Title: ABSENCE OF HEMATOGENOUS MEDIATED PULMONARY INJURY WITH SMOKE INHALATION IN SHEEP

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Introduction. Inhalation is a serious clinical problem that increases the already high mortality associated with thermal injury.[1] The damage to the lung includes the parenchymal areas as well as the airways.[2] We have studied this in an ovine model and have determined that the injury is associated with deposition of polymorphonuclear cells in the pulmonary microvascular.[3] We propose that these cells are attracted to the parenchyma of the lung by the release of chemotactic factors from the tracheobronchial areas. We tested this hypothesis by creating an inhalation injury to one lung and evaluating the status of both lungs.

Methods. Eighteen sheep were prepared for study by implantation of catheters. Forty-eight hours after the recovery from the surgical procedure these animals were anesthetized with halothane and their lungs intubated with a modified Carlings tube. The lungs were then ventilated utilizing two Servo 600C ventilators which were slaved together. For the smoking procedure, the ventilator was disconnected from one side and the lung to be smoked insufflated with smoke from burning cotton material. Six animals had smoke put into the left lung and six in the right. In addition six animals were insufflated with air rather than smoke. The smoke was created with a bee smoker and insufflated into the sheep until a carboxyhemoglobin value of 50% was attained. Twenty-four hours after injury, hemodynamic variables were measured, the animals were sacrificed, and the lungs harvested for gravimetric measurement of extravascular lung water and histological evaluation. Lower lobes of each lung were isolated, a portion of each was lavaged and the cellular control of the lavage material evaluated.

Results. Following inhalation injury, the PaO_2 fell from 109±3 to 75±4 mm of mercury. This was accompanied by a change in shunt fraction from 10±1 to 30±3%. Extravascular lung water was elevated only in the smoked lung. The smoked lungs likewise showed histological evidence of alveolar edema with infiltration of polymorphonuclear cells. The lavage materials obtained from the smoked lungs contained an increased number of polymorphonuclear cells.

Discussion. Inhalation injury to one lung does not lead to damage to the contralateral unsmoked area, but is restricted only to the side of injury. The damage was associated with the infiltration of polymorphonuclear cells into the parenchymal areas as well as the lavage materials. Consequently, chemotactic substances must have been released by the injury. This chemotaxis is restricted to the injured lung in our present study.

This study was supported by National Institutes of Health Grant #GM33324 and the Shriners of North America.

	Bloodless Sham-Group Lungs	Smoke-Group Contralat. Lungs	Smoke-Group Injured Lungs
W/D	4.0 ± 0.15	4.3 ± 0.15	5.4 ± 0.32*
% PMN	30 ± 7	34 ± 6	55 ± 3*

Values are mean ± SE. * Significant difference by one way analysis of variance (p is less than or equal to 0.05). Bloodless wet-weight/dry-weight (W/D) ratios and PMN control of the lavage materials from sham-group, injured, and contralateral lungs measured hours after sham or smoking procedure.

References.

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