題名:機械通氣導致肺損傷.

作者:林蕙鈴; 江玲玲; 施崇鴻; 楊式興; 蘇千玲;

貢獻者:呼吸治療學系

上傳時間:2009-08-24T03:32:56Z

摘要:機械性通氣已經廣泛運用在急重症病人維持其生命，機械性通氣具有許多潛在的併發症，最被深入探討的併發症－呼吸器導致肺損傷(Ventilator-induced lung injury, VILI)，VILI過去常被稱爲壓力性損傷(barotrauma)顯微鏡的觀察下，機械通氣引發肺泡滲透性增加，導致肺水腫及嚴重的細胞損傷。生化損傷"Biotrauma"是用來描述肺部細胞在重複的機械性換氣強迫性的擴張及塌陷或過度擴張之下，導致肺損傷並引發炎物質的釋放，經由循環引起全身性的發炎反應。而這種生化損傷的機轉，構成了急性呼吸窘迫症候群與多重器官衰竭(multiple organ failure, MOF)之間的橋樑。急性肺損傷時，肺泡的表面張力即失去維持穩定肺泡大小的功能，肺泡大小的劇烈變化以及大範圍持續重複性的肺泡及氣道塌陷與再開啓，所形成的剪力(shear force)會造成肺泡進一步的損傷。機械通氣導致肺損傷相關機轉包括：容積損傷(Volutrauma)、塌陷損傷(Atelectrauma)、生化損傷(Biotrauma)、壓力損傷(Barotrauma)、氧氣毒性效果(Oxygen toxicity)等，本文獻回顧主要探討其相關作用機轉，並討論如何預防機械通氣導致肺損傷。

Mechanical ventilation has been used in critical care for decades to save patients' lives in critical settings, but it has potential complications, such as ventilator-induced lung injury (VILI). Barotrauma is widely referred to as the VILI, but, with advanced microscopic technology, researchers have found that lung injury might also be the consequence of inflammatory response to mechanical ventilation. During normal gas exchange, alveoli structure is maintained by surface tension and connective tissues; however, when the lungs are injured, the surface tension loses its function of maintaining the alveolar structure. For instance, the repeating recruitment and derecruitment of the lungs creates inflammatory responses. The mechanisms causing acute lung injury during mechanical ventilation include

volutrauma, atelectrauma, biotrauma, barotrauma, and oxygen toxicity. Understanding the mechanisms of VILI facilitates the prevention of worsening disease.