TOPICS IN ENVIRONMENTAL PHYSIOLOGY AND MEDICINE

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High Altitude Physiology and Medicine



Hypoxic Pulmonary Vasoconstriction and Ambient Temperature

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It has long been known that hypoxia and arteriolar structural changes were the main factors in altitude pulmonary hypertension. However, other environmental factors such as cold may play a contributing role.

In order to test the effects of exposure to heat and cold on highlanders' pulmonary hemodynamics, 21 volunteers, born and residing between 3800 and 4200 m, were studied at 3750 m ($P_B = 493 \text{ mmHg}$).

Cardiac output (measured by indocyanine green dilution), brachial and pulmonary (free and wedge) arterial pressures, heart rate (EKG), and blood gases were measured in different environments.

- 1) In 21 subjects at room temperature (Tgl 25°C, 40-45% humidity) breathing ambient air.
- 2) In 13 subjects after a 30-min exposure to radiant heat (5 kW).
- 3) In 9 of the proceding subjects for whom the heat stress was prolonged, and the hypocapnia induced by thermal hyperventilation was corrected by breathing hypercapnic mixture.
- 4) In 8 subjects after 30-min exposure to cold from ice bags on the limbs and the

chest. In no case was shivering observed.

Heat exposure increases cardiac output with no significant change in pulmonary pressures. This situation results in a significant decrease in the so-called pulmonary arteriolar resistance; correction of the hypocapnia does not significantly affect the effect of heat.

Conversely, cold stress decreases cardiac output and induces an increase, although not significant, in pulmonary artery pressures. Pulmonary vascular resistance increases significantly (Fig. 40-1).

The effect of elevated ambient temperature on pulmonary hemodynamics has already been studied at sea level. As far as cold is concerned, very little has been published concerning humans; Viswanathan failed to find any change in pulmonary arterial pressure during cold pressure tests at sea level in subjects with a prior history of HAPE, whereas Atterhög et al. measured an increase central blood volume after exposure to a cold wind. However, there are several publications on the effects of both cold and altitude upon the

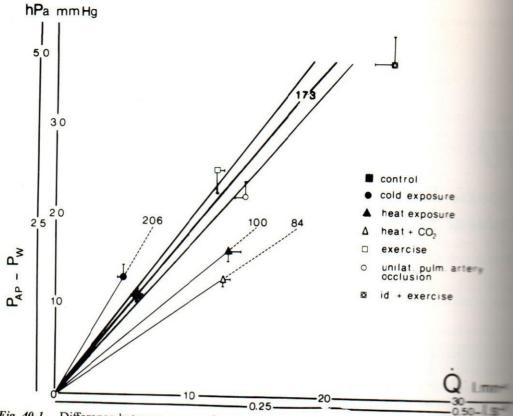


Fig. 40-1. Difference between mean pulmonary artery (P_{AP}) and pulmonary artery (P_{W}) as function of cardiac output (Q). Data obtained during thermal stress are comparable by Lockhart et al. (2) during exercise and during unilateral pulmonary artery occlusion have been multiplied by 2 (mean and standard error). Isoresistance lines have with corresponding values expressed in $kPa \cdot ml^{-1} \cdot s$; shaded area corresponds to 95% arteriolar resistance measured in La Paz, Bolivia, in normal subjects at rest.

pulmonary circulation in animals (1,3) which show the potentiation of the vasomotor effect of hypoxia by cold.

Summary

Thermal environment has to be taken into account in the interpretation of changes in the pulmonary circulation induced by altitude hypoxia, even in resting conditions. For instance, at 3750 m, mean pulmonary artery pressure measured routinely in a catheterization room at 19°C was found to be 21 ± 4 mmHg (n = 213) whereas it is 19 ± 4 mmHg (n = 118) when measured at 25°C room temperature.

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