



Dispersal of Respiratory Droplets With Open vs Closed Oxygen Delivery Masks*

Implications for the Transmission of Severe Acute Respiratory Syndrome

Ron Somogyi, BSc; Alex E. Vesely, MSc;
Takafumi Azami, MD, PhD; David Preiss, MSc;
Joseph Fisher, MD; Joe Correia, RT; and
Robert A. Fowler, MD, MS

Nosocomial transmission of droplet-borne respiratory infections such as severe acute respiratory syndrome (SARS) may be influenced by the choice of oxygen face mask. A subject inhaled saline mist and exhaled through three oxygen masks to illustrate the pattern of dispersal of pulmonary gas. In two commonly used masks, exhaled gas formed a plume emanating from the side vents, while a third mask with a valved manifold, which was modified by adding a respiratory filter, retained the droplets. Maintaining respiratory isolation during the administration of oxygen may reduce the risk of the nosocomial transmission of respiratory infections such as SARS. (*CHEST* 2004; 125:1155–1157)

Key words: droplets; nosocomial; oxygenation; oxygen mask; severe acute respiratory syndrome; ventilation

Abbreviation: SARS = severe acute respiratory syndrome

Severe acute respiratory syndrome (SARS) is a newly recognized illness that has spread rapidly throughout Asia, North America, and Europe. As of June 20, 2003,

*From the University Health Network (Mssrs. Somogyi, Vesely, Preiss, and Correia, and Drs. Azami and Fisher), Toronto General Hospital, and the Department of Medicine (Dr. Fowler), Sunnybrook & Women's College Health Sciences Centre, University of Toronto, Toronto, ON, Canada.

This study was funded by the Ontario Thoracic Society. Dr. Fisher and Mssrs. Vesely, Somogyi, and Preiss participated in the development of the Hi-Ox80 and receive royalties on sales. Manuscript received July 10, 2003; revision accepted November 18, 2003.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: permissions@chestnet.org).

Correspondence to: Robert Fowler, MD, MS, Assistant Professor, University of Toronto, Departments of Medicine (General Internal Medicine) and Critical Care Medicine, Sunnybrook & Women's College Health Sciences Centre, 2075 Bayview Ave, Room D478, Toronto, ON, Canada M4N 3M5; e-mail: rob.fowler@stw.ca

8,461 people in 30 countries had developed SARS, leading to 804 deaths.¹ The transmission of SARS from patients to health-care workers and other patients has been a prominent and worrisome feature of existing SARS outbreaks.^{2,3} The transmission of infection is thought to be through exhaled viral-laden droplets.^{2,3} Because many patients with SARS require supplemental oxygen administration by face mask,⁴ we imaged patterns of droplet dispersal with various oxygen masks to investigate the potential for possible nosocomial spread and reported our preliminary observations.



FIGURE 1. Subject exhaling previously inhaled saline aerosol mist while wearing nonrebreathing oxygen mask (top, A) and Venturi-type oxygen mask (bottom, B). The plume represents only exhaled breath because there was no oxygen flow into the masks.

MATERIALS AND METHODS

After ethics board approval was obtained, the subject inhaled aerosolized 3% saline fluid droplets that were produced by an ultrasonic nebulizer (DeVilbiss Ultra-Neb-99 Large-Volume Ultrasonic Nebulizer; Sunrise Medical; Somerset, PA). The breath was then held briefly (approximately 2 s), while one of three oxygen masks was fitted to the face. The subject then exhaled smoothly for > 2 s through the mask while a series of photographs were taken. Photographs were not altered except for adjustments to brightness and contrast.

The procedure was repeated three times with each of three oxygen masks. Two of the masks had side vents for exhaled gas (a nonbreathing mask [Airlife Adult Oxygen Mask; Allegiance Healthcare Corp; McGaw Park, IL] and a Venturi-type mask [Airlife Percento₂ Mask; Allegiance Healthcare Corp]). The third mask had no vents. All exhaled gas exited through a single 22-mm port in the attached manifold (Hi-Ox80; Viasys; Yorba Linda, CA). The Hi-Ox80 was tested without and with a respiratory filter (Barrierbac "S"; Mallinckrodt; Mirandola, Italy) on the expiratory port. No oxygen flow was supplied to any of the masks during testing.

RESULTS

A good fit was achieved with all masks: no exhaled gas was observed leaking between the edge of the mask and the face. With the nonbreathing and Venturi-type masks, plumes of exhaled droplets could be seen exiting the side vents (Fig 1). In the mask without vents, all

exhaled vapor was directed through the exhalation port (Fig 2, *left, A*). Placing the respiratory filter on the exhalation port appeared to prevent droplets from leaving the mask (Fig 2, *right, B*). There was no increased positive pressure or increased end-tidal carbon dioxide discovered using the modified oxygen delivery system.

DISCUSSION

These preliminary observations from an ongoing study suggest the presence of a mechanism by which the method of oxygen administration can contribute to the spread of droplet-borne respiratory infection, including SARS (<http://isocapnia.com/SARS.htm>). Throughout exhalation, both the nonbreathing mask and the Venturi-type mask channeled the exhaled gas through the side vents, forming a plume of exhaled gas (marked here by the previously inhaled aerosol) that was directed to the side of the patient. This potentially infectious plume could be directed toward caregivers, visitors, and other patients at face level. The extent of the plume might be greater with oxygen flows or when expiratory flows are increased by coughing, sneezing, or hyperventilation.

Does this model accurately reflect the droplet spread associated with respiratory infections such as SARS? The plumes visible in the figures are composed of the previously inhaled mist that marks the distribution of exhaled



FIGURE 2. Subject exhaling previously inhaled saline aerosol mist while wearing Hi-Ox80 oxygen mask without an added filter placed on the exhalation port (*left, A*) and with added filter placed on exhalation port (*right, B*).

gas. We assumed that endogenously produced droplets would be within the distribution of the exhaled gas. The spread of the exhaled gas may be greater than shown, as evaporation and reduction in density of the droplets at the margin of the plume may limit their effectiveness as markers. On the other hand, the visualized cloud does mark the minimum distribution of gas that originated in the lung.

In the mask without side vents, all exhaled gas traverses a manifold containing three valves and exits through a single port. Placing a bacterial/viral filter on this port may provide effective respiratory isolation during oxygen administration to spontaneously breathing patients.

The current recommendations for the management of SARS patients acknowledge the importance of the isolation of exhaled gas to prevent the release of infected droplets into the atmosphere.⁵ This can be accomplished with N95 or equivalent masks in spontaneously ventilating patients who do not require oxygen supplementation, and by the placement of bacterial/viral hydrophobic filters on the end of the endotracheal tube and/or the exhalation port of the self-inflating bag or breathing circuit in ventilated patients. However, when oxygen supplementation is required for spontaneously breathing patients, guidelines often condone open oxygen delivery systems that do not retain respiratory droplets.⁶ In light of reports of SARS patients infecting other patients and health-care workers during the pre-intubation phase of their treatment, despite the use of protective equipment by health-care workers, we think that additional measures should be considered.³ The administration of oxygen using the delivery systems described in this article may further reduce the risk of the nosocomial transmission of respiratory infections such as SARS.

ACKNOWLEDGMENTS: We thank Steve Iscoe, PhD, and Arthur S. Slutsky, MD, for their review of the manuscript, Allan Coates, MD, and Gerald Smaldone for their guidance on droplet dispersal, and Agnes Ryzynski and Jon Hendrix for photography services.

REFERENCES

- 1 World Health Organization. Communicable disease surveillance and response: cumulative number of reported probable cases of SARS. Available at: http://www.who.int/csr/sars/country/2003_06_20/en/. Accessed February 17, 2004
- 2 Lee N, Hui D, Wu A, et al. A major outbreak of severe acute respiratory syndrome in Hong Kong. *N Engl J Med* 2003; 348:1986–1994
- 3 Centers for Disease Control and Prevention. Cluster of severe acute respiratory syndrome cases among protected health-care workers—Toronto, Canada, April 2003. *MMWR Morb Mortal Wkly Rep* 2003; 52:433–436
- 4 Fowler RA, Lapinsky SE, Hallett D, et al. Critically ill patients with severe acute respiratory syndrome (SARS). *JAMA* 2003; 290:367–373
- 5 Li TS, Buckley TA, Yap FH, et al. Severe acute respiratory syndrome (SARS): infection control [letter]. *Lancet* 2003; 361:1386
- 6 Ontario Medical Association. Directives to all Ontario acute care hospitals for high-risk procedures involving SARS pa-

tients critical care areas: SARS Provincial Operations Centre; May 13, 2003. Available at: http://www.oma.org/phealth/sars/high_risk_procedures_in_critical_care.pdf. Accessed February 17, 2004

Identical Twins With Primary Pulmonary Hypertension*

Beraprost vs Epoprostenol

Erika Berman Rosenzweig, MD; Kelly A. Schmitt, RN; Robert Garofano, PhD; and Robyn J. Barst, MD

Background: The course of 12-year-old, homozygotic twins with primary pulmonary hypertension (PPH) treated with different vasoactive agents, beraprost vs epoprostenol, is described.

Methods: Clinical, exercise, and hemodynamic assessments were made at baseline, and at 9 months and 24 months of treatment.

Findings: Twin A had a rapid improvement with epoprostenol. In contrast, twin B, initially treated with beraprost, had progressive worsening with subsequent improvement on epoprostenol.

Interpretation: Epoprostenol was efficacious for identical twins with PPH. A 9-month delay in initiating epoprostenol for twin B did not appear to have irreversible short-term detrimental effects.

(*CHEST* 2004; 125:1157–1160)

Key words: beraprost; epoprostenol; pulmonary hypertension

Abbreviations: PPH = primary pulmonary hypertension; $\dot{V}O_2$ = oxygen consumption; WHO = World Health Organization

The course of 12-year-old, homozygotic twins with primary pulmonary hypertension (PPH) treated with different vasoactive agents is described (beraprost vs epoprostenol). Twin A was treated with continuous IV epoprostenol, and twin B was initially treated with the oral prostacyclin analog beraprost, and was subsequently transitioned to epoprostenol.

*From Children's Hospital of New York, Columbia University, College of Physicians and Surgeons, New York, NY. Although none of the authors have stock ownership or other equity interests or patent licensing arrangements that might pose a conflict of interest in connection with this work, Dr. Barst has served as a consultant to United Therapeutics Corporation, the sponsor for the beraprost trial that twin B participated in. Manuscript received June 24, 2003; revision accepted October 8, 2003.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: permissions@chestnet.org).

Correspondence to: Erika Berman Rosenzweig, MD, Children's Hospital of New York-Presbyterian, 3959 Broadway, 2 North, New York, NY 10032; e-mail: esb14@columbia.edu