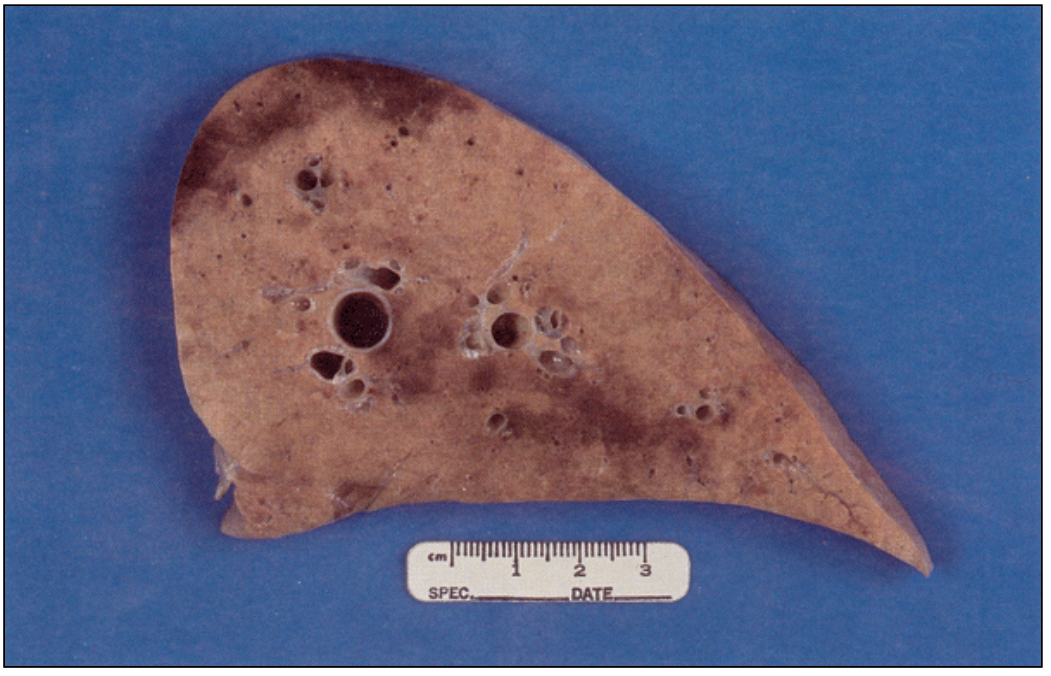

Fig. 8-5. In this cross-sectional wedge from the right diaphragmatic lung lobe from a sheep exposed tu a single blast, note the dark-brown areas of subpleural and peribronchial hemorrhage.
Source: Walter Reed Army Institute of Research

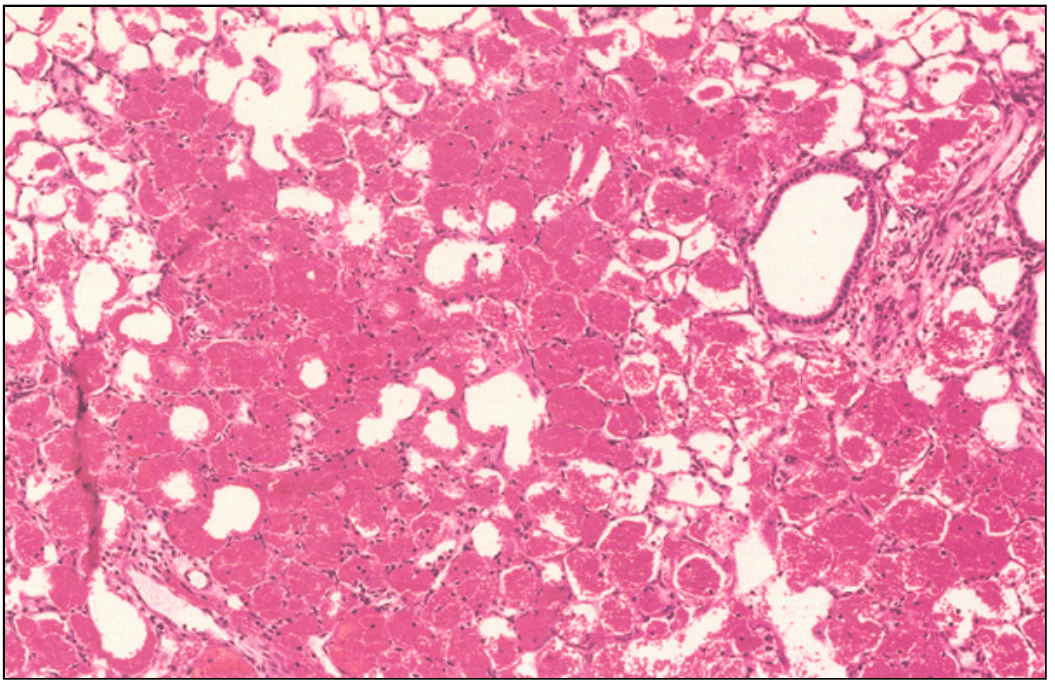

Fig. 8-6. In this histological section of lung parenchyma from a sheep exposed to a single blast, the alveoli are slightly dilated and filled with erythrocytes.
Source: Walter Reed **Army** Institute of Research

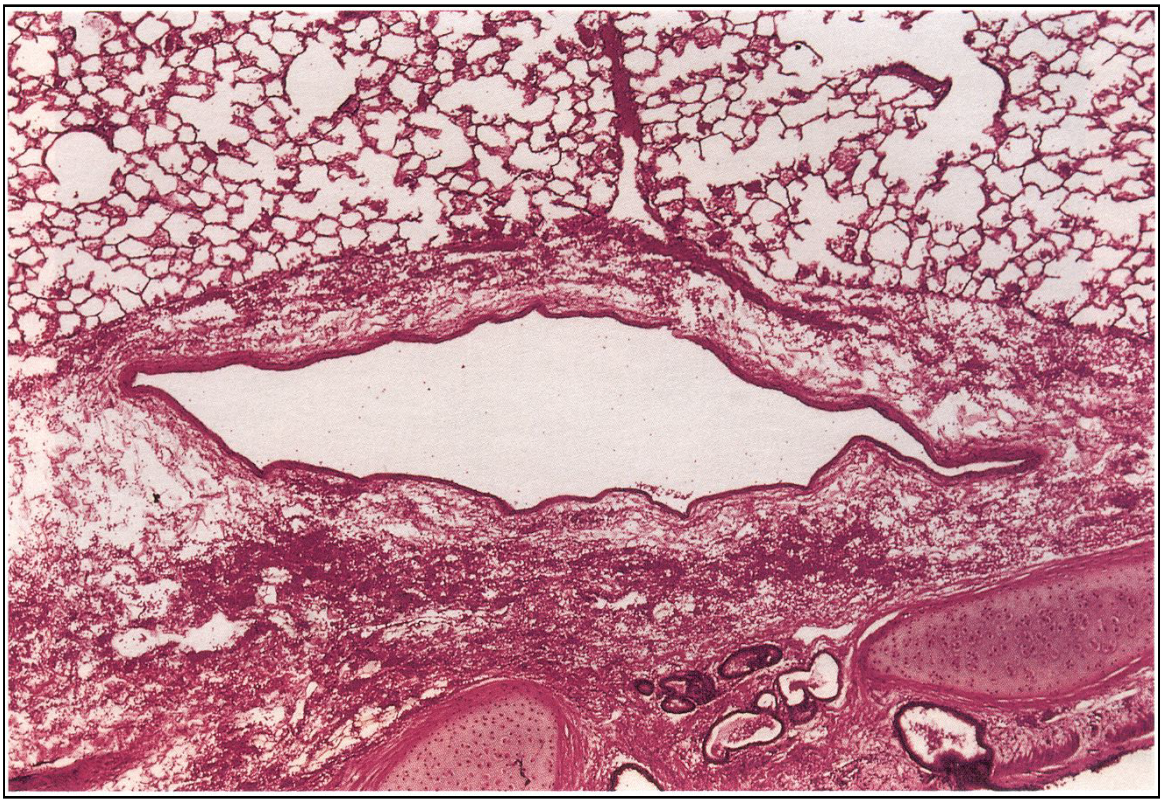

Fig. 8-7. In this histological section of lung parenchyma from a sheep exposed to a single blast, note the hemorrhage within the connective tissue that surrounds this blood vessel. This phenomenon is called ring hemorrhage.
Source: Walter Reed Army Institute of Research

diate death in blast victims, and their generation and pathophysiological effects are discussed in the next section of this chapter.)

A third site of pulmonary hemorrhage is in the thin interstitial band that surrounds vascular and airway structures. When damaged by blast, the interstitium may be filled with erythrocytes and plasma, forming hemorrhagic bands of erythrocytes surrounding airways and blood vessels. These are often called *ring hemorrhages.* They may occur far from the foci of alveolar hemorrhage, and thus they appear to develop independently. This site is particularly vulnerable to hemorrhage because of the difference in density between *(a)* the relatively rigid airways and blood vessels and *(b)* the fine meshwork of surrounding connective tissue and alveolar septa. When the bronchovascular structures (which have more inertia) resist displacement, the surrounding connective tissues are stretched and some of the capillaries are broken, with resultant hemorrhage (Figure 8-7).

Although blast researchers would like to be able to correlate the amount of hemorrhage with other blast-induced changes (such as alterations of the lung's physiological parameters, the presence of air emboli, or even mortality), the amount of hemorrhage within the lungs is difficult to quantitate. The most common way to indicate the extent of blast-related lung hemorrhage is by measuring the increase in lung weight after blast exposure. In experimental animals, the increase in gross lung weights is proportionate to the intensity of the blast, and is caused primarily by hemorrhage.

To standardize lung weights for various species and sizes of experimental subjects, the lung weight is commonly expressed as a percentage of the whole-body weight. Most uninjured lungs can be expected to fall within the normal range, usually 0.6%–1.2% of the whole-body weight. After blast exposure, lung-weight percentages that are higher than those in the normal range indicate the presence primarily of hemorrhage, with edema fluid and congestion (the pooling of blood within pulmonary blood vessels) being much less significant. In the larger proximal airways, only a small amount of blood-tinged froth (indicating hemorrhage) will typically be present; most of the hemorrhage will be in the lung parenchyma, especially in the dependent distal lobules, suggesting a gravitational

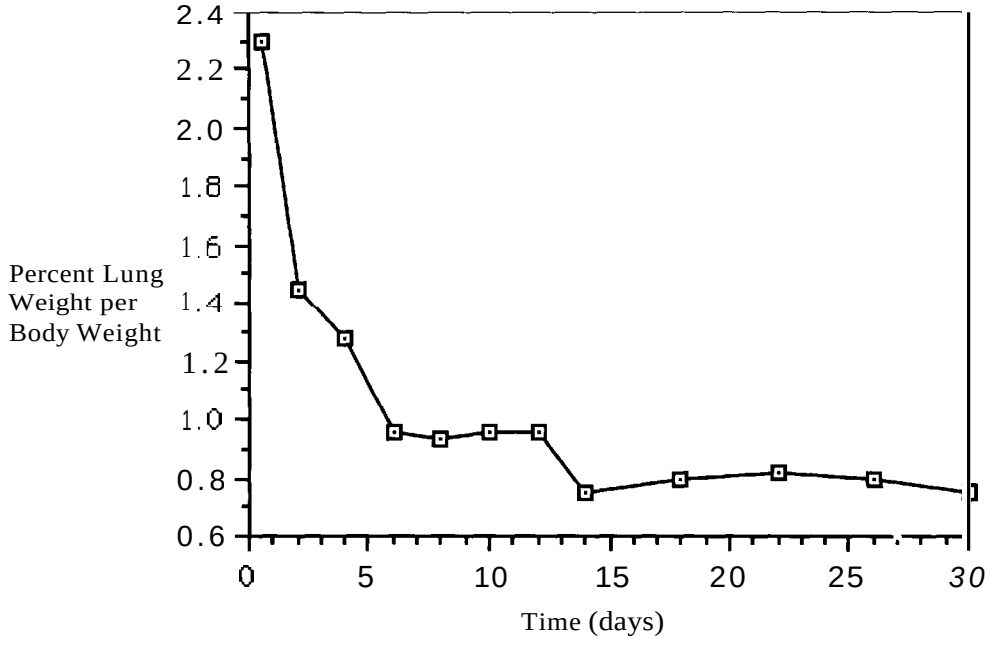

Fig. 8-8. Lung weight (expressed as a percentage of body weight) plotted against the time after a blast
Source: Redrawn from reference **9**

pooling of blood.

The lung weight reaches its peak almost immediately after the blast (Figure 8-8) and returns to a near-normal level several days later. Similarly, most histological evidence of hemorrhage will be gone by the sixth day after the blast. However, laden alveolar macrophages will remain and may not be cleared for several weeks. Months after a blast exposure, experimental animals have exhibited multifocal fibroplasia around alveolar ducts.[9] These residual scars are the only evidence that the animal was ever exposed to a blast.

The extent of parenchymal hemorrhage is an important determinant of mortality (Figure 8-9). The hemorrhage within the acini may lead to increased mortality from suffocation in some subjects because it reduces the surface area that is available for gas exchange. However, the connection between parenchymal hemorrhage and increased mortality may more likely be explained by the fact that a blast that produces more severed alveolar septa is likely also to produce more alveolovenous fistulae, and subsequently more air emboli. Most PBI-related deaths are caused by air embolism, rather than by hemorrhage.

Emphysema. When the alveolar septa tear, the alveoli themselves coalesce, producing giant air spaces and causing pulmonary emphysema. Subpleural cysts are formed if the air accumulates near the surface of the lung and the pleura If the pleura ruptures, a pneumothorax may occur.

Pulmonary Edema. Some blast researchers believe that diffuse edema is a significant component of PBI.[15,16] In theory, the same blast forces that sever alveolar septa and cause hemorrhage in the regions that are subject to the greatest distortion may also compromise the functional integrity of the endothelial-epithelial fluid barrier of the alveolar walls throughout the lungs. Then, although the barrier would remain intact enough to prevent erythrocytes from leaking, plasma would escape, producing alveolar edema and resulting in a condition that would, if the edema were diffuse, be identical to *adult respiratory distress syndrome* (ARDS). ARDS refers to the clinical syndrome that is evident when noncardiogenic pulmonary edema results from diffuse damage to the alveolar wall. It does not connote a specific etiology,

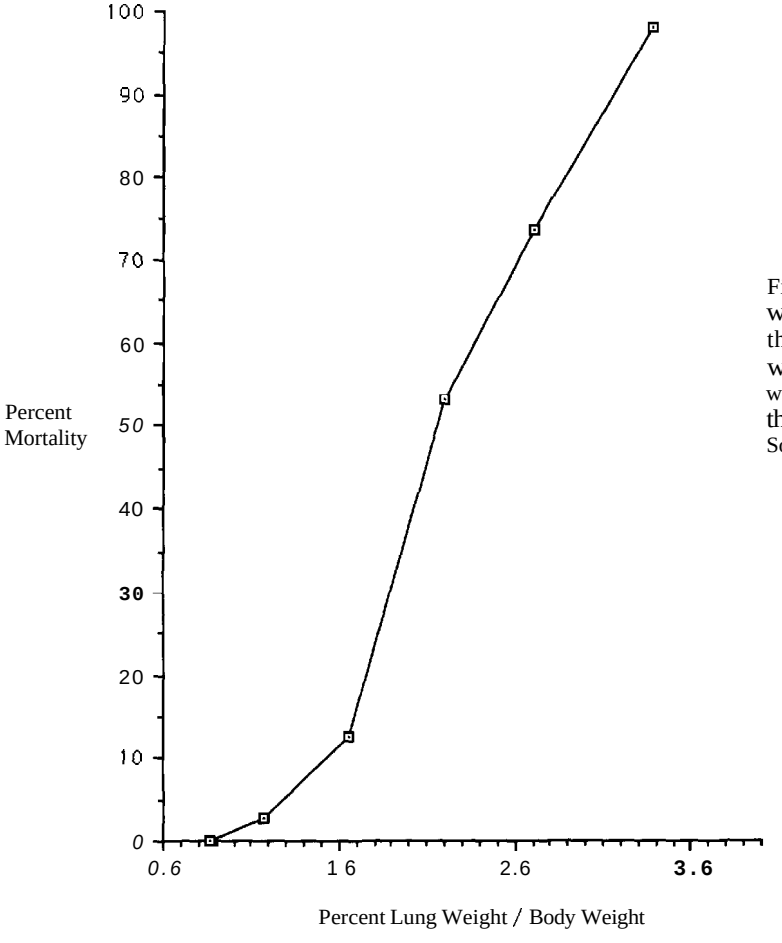

Fig. 8-9. In a study of 556 guinea pigs that were exposed to fast-rising overpressures that lasted 6–8 seconds, the animals' lung weights (expressed as percentages of their whole-body weights) were proportionate to their mortality.
Source: Redrawn from reference 9

but rather has been associated with a variety of causes, such as *(a)* oxygen toxicity, *(b)* pneumotoxicity from substances like paraquat, and *(c)* septic or traumatic shock.

However, most blast studies do not indicate that this kind of widespread pulmonary edema exists.[9,10] One study, for example, estimated the amount of pulmonary edema by measuring the amount of fluid delivered to a cannula placed in the caudal mediastinal lymph node of a sheep.[17] Blast doses that produced pleural petechiae did not increase the amount of fluid in the cannula, indicating that the permeability of the pulmonary capillaries was not significantly increased. In other studies, animals that were exposed to higher blast doses did not show significant alveolar edema, and histological sections of sites away from the foci of hemorrhage were devoid of fluid accumulation.[9,18]

Some blast effects (such as hemorrhage) are obviously localized, but other effects (such as edema), which some investigators may have assumed to be diffuse, may be localized as well. The same mechanistic aspects of blast injury that are responsible for the multifocal nature of lung hemorrhage may also cause edema that is usually localized in the same regions as the hemorrhage. In animal studies, for example, edema has been seen in the hemorrhagic regions of the lung parenchyma at autopsy (Figure 8-10).

Blast casualties in whom death is delayed may show diffuse alveolar edema at autopsy, which could be explained by *(a)* the presence of multifactorial cardiopulmonary distress or *(b)* therapeutic interventions, such as fluid resuscitation or mechanical ventilation.

Stripped-Epithelium Lesions. The tissues that line the larger conducting airways within the lungs are not immune from the consequences of the passing blast wave.

The most commonly reported lesion in airways is a *stripped-epithelium lesion,* which results when bronchial or bronchiolar epithelium is stripped from the underlying basal lamina (Figure 8-11). When these lesions are severe, they may manifest as ulcerations

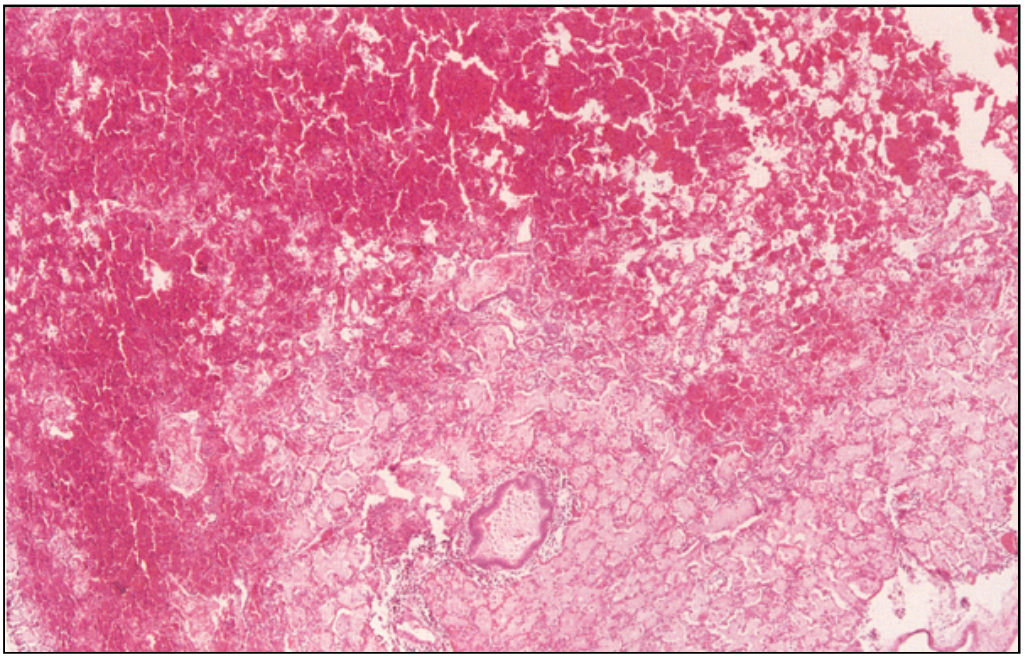

Fig. 8-10. In this histological section of lung parenchyma from a sheep exposed to a single blast, note the large area of alveolar edema (lower right) adjacent to the area of alveolar hemorrhage (upper left).
Source: Walter Reed Army Institute of Research

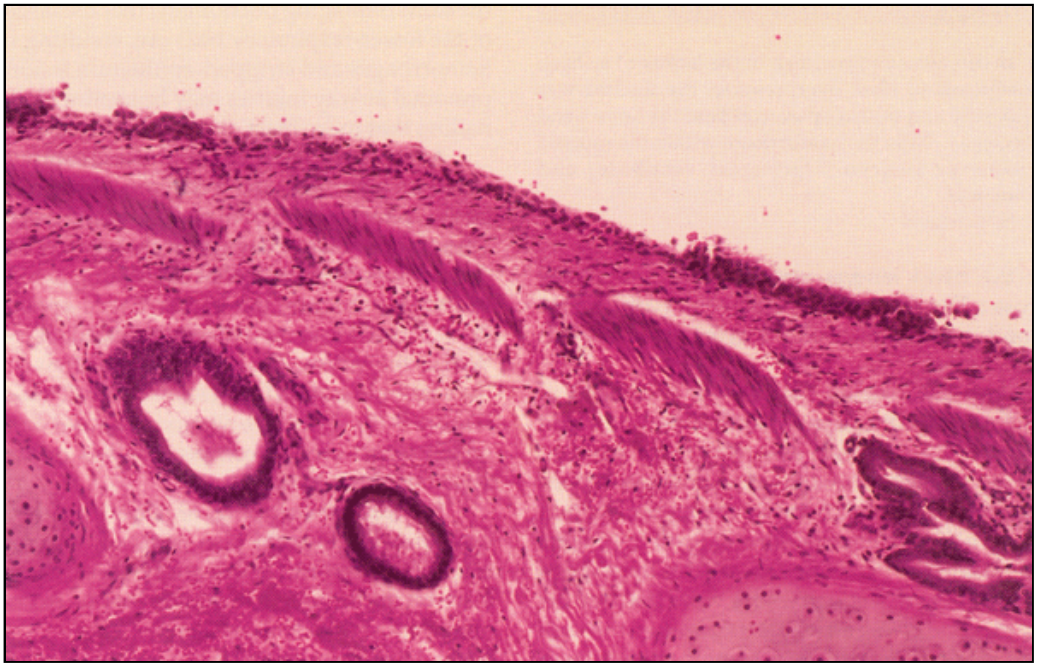

Fig. 8-11. In this histological section of lung parenchyma from a sheep exposed to a single blast, note the loss of pseudostratified ciliated epithelium from the bronchiolar surface and the hemorrhage in the underlying submucosa.
Source: Walter Reed Army Institute of Research

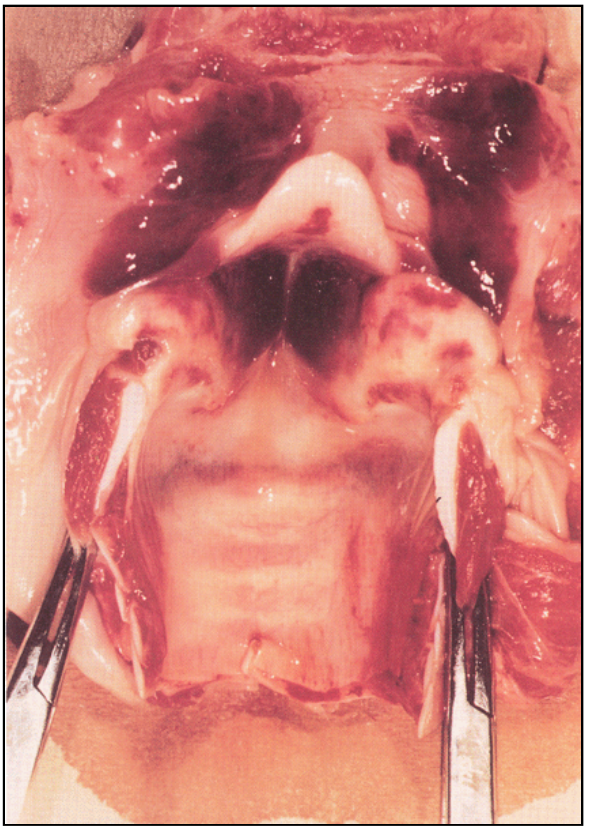

Fig. 8-12. In this autopsy specimen of the larynx of a sheep that was cxposcd to blast ovcrpressurc, the trachea was bisected dorsally and pulled apart to expose the larynx and proximal trachea. Note the hemorrhage within the mucosa of the posterior pharynx, laryngeal vestibule, and aryepiglottic fold.
Source: D. R. Richmond

through the basal lamina and into the underlying submucosa.[9] The hemorrhage that results contributes to the blood and fluid that collects in the distal airways and parenchyma.

Although the injuring mechanism is difficult to determine, one hypothesis suggests that spalling might be responsible for epithelial lesions in these airways. In iioiiliving materials, syalling occurs when a pressure wave passes through material of one density into material of a different density, throwing off fragments at the interface of the two materials.[19] If this hypothesis is applied to living tissue, a pressure wave that hits the tissues of the conducting airway is transmitted through the denser supporting cartilage or smooth-muscle layer before it reaches the less-dense attached mucosa. At the interface of the two, according to this hypothesis, spalling causes fragmentation of the mucosa. Although the applicability of this hypothesis to living tissue has never been validated, blast researchers have seen multifocal stripping of epithelium from the basal lamina. A more current explanation of injuring mechanisms in the respiratory system is discussed in some detail in Chapter Seven.

Regardless of the precise nature of the injuring mechanism, these lesions are caused by the blast's distortion of the airway, which stretches the mucosal layer and results in both the stripping of the epithelium **and** the rupture of small submucosal blood vessels.

Stripped-epithelium lesions can occur even when blast dose levels are below those associated with parenchymal hemorrhage, particularly if exposures are repeated.[18] In fact, these lesions may represent the most significant type of respiratory-tract injury following low-intensity blast doses. The denudation of airways and the loss of portions of the mucociliary apparatus may significantly inhibit the ability of the lung to clear particulate material, thereby creating an environment that is conducive to secondary infections after blast injury.

The Upper Airways

The upper-respiratory passages are vulnerable to the same damaging phenomena that the large airways of the lower-respiratory tract are, resulting in similar hemorrhages and stripped-epithelium lesions. These proximal airway injuries may be sentinel injuries, signalling that more blast damage has occurred distally.

Autopsy studies reveal multifocal submucosal and mucosal petechiae and ecchymoses in the trachea, larynx, pharynx, and both paranasal and nasal sinuses. These hemorrhages have been shown to occur at blast doses lower than those that cause lung hemorrhage.[20] Within the trachea, the hemorrhages appear at random or in a transverse pattern over cartilaginous rings.[9] Within the larynx, the hemorrhages often occur over the vestibule and on the posterior face of the epiglottis (Figure 8-12). There are no reported cases or studies in which upper-airway competence has been compromised by these lesions.

The loss, or stripping, of surface epithelium that occurs in the bronchi also commonly occurs in the trachea and larynx. In one study, researchers used an electron microscope to examine lesions from rats that were exposed to 20 repetitions of blast.[21] The lesions ranged from *(a)* a flattening of epithelial cells without cell loss to *(b)* the focal loss of epithelial cells with disruption of the basal lamina. When ulceration occurred, the defects were covered by fibrinocellular

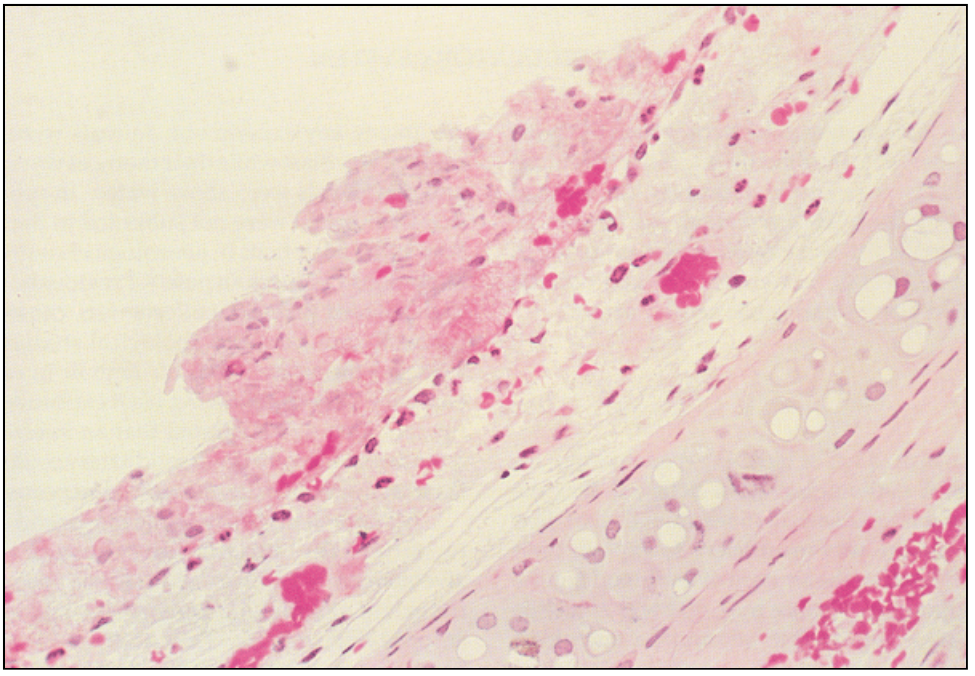

Fig. **8-13.** In this histological section of the trachea from a rat that was exposed to twenty repetitions of blast at 27.5 psi on *2* consecutive days, the epithelium is focally flattened and partially denuded, with a fibrinocellular clot overlying the area of injury.

clots with admixed leukocytes (Figures 8-13 and 8-14). These lesions first appeared at or below the threshold blast dose for hemorrhage within the rats' lungs. Similarly, sheep that were exposed to fifty repetitions of blast overpressure developed tracheal lesions at peak pressures of 5 psi, although they did not develop significant lung hemorrhages until peak pressures exceeded 15 psi.[18]

The protective mucociliary apparatus extends from the larynx to the terminal bronchioles. It is made up of the cilia of the epithelial cells, which continuously carry mucus from the lungs to the pharynx. The stripped-epithelium lesions of the trachea are similar to those in the conducting airways within the lung (that is, the bronchi and bronchioles), and whether the blast wave's disruption of either of these portions of the protective mucociliary apparatus is significant has yet to be determined.

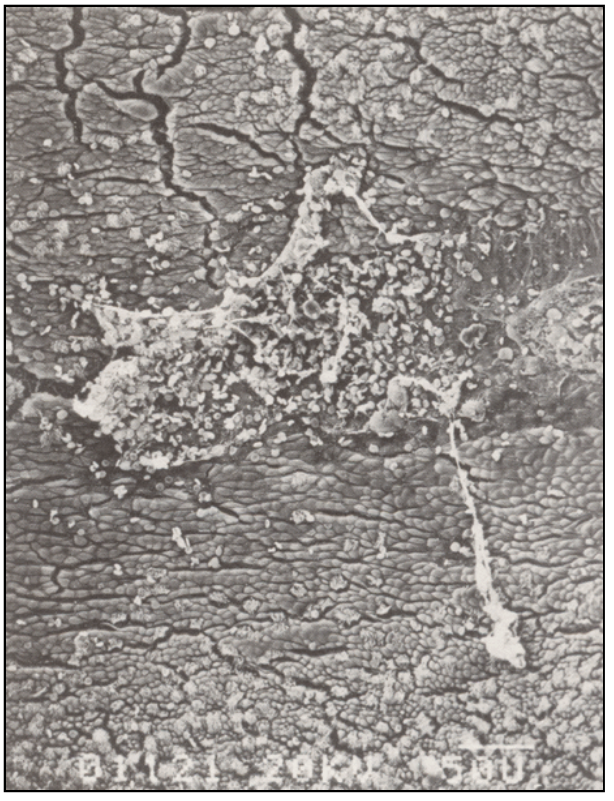

Fig. 8-14. In this scanning electron micrograph of the tracheal epithelium from a rat that was exposed to twenty repetitions of blast at 22.5 psi, the focal denudation of the epithelial lining is covered by a fibrinocellular clot.
Source: Walter Reed Army Institute of Research