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Helium–Oxygen in Treatment of Upper Airway Obstruction

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The therapeutic use of helium–oxygen for treatment of upper airway obstruction is dependent upon the low density of helium.[1-4] A helium–oxygen mixture may be indicated when the obstruction is unresponsive to conventional techniques or in cases of obstructive pulmonary disease in which respiratory fatigue is marked.[2,3]

REPORT OF A CASE

A 49-year-old black woman received a right pneumonectomy for bronchogenic squamous-cell carcinoma. Three weeks later she was readmitted with marked dyspnea and inspiratory stridor.

Bronchoscopy was performed the next day with anesthesia standby. No premedication was given. Two milliliters of 4 per cent lidocaine were administered transtracheally and the vocal cords sprayed. Diazepam, 5 mg, iv, was given for sedation, and bronchoscopy was performed in 15 min. A marked narrowing of the left mainstem bronchus (estimated 4 mm diameter) just distal to the carina was visualized. The narrowing was the result of extrinsic compression of the left mainstem bronchus. Following withdrawal of the bronchoscope, the patient experienced increasing difficulty in breathing, and she became unconscious during the ensuing 10 min. Attempts to ventilate her with an anesthetic mask and 100 per cent oxygen were unsuccessful. The trachea was intubated without difficulty and without the use of any drug. An attempt was made to ventilate her with a Bennett MA-1 ventilator with 100 per cent oxygen. Ventilation became progressively more difficult, and tidal volumes became unmeasurable before the maximum inspiratory pressure limit of the ventilator was reached.

Analysis of arterial blood drawn at this time revealed Pa_{O_2} 190 torr, Pa_{CO_2} more than 100 torr, and pH 7.01. Sodium bicarbonate, 90 mEq, iv, was administered to treat the severe acidosis. Meanwhile, a tank of helium was summoned to the scene. A helium–oxygen (3.5–1.5 l) mixture, FI_{O_2} = 0.3, was delivered by the Bennett MA-1 ventilator through the medication chamber to the patient.

In a matter of a few breaths, ventilation showed a dramatic improvement, with measurable tidal volumes as much as 175 ml, minute volume about 4,900 ml, with a peak inspiratory pressure of 56 cm H_2O. After 45 minutes Pa_{O_2} was 82 torr, Pa_{CO_2} 56 torr, pH 7.41. The patient had regained consciousness.

To substantiate our clinical impression, the following laboratory study simulating the pulmonary abnormality in the patient was performed.

METHOD

The tracheas of six unpremedicated mongrel dogs weighing 20–30 kg were intubated following thiopental, 12–15 mg/kg, iv, and the dogs were anesthetized with halothane–oxygen.

A central arterial line was inserted via a femoral artery for pressure and blood-gas

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TABLE 1. Summary of Respiratory Data from Six Dogs, Means ± SE

	Chest Open	Right Mainstem Bronchus Clamped	Control, Right Mainstem Bronchus Clamped Left Mainstem Bronchus Partially Clamped	He, 20 Per Cent O_2, 80 Per Cent	He, 40 Per Cent O_2, 60 Per Cent	He, 60 Per Cent O_2, 40 Per Cent	He, 80 Per Cent O_2, 20 Per Cent
Peak airway pressure (cm H_2O)	19 ± 0.69	26 ± 0.97	40 ± 2.84	39 ± 2.82	36 ± 2.82	32 ± 3.06	28* ± 2.84
Pa_{CO_2} (torr)	23.9 ± 2.57	37.0 ± 5.05	63.3 ± 5.61	51.9 ± 5.61	47.6 ± 5.96	44.1* ± 5.72	45.3 ± 7.46
Pa_{O_2} (torr)	232 ± 44.99	267 ± 53.72	146 ± 30.47	155 ± 33.87	149 ± 41.24	80 ± 15.55	50* ± 6.68
*p*H	7.36 ± 0.024	7.33 ± 0.036	7.13 ± 0.057	7.22 ± 0.034	7.23 ± 0.028	7.24 ± 0.039	7.25 ± 0.037
Negative base excess	10.5 ± 0.72	6.3 ± 1.27	10.3 ± 1.61	7.9 ± 1.20	8.8 ± 1.60	9.1 ± 2.03	8.3 ± 1.89

* Significantly different ($P < 0.05$) from control using t test for paired data.

monitoring. The arterial pulse contour was analyzed to provide hemodynamic measurements of cardiac output (CO), stroke volume (SV), heart rate (HR), mean arterial pressure (MAP), and peripheral resistance (PR), by the method of Warner.[5] The lungs were mechanically ventilated with a Bird Mark 9 anesthesia ventilator with 10–15 ml/kg tidal volume, maintaining a Pa_{CO_2} of 30–35 torr.

1) A midline sternotomy was performed, followed by 2) occlusion of the right mainstem bronchus and right pulmonary circulation, followed by 3) partial occlusion of the left mainstem bronchus. The endpoint of the partial occlusion was the maximum occlusion tolerated without resulting in severe hypotension and subsequent bradycardia. 4) Helium was introduced through the helium flowmeter in 20 per cent increments until 80 per cent, with 20 per cent oxygen, had been reached.

Arterial blood gases, peak airway pressure, CO, SV, HR, MAP, and PR were measured following steps 1, 2, 3, and 4.

At least 20 minutes of equilibration time were allowed before each measurement.

RESULTS

The respiratory data are summarized in table 1. Mean peak airway pressure increased from 19 to 40 cm H_2O when the right mainstem bronchus was occluded with partial occlusion of the left mainstem bronchus.

With constant flow and tidal volume, the addition of helium to oxygen reduced peak

TABLE 2. Densities and Relative Gas Flow Rates of Oxygen, Air, and Helium

	Per Cent	Density	$\sqrt{\text{Density}}$	$\frac{1}{\sqrt{\text{Density}}}$	Relative Gas Flow Rate (Air Assigned an Arbitrary Value of 1.00)
Oxygen	100	1.429	1.182	0.846	0.96
Air	100	1.293	1.135	0.881	1.00
Helium	100	0.179	0.423	2.364	2.68
He–oxygen	20/80	1.178	1.085	0.922	1.048
He–oxygen	60/40	0.678	0.823	1.215	1.381
He–oxygen	80/20	0.429	0.655	1.527	1.73

airway pressure from 40 to 28 cm H_2O as the helium concentration was increased from 20 to 80 per cent. Pa_{CO_2} was also reduced, from 63 to 45 torr. Pa_{O_2} decreased proportionally with reduction in FI_{O_2}.

After total occlusion of the right mainstem bronchus and partial occlusion of the left mainstem bronchus, the following hemodynamic data (mean ± SE) were obtained (n = 6): CO = 2.7 ± 0.65 l/min; SV = 23.1 ± 6.40 ml; HR = 120 ± 5 beats/min; MAP = 115 ± 7 torr; PR = 2.56 ± 0.91 torr/ml/sec. These variables were not significantly altered during administration of the helium–oxygen mixture.

Discussion

The low-density helium–oxygen mixture promoted better gas flow through the narrow orifice, as indicated by the reduction in peak airway pressure. As ventilation improved, the Pa_{CO_2} decreased.

Graham's law of diffusion states that the gas flow rate through a narrow orifice is inversely proportional to the square root of its density.[6] Hence the lighter the gas mixture, the greater the flow rate through a narrow orifice. This was demonstrated in our animal studies, in which ventilation was improved after helium was added. The densities and relative gas flow rates are summarized in table 2.

Forty per cent helium with 60 per cent oxygen was the optimal mixture for maintaining desirable arterial blood-gas values in the acutely airway-obstructed dogs. The low density of helium made it possible to ventilate the lungs through a restricted orifice, requiring less driving pressure to fill the lung. An oxygen–helium mixture can substantially improve the efficiency of breathing during upper airway obstruction.

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Esophageal Perforation of Unusual Etiology

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When perforation of the esophagus occurs during anesthesia the mechanism by which it was produced is not always apparent. It may be associated with difficulty in laryngoscopy and insertion of the endotracheal tube. The following report presents an unusual etiologic factor of this complication—the presence of a pharyngoesophageal, or Zenker's, diverticulum.

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Report of a Case

A 40-year-old white woman was admitted for removal of chronically infected adenoidal tissue. Past medical history was noncontributory. Physical examination disclosed no abnormality except the presence of excess adenoidal tissue, and results of routine laboratory determinations were within normal limits.

Hydroxyzine, 50 mg, meperidine, 75 mg, and atropine, 0.4 mg, were given im at 9:35 AM. At 10:30 AM anesthesia was induced with 250 mg thiopental followed by succinylcholine, 70 mg, iv, and 4 ml of