Introduction:
Hemodynamic resuscitation by means of fluids and norepinephrine (NE) is currently considered as a cornerstone of the initial treatment of septic shock. However, there is growing concern about the side effects of this treatment. The aim of this study was to assess the relationship between the hemodynamic resuscitation and the development of the ARDS.

Methods:
18 New Zealand rabbits. Animals received placebo (SHAM=6) or lipopolysaccharide (LPS) with or without (EDX-R, n=6; EDX-NR, n=6) hemodynamic resuscitation (fluids: 20 ml/kg of Ringer’s lactate; and later NE infusion titrated up to achieve their initial arterial pressure). Animals were monitored with an indwelling arterial catheter and an esophageal Doppler. Respiratory mechanics were continuously monitored from a side-stream spirometry. Pulmonary edema was analyzed by the ratio between lung wet and lung dry weight (W/D), and the histopathological findings.

Results:
SHAM group did not show any hemodynamic or respiratory changes. The administration of the LPS aimed at increasing cardiac output and arterial hypotension. In the LPS-NR group, animals remained hypotensive until the end of experiment. Infusion of fluids in LPS-R group increased cardiac output without changing arterial blood pressure, while the norepinephrine reversed arterial hypotension. Compared to the LPS-NR group, the LPS-R group had more alveolar neutrophils and pneumocytes with atypical nuclei, thicker alveolar wall, non-aerated pulmonary areas and less lymphocyte infiltrating the interstitial tissue. In addition, the airway pressure increased more in the group LPS-R, and the W/D, although slightly higher in the LPS-R, did not show significant differences.

Conclusion:
In this model of experimental septic shock resuscitation with fluid bolus and norepinephrine increased cardiac output and normalized blood pressure but worsened lung damage.