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Surfactant deactivation in a pediatric model induces hypovolemia and fluid shift to the extravascular lung compartment.

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Abstract

BACKGROUND:

Surfactant deficiency is the pivotal abnormality in Neonatal and Acute Respiratory Distress Syndrome. Surfactant deactivation can produce hypoxemia, loss of lung compliance, and pulmonary edema, but its circulatory consequences are less understood.

OBJECTIVE:

To describe the sequential hemodynamic changes and pulmonary edema formation after surfactant deactivation in piglets.

METHODS:

Surfactant deactivation was induced by tracheal instillation of polysorbate 20 in 15 anesthetized and mechanically ventilated Large White piglets. The hemodynamic consequences of surfactant deactivation were assessed at 30, 120, and 240 min by transpulmonary thermodilution and traditional methods.

RESULTS:

Surfactant deactivation caused hypoxemia, reduced lung compliance, and progressively increased lung water content ($P < 0.01$). Early hypovolemia was observed, with reductions of the global end-diastolic volume and stroke volume ($P < 0.05$). Reduced cardiac output was observed at the end of the study ($P < 0.05$). Standard monitoring was unable to detect these early preload alterations. Surprisingly, the bronchoalveolar protein content was greatly increased at the end of the study compared with baseline levels ($P < 0.01$). This finding was inconsistent with the notion that the pulmonary edema induced by surfactant deactivation was exclusively caused by high surface tension.

CONCLUSIONS:

Hypovolemia develops early after surfactant deactivation, in part due to the resulting fluid shift from the intravascular compartment to the lungs.