Chapter 2

The Morphology of the Adult Respiratory Distress Syndrome

G. Schlag and H. Redl

The morphology of adult respiratory distress syndrome (ARDS) essentially involves two distinct aspects: aetiology and pathophysiology. Many different pathophysiological mechanisms may be important in the development of ARDS. However, although during the initial phase the morphological appearances may be somewhat disparate, eventually the morphological picture is that of classical ARDS.

The primary changes are of great relevance, since they represent a frequent, often reversible process and, if correctly treated at the appropriate time, organ function can be fully restored. The most important part of the lung in morphological terms is the alveolar septum, where gas exchange takes place. This is also where early pathological changes occur, while extension to the interstitial spaces is observed as a later event. Light microscopy is not always able to demonstrate this change in cellular structure and the initiation of cellular damage, and only the advent of electron microscopy has resolved some of these problems.

The alveolar septa divide the alveolar region into individual alveoli. The alveolar septum consists of: epithelium (type I and type II pneumocytes), interstitium, basal membrane and the endothelium of the alveolar capillary vessels (Fig. 2.1).

Pathogenesis of Post-traumatic ARDS: Direct Versus Indirect Lung Injury

In the pathogenesis of ARDS a distinction is made between direct and indirect lung injury. Injury may also occur from a combination of both these processes. According to its aetiology the pulmonary damage differs primarily in morphology.
Direct lung lesions are most frequently due to lung trauma, such as contusion, aspiration, inhalation and burn injuries. Iatrogenic damage such as fluid overload and overtransfusion may also cause similar morphological alterations in the lungs.

Indirect lung injuries are mainly assigned to the broad pathological spectrum of shock (hypovolaemia, trauma, endotoxin, sepsis) and are related to the release of mediators with cytotoxic effects (see Chapter 3). The neurogenic interstitial lung oedema associated with craniocerebral trauma is also considered as indirect lung damage. It is probably caused by sympathico-adrenergic over-regulation.

**Direct Lung Trauma**

**Contusion**

One example of direct lung injury is pulmonary contusion as a consequence of trauma with or without rib fractures. Both the vascular system and the alveolar area are injured. The pathological changes are caused by damage to the alveoli and pulmonary capillaries, with subsequent interstitial and alveolar haemorrhage and oedema. The oedema and haemorrhage produce collapse of airways in the form of microatelectasis and segmental atelectasis. Surrounding these areas is a layer of less severely injured tissue with interstitial oedema and inflammatory cells (Mecca 1986).

**Aspiration and Inhalation**

Aspiration and inhalation damage primarily involves not only the proximal branches of the tracheobronchial tree but also the alveolar area and thus directly injures the gas exchange surface (Glause et al. 1979).