Editorial

The Preterminal Arterioles in the Pulmonary Circulation of High-Altitude Natives

NOTABLE advances in knowledge of respiratory physiology and pulmonary hemodynamics have resulted from the application of newer technics. Special attention has been given to the alterations produced by hypoxia in the pulmonary circulatory system. In 1940 Von Euler and Liljestrand showed that the pulmonary arterial pressure in cats increased when they were maintained in low-pressure chambers. The anatomic studies performed on the inhabitants in high-altitude regions of the Peruvian Andes have shown some of the characteristics of the pulmonary circulation and respiratory function in these individuals. The anatomic studies performed in this field have provided the basis for further understanding of the functional changes observed. Close correlation has been found between the hemodynamic alterations and the histologic structural features in pulmonary vasculature.

The vascular channels designated “pre-terminal arterioles,” (“capillary arterial collaterals”) an important component of the pulmonary vascular tree, have been assigned little functional significance. Some of the hemodynamics and oxygen blood saturation changes appearing in high-altitude natives during exercise, as well as in subjects suffering from acute pulmonary edema at high altitude, are better understood when the role played by these vessels is appreciated.

The “preterminal arterioles” are vessels whose walls consist of a single elastic layer and a few subintimal smooth-muscle fibers. They do not belong to the end branchings of the pulmonary vascular tree. They arise from small and medium-sized muscular arteries, from which they emerge at right angles and open directly into the venous side of the alveolar septal capillaries, forming a bypass between the arterial and venous circulation of the lungs (fig. 1). In some instances a neuromuscular apparatus can be demonstrated at the arterial stoma.

These vessels carry unoxygogenated blood and they cannot participate in gaseous exchange because they are thick walled and are not located in the alveolar septal walls. Arias-Stella designated these vessels “preterminal arterioles,” when studying the histologic characteristics of the lung in primary pulmonary hypertension. Costero and associates verified the existence of these communications in normal subjects. More recently, Heard and Reid, using India ink injection, clearly demonstrated the existence of the same structures.

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The physiologic role attributable to the preterminal arterioles in the pulmonary circulation may be presumed from (a) the hemodynamics of the lungs in the high-altitude subject during exercise and (b) the features of acute high-altitude pulmonary edema.

**Preterminal Arterioles and Pulmonary Hemodynamics in the High-Altitude Native during Exercise**

The existence of a mild degree of pulmonary arterial hypertension has been clearly demonstrated in natives at high altitudes. Anatomic studies in these subjects have revealed that the basic structural feature associated with this hemodynamic state is a persistence of the fetal pulmonary vascular pattern. Another pertinent observation is that individuals born and living at high altitudes exhibit arterial blood desaturation at rest.

Cruz in Morococha (14,900 feet above sea level) has shown that pulmonary arterial pressure and arterial blood desaturation increase significantly during exercise in relation to levels at rest. West and associates and Vogel and associates have obtained similar results.

It has been widely held that pulmonary arteriolar constriction and increased pulmonary blood flow during exercise are fundamental contributory factors in the rise of pulmonary arterial pressure. On the other hand, several interpretations have been offered to explain the significant increase in peripheral blood desaturation exhibited by high-altitude natives when performing exercise. Cruz suggested that the supine position assumed by the experimental subject causes a deficient gaseous exchange in the posterior portion of the lungs. West and collaborators thought that the permeability of the capillary alveolar membrane underwent alteration. Vogel and associates, considering the reduction of the respiratory equivalent, have recently taken the stand that at high altitude, exercise produces a condition of relative hypoventilation.

Knowledge of the vascular structure in the "high-altitude lung" aids in interpretation of the results obtained from hemodynamic studies performed in the exercising high-altitude subject. The increase of arteriolar smooth muscle suggests that these vessels are capable of an intense degree of contraction. This could thus be a basis for the marked rise in pulmonary arterial pressure noted during exercise.

Two factors appear to indicate that "preterminal arterioles" are involved in the production of blood desaturation in the high-altitude native performing exercise. The first relates to the occurrence of pulmonary arterial hypertension in the high-altitude subject during exercise, which could be a determining factor in the opening of a large number of these channels connecting the arterial and venous side of the capillary bed. Such bypasses would greatly reduce the transit time for gaseous exchange in venous capillaries, partially in view of the increased blood stream velocity resulting from pulmonary arterial hypertension. By this mechanism a considerable volume of unoxygenated blood may enter pulmonary veins and ultimately the systemic circulation, thereby increasing the desaturation of peripheral arterial blood. The opening of the preterminal arterioles would serve as a compensatory safety valve to reduce the pulmonary hypertension.

The patency of the collateral channels may
have further significance; the elevated pressure in the pulmonary arterial system may be transferred unaltered into capillary network (fig. 2). The existence of pulmonary capillary hypertension during exercise provides a second point of support for our hypothesis. In catheterization experiments in exercising high-altitude natives, 17 of 35 subjects showed increases of capillary pressure, some of them registering rises of over 100 per cent.

**High-Altitude Pulmonary Edema and the Preterminal Arterioles**

High-altitude pulmonary edema appears in young people native to or residing at high altitudes for long periods of time when these individuals return to high altitudes after a brief period in the lowlands. Hemodynamic studies performed in these individuals have shown higher pulmonary arterial pressure levels than in ordinary high-altitude subjects; there are increased desaturation of the peripheral arterial blood and an apparently normal pulmonary capillary pressure.

The degree of pulmonary arteriolar muscularization that these patients exhibited correlates closely with the level of pulmonary hypertension that appeared during the attack of pulmonary edema.

It appears also that the hypertension is sufficient to open the preterminal arterioles and thus permit the pulmonary arterial hypertension to be transmitted into the extensive pulmonary capillary bed. The resulting increment in capillary hydrostatic pressure and the increased capillary permeability, sec-
ondary to hypoxia, are the fundamental factors in the production of edema. It is interesting to note that hemodynamic measurements indicating normal capillary pressure in people affected by high-altitude pulmonary edema were actually taken after the edema existed. It would then appear that the passage of fluid from the vascular space to the alveoli had already occurred and, therefore, a fall in capillary hydrostatic pressure would be anticipated.

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References

Servetus and the Lesser Circulation

The most important physiological contributions of Michael Servetus are these: first, the passage of blood through the lungs, its mixing there with air, and its change in color; second, his emphasis on the size of the pulmonary artery, from which he deduced that it must serve some purpose other than transporting blood for the nourishment of the lungs; and third, his twice-repeated statement that there is no communication allowing blood to move through the interventricular septum.—André Courmand, M.D. Circulation of the Blood. Edited by Alfred P. Fishman, M.D., and Dickinson W. Richards, M.D. New York, Oxford University Press, 1964, p. 23.
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