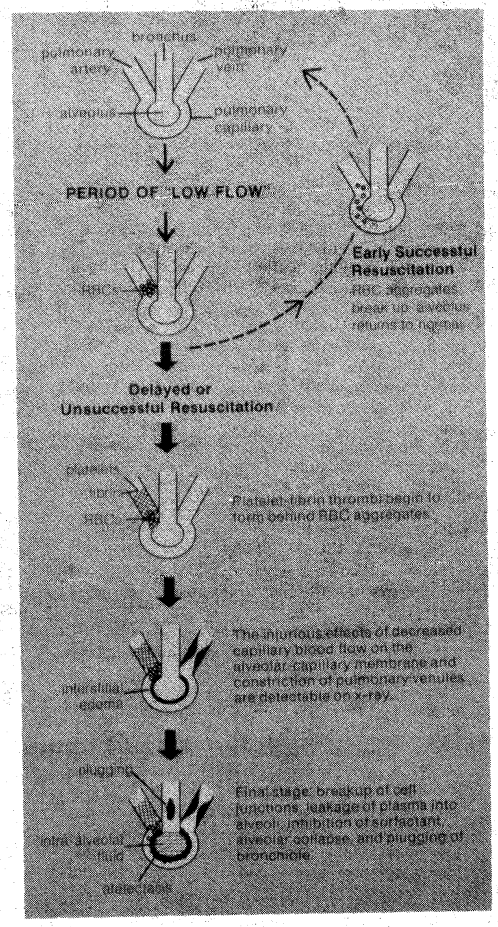
Fig. 4.—Microphotograph of animal lung (*in vivo*) following hypovolemic shock. Capillaries around the central alveolus are engorged with erythrocyte aggregates.

Fig. 5.—Pathophysiology of low-flow lung.



with the probable exception of cardiogenic shock. In our laboratories, we have observed the onset of the disease process in animals only five minutes after hypotension had been induced by hypovolemia. Microscopic studies of intravascular events reveal the aggregation of formed elements of the blood—particularly the erythrocytes—into progressively larger clumps (Fig. 4). Such aggregates tend to settle in relatively dependent lung areas, while plasma streams toward the upper areas. Pulmonary capillaries may then become blocked by the aggregates. If the patient is resuscitated in time, the aggregates will break up and reenter the circulation. However, platelet-fibrin thrombi may form behind the aggregates leading to a reactional edema of the perivascular tissues; this process goes on to destroy the endothelial and epithelial cells of the lung, producing exudation of plasma into alveoli, loss of surfactant function, decreased compliance, and atelectasis (Fig. 5). Occasionally, the process may actually be aggravated by resuscitative efforts.[8] Experiments in our laboratory suggest that simply by administering large volumes of crystalloid solution (eg, glucose, saline), one can trigger the aggregation of erythrocytes to cause interstitial edema of the lung.

In addition to the cellular effects of the low-flow state described, it seems likely that a humoral component causes constriction of the small veins.[11] The combination of all these factors produces a maldistribution of air in the lung and progressive alveolar-capillary block. As in patients with fat embolism, the clinical onset of post-traumatic pulmonary failure may be insidious because the initial pulmonary reactions are interstitial; few, if any, physical abnormalities are apparent. Characteristically, the patient experiences arterial oxygen desaturation following an apparently successful resuscitation from the initial shock state (Fig. 6a). Hypoxia supervenes within hours, and even though one hears normal chest sounds on auscultation, the x-ray shows a diffuse infiltration of both lungs (Fig. 6b). The patient breathes deeply and rapidly; there is marked metabolic acidosis, arterial oxygen desaturation and, terminally, the carbon-dioxide level increases in arterial blood. The most prominent finding is hypoxia which is secondary to a severe diffusion block produced by intra-alveolar septal edema (see Fig. 5). As the process continues, atelectasis and pulmonary congestion become superimposed.