Estimation of oxygen concentration in the airway during inspiration and pre-inspiration with a nasal cannula

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Abstract

BACKGROUND: In low flow oxygen therapy, FIO2 is difficult to measure in spontaneously breathing patients due to room air dilution and dead space rebreathing, especial in impairments respiratory mechanics. This study determined oxygen concentrations with different tidal volumes and respiratory rates among different lung mechanics and provided equations to estimate oxygen concentrations during standard nasal cannula oxygen therapy. METHODS: Two Training & Test Lung models were used in this study. One simulated spontaneous breathing, whereas the other included an expiratory gas modification bellow. Three lung mechanics [normal (R5/C60), restrictive (R20/C80) and obstructive (R5/C40)] were designed, and spontaneous breathing settings for different tidal volumes(VT) and respiratory rates(f) were simulated by the mechanical ventilator. The nasal cannula used flows of 1, 3 and 5L/min; peak inspired oxygen concentration (FO2 insp.) and pre-inspired oxygen concentration (FO2 pre-insp.) were measured. RESULTS: Increased VT caused a decreased FO2 insp. and FO2 pre-insp., except at 1L/min oxygen flow with a high f (30breaths/min). Multiple regression analysis showed oxygen flow rate, VT and f as the most important factors in predicting oxygen delivery during nasal cannula therapy. Therefore, we provided equations to predict oxygen concentration for managing patients with acute and chronic lung diseases. CONCLUSIONS: Our study suggested that under low-flow nasal cannula therapy, various lung mechanics and respiratory patterns in the normal, restrictive and obstructive lung models will affect the oxygen concentration.

Introduction

In contemporary clinical practice, 50–84% of patients are exposed to excess oxygen and hyperxemia as a result of efforts to prevent or reverse hypoxemia via various interfaces such as a nasal cannula, nasal catheter, venturi mask and non-rebreathing mask.[1-4]Recently, a systematic review and meta-analysis provides high-quality evidence that hyperoxia is life-threatening in acutely ill adults with acute myocardial infarction, sepsis, critical illness, stroke, trauma, cardiac arrest.[5]

Oxygen treatment can be divided into high-flow and low-flow systems. The high-flow system provides a flow that is equal to or exceeds the patient's peak inspiratory flow and ensures a fixed F_{IO2}, while the low-flow system is variable due to the entrained indoor air that dilutes the oxygen, resulting in a low F_{IO2}.[1,2,4] The precise F_{IO2} amount delivered is difficult to determine in spontaneously breathing patients because it is influenced by the breathing pattern, including the patient's minute ventilation, f, V_T, inspiratory time (T_I), expiratory time (T_E), functional apparatus dead space, inspiratory flow rate, expiratory flow rate and impact of open or closed mouth,[1,2,4,6-9] particularly in patients with respiratory failure, which manifests as hypoventilation or hyperventilation.

Although oxygen therapy can reduce the symptoms of hypoxemia,[10] but high concentrations of oxygen cause adverse effects such as chronic obstructive pulmonary disease(COPD)[11] and type II respiratory failure.[12] Clearly, more precise control of the inspired oxygen concentration is very important for patients have a

high respiratory rate (f) with shallow or deep breathing in lower lung compliance such as acute respiratory distress syndrome,[13] left ventricular failure[14] and pulmonary fibrosis[15]and increased airway resistance such as COPD[16-18] and asthma.[19,20] The nasal cannula provides oxygen flow at a rate of 1–5L/min,[3] is the most widely used low-flow oxygen device for adults, children and infants.[1-4]Modern guidelines and the literature lack much data regarding the correlation between how much to give and breathing patterns in respiratory failure during low-flow oxygen therapy with a nasal cannula.[1,2,6,21-26]

We conducted this bench study to investigate the performance of nasal cannula in a manikin head-test lung-ventilator system to simulate a spontaneous breathing pattern in normal, restrictive and obstructive lung models. To describe the effects of various V_T and f on the measured F_{IO2} near the carina and provide equations to estimate F_{IO2} during standard nasal cannula oxygen therapy.

Materials and Methods

Lung model and testing protocol

Spontaneous breathing was simulated by a lung model using a mechanical ventilator (LTV 1200, CareFusion, San Diego, CA) and two Training & Test Lungs (TTLs) (Dual Adult Lung Simulator 5600i, Michigan Instruments, Inc.; Grand Rapids, MI). In the first TTL, paired bellows called the driving bellow and ventilation bellow were linked together by a rigid metal strip. In the second TTL, one bellow called the expiratory gas modification bellow was linked to the first TTL by a rigid metal strip to wash out previous gas in the anatomic dead space in the airway trainer. During simulation, the driving bellow was connected to LTV1200 ventilator and the ventilation bellow was connected to a human-like anatomy model (Laerdal Airway Management Trainer 25 00 00) with oxygen therapy applied via a nasal cannula (VADI Medical Technology, Taoyuan, Taiwan). The carina position of the human-like anatomy model was connected to the oxygen analyser (MiniOX I*;* MSA Medical*,*Gurnee, Ill.). Between the TTLs, we incorporated four one-way valves (valves 1 to 4) to prevent the mixing of inspired and expired gases (Fig. 1). When inspiration was simulated by the LTV 1200, the ventilator delivered a TV to the driving bellow, causing the bellow to expand and force the metal strap to pull on the ventilation bellow, which expanded passively. This action was detected as an "inspiratory" effort, which in turn triggered spontaneous breathing and drew in gas inhaled only from the nose and mouth into the "ventilation bellows" of the TTL through the in-line one-way valve 1 (directed to the ventilation bellows). Thus, the expiration washing out ventilation bellow was also inflated simultaneously and aerated through valve 4. During expiration, the air was exhaled from the "ventilation bellow" through the other one-way valve 2 (directed to the outside), and the expiration washing out ventilation bellow passively deflated. Consequently, the air was exhaled to the anatomic dead space in the airway trainer through the in-line one-way valve 3.

This experiment included three levels of resistance(R) and compliance(C) of TTLs to represent the lung mechanics of normal, obstructive and restrictive lung diseases (Fig. 2), as suggested by the manufacturer and previous studies.[27-30] In the normal lung model was R5/C60, R20/C80 and R5/C40 were represented to obstructive and restrictive lung diseases, respectively. The protocols were performed with a wide-ranging change in the ventilatory pattern of the LTV1200 ventilator [V_T:300, 500 and 700mL; f:10, 20 and 30breaths/min]; Oxygen flow rates of 1, 3 and 5L/min were set for the nasal cannula.

Variables and measurements

The main outcome variable of this study was F_{IO2}, prior to beginning the experiment, the oxygen analyzer was calibrated according to the manufacturer's instructions using 50 psi wall oxygen and air source. Oxygen concentrations were recorded as FO_2 insp. and FO_2 pre-insp., which are defined as the peak inspiratory and pre-inspiratory phase, respectively (Fig. 3). Oxygen concentrations measurements were obtained at equilibrium, which was assumed to occur when the reading was steady over the 3-min period. FO_2 insp. and FO_2pre-insp. were recorded for 10breaths and triplicate in each experimental condition.

Statistics

FO_2 insp. and FO_2 pre-insp. measurements are expressed as mean and SD. ANOVA was performed with

a post-hoc Tukey's test to analyse between-group differences. In addition, to test the predictive factors associated with FO_2 insp. and FO_2 pre-insp., multiple regression analyses were performed to determine whether V_T, f and oxygen flow rate were significant predictors. Statistical analyses were performed using SPSS 17.0 software (SPSS Inc., Chicago, IL). A p value of <0.05 was considered statistically significant.

Results

Effect of different TVs on muscle pressure

In our simulated lungs, measured driving pressure at the carina was represent inspiratory muscle activity. We observed that muscle activity in the obstructive lung model was greater than that in the normal and restrictive lung disease models, regardless of f and $V_T$settings in our experiment. However, the trend of decreased driving pressure was more profound in the largest V_T (700mL) than in the small (300mL) and normal (500mL) V_T. For example, with an RR of 30breaths/min, driving pressure decreased from -2.1 to -7.4cmH_2O, -2.4 to -9.3cmH_2O and -3.5 to -15.4cmH_2O in the normal, restrictive and obstructive lung mechanics settings, respectively, for V_T of 300, 500 and 700mL (Fig. 4).

FO_2 insp. and FO_2 pre-insp. measurements

For all experimental conditions, the actual oxygen concentrations of FO_2 insp. and FO_2 pre-insp. with different lung conditions from our spontaneous breathing lung model are shown in Table 1. The data indicate that FO_2 pre-insp. was higher than FO_2 insp. under different conditions. With the same V_T as normal lung mechanics, the measured FO_2 insp. and FO_2 pre-insp. decreased as the f increased. However, at the same f, a smaller FO_2 insp. and FO_2pre-insp. were recorded when a larger V_T was present. The same outcomes were reported in the restricted and obstructed lung models.

Effect of TVs and oxygen flow rate on FO_2 insp.

In the normal lung mechanics settings, a low f (10breaths/min) with an oxygen flow of 1L/min displayed a measured FO_2 insp. that was 1.116 times greater at a low V_T than at the normal V_T; conversely, the lowest measured FO_2 insp. at the high V_T was 0.966 times that of the normal V_T. Similar trends appeared with oxygen flow rates of 3 and 5L/min. The post-hoc analysis showed significant differences in performance as the f increased to 20breaths/min ($p < 0.05$) and 30breaths/min ($p < 0.05$), except at 1L/min of oxygen flow with 30breaths/min (p =0.342). When we tested the restrictive and obstructive lung mechanics, the FO_2 insp. values during different V_T at 1, 3 and 5L/min oxygen delivery also showed the same patterns as normal lung mechanics (Fig. 5).

Effect of TVs and oxygen flow rate on FO_2pre-insp.

In normal, restrictive and obstructive lung conditions, as V_T increased, the measured FO_2 pre-insp. showed a statistically significant difference at all gas flow rates ($p < 0.05$) during low, normal and high f settings, except at the lowest oxygen flow (1L/min) with the highest f (30breaths/min) (normal: p =0.364, restrictive: p =0.104, obstructive: p =0.512) (Fig. 6). There was a greater reduction in performance at a $V_T$of 700mL than at 500mL and 300mL.

Multivariate analyses and predictive equations

According to the above results, V_T, f and oxygen flow rate affect FO_2 insp. and FO_2 pre-insp. measurements; thus, we aimed to approximate their relationship using a linear regression analysis. In normal lung conditions, both V_T and oxygen flow rate have significant ($p < 0.05$) effects on FO_2 insp. and FO_2 pre-insp., but f only affects FO_2pre-insp. (Table 2a). In addition, the restrictive (Table 2b) and obstructive (Table 2c) lung model display the same regression pattern. Finally, simple linear regression models using V_T, f and oxygen flow rate as predictor variables were found to provide the equations that are presented in Table 3.

Discussion

In our experiment, we developed an expiratory gas modification bellow, which modified the expiratory flow during the exhalation phase. This model was developed to sample and measure tracheal oxygen concentra-

tions with various breathing patterns of both V_T and frequency under normal, restrictive and obstructive lung mechanics. In addition, we provided equations for estimating inspired oxygen in the trachea for clinical reference.

During oxygen therapy with nasal cannula, the rule of thumb is that for patients with a normal rate and depth of breathing, each 1L/min of oxygen supplied increases F_{IO2} by approximately 4%;[1,2,4] therefore, the expected delivery is a F_{IO2} of 0.24–0.44. However, if the patient's f approaches or exceeds 20breaths/min, F_{IO2} will likely be well below estimates, and the delivered F_{IO2} will only increase by approximately 0.025 for each 1L/min above the ambient oxygen level.[1,31] In those studies, F_{IO2} was measured by an oxygen analyzer placed in the low airway, such as an oxygen analyzer port of the TTL bellow, which is the distal airway. In our experiment in which we designed the test lung, there was no effect of fresh oxygen flow from the exhaled carbon dioxide because the expiratory gas modification compartment was designed to produce a modified expired gas. In addition, we measured FO_2 insp. and FO_2 pre-insp. at the carina, which is the proximal airway. Lower FO_2 insp. and FO_2pre-insp. values than estimated were found at all settings, which may be due to differences in monitoring sites and the experimental design. By contrast, Gibson et al. reported that in healthy subjects with a percutaneously placed tracheal sensing catheter, the highest absolute inspired tracheal oxygen concentration with a nasal cannula was 23.6% at 3L/min and 25.4% at 5L/min during normal breathing [V_T:690mL, f:17breaths/min, minute ventilation (MV):11L/min, peak inspiratory flow rate (PIFR):37L/min], 22.4% at 3L/min and 23.8% at 5L/min during quiet breathing (V_T:400mL, f:16breaths/min, MV:6.4L/min, PIFR:21L/min), and 22.7% at 3L/min and 25.2% at 5L/min during hyperventilation (V_T:1400mL, f:14breaths/min, MV:19.5L/min, PIFR:63L/min) .[32] In another report, in patients with 97% oxygen supply via nasal cannula, the effective F_{IO2} was 22.8 ± 0.1%, 27.6 ± 0.5% and 31.8 ± 0.5% with 1, 3 and 5L/min based on trachea sampling, respectively.[33] Our findings concur reasonably well with those data, but contrast with a previously published formula.

Oxygen concentration is also influenced by V_T in our model in the same sense as previously described by Chikata et al., who reported that statistically significantly changes in $V_T$affected measured F_{IO2} at all flow levels: at 2L/min and V_T of 300, 500 and 700mL, F_{IO2} was 0.37 ± 0.01%, 0.32 ± 0% and 0.29 ± 0%, respectively; at 4L/min, F_{IO2} was 0.45 ± 0.01%, 0.39 ± 0.01% and 0.34 ± 0%, respectively.[34] Indeed, various authors have reported decreasing F_{IO2} values during increasing MV and f.[24] Our model clearly demonstrates that changes in V_T affect measured oxygen concentration. Specifically, as V_T increases with a fixed f in the normal, restrictive and obstructive lung models, there is a reduction in the effective oxygen concentration due to more room air inhalation and dilution of the oxygen concentration from the nasal cannula at all flow levels. For example, the measured FO_2 insp. in normal lung mechanics at a flow rate of 1L/min, V_T of 300mL and f of 10breaths/min was 22.7 ± 0.10%, whereas it was 22.33 ± 0.15% and 21.60 ± 0.20% at V_T of 500mL and 700mL, respectively.

Our study found that FO_2 insp. and FO_2pre-insp. differed in different respiratory cycles. During the inspiratory phase, FO_2 insp. measured at the carina was significantly lower than that measured during the expiratory period. In the previously described by O'Reilly-Nugent et al, the variability of F_{IO2} in different respiratory cycles during low-flow nasal cannula at oxygen flow rates of 2–4L/min. Their novel method of F_{IO2} measurement involved sampling via a catheter placed at the distal trachea; this has clarified the uncertainties of other studies that were sampled near the proximal airway where inadequate gas mixing had occurred. The researchers found that because of the bolus of oxygen from the nasopharynx or relatively low inspiratory flow at the beginning of each breath, the peak F_{IO2} is significantly higher than the remainder of inspiration and then declines rapidly as the inspiratory flow reaches its peak. After inspiration, F_{IO2} then increases steadily, coinciding with a reduction in inspiratory flow.[35] Conversely, the oxygen delivered from the nasal cannula is inhaled into the lungs for gas exchange, and the oxygen concentration measured at the carina is relatively low. However, during the expiratory period, carbon dioxide in the lungs is exhaled, but oxygen inhalation does not occur. Therefore, during this period, the measured oxygen concentration is higher, which is most pronounced particularly between the end-expiratory period and the next inspiratory period. Thus, any variation in the total expiratory time affects the composition of the next inspiration.[4,21,27]

Finally, our study provides scientific data that f, $V_T$and oxygen flow rate significantly influence the inspired oxygen concentration under different lung mechanics. In addition, during spontaneous breathing, we have successfully proposed equations for clinical practice for nasal cannula oxygen therapy. These equations are based on different lung mechanics, V_T and f to estimate the actual patient oxygen concentration; they will provide clinicians a reference when using nasal cannula among patients with normal, restrictive and obstructive lung diseases.

Although we devised a spontaneously breathing lung model with three lung mechanics conditions and different V_T and f, but our study was limited to did not consider open or closed mouth breathing states even though oxygen concentration is influenced by the reservoir space in the oral cavity during nasal cannula. In addition, we did not investigate the effect of water vapours on oxygen concentration.

Conclusion

Our study assessed FO_2 insp. and FO_2pre-insp. during nasal cannula oxygen therapy in a spontaneous breathing lung model and suggested that multiple factors influence the measured FO_2 insp. and FO_2 pre-insp. under low-flow nasal cannula, such as respiratory patterns, additional V_T, f and inspiratory or expiratory time, which indicate the short-term storage of oxygen in an anatomic space between the end-expiratory period and the next inspiratory period, as in the normal, restrictive and obstructive lung models. As such, an increased V_T cause a decrease in FO_2 insp. and FO_2 pre-insp. measured in the carina. As V_T and f decrease, there is less difference between FO_2 pre-insp. and FO_2 insp.; therefore, any variation in the total expiratory time will affect the oxygen reservoir in the next inspiration. The results of this bench study provide a reference for clinicians and researchers regarding various lung mechanics and respiratory patterns under low flow nasal cannula.

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Figure legends

Figure 1 Experimental apparatus of the connection test lung model

The designed spontaneous breathing lung model essentially contains two TTL, an airway management trainer and a ventilator. The driving bellows (A) with a lifting bar (coupling clip) in the site is connected to a LTV1200. The ventilation bellow (B) is connected to the tracheal of an airway management trainer through a set of a Y piece (Y1) adaptor and V1 and V2, which bring together through Y2). The expiratory gas modification bellow(C) is connected to the bellow B through a rigid metal strip and is connected to Y1 through V3 and V4. The open end of the main bronchus, which the carina position was to join together through the Y piece (Y3), and oxygen analyzer is connected between the Y1 and Y3. When the driving ventilator enlarges the bellow A of the TTL, which results in negative pressure within the bellow B and bellow C, and inspiration flow into the bellow B and C through the V1 and V4, respectively, and airway trainer, just like spontaneous breathing. During the expiration phase, the bellows B and bellow C passively deflates the gas outside through V2 and V3, respectively. The gas from bellow C through V3 was used to wash out the previous gas in the anatomic dead space in the airway trainer. An oxygen analyzer is position near the carina to monitor the FO_2 insp. and FO_2 pre-insp. of gas during the inspiratory and expiratory phases, respectively. The airway trainer is equipped with nasal cannula.

Figure 2 Simulated spontaneous breathing lung model and nasal cannula setting. V_T: tidal volume, f: respiratory rate, T_I: inspiratory time.

Figure 3 The friction of oxygen waveform during respiratory cycle. FO_2 insp.: peak inspired oxygen concentration, FO_2 pre-insp.: end-expired oxygen concentration.

Figure 4 Effect of V_T on muscle pressure

Figure 5 Effect of V_T on FO_2insp. during the same f

Figure 6 Effect of tidal volume on FO_2pre-insp. during the same f

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