PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Unintended inhalation of nitric oxide by contamination of compressed air

ArticleInfo		
ArticleID	:	4163
ArticleDOI	:	10.1186/ccf-1999-2221
ArticleCitationID	:	2221
ArticleSequenceNumber	:	22
ArticleCategory	:	Paper Report
ArticleFirstPage	:	1
ArticleLastPage	:	3
ArticleHistory	:	RegistrationDate : 1999–11–16 OnlineDate : 1999–11–16
ArticleCopyright	:	Current Science Ltd1999
ArticleGrants	:	
ArticleContext	:	1305422

Adrian Mellor

Keywords

Acute respiratory distress syndrome, air pollution, mechanical ventilation

Comments

This paper is of great interest to anyone involved in studying the effects of NO. The important findings are that the hospital supply reached 1270 ppb of NO (higher readings have been found in more industrialised cities) and also that there seemed to be a therapeutic benefit to inhaling hospital air. The clinical relevance of the study is less clear as most patients receiving NO will have such severe lung injury that a high FiO_2 is required.

Introduction

Inhaled nitric oxide is a selective pulmonary vasodilator that improves arterial oxygen tension in acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). Very low concentrations are effective (60 parts per billion [ppb]). Compressed air in hospitals contains a variable amount of nitric oxide (NO), as a result of air pollution, and this compressed air is added to oxygen to ventilate critically ill patients.

Aims

To determine whether contamination of compressed air by NO affects haemodynamics and gas exchange in patients with ALI and ARDS, and whether unintended NO inhalation interferes with the therapeutic use of inhaled NO.

Methods

During two periods, the hospital supply of compressed air was continually monitored for NO by chemiluminescence (allowing the detection of less than 10 ppb). Environmental NO was also measured at a distant site. The effect of this unintentional NO inhalation was studied in 15 patients with ALI during the working day, when NO levels were highest. In the first ten patients the source of compressed air was changed from the central supply to a gas tank containing air free of NO. Measurements were made before, 15 min after the change, and 15 min after the return to baseline. In the final five patients the effects of 5 ppm inhaled NO were evaluated when the ventilator was connected to the central gas supply and to the gas tank.

Results

NO levels in the hospital air supply varied from less than 10 ppb to 1270 ppb. They were above 80 ppb for 40% of the time. Replacement of the hospital compressed air with tank compressed air decreased PaO_2 by 10 ± 5%, and increased pulmonary vascular resistance (PVR) by 13 ± 7%. Adding 5 ppm NO to the hospital supply of air did not affect PaO_2/FiO_2 or PVR but adding 5 ppm to tank air increased PaO_2/FiO_2 by 16 ± 11% and decreased PVR by 14 ± 8%.

Discussion

This study shows that unintended inhalation of NO in industrialised areas may alter the PaO_2 and may make the therapeutic use of NO less successful. Although NO is oxidised in air to nitric dioxide (NO_2) it is stable enough to persist for some time in compressed air. The NO levels observed were highest during working days, and were slightly less than the local enviromental pollution. The withdrawal of small amounts of NO in the hospital compressed air system produced measurable changes in PaO_2. It is not thought that this could be due to methodology, as the air supply tubing was very rapidly swapped between supplies. Intentional inhalation of 5 ppm of NO resulted in no improvements when added to hospital-supplied air, suggesting that patients who were going to respond to NO had already done so (to the small amount of enviromental contamination). NO is often used therapeutically when the patient is receiving high concentrations of oxygen and hence little unintentional NO. There may be implications for future prospective trials on NO as unintentional inhalation would have to be considered.

References

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