Shock Lung

A Distinctive Nonentity

BY THE CLOSE of World War II, a syndrome of “wet lung” had been identified in which life-threatening respiratory distress unexpectedly interrupted convalescence from hemorrhagic and traumatic shock incurred during combat. During the recent war in Vietnam, as the salvage rate after circulatory collapse on the battlefield increased, the syndrome became even more familiar, but under new eponyms. Thus, “wet lung,” “shock lung,” or “DaNang lung” became synonyms for lung failure that followed successful resuscitation from circulatory collapse.

But no matter what it was called, the sequence was the same: severe nonthoracic injury, blood loss, and hypotension during combat; successful resuscitation using tourniquets, transfusions, and opiates on the battlefield; prompt evacuation to a sophisticated medical facility for more deliberate management; and then, a few days later, the calamitous interruption of convalescence by progressive respiratory distress and failure.

Only a few who reached the hospital developed shock lung, but in those who did, the pattern of evolution was consistent: insidious onset of rapid shallow breathing, breathlessness, and productive cough; rales and wheezes; refractory cyanosis. X-ray appearance of enlarging interstitial and alveolar infiltrates continued to extend and to coalesce until the entire lung was enveloped in a diffuse haze. Enriched oxygen mixtures and assisted ventilation became less and less effective in achieving tolerable levels of oxygenation. Finally, death resulted from respiratory insufficiency often complicated by the recurrence of circulatory collapse. And, at autopsy, a stereotyped morbid anatomy: vascular congestion, interstitial and alveolar edema, and focal atelectasis (“congestive atelectasis”). Other anatomic changes were common but not quite as consistent: hemorrhage in the interstitial spaces and alveoli, vascular thrombi, fat emboli, fibrin deposits, and hyaline membranes.

The predominant physiologic derangements were as anticipated from the clinical picture and from the pathology at autopsy, i.e. reduced compliance and arterial hypoxemia. The reduced compliance reflected the stiff lungs; whether the stiffness was primarily due to congestive atelectasis or to loss of surfactant was, and remains, enigmatic. The progressive arterial hypoxemia (without hypcapnia) was a consequence of increasing “venous admixture” as blood flow continued through parts of the lungs which became functionless in gas exchange.

Unfortunately, insights into pathogenesis did not keep pace with the clarification of the clinical syndrome and the morbid anatomy. Consequently, many of the links between injury and resuscitation on the battlefield and respiratory insufficiency during convalescence have remained speculative. Some believe that hypoperfusion of parts of the lungs during the hypotensive episode damages the lung so that it leaks after the circulation is restored; more specifically, they propose interference with the production of alveolar surfactant, or an upset in the balance between local metabolism and blood flow in the lung, or selective injury to lung vessels by toxic substances from afar. On the other hand, many doubt that the hypotension after blood loss and trauma is, per se, sufficient explanation; they invoke complications, including endotoxinemia, fat emboli, release of injurious fatty acids and proteolytic agents, discharge of leukocyte lysosomes in the pulmonary capillaries, and disseminated intravascular coagulation or microemboli.

No explanation for the origins of “DaNang lung” is entirely convincing. Most hypotheses include the period of pulmonary hypoperfusion during the episode of circulatory collapse as an important, possibly central, initiating mechanism. However, great uncertainty persists concerning the augmenting influences which convert a transient episode of diminished pulmonary blood flow into a respiratory disaster. Most disconcerting in this regard is the

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grim prospect that the lifesaving therapeutic measures may be a watershed for subsequent respiratory distress. Thus, on the battlefield, overzealous administration of liquids, particularly of crystalloidal solutions, predisposes to pulmonary congestion and edema; thrombi in bank blood may embolize the lungs, thereby contributing to congestive atelectasis. Even in the hospital, where respiratory distress characteristically interrupts an otherwise uneventful convalescence, iatrogenic influences may become operative—injudicious administration of enriched-oxygen mixtures may add to the lung damage; oversedation may predispose to alveolar hypoventilation and atelectasis. Once the syndrome is full blown, distinctions between cause and effect tend to blur.

Military physicians, faced with the formidable problem of acute respiratory distress which they could describe but whose origins they could not explain, naturally resorted to empirical measures. Drawing heavily on a burgeoning civilian experience with acute respiratory distress in intensive care units, parallels were quickly uncovered between the “shock lung” in Vietnam and the variety of “wet lungs,” “stiff lungs,” and “pump lungs” in the nonmilitary hospitals. Furthermore, as the designation “shock lung” became further and further removed from its origins in hemorrhage and hypotension, it blended into the mélange of “adult respiratory distress syndromes” in which clinical, X-ray, and autopsy features, rather than pathogenetic routes, are the common denominators. The management of shock lung then became that of the adult respiratory distress syndrome, i.e. volume-cycled respirators, positive pressure breathing throughout the respiratory cycle, judicious oxygen therapy, and a wide variety of pharmacologic agents, some of dubious value.

Although it is difficult to quantify the contribution of intensive care to recovery from the various adult respiratory distress syndromes, few experienced observers would deny that many lives have been saved by the standardized approach to improving the gas-exchanging functions of the stiff lungs. On the other hand, it does seem reasonable that this record could be considerably improved if candidates for respiratory distress could be identified for therapeutic intervention soon after the stage of circulatory collapse rather than during the stage of irreversible pulmonary dysfunction.

Important steps in this direction have already been taken. Some have helped to clarify the role of pulmonary hypoperfusion. For example, respiratory distress may terminate a sequence beginning either with hemorrhagic or cardiogenic shock. Both are characterized by systemic hypotension and pulmonary hypoperfusion, but because of differences in the levels of left atrial pressure they differ strikingly with respect to the pattern of distribution of the pulmonary blood flow. Indeed, “shock lung” after cardiogenic shock represents pulmonary edema from left ventricular failure complicated by the consequences of desperate resuscitative efforts as the lungs grow stiffer and arterial hypoxemia intensifies. On the other hand, if pulmonary hypoperfusion is involved in the pathogenesis of “shock lung” after hemorrhage it must operate in a different way since left ventricular failure (and pulmonary venous hypertension) cannot be implicated. “Shock lung” after septic shock must have entirely different origins since pulmonary blood flow may remain high throughout—from the stage of sepsis, through the stage of shock, to the advent of respiratory distress. Finally, it is difficult to implicate abnormal pulmonary hemodynamics of any sort in instances of adult respiratory distress which do not include an episode of systemic hypotension in their evolution.

Leaky pulmonary vessels, often in conjunction with abnormal pulmonary hemodynamics, could contribute to the pathogenesis of “shock lung.” The prototype is septic shock in which bacterial products released at the site of sepsis reach the lungs to modify the permeability, as well as the caliber of lung vessels, and to damage lung cells. Noxious agents reaching the lungs are, however, not unique to septic shock. Remote organs and tissues, damaged and inflamed by a variety of agents and mechanisms, may contribute injurious by-products for the circulation to transmit to the lungs. These may not only act directly but may act by injuring blood constituents, such as platelets, or by activating blood systems, such as that which produces kinins, to modify vascular permeability within the lungs. Even the “shock lung” after hemorrhage may involve seepage into the circulation of endotoxins from ischemic bowel so that a combination of pulmonary hemodynamic and permeability factors enter into its pathogenesis.

The prospect of different pathogenetic routes to the same final clinical and anatomic picture gives pause to the current practice of homogenizing “shock lung” into the mix of “adult respiratory distress syndromes.” Would it not be preferable to retrieve “shock lung” for the respiratory distress syndrome
that relates to hemorrhagic shock on which we have a half century of clinical and experimental observations? Perhaps we could then identify the contribution of nonthoracic trauma to the picture of "DaNang lung." Isn't "wet lung" most applicable to the respiratory distress syndrome after myocardial infarction even though interstitial and alveolar edema may play important roles in other types of respiratory distress? Perhaps "pump lung" will prove to differ from the "wet lung" after myocardial infarction even though it may share the hemodynamic abnormalities of pulmonary hypoperfusion and high left atrial (and pulmonary venous) pressures? Nor is there obvious advantage, other than expediency for emergency care, in losing sight of oxygen toxicity or gastric aspiration or acute pancreatitis as distinct etiologic entities which, if uncontrolled, converge upon the final syndrome of respiratory distress.

This separatist approach should not be misinterpreted as an aspersion on the success of intensive care for respiratory distress syndromes in which the stiff lungs and abnormal gas exchange are the center of attention. But it would be misconstrued if it failed to distinguish between management based on pathophysiologic insights concerning end-stage disease versus management based on pathogenetic understanding of disparate sequences that end the same way.

Indeed, it may even be questioned if they all end the same way despite the final label of "congestive atelectasis." After cardiogenic shock, pulmonary edema predominates. In other disorders, edema may play a lesser role. In some disorders, interstitial edema is the first detectable morphologic disturbance. Indeed, "congestive atelectasis" provides no more than a list of principal pathologic components, making no commitment concerning the degree of edema, congestion, atelectasis, thrombosis, pneumonia, emboli, or other morphologic abnormalities.

In essence, ambiguity that hampers both understanding and treatment characterizes the clinical and pathologic descriptions of the various respiratory distress syndromes. This confusion is apt to grow in the years ahead since opportunities for developing these syndromes in civilian life are multiplying—more and more vehicular accidents, increasing salvage from circulatory collapse of patients who previously would have died, and proliferation of mechanical bypass devices in cardiac surgery. Would it not be reasonable to encourage the dissection of the adult respiratory distress syndromes according to etiologic entities, each with its own pathogenesis and natural history, so that clinicians will find it easier to compare bedside observations, physiologists will be encouraged to produce meaningful experimental replicas of the human disorders, and pathologists will be abetted in their efforts to relate structural abnormalities to antecedent functional derangements?

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References

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