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Supplemental Oxygen Impairs Detection of Hypoventilation by Pulse Oximetry*

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Study objective: **This two-part study was designed to determine the effect of supplemental oxygen on the detection of hypoventilation, evidenced by a decline in oxygen saturation (Spo_2) with pulse oximetry.**

Design: **Phase 1 was a prospective, patient-controlled, clinical trial. Phase 2 was a prospective, randomized, clinical trial.**

Setting: **Phase 1 took place in the operating room. Phase 2 took place in the postanesthesia care unit (PACU).**

Patients: **In phase 1, 45 patients underwent abdominal, gynecologic, urologic, and lower-extremity vascular operations. In phase 2, 288 patients were recovering from anesthesia.**

Interventions: **In phase 1, modeling of deliberate hypoventilation entailed decreasing by 50% the minute ventilation of patients receiving general anesthesia. Patients breathing a fraction of inspired oxygen (Fio_2) of 0.21 (n = 25) underwent hypoventilation for up to 5 min. Patients with an Fio_2 of 0.25 (n = 10) or 0.30 (n = 10) underwent hypoventilation for 10 min. In phase 2, spontaneously breathing patients were randomized to breathe room air (n = 155) or to receive supplemental oxygen (n = 133) on arrival in the PACU.**

Measurements and results: **In phase 1, end-tidal carbon dioxide and Spo_2 were measured during deliberate hypoventilation. A decrease in Spo_2 occurred only in patients who breathed room air. No decline occurred in patients with Fio_2 levels of 0.25 and 0.30. In phase 2, Spo_2 was recorded every min for up to 40 min in the PACU. Arterial desaturation ($Spo_2 < 90\%$) was fourfold higher in patients who breathed room air than in patients who breathed supplemental oxygen (9.0% vs 2.3%, p = 0.02).**

Conclusion: **Hypoventilation can be detected reliably by pulse oximetry only when patients breathe room air. In patients with spontaneous ventilation, supplemental oxygen often masked the ability to detect abnormalities in respiratory function in the PACU. Without the need for capnography and arterial blood gas analysis, pulse oximetry is a useful tool to assess ventilatory abnormalities, but only in the absence of supplemental inspired oxygen.**

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Key words: hypoventilation; pulse oximetry; supplemental oxygen

Abbreviations: Fio_2 = fraction of inspired oxygen; HR = heart rate; $Paco_2$ = alveolar carbon dioxide tension; PACU = postanesthesia care unit; Pao_2 = alveolar oxygen tension; $Petco_2$ = end-tidal carbon dioxide; RR = respiratory rate; Spo_2 = oxygen saturation; $\dot{V}E$ = minute ventilation; $\dot{V}/\dot{Q}$ = ventilation-perfusion; VT = tidal volume

Although the physiologic consequences of moderate hypoventilation have not been clearly elucidated, profound hypoventilation with the development of carbon dioxide narcosis can cause coma, respiratory arrest, and circulatory failure.[1,2] Various studies[2–5] have reported the difficulty in detecting hypoventilation in patients undergoing sedation for GI, dental, and other endoscopic procedures. Moreover, several reports[6,7] have discussed the failure to diagnose severe hypoventilation in the perioperative period.

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The early postoperative period may be associated with hypoventilation caused by respiratory depression and inability to maintain an adequate airway.[8,9] In addition, ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch also may occur secondary to atelectasis. Currently, accurate measurement of Pa_{CO_2} or end-tidal carbon dioxide (P_{ETCO_2}) to assess the adequacy of ventilation is not routine outside of the operating room environment, or in patients not intubated.[10] Although pulse oximetry is used widely to monitor arterial blood oxygenation, it is possible that pulse oximetry can be used to detect abnormalities in ventilation, by quantifying changes in oxygen saturation (Sp_{O_2}).[10,11] The objective of this study was to determine if pulse oximetry would indicate a decrease in ventilation, with and without administering supplemental oxygen.

For editorial comment see page 1399

Materials and Methods

The Institutional Review Board of the University of South Florida College of Medicine, Tampa, approved the study protocol, and consent was obtained in patients scheduled to undergo surgical procedures.

Phase 1

Forty-five patients gave informed consent to undergo a trial of controlled deliberate hypoventilation receiving general anesthesia and mechanical ventilation. Patients underwent abdominal, gynecologic, urologic, and lower-extremity vascular operations. We used a pulse oximeter, capnograph, arterial catheter, and ECG to measure Sp_{O_2}, P_{ETCO_2}, BP, heart rate (HR), and arterial blood gas data. General anesthesia was induced with thiopental, 3 to 5 mg/kg IV. IV administration of succinylcholine chloride, 1 mg/kg, or vecuronium bromide, 0.1 mg/kg, facilitated tracheal intubation. Anesthesia was maintained with isoflurane using a semiclosed-circle absorber system. IV vecuronium bromide was administered to maintain muscle relaxation.

Patients received mechanical ventilation with a tidal volume (V_T) of 8 mL/kg and a respiratory rate (RR) sufficient to produce an P_{ETCO_2} of 30 to 40 mm Hg before initial data collection. Data collection started 10 min after administering the desired fraction of inspired oxygen (F_{IO_2}). F_{IO_2} was determined by measurement of the inspired oxygen concentration on the oxygram. Three patient groups with F_{IO_2} levels of 0.21, 0.25, or 0.30, respectively, were studied. We recorded HR, BP, Sp_{O_2}, P_{ETCO_2}, RR, and V_T. Minute ventilation ($\dot{V}_E$) was determined as the product of RR and V_T. Hypoventilation was instituted by reducing RR to decrease the $\dot{V}_E$ by 50%. Patients breathing room air (F_{IO_2} of 0.21) underwent induced hypoventilation for up to 5 min, or until Sp_{O_2} was $< 90\%$. Patients with F_{IO_2} levels of 0.25 or 0.30 underwent hypoventilation for up to 10 min.

Intraoperative data were collected each minute until the end of the hypoventilation trial, or until Sp_{O_2} fell below 90%, whichever occurred first. Arterial blood was sampled to measure pH, Pa_{O_2}, and Pa_{CO_2} before hypoventilation and during final data collection. Intraoperative data were summarized as mean ± SD and evaluated with Student *t* test for paired observations, or with analysis of variance for repeated measurements and Scheffé test, when appropriate. A Pearson χ^2 test with Yates correction for continuity was used to determine probability under the null hypothesis of increase or decrease from initial to final value of Sp_{O_2}, arterial pH, Pa_{CO_2}, and P_{ETCO_2}.

Phase 2

Patients gave informed consent to be randomized to breathe room air, or to receive supplemental oxygen on arrival to the postanesthesia care unit (PACU). All surgical patients except those undergoing thoracotomy were eligible for participation. Baseline Sp_{O_2} measurements were recorded preoperatively in all patients breathing room air, prior to entering the operating room. Patients who received general anesthesia, regional anesthesia, or monitored anesthesia care were included in the study. Patients who received general anesthesia and who were not extubated in the operating room were excluded from the study. On leaving the operating room, patients with $Sp_{O_2} \geq 90\%$ breathing room air were transported to the recovery room without supplemental oxygen. On arrival in the PACU, while breathing room air, patients with $Sp_{O_2} \geq 90\%$ were randomized to continue to breathe room air, or to receive 30% oxygen with a facemask. Patients who experienced $Sp_{O_2} < 90\%$ before or on arrival to the PACU were administered supplemental oxygen and discontinued from the study.

In the PACU, Sp_{O_2} measurements were recorded every minute. Patients who experienced $Sp_{O_2} < 90\%$ for 2 consecutive min in the PACU received a "stir-up" regimen consisting of verbal and tactile stimulation. Patients in the supplemental oxygen group received a stir-up regimen, in addition to receiving oxygen by facemask. Patients in the room-air group initially received a stir-up regimen, without oxygen administration. If the Sp_{O_2} did not increase to $\geq 90\%$ within 2 min, patients then were administered supplemental oxygen with a facemask. Patients who initially breathed room air and subsequently received oxygen were classified as room-air dropouts. However, data collected until the time of dropout were recorded. Logistic regression analysis was used to study the effects of age, gender, and weight. Intergroup comparisons of the incidence of desaturation, use of intraoperative narcotics and muscle relaxants, and use of narcotics in the PACU were made with a Pearson χ^2 test.

Results

Phase 1

There were no intergroup differences in age or weight (Table 1). There were no differences between the V_T measured at the start of the hypoventilation trial (initial) and the V_T measured at the end of the trial (final). The final RR and $\dot{V}_E$ were approximately 50% of initial values. Initial and final arterial blood analysis data collected during induced hypoventilation are shown in Tables 2–4. Every patient had an increase in Pa_{CO_2} and P_{ETCO_2} and a decrease in arterial pH and Pa_{O_2} ($p < 0.001$). Patients with an F_{IO_2} of 0.21 had a significant decrease in Sp_{O_2} when initial and final Sp_{O_2} values were compared (97 ± 2% vs 91 ± 3%, $p < 0.001$). All patients with an F_{IO_2} of 0.21 had an immediate decrease in mean Sp_{O_2} and increase in mean

Table 1—*Demographic and Ventilatory Data in Patients Undergoing Induced Hypoventilation Under General Endotracheal Anesthesia**

Variables	Fio$_2$ 0.21	Fio$_2$ 0.25	Fio$_2$ 0.30
Patients, No.	25	10	10
Age, yr	56 ± 19	65 ± 12	68 ± 10
Weight, kg	68 ± 17	71 ± 17	77 ± 13
Initial Vt, mL	664 ± 210	599 ± 153	744 ± 101
Final Vt, mL	644 ± 155	613 ± 145	752 ± 102
Initial RR, breaths/min	7 ± 1	7 ± 1	7 ± 1
Final RR, breaths/min	4 ± 1†	4 ± 1†	4 ± 1†
Initial $\dot{V}$e, L/min	4.6 ± 1.6	4.4 ± 1.6	5.2 ± 1.4
Final $\dot{V}$e, L/min	2.4 ± 0.9*	2.3 ± 0.7*	2.7 ± 0.7*

*Data are summarized as mean ± SD unless otherwise indicated.
†$p < 0.001$ compared to initial value.

Table 3—*Variables Reflecting Gas Exchange and Cardiovascular Function for Patients Breathing an Fio$_2$ of 0.25**

Variables	Initial Data†	Final Data‡
Spo$_2$, %	97 ± 2	95 ± 3
Petco$_2$, mm Hg	34 ± 2	44 ± 3§
Arterial pH	7.40 ± 0.07	7.35 ± 0.05§
Paco$_2$ (range), mm Hg	34.7 ± 5.0 (23–42)	42.0 ± 3.3§ (36–50)
Pao$_2$ (range), mm Hg	105 ± 26 (73–49)	90 ± 27§ (59–130)
HR, beats/min	85 ± 15	87 ± 18
Mean arterial pressure, mm Hg	93 ± 14	94 ± 17

*Data are summarized as mean ± SD (n = 10) unless otherwise indicated.
†Initial data were collected before induction of hypoventilation.
‡Final data were collected 10 min after hypoventilation.
§$p < 0.001$ compared to initial value.

Petco$_2$ during hypoventilation ($p < 0.001$) [Fig 1]. Over half of these patients had Spo$_2$ < 90% within 5 min of hypoventilation, which accounts for the variable number of patients after 3 min of hypoventilation. Nine of 10 patients with an Fio$_2$ of 0.25 maintained Spo$_2$ > 90% throughout the 10-min study period. Every patient with an Fio$_2$ of 0.30 maintained Spo$_2$ > 90% throughout the study. Changes in Spo$_2$ during hypoventilation in patients with Fio$_2$ levels of 0.25 or 0.30 were insignificant (Fig 1).

Phase 2

Three hundred eleven surgical patients consented to participate in this phase of the study. Six patients had cancellation of the operation, or were transported to the ICU postoperatively and were not included. Another 16 patients were not able to participate in the study because they remained intubated (n = 8) or received supplemental oxygen (n = 8) on arrival to the PACU. The thoracic surgeon would not permit inclusion of his patients in the study. Of the 289 eligible patients, 155 patients were randomized to breathe room air and 134 patients were randomized to receive supplemental oxygen. One patient who was randomized to receive supplemental oxygen was dropped from the study due to noncompliance and refusal to wear the oxygen mask.

There were no intergroup differences in age, gender, use of intraoperative narcotics or muscle relaxants, and the use of narcotics in the PACU (Tables 5, 6). There was an intergroup difference in weight. There were no intergroup differences in preoperative Spo$_2$ (98 ± 2%) or in the Spo$_2$ measurement on arrival to the PACU (97 ± 3%). Seventeen of 289 patients experienced episodes of Spo$_2$ < 90% (5.9%). Fourteen of 155 patients who breathed room air had episodes of Spo$_2$ < 90% compared to 3 of 133 patients who breathed 30%

Table 2—*Variables Reflecting Gas Exchange and Cardiovascular Function for Patients Breathing an Fio$_2$ of 0.21**

Variables	Initial Data†	Final Data‡
Spo$_2$, %	97 ± 2	91 ± 3§
Petco$_2$, mm Hg	35 ± 3	41 ± 3§
Arterial pH	7.37 ± 0.06	7.34 ± 0.06§
Paco$_2$ (range), mm Hg	38.5 ± 3.9 (30–48)	43.5 ± 3.8§ (37–52)
Pao$_2$ (range), mm Hg	89 ± 15 (63–113)	62 ± 12§ (47–94)
HR, beats/min	85 ± 14	84 ± 13
Mean arterial pressure, mm Hg	87 ± 19	87 ± 18

*Data are summarized as mean ± 1 SD (n = 25) unless otherwise indicated.
†Initial data were collected before induction of hypoventilation.
‡Final data were collected 5 min after hypoventilation or when Spo$_2$ became < 90%, whichever occurred first.
§$p < 0.001$ compared to initial value.

Table 4—*Variables Reflecting Gas Exchange and Cardiovascular Function for Patients Breathing an Fio$_2$ of 0.30**

Variables	Initial Data†	Final Data‡
Spo$_2$, %	98 ± 2	97 ± 2
Petco$_2$, mm Hg	36 ± 2	45 ± 2§
Arterial pH	7.38 ± 0.03	7.32 ± 0.04§
Paco$_2$ (range), mm Hg	39.2 ± 3.9 (36–44)	46.7 ± 3.8§ (41–53)
Pao$_2$ (range), mm Hg	112 ± 25 (68–153)	100 ± 17§ (76–126)
HR, beats/min	82 ± 20	82 ± 19
Mean arterial pressure, mm Hg	86 ± 14	87 ± 13

*Data are summarized as mean ± SD (n = 10) unless otherwise indicated.
†Initial data were collected before induction of hypoventilation.
‡Final data were collected 10 min after hypoventilation.
§$p < 0.001$ compared to initial value.

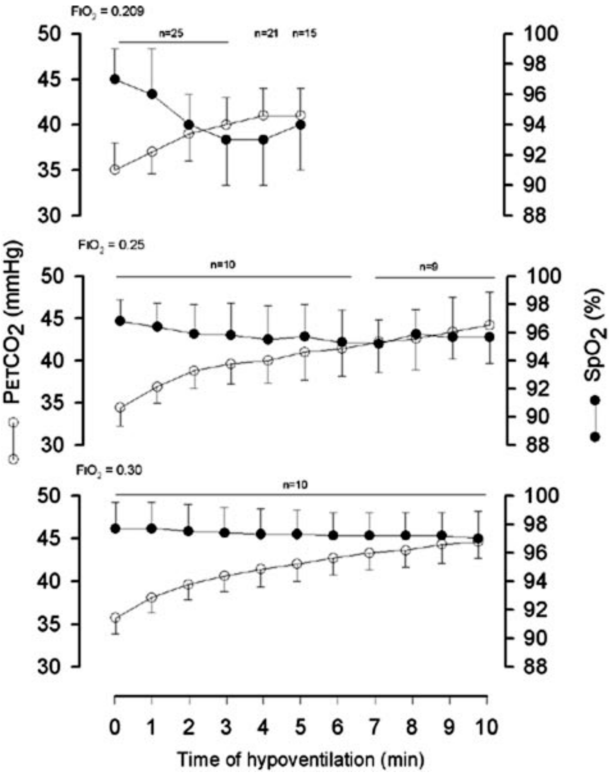

FIGURE 1. Changes in Spo_2 (closed circles) and Petco_2 (open circles) during hypoventilation in patients receiving Fio_2 concentrations of 0.21, 0.25, and 0.30. Baseline values were measured at time = zero minutes. Data are presented as mean ± SD. Data points are connected for clarity. n = No. of patients at each data collection point.

oxygen ($p = 0.02$). The three patients who experienced $\text{Spo}_2 < 90\%$ while receiving supplemental oxygen had immediate restoration of $\text{Spo}_2 \geq 90\%$ with the stir-up regimen. Of the 14 patients who inhaled room air and experienced desaturation, 9 patients had an immediate increase in Spo_2 with the stir-up regimen. The other five patients received supplemental oxygen to restore $\text{Spo}_2 \geq 90\%$. All patients ultimately had $\text{Spo}_2 \geq 90\%$ (Table 7).

DISCUSSION

In the phase 1, the effect of hypoventilation on Spo_2 was modeled in patients under general anesthesia and mechanical ventilation. Increases in Petco_2 and Paco_2 occurred in every patient who underwent deliberate hypoventilation. These changes were accompanied by an immediate decrease in Spo_2 in patients with Fio_2 of 0.21, but not in patients with Fio_2 levels of 0.25 or 0.30. Although a significant decrease in mean Pao_2 occurred during 10 min of hypoventilation in patients with Fio_2 levels of 0.25 or 0.30, mean Pao_2 remained sufficiently elevated to prevent a detectable decrease in Spo_2. When such a decline in Spo_2 occurred, it was not consistent or sufficient to detect a significant change.

Table 5—*Patient Demographics**

Variables	Age, yr	Weight, kg	Male/Female Gender, No.
Air group (n = 155)	57 ± 15	75 ± 18	106/49
Oxygen group (n = 133)	57 ± 13	80 ± 19	87/46

*Data are summarized as mean ± SD unless otherwise indicated. Using Student *t* test, there was no significant intergroup difference in age ($p = 0.63$), but there was a significant intergroup difference in weight ($p = 0.02$). Using the Pearson χ^2 test, there was no significant intergroup difference in sex distribution ($p = 0.59$).

Table 6—*Intergroup Contrasts of Administered Pharmaceuticals**

Variables	Intraoperative Narcotic		Muscle Relaxant		PACU Narcotic	
	Yes	No	Yes	No	Yes	No
Air group (n = 155)	129	26	61	94	81	74
Oxygen group (n = 133)	112	21	70	63	69	64

*Data are presented as No. Using a Pearson χ^2 test, there were no significant intergroup differences in the use of intraoperative narcotics ($p = 0.22$) and muscle relaxants ($p = 0.14$) or PACU narcotics ($p = 0.42$).

Table 7—*Summary of Patients Who Experienced $\text{Spo}_2 < 90\%$ in the PACU**

Patients	Stir-up With Oxygen	General Anesthetic	Intraoperative Narcotic	Muscle Relaxant	PACU Narcotic
Oxygen group					
1	Yes†	Yes	Yes	Yes	Yes
88	Yes†	Yes	Yes	No	No
119	Yes†	Yes	Yes	Yes	Yes
Room air group					
30	Yes‡	Yes	Yes	Yes	Yes
98	No	Yes	Yes	No	Yes
111	No	Yes	Yes	No	No
126	Yes‡	Yes	Yes	Yes	Yes
134	No	Yes	Yes	Yes	Yes
150	No	Yes	Yes	Yes	No
152	No	Yes	Yes	No	Yes
203	Yes‡	Yes	Yes	Yes	No
211	No	Yes	Yes	Yes	No
215	Yes‡	Yes	Yes	No	No
246	Yes‡	Yes	Yes	Yes	No
270	No	Yes	Yes	Yes	Yes
292	No	Yes	Yes	No	Yes
303	No	Yes	No	Yes	Yes

*Nine of 14 patients who experienced $\text{Spo}_2 < 90\%$ breathing room air had a stir-up maneuver without oxygen administration, resulting in immediate restoration of Spo_2 to $> 90\%$.

†Patients who were randomized to receive supplemental oxygen by facemask upon arrival to the PACU ($\text{Fio}_2 = 0.30$).

‡Patients randomized to room air initially, but who received supplemental oxygen via nasal cannula by PACU nursing staff, all of whom sustained $\text{Spo}_2 > 90\%$.

Thus, administration of even small amounts of supplemental oxygen masked our ability to detect hypoventilation with pulse oximetry in anesthetized patients receiving mechanical ventilation.

In healthy volunteers, $PaCO_2$ has been shown to rise at a logarithmic rate of 8 to 25 mm Hg/min after the onset of apnea.[12] A decrease in alveolar ventilation of at least 50%, such as we produced, caused a small, but significant increase in $PaCO_2$ during the brief period of data collection. Given sufficient time for equilibrium, $PaCO_2$ would have doubled, at least. In contrast, PaO_2 fell relatively precipitously. Acute hypoventilation is known to decrease the volume of oxygen delivered to gas-exchanging lung units. However, the rate at which oxygen is removed from the lung by pulmonary capillary blood proceeds at a normal rate. During hypoventilation with room air, the disequilibrium between oxygen extracted from the alveoli and oxygen delivered to the alveoli causes a concentrating effect of nitrogen and carbon dioxide, which further exaggerates the decrease in alveolar oxygen tension (P_AO_2). This sequence explains why, during hypoventilation with room air, mean PaO_2 fell rapidly and markedly by 30 mm Hg while mean $PaCO_2$ increased only 5 mm Hg. The differences in the rate of change of P_AO_2, PaO_2, and $PaCO_2$ during hypoventilation have clinical importance. The rate of decline in PaO_2 and SpO_2 is greater than the increase in $PaCO_2$. Therefore, changes in oxygenation, as measured by pulse oximetry, will provide an earlier indication of hypoventilation than will capnography, but only when breathing room air.[11]

Changes in oxygenation during hypoventilation may be further clarified by examining the equation used to estimate alveolar gas content:

$$P_AO_2 = P_IO_2 - PaCO_2\,[(F_IO_2 + (1 - F_IO_2)/R)]$$

where P_IO_2 is the product of F_IO_2 and barometric pressure minus water vapor pressure, and R is the respiratory gas exchange ratio. This ratio results when the amount of carbon dioxide eliminated by alveolar ventilation is divided by the amount of oxygen taken up by the pulmonary capillary blood, which, under normal metabolic and ventilatory conditions, is typically 0.8. As indicated by the alveolar gas equation, P_AO_2 will decrease in response to increasing $PaCO_2$. During hypoventilation, elimination of carbon dioxide decreases transiently, causing a rapid and significant increase in the volume of oxygen removed relative to the volume of carbon dioxide eliminated. PaO_2 varies directly with P_AO_2. Under unsteady-state conditions, there is a temporary decrease in the respiratory gas exchange ratio to produce a marked fall in PaO_2 accompanied by a modest rise in $PaCO_2$.[11]

When supplemental oxygen is administered, hypoventilation will have a similar, but far less significant effect on P_AO_2 and PaO_2. As we observed, only small increases in inspired oxygen are needed to alleviate any desaturation that might occur secondary to hypoventilation. With supplemental oxygen, relatively less nitrogen in the alveolar gas mixture partially negates the concentrating effect of nitrogen and carbon dioxide in the alveoli, as oxygen is consumed. The effect of supplemental oxygen on masking hypoventilation can be demonstrated further with mathematical modeling of P_AO_2 and PCO_2 as a function of alveolar ventilation and varying F_IO_2 (Fig 2). With an F_IO_2 of 0.30, P_AO_2 is approximately 100 mm Hg (point *a*) when alveolar carbon dioxide tension (P_ACO_2) approaches 90 mm Hg (point *b*), thereby making detection of profound hypoventilation impossible with pulse oximetry. But with an F_IO_2 of 0.21, as P_ACO_2 rises > 65 mm Hg (point *c*), P_AO_2 will decrease to < 60 mm Hg (point *d*), resulting in a $SpO_2 < 90\%$. While breathing room air, a patient cannot hypoventilate sufficiently to elevate $PaCO_2 > 70$ mm Hg without a pulse oximeter reading $< 90\%$, thus precluding the possibility of carbon dioxide narcosis and undetected apnea.[1] With supplemental inspired oxygen as low as 0.25, P_ACO_2 could be nearly 100 mm Hg (point *e*) when P_AO_2 approaches 60 mm Hg (point *f*). Supplementation of inspired oxygen with an $F_IO_2 > 0.25$ could put a patient at risk for carbon dioxide narcosis, before SpO_2 would fall below 90%.

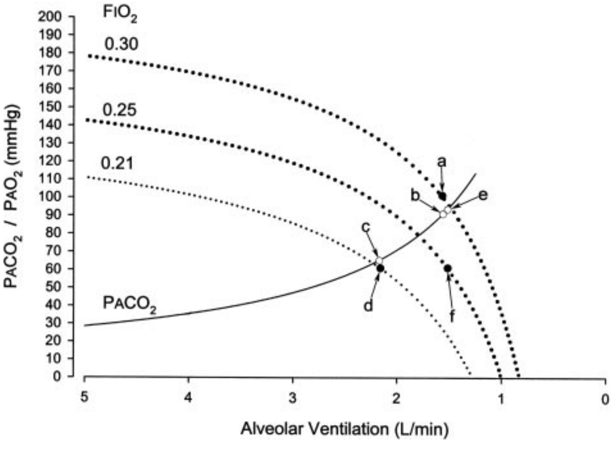

FIGURE 2 Mathematical modeling of P_AO_2 (closed circles) and P_ACO_2 (solid line, open circles) as a function of alveolar ventilation with varied F_IO_2. This model assumes a respiratory gas exchange quotient of 0.8, no physiologic shunting of blood, and a steady state. With an F_IO_2 of 0.3, P_AO_2 is still 100 mm Hg (point *a*) when P_ACO_2 is 90 mm Hg (point *b*). With an F_IO_2 of 0.21, by the time P_ACO_2 has increased to 65 mm Hg (point *c*), P_AO_2 has decreased to 60 mm Hg (point *d*). With an F_IO_2 of 0.25, P_ACO_2 can rise to 100 mm Hg (point *e*) before P_AO_2 decreases to 60 mm Hg (point *f*).

In phase 1, all patients with an FIO_2 of 0.30 maintained $SpO_2 > 90\%$ throughout 10 min of hypoventilation during general anesthesia. In addition, 9 of 10 patients with an FIO_2 of 0.25 maintained $SpO_2 > 90\%$. Based on our modeling of induced hypoventilation in anesthetized patients receiving mechanical ventilation, we hypothesized that in spontaneously breathing patients, the detection of hypoventilation with SpO_2 monitoring may be ineffective when patients are administered supplemental oxygen. Prior publications support this concept.[2,5] The clinical applicability of using pulse oximetry to detect hypoventilation has been limited by the use of supplemental oxygen.[10,13,14] Therefore, in the second phase of the study, we sought to evaluate the clinical utility of pulse oximetry to detect hypoventilation in spontaneously breathing patients in the PACU, with and without administering supplemental oxygen.

Patients who were able to maintain $SpO_2 \geq 90\%$ during room-air breathing on arrival to the PACU were randomized for further data collection. There was an intergroup difference in mean body weight that was small but statistically significant. It is unlikely that this difference confers any clinical significance. The vast majority of the patients who breathed room air did not experience $SpO_2 < 90\%$, yet the administration of supplemental oxygen did not maintain $SpO_2 \geq 90\%$ in all patients. The incidence of $SpO_2 < 90\%$ was four times higher in patients who breathed room air (9.0% vs 2.3%). All but a few patients who breathed room air had immediate restoration of SpO_2 following the stir-up regimen, suggesting that hypoventilation may be an etiologic factor in the decline in SpO_2.

Measurements to confirm hypoventilation with the presence of hypercarbia during episodes of desaturation in the PACU were not undertaken. Drawing arterial blood to measure $PaCO_2$ would have been impractical from a clinical standpoint, since episodes of desaturation were transient. Other methods that quantify carbon dioxide, such as capnography or transcutaneous carbon dioxide, also are fraught with difficulties in the PACU setting.[3,4] Since all other variables and anesthetic management were equivalent, we surmise that respiratory depression and hypoventilation occurred with equivalent frequency in both groups of patients. However, the lower incidence of desaturation in the group of patients who received supplemental oxygen likely was due to a masking effect by the increased FIO_2. In all patients, the transient decrease in SpO_2 easily was managed.

There are six physiologic/pathologic conditions that may lead to arterial oxyhemoglobin desaturation: (1) $FIO_2 < 0.21$; (2) diffusion defect; (3) barometric pressure < 760 mm Hg; (4) right-to-left intrapulmonary shunting of blood; (5) low, but finite, $\dot{V}/\dot{Q}$ ratio; and (6) hypoventilation. In our investigation, only the last three are potential etiologies of $SpO_2 < 90\%$. Since arterial desaturation was transient and reversed by either a stir-up regimen, or an increase in FIO_2, it is unlikely that right-to-left intrapulmonary shunting was a significant cause. Only by analysis of an arterial blood sample could we verify hypoventilation, rather than decreased $\dot{V}/\dot{Q}$ ratio, as the source of arterial desaturation. Nevertheless, increase in FIO_2 to 0.25 or 0.3 will increase PaO_2 and SpO_2, thereby masking the detection of respiratory abnormalities, either hypoventilation or low $\dot{V}/\dot{Q}$. Thus, whether the decrease in SpO_2 is secondary to low $\dot{V}/\dot{Q}$ ratio, a form of "regional hypoventilation," or global hypoventilation resulting in hypercarbia, pulse oximetry monitoring during room air breathing will permit earlier detection of gas exchange abnormalities.

Our findings concur with others[6,13] who have reported the limitation of pulse oximetry in monitoring ventilatory status when supplemental oxygen is administered. Hypercarbia secondary to respiratory depression and not reliably detected by pulse oximetry has been reported during GI endoscopy and bronchoscopy.[2,5] One case report[7] describes a patient receiving morphine patient-controlled anesthesia with high-flow oxygen administration by facemask in whom carbon dioxide narcosis and apnea developed. Pulse oximetry readings between 92% and 95% were recorded, despite a $PaCO_2$ of 102 mm Hg and an arterial pH of 7.08. The authors[7] concluded that pulse oximetry failed to permit detection of opioid-induced respiratory depression, in the presence of supplemental oxygen.

Based on our findings, we advocate the application of supplemental oxygen only in patients who are unable to maintain an acceptable SpO_2 while breathing room air. In patients able to maintain $SpO_2 > 90\%$ on an FIO_2 of 0.21, pulse oximetry monitoring during room air breathing is a useful tool to assess ventilation, without the need for capnography or arterial blood gas analysis. While our data were obtained in the operating room and PACU setting, our results suggest that this type of monitoring also could be utilized in any environment where monitoring of ventilation is needed, such as procedural suites for bronchoscopy and GI endoscopy, where sedation is utilized. Pulse oximetry during room-air breathing also will be useful in guiding and/or limiting the administration of opioids and other respiratory-depressant drugs. Assessment of ventilatory abnormalities in patients receiving epidural, intrathecal, and IV patient-controlled anesthesia narcotics could be achieved with pulse oximetry, but only during room-air breathing.

Historically, an $\mathrm{Spo}_2 < 90\%$ has been used to define "arterial hypoxemia."[15,16] Accordingly, clinicians often will administer supplemental oxygen out of habit to ensure "adequate" oxygenation and to avoid reaching the 90% threshold.[15] But is this clinical practice warranted? Currently, there is no consensus in the literature regarding recommendations on the prophylactic administration of supplemental oxygen to all postoperative patients, and some communications have stressed the dangers of masking severe hypoventilation with supplemental oxygen.[6,7,13] We suggest that the decision to administer supplemental oxygen not be based on routine, but should entail consideration of the risk of masking undetected hypoventilation, or mismatching of ventilation and perfusion, in accordance with the patient's need for increased Spo_2. If persistent, decreased Spo_2 may indicate the need for arterial blood analysis to determine if the arterial hypoxemia is due to hypoventilation, or mismatching of ventilation and pulmonary perfusion. Then, appropriate treatment may be administered.

Sedation may cause profound respiratory depression and hypoventilation. Thus, accurate monitoring of ventilatory status of sedated patients is desirable. Methods to detect hypoventilation in the spontaneously breathing patients receiving respiratory-depressant drugs are limited. Pulse oximetry primarily has been used to assess oxygenation, but not ventilation. A decline in Spo_2 during room-air breathing appears to be a reliable indicator of ventilatory abnormalities, whether occurring at a global or regional level; the presence of such abnormalities will go undetected in the presence of supplemental oxygen. Without the need for capnography and arterial blood gas analysis, pulse oximetry is a useful tool to assess ventilation in the absence of supplemental inspired oxygen.

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