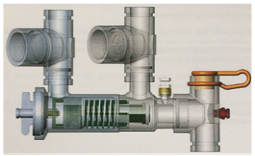

Clinical and pathophysiologic problems associated with smoke inhalation injury

[Article in Japanese]

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Smoke inhalation injury is one of the primary determinants of survival following major burn injury. The primary site of injury in smoke inhalation appears to be the small airway rather than the alveoli, and thus small airway occlusion caused by edema and pseudomembrane formation are the primary mechanisms of progressive hypoxia. Ventilation-perfusion (VA/Q) alterations after smoke inhalation are characterized by increased blood flow to low VA/Q compartments, although an increase in true shunt (VA/Q = 0) was not a consistent finding. This differs considerably from most adult respiratory distress syndrome (ARDS) patients or oleic acid-induced lung edema models, in which an increase in true shunt is the major mechanism of hypoxia. Such differences lead to different responses to nitric oxide (NO_2) inhalation therapy, and NO_2 does not improve oxygenation and outcome in patients with smoke inhalation injury. In the treatment of inhalation injury, meticulous removal of pseudomembrane by fiberoptic bronchoscopy is essential; the use of high concentrations of oxygen should be avoided since it can cause absorption atelectasis. High-frequency percussive ventilation is a suitable treatment for inhalation injury, as it improves oxygenation and facilitates removal of pseudomembrane.

PMID: 9547747 [PubMed - indexed for MEDLINE]

Nippon Geka Gakkai Zasshi - 1998 Jan;99(1):46-51.