was from 22.9 to 18 mmHg and there was no change in the pulmonary wedge pressure.

Coudert and the French-Bolivian team (4) studied 67 normal male high altitude natives with a mean age of 24 years and compared the results with those obtained by Banchero et al. from a comparable group in Lima (2). The results, which are similar to those of the first study, are shown in Tables 19-4–19-7. During exercise (75 W) cardiac output increased from 7.0 ± 1.5 to 11.9 ± 1.8 liter/min and mean pulmonary artery pressure increased from 20.0 ± 3 to 32 ± 8 mmHg. Pulmonary wedge pressure remained unchanged (7 ± 4 at rest; 7 ± 3 mmHg during exercise).

It could be supposed that the increase in pulmonary artery pressure would favor the perfusion of the upper lung zones and thus improve the ventilation-perfusion rate, especially in the sitting and standing positions. In order to establish whether or not there exists a relationship between pulmonary artery pressure and regional distribution of pulmonary blood flow, two studies were undertaken (5,10).

The first study compared 15 normal male high altitude natives to five normal lowlanders. The observations were confined to the right upper lung (RUZ) and right lower lung zones (RLZ). Simultaneous isotope dilution curves derived from these lung zones by surface scanning in the sitting and recumbent positions showed in

Table 19-2. *Pulmonary and Systemic Vascular Resistances of 11 Normal High Altitude Natives at La Paz (3600 m).*

	$\dot{Q}$ FICK (liter/min)	TPR (dyn/s/cm^{-5})	PAR (dyn/s/cm^{-5})	SAP			SVR (dyn/s/cm^{-5})
				S	*D*	$\bar{X}$	
$\bar{X}$	5.71	312.0	219.7	127	80		2207
SE	0.32	21.4	21.6	3.5	2.5	2.7	82.7

TPR, total pulmonary vascular resistance; PAR, pulmonary arteriolar resistance; SAP, systemic artery pressure; SVR, systemic vascular resistance; Q Fick = cardiac output by direct Fick method; S = systolic; D = diastolic; $\bar{X}$ = mean.