Pulmonary Dysfunction Secondary to Heart Disease: Aspects Relevant to Anesthesia and Surgery

Myron B. Lauer, M.D., Phillips Hallowell, M.D., Allan Goldblatt, M.D.

SYMPTOMATIC HEART DISEASE, be it congenital or acquired, is frequently accompanied by abnormal lung function. Unlike chronic lung disease in which parenchymal destruction is prominent, changes secondary to cardiac dysfunction are predominantly vascular in nature. Pulmonary vessels are known to react to one of three principal stimuli: 1) passive congestion (e.g., left ventricular failure or mitral stenosis); 2) hypoperfusion (e.g., right ventricular outflow tract obstruction); 3) hyperperfusion (i.e., left-to-right shunt). We are generally agreed that the degree of respiratory failure caused by passive congestion is related to the amount of water accumulated in the lung. Contrast this effect, usually the consequence of acquired heart-valve disease, with the changes that arise from congenital heart disease. Pulmonary edema leads to hypoxemia as well as an alteration in lung mechanics; congenital defects characterized by a right-to-left shunt are associated with hypoxemia and little change in lung mechanics. Pulmonary hypoperfusion secondary to severe right ventricular outflow tract obstruction and cyanosis owing to a right-to-left shunt (e.g., tetralogy of Fallot) is associated with no specific ventilatory impairment apart from the expected sequelae of diminished blood flow. Similarly, a left-to-right shunt has a minimal effect on respiratory function early in life, but does lead eventually to severe vascular deformities and limitation of appropriate perfusion.

The present review considers: 1) the altered mechanics of ventilation in acquired heart-valve disease and the effect of vascular changes on blood-gas exchange; 2) the effects of extracorporeal circulation; 3) the problems attendant upon pulmonary embolism; and 4) the abnormalities characteristic of congenital heart disease. Preference is given to common forms of heart disease to assist the clinician charged with the care of these patients in and outside the realm of cardiac surgery.

Pulmonary Dysfunction in Acquired Heart-valve Disease

BLOOD FLOW AND MECHANICS

Dyspnea, or "breathlessness," is the hallmark of abnormal pulmonary function in heart disease, and a direct result of acute or chronic vascular congestion. A rise in pulmonary venous pressure increases the transmural hydrostatic force that promotes translocation of water from the intra- to the extravascular space; all abnormalities in pulmonary function stem from this increase in pulmonary blood volume and water content.

To understand better the mechanics involved, we need to examine how vascular and airway (transpulmonary) pressures influence the distribution of blood flow within the lung. Regardless of position (upright, supine, or lateral) the lung is always subject to the effects of gravity. This will be influenced primarily, but not exclusively, by the magnitudes of both pulmonary arterial and pulmonary venous pressures. Let us assume for the moment that we are dealing with an upright lung in which alveolar pressure (P\(_{a\upsilon}\)) is the same throughout (for details see figure 1). We note that above a certain level near the apex of the lung there is little or no perfusion, since pulmonary arterial pressure (reflected by the height of the hydrostatic column) is not quite high enough. Keep in mind that 1) we are dealing with collapsible vessels at the alveolar level and 2) left atrial pressure is normally lower than pulmonary arterial pressure. Vessels at the top, or the nonperfused portion of the lung, are permanently collapsed (at least in the model) because alveolar or transpulmonary pressure at end-expiration is greater...
than pulmonary arterial pressure. This area, which is ventilated but not perfused, characterizes the physiologic deadspace (VD) in the absolute sense and has been defined as Zone I. Immediately below this level, the hydrostatic pressure within the pulmonary artery (Ppa) is higher than Ppa and the collapsible vessel (capillary) is open (Zone II). Note, however, that the capillary is collapsed at the effluent end of its alveolar portion. This phenomenon, caused by an alveolar pressure (Ppa) that is higher than effluent pressure (Ppa).

**Fig. 1A.** The effects of pulmonary arterial (Ppa), alveolar (Ppa), and left atrial (Ppa) pressures on flow in a collapsible blood vessel. Zones I, II, and III represent well-defined areas of progression from the top to the bottom of the lung, where changes in hydrostatic pressure (indicated by dashed lines) will affect both flow and patency of the intra-alveolar vessel. In Zone I (top of the lung) alveolar pressure (Ppa) exceeds both pulmonary artery (Ppa) and left atrial (Ppa) pressures. There is no blood flow, and this area represents physiologic deadspace, i.e., ventilation but no perfusion. Zone II (mid-lung) is characterized by Ppa that is higher than Ppa; however, flow through the capillary is not continuous since Ppa is higher than Ppa. Patency of the effluent portion is regulated by the fluctuation in the three pressures. This area is the main source of resistance to flow within the lung. In Zone III (bottom of the lung) Ppa exceeds Ppa and the collapsible vessel is open at all times. Blood flow is regulated by the Ppa-Ppa gradient as well as the vessel diameter. Heavy cross-hatching: mixed venous blood; light cross-hatching: arterialized blood. Zone IV is hypothetical and postulated to appear only with an abnormally high Ppa. The associated pulmonary hypertension is the result of vascular changes, i.e., medial hypertrophy and intimal proliferation at the arteriolar level. Reversal of zonal flow characteristic of mitral-valve disease is secondary to these changes: pulmonary hypertension enhances apical flow while vascular changes, most prominent at the base, increase resistance and hinder flow. When Ppa reaches a critical value, colloid osmotic and tissue hydrostatic forces are overcome and fluid transudation takes place (arrows). Alveoli in Zone IV are subject to early collapse.