

TITLE: PULMONARY INJURY IN THE PIG LUNG PERFUSED WITH CARDIOGENIC SHOCK BLOOD

AUTHORS: Toung, T.J.K., M.D., Sendak, M., M.D., Grayson, R., M.D., Rosenfeld, B., M.D., Porta, J., M.D., Susman, M., M.D., Bulkley, G., M.D., and Traystman, R.J., Ph.D.

AFFILITATION: Department of Anesthesiology and Critical Care Medicine and Department of Surgery, Johns Hopkins Medical Institutions, Baltimore, Maryland 21205.

Introduction: Adult respiratory distress syndrome (ARDS) is a non specific response to a variety of insults among which hemorrhagic hypotension has been reported frequently. A variety of explanations have been advanced including ischemic injury to pulmonary parenchyma. However, recent evidence suggest that ischemic injury to pancreas during hypotension and the subsequent liberation of pancreatic enzymes into systemic circulation to be the cause of lung injury. Blood from hypotensive animals could therefore be expected to cause lung damage if transfused into normotensive animals. We investigated the effects of perfusing normal pig lungs in situ with blood from other animals previously subjected to prolonged hypotension.

Methods and Materials: Eleven 20-25 kg pigs of both sexes were anesthetized with Ketamine 10 mg/kg IM and 30 mg/kg sodium pentobarbital IV. Tracheostomies were performed and animals ventilated with room air. The femoral artery was cannulated and the animal exsanguinated following heparinization. The mediastinum was opened with care taken not to enter the pleural cavity. The pulmonary artery and left atrial appendage were cannulated. Perfusion of the lungs was established with autologous blood at a flow of 20 ml/kg/min with a Sarns roller pump. This rate was kept constant throughout. A Swank micro filter (20 u) was placed in the arterial site of the perfusion circuit. Temperature of the perfusate was maintained at 38°C, and the lungs were now ventilated with a gas mixture containing 5% CO₂ and compressed air to maintain the PCO₂ of the perfusate between 35-45 torr. NAHCO₃ was added as needed to maintain pH between 7.35-7.45. After 30 minutes of stabilization, the lung perfusate of all animals was changed to heterologous blood. In the four control animals (group 1) the lungs were perfused with normal, unshocked heterologous blood. In the other seven animals that served as the study group (group 2), the lungs were perfused with heterologous blood from other animals (doner group) that had undergone 4 hours of cardiogenic shock followed by 4 hours of recovery. (Cardiogenic shock was produced in this doner group by infusing the pericardium with Dextran-40 to depress the cardiac output to 40% of the baseline output). Lung weight change was recorded as the inverse of weight of the perfusate reservoir. Lung weight change, perfusion pressure (Ppa), and airway pressure (Paw) were recorded at baseline and at 1,2,3, and 4 hours after perfusion with heterologous blood. Mean values between the groups were compared at each time interval by unpaired t-test. P<0.05 was considered significant.

Results: The changes in mean lung weight and Ppa are shown in figures 1 and 2 respectively. Lung weight was stable over time in the control group. The study group however, showed a statistically significant increase at all time intervals (P<0.05). The study group also showed a significant increase

in Ppa over the control group at the 1,2,3 and 4 hour intervals (P<0.05).

Discussion: Using an isolated, flow controlled porcine lung model, lung injury was induced by perfusion with blood from animals having recovered from shock. It appears that the injury was caused either by humoral or mechanical elements contained in the blood. Since the possibility of the injury induced by blood aggregates was eliminated, we speculate pancreatic enzymes to be the most likely agent. However, leucocyte and their metabolites could also be involved in the injury process. Further investigation is underway to determine the factor responsible for the injury.

