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Discussion

Dr. Watts R. Webb (Dallas): I think these studies further establish that the lung is one of the most vulnerable end organs in protracted shock, or, in fact, even in acute stages of low flow states. Dr. Sugg, working in our laboratory with hemorrhagic shock, and Dr. Baxter and Dr. Cook, working with burn shock, have shown changes somewhat different from those Dr. Clowes has shown you in his microscopic slides. The acute changes consist of congestive atelectasis, with collapse of the lung, red cell infiltration, early leukocytic infiltration, and alveolar filling with red cells.

This is not a bacteremic phenomenon, in the acute stages at least, because this picture can be produced in the completely sterile situation after 2 hours of hemorrhagic shock at a pressure of 40 mm. Hg, which is not a very severe shock preparation. The question of whether or not this is heart failure, we believe, has been ruled out, because the heart is very resistant to shock, and fails only in the very late stages. Hemodynamic studies reveal that left atrial pressures throughout this period are always low, rather than high.

Catheterization of the pulmonary circulation from both sides (that is, both the venous and the arterial) confirms the work of Dr. Al Hyman, of New Orleans, and Dr. Oscar Creech, to demonstrate that there is no constriction of the pulmonary arteries or arterioles, or even the arterial wedge position. Instead, there is a constriction of the pulmonary small veins—not the large veins, but the small veins, and not of the pulmonary capillary bed.

In addition, it is not a sludging phenomenon, because ordinarily, in the normal lung, there is an active recruitment of only a very small proportion of the pulmonary capillary bed. However, in this phenomenon virtually all of the capillary bed is very widely dilated, as can be demonstrated by dye or microscopic studies. In attempts at prophylaxis of this, we have not found that atropine or acetylcholine or cortisone or the antihistaminics or vagotomy or hilar stripping have been of any value in protecting the lung against the development of congestive atelectasis in shock.

The only way we have been able to prevent this is by resecting the lung and reimplanting it; the reimplanted lung does not develop the picture of congestive atelectasis, as does the contralateral lung. This suggests to us that this is a neurally mediated phenomenon, and not mediated (at least acutely) by the vascular end products of end metabolism.

Again, I congratulate Dr. Clowes on a very excellent study. These studies will be of great help in the further treatment of our patients.

Dr. George H. A. Clowes, Jr. (Closing): I would like to thank Dr. Webb for his discussion of the shock aspects of this subject. While I was discussing sepsis, time did not permit the discussion of the details concerning the slide which I presented on the shock picture. It shows exactly the difference that Dr. Webb mentioned between wedge pressure and left atrial pressure. We think this may have something to do with the phenomenon of red cell accumulation.

At present I can not say whether red cell congestion of the septa is due to shut-off of vessels and skimming; whether it is due to ischemia of parts of the lung in the low flow state, or to the formation of clots and thrombi, as has been suggested by Colonel Hardaway. Further, it is possible that this "red cell packing" is related to changes in membrane charges on the surface of cells and vessels. If so, this could represent the effect of a circulating agent released by deteriorating tissue or killed bacteria.

The fact remains that we see this red cell packing and hemorrhage in the septic states as well as in shock. The one thing that has come out of this study, in my mind, is that we are looking at a common phenomenon. It is the lung's reaction to this kind of stress that is common to shock, to sepsis, and to trauma.