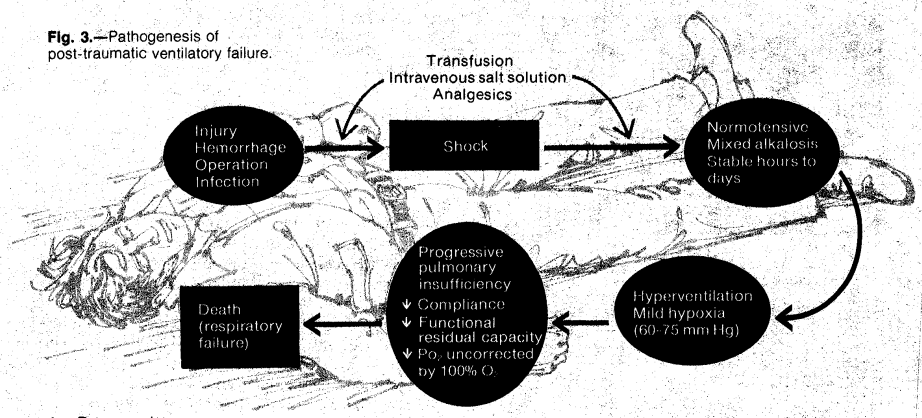
Fig. 3.—Pathogenesis of post-traumatic ventilatory failure.



↓ = Decreased

Adapted from Rogers, RM: Shock lung, or respiratory distress syndrome of adults. *Continuing Educ* 3:26, 1975.

by detecting free fat in the urine or increased activity of serum lipase. Early diagnosis may be lifesaving. Administration of heparin is recommended on the same basis as in thromboembolic disease.[4] Besides increasing blood flow through the pulmonary capillaries, heparin may help to reduce the aggregation of chylomicrons and fat globules so that they can be transported through the pulmonary bed.

As the fat embolism breaks down, it produces a *second* stage of pulmonary involvement in the form of exudative pneumonia. The latter results from a reaction of the pulmonary parenchyma to the products of lipid breakdown.[2] Even though x-rays clearly reveal exudative pneumonia (Fig. 2b), physical examination of the patient may be remarkably unrevealing, since the pulmonary involvement is primarily interstitial. Arterial hypoxemia usually is present and often progressive. Pulmonary function tests reveal a marked diffusion block, ie, alveolar-capillary block due to an inflammatory response. The latter may be controlled by administering massive doses of steroids which purportedly stabilize the cell membrane and thereby lessen the release of lysosomal enzymes. However, if the condition progresses, a decrease in pulmonary compliance and the closure of small airways leads to progressive hypoxia which, together with the oxygen-diffusion block, results in arterial oxygen desaturation. This condition is extremely difficult to treat, even by controlled mechanical ventilation. Some benefit may be obtained by administering concentrated oxygen and continuous positive end-expiratory pressure.[12]

Low-Flow Lung Syndrome

During the Vietnamese war, casualties were rapidly evacuated from the battlefield to medical units at which they were given resuscitative as well as definitive care by surgeons and paramedical personnel. This led to the highest ratio of survivors to fatalities documented during wartime. But a new clinical problem then became apparent: after successful resuscitation from shock, some of the wounded died of pulmonary complications 4-7 days following injury—despite effective ventilatory support. Often patients without chest injuries—but with extensive injuries to other parts of the body—were affected. This lethal syndrome of post-traumatic pulmonary failure (shock lung or low-flow lung) is today well recognized as a potential complication of any severe injury that leads to shock.

Moore and coworkers[9] classified the pathophysiologic phases of post-traumatic ventilatory failure as follows:

Phase I: Injury, resuscitation, and alkalosis
Phase II: Circulatory stabilization and beginning of respiratory difficulty
Phase III: Progressive pulmonary insufficiency
Phase IV: Terminal hypoxia with hypercarbia and asystole

The lethal progression from injury through resuscitation to the state of terminal hypoxia is illustrated in Figure 3.

The most important diagnostic clue to low-flow lung is a history either of shock or low cardiac output following trauma. Pulmonary complications may follow any condition that leads to low cardiac output,