

The Utility of Supplemental Oxygen During Emergency Department Procedural Sedation and Analgesia With Midazolam and Fentanyl: A Randomized, Controlled Trial

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Study objective: To determine whether supplemental oxygen reduces the incidence of hypoxia by 20% in study patients receiving midazolam and fentanyl for emergency department procedural sedation and analgesia.

Methods: Patients were randomized to receive either supplemental oxygen or compressed air by nasal cannula at 2 L per minute. Physicians were blinded to the gas used and end-tidal carbon dioxide (ETCO₂) data. Respiratory depression was defined a priori as oxygen saturation less than 90%, ETCO₂ level greater than 50 mm Hg, an absolute change from baseline of 10 mm Hg, or loss of the ETCO₂ waveform.

Results: Of the 80 patients analyzed, 44 received supplemental oxygen and 36 received compressed air. Twenty supplemental oxygen patients and 19 compressed air patients met at least 1 criterion for respiratory depression. Six supplemental oxygen patients and 5 compressed air patients experienced hypoxia ($P=.97$; effect size 0%; 95% confidence interval -15% to $+15\%$). Fourteen patients in each group met ETCO₂ criteria for respiratory depression but were not hypoxic. Physicians identified respiratory depression in 8 of 11 patients who became hypoxic and 0 of 28 patients who met ETCO₂ criteria for respiratory depression but who did not become hypoxic. There were no adverse events.

Conclusion: Supplemental oxygen did not reduce (or trend toward reducing) the incidence of hypoxia in patients moderately sedated with midazolam and fentanyl. However, our lower-than-expected rate of hypoxia limits the power of this comparison. Blinded capnography frequently identified respiratory depression undetected by the treating physicians. [Ann Emerg Med. 2007;49:1-8.]

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INTRODUCTION

Background

Procedural sedation and analgesia is the use of analgesic, dissociative, and sedative agents to prevent the pain, anxiety, and unpleasant memories associated with painful diagnostic and therapeutic procedures. All the agents used for procedural sedation and analgesia have the potential for serious adverse effects, including respiratory depression.¹

The benefits of supplemental oxygen during procedural sedation and analgesia are unknown. In theory, supplemental oxygen may prevent hypoxemia in some patients, which has prompted the American Society of Anesthesiology to recommend supplemental oxygen for patients undergoing deep

sedation (and to suggest that it be considered during moderate sedation).² However, patients receiving supplemental oxygen may have normal oxygen saturation despite significant ventilatory depression.³ This could hinder the physician's ability to recognize respiratory depression, leading to serious adverse events. The utility of supplemental oxygen during emergency department (ED) procedural sedation and analgesia has not been adequately studied. As a result, the use of supplemental oxygen is generally in accordance with institutional protocols or at the discretion of the treating physician.

Importance

Procedural sedation and analgesia has become an integral part of emergency medicine practice. Agents such as propofol

Editor's Capsule Summary*What is already known on this topic*

There is no current evidence to support or refute the use of prophylactic oxygen during procedural sedation and analgesia, and such supplementation may delay clinical recognition of respiratory depression by delaying oxygen desaturation.

What question this study addressed

Do oxygen desaturation and respiratory depression occur more or less frequently when supplemental oxygen is administered during moderate sedation with midazolam and fentanyl?

What this study adds to our knowledge

The incidence of hypoxia and respiratory depression was similar with and without supplemental oxygen, although these comparisons are not powerful enough to exclude clinically important differences.

How this might change clinical practice

Although not definitive, this best available study does not show any apparent advantage (or any trend toward an advantage) to the use of supplemental oxygen during moderate sedation.

Research we'd like to see

Replication of this study would be useful in the setting of deep sedation with propofol or etomidate, in which a higher baseline rate of respiratory depression might better define any marginal benefit from supplemental oxygen.

and etomidate, once reserved for the operating suite, are now routinely used in the ED setting.¹ These agents, as well as others used for ED procedural sedation and analgesia (eg, barbiturates, benzodiazepines, opiates), may cause significant respiratory depression.¹ If supplemental oxygen can limit the incidence or severity of hypoxia without masking the presence of underlying respiratory depression, it should be incorporated into standard ED procedural sedation and analgesia protocols. If supplemental oxygen prevents hypoxia but masks respiratory depression, then additional precautions such as monitoring end-tidal carbon dioxide (ETCO₂) may be indicated.⁴ If supplemental oxygen does not prevent hypoxia, its use should be abandoned.

Goals of This Investigation

The goal of this study was to determine whether supplemental oxygen delivered at 2 L per minute by nasal cannula would reduce the incidence of hypoxia by 20% in study patients receiving midazolam and fentanyl for ED procedural sedation and analgesia.

MATERIALS AND METHODS**Study Design**

This was a prospective, randomized, double-blind, placebo-controlled study conducted between October 1, 2004, and March 1, 2005. The institutional review board at the Albert Einstein Medical Center approved the study.

Setting and Selection of Participants

The study was performed in the ED at the Albert Einstein Medical Center, a Level I trauma center located in Philadelphia, PA. The ED features a well-established emergency medicine residency program and has an annual census of approximately 70,000 patient visits.

All patients older than 2 years and receiving fentanyl and midazolam to facilitate a painful procedure were eligible for the study. Enrollment occurred after the attending physician made the decision that fentanyl and midazolam would be safe and appropriate for procedural sedation and analgesia. Consecutive patients who met inclusion criteria were enrolled 24 hours a day, 7 days a week during the study period.

Patients were excluded if they had severe chronic obstructive pulmonary disease, long-term oxygen use, hemodynamic instability, respiratory distress, pregnancy, allergy to any of the study drugs, or inability to provide informed consent.

Informed consent was obtained from each subject or, in the case of minors, the subject's parent or legal guardians. School-aged children were also asked to sign an assent form.

Patients were randomized to receive either supplemental oxygen or room air at 2 L per minute by nasal cannula. Randomization was done with a computerized randomization table. Patients were assigned to their respective groups sequentially down a numbered list.

Procedural sedation and analgesia was performed according to our standard ED protocol, which includes a medical history and focused physical examination, placement of an intravenous line, and mechanical monitoring of ECG, oxygen saturation, pulse rate, respiratory rate, and noninvasive blood pressure. Procedural sedation and analgesia in our department requires the presence of an emergency attending physician and an ED nurse. The physician is responsible for ordering procedural sedation and analgesia drugs and performing the procedure, whereas the nurse is responsible for administering the drugs and monitoring the patient. An emergency medicine resident is also generally present but this is not an institutional or departmental regulation. For this study, a research associate was also present and was responsible for ensuring appropriate patient selection, randomization, and data collection. The research associates are non-board-certified physicians who received specific training about procedural sedation and analgesia, the study protocol, and data collection techniques, including identifying interventions by the treatment team to improve oxygenation or ventilation.

After collection of baseline data, patients were randomized to receive room air or oxygen by nasal cannula at 2 L per minute. To ensure that the treatment team was blinded to the type of gas being administered, the gases were delivered from one of 2

Table 1. Ramsay scale.

Level	Description
1	Anxious, agitated, restless
2	Cooperative, oriented, tranquil, accepts mechanical ventilation
3	Responds to commands only
4	Brisk response to light glabellar tap or loud noise
5	Sluggish response to light glabellar tap or loud noise
6	No response to light glabellar tap or loud noise

identical-appearing D-tanks marked “A” and “B.” Intravenous midazolam and fentanyl were titrated to the desired level of sedation and analgesia, and the procedure was performed. Patients were monitored until back to their baseline alertness.

Data Collection

Age, sex, medical history, medications, and allergies; type of procedure performed; and sedation and procedure times were recorded by the research associates with a standardized data collection instrument. Procedure time was defined as the time from initial medication administration until the patient returned to baseline alertness. The research associates measured alertness levels using a 6-point Ramsay scale, with 1 indicating agitation and 6 indicating unresponsiveness (Table 1). This scale has been validated in ICU patients and used in a number of studies of ED procedural sedation and analgesia.⁵⁻⁷ A Ramsay score was recorded at baseline, 90 seconds after completion of drug administration, and when it appeared the patient was back to baseline alertness.

Vital signs (pulse rate, respiratory rate, and blood pressure), oxygen saturation, and ETCO_2 levels were recorded at baseline and every 5 minutes until the patient returned to baseline alertness. ETCO_2 was monitored with the NPB-Microstream 75 ETCO_2 monitor (Nellcor Puritan Bennett Inc., Pleasanton, CA) connected to a nasal cannula capable of delivering compressed gases and fitted with an oral ETCO_2 sampler to accommodate for mouth breathers (Smart Capnoline O₂ Nasal Cannulas; Oridion Inc., Jerusalem, Israel). The NPB-Microstream 75 ETCO_2 monitor samples continuously at a rate of 50 mL per minute and can process up to 150 breaths/min. Nasal cannula-derived ETCO_2 values are as accurate as those derived from endotracheal tubes.^{4,8} Studies have shown that the ETCO_2 levels measured by nasal cannula closely approximate arterial CO_2 levels.⁹⁻¹³ In an effort to determine whether procedural sedation and analgesia providers recognize respiratory depression using standard monitoring techniques (ie, clinical observation, pulse oximetry, continuous ECG, and noninvasive blood pressure, pulse rate, and respiratory rate), the treatment team was kept blinded to ETCO_2 levels.

Outcome Measures

Respiratory depression was defined a priori as oxygen saturation less than 90%, ETCO_2 greater than 50 mm Hg, an absolute ETCO_2 change from baseline of greater than 10 mm Hg,

or loss of the ETCO_2 waveform. These criteria were considered present if they occurred any time during the procedure, regardless of their duration. When capnographic evidence of respiratory depression occurred, the patient’s vital signs, oxygen saturation, and ETCO_2 level were recorded. The research associates also recorded these parameters if any member of the treatment team verbalized that the patient was experiencing respiratory depression or provided an intervention to assist breathing, including verbal or physical stimulation, airway realignment, use of additional oxygen (from a wall source) or airway adjuncts, assisted ventilation, or intubation. Other adverse events, including hypotension, bradycardia, vomiting, prolonged ED stay (>2 hours after the procedure), or admissions, were also recorded on the data collection instrument.

Clinical staff were unaware that the research associates were evaluating their (ie, the clinical staff’s) ability to recognize respiratory depression using standard procedural sedation and analgesia monitoring. Members of the clinical staff were not asked to sign an informed consent because we felt that this could result in significant bias. The institutional review board agreed that because individual staff members would not be identified in any way, staff consent was not necessary.

Primary Data Analysis

Data analysis was performed using SPSS statistical software (SPSS, Inc., Chicago, IL). The incidence of hypoxia in each group was compared by using the 2-sample test of proportions. The number of interventions to treat respiratory depression in each group was compared by using the χ^2 test. Data are presented with 95% confidence intervals where appropriate. A $P < .05$ was used to denote statistical significance. The study was powered to test the null hypothesis that there is no difference in the incidence of hypoxia in patients receiving supplemental oxygen and in those receiving room air during ED procedural sedation and analgesia using midazolam and fentanyl. Previous procedural sedation and analgesia studies evaluating midazolam and fentanyl have found that hypoxia occurs in 10% to 30% of patients.^{5,14,15} For our power calculation, we assumed that lowering the absolute incidence of hypoxic events by 20% would be clinically significant. Using Fischer’s exact test of 2 means (1-tailed test), with group 1 at 25% and group 2 at 5%, a power of 80%, and an α of 0.05, we calculated that the study would require approximately 48 patients per group. We decided to analyze our data at 80 patients because of the American College of Emergency Physicians abstract submission deadline, without breaking the blind.

RESULTS

Of 140 patients screened, 83 patients were enrolled in the study. Three patients were subsequently excluded, leaving 80

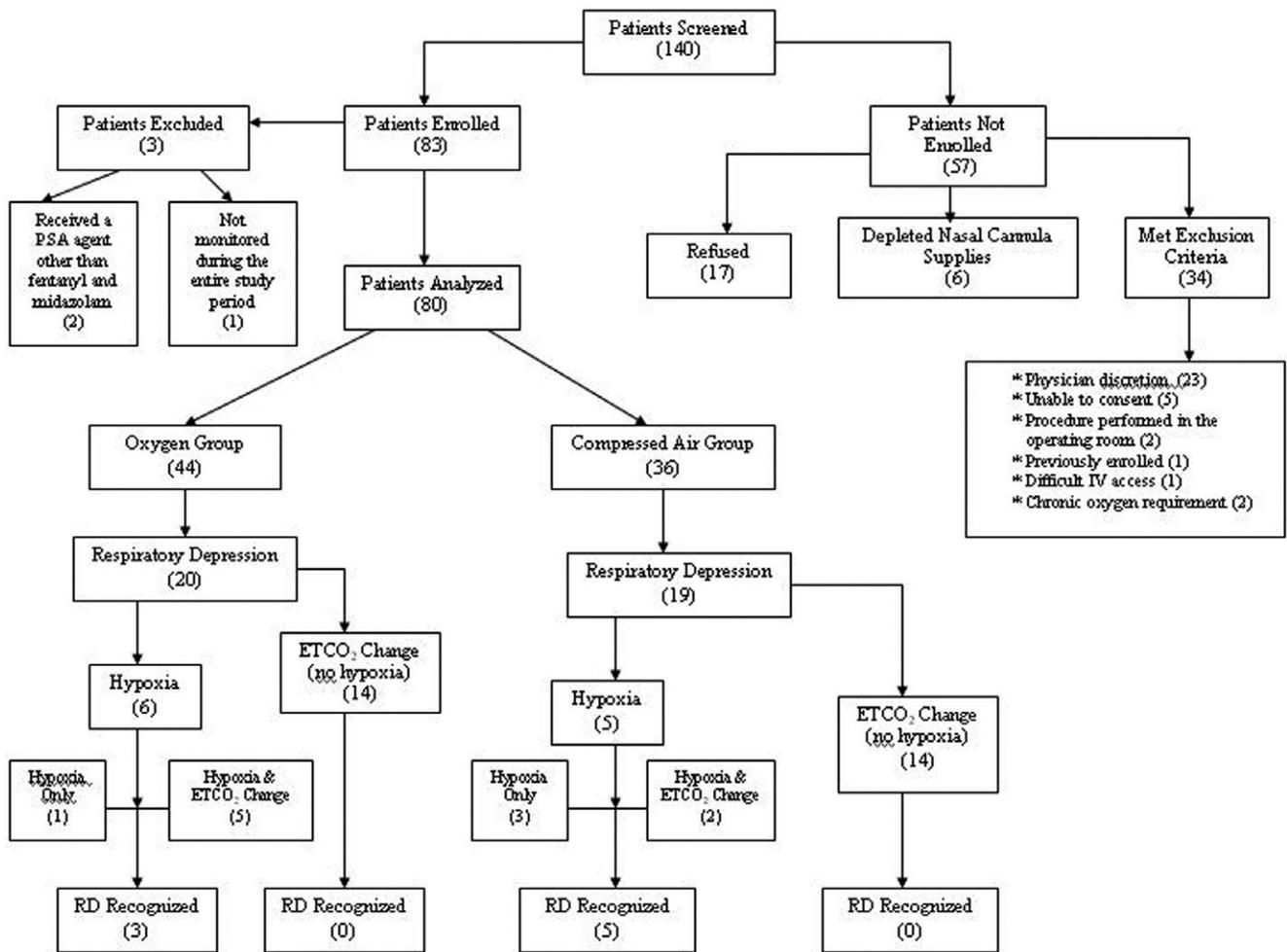


Figure. Schematic representation of study results. PSA, Procedural sedation and analgesia; RD, respiratory depression.

Table 2. Patient characteristics.

Characteristics	Supplemental Oxygen (n=44)	Room Air (n=36)
Median age, y	36 (Range 2–77)	32 (Range 13–68)
Sex (female), No.	24	24
Children by ages, y	0–2: 0 2–6: 2 12–18: 4	0–2: 0 2–6: 0 12–18: 6
Median weight, kg	77 (Range 18–109)	80 (Range 45–147)
Abscess incision and drainage (%)	26 (59)	24 (67)
Fracture/joint reduction (%)	17 (39)	10 (28)
Other procedures* (%)	1 (2)	2 (5)
Median fentanyl dose, µg	150.0 (Range 25–400)	100.0 (Range 50–400)
Median midazolam dose, mg	5.0 (Range 0.5–10.0)	4.0 (Range 1.0–8.0)
Median Ramsey scores (90 s after the last dose of preprocedural medication)	4 (Range 2–5)	4 (Range 3–5)
Median time from first dose of medication to baseline alertness, min	35 (Range 5–100)	38 (Range 9–75)

*Tube thoracostomy, lumbar puncture, and hemorrhoid thrombectomy.

patients for analysis (Figure). The 2 groups were similar with respect to age, sex, and weight (Table 2). Abscess incision and drainage and fracture and joint reduction accounted for the

majority of procedures (Table 2). There were no differences between the groups in the type or duration of procedures performed, the median doses of midazolam and fentanyl

Table 3. Patients experiencing respiratory depression.

Patients	Supplemental Oxygen (n=44), No. (%)	Room Air (n=36), No. (%)	Effect Size, % (95% Confidence Interval)
Total number of patients meeting 1 or more criteria* for RD	20 (45)	19 (52)	7 (-29 To 15)
Patients meeting both oxygen saturation and ETCO ₂ criteria* for RD	5 (11)	2 (6)	6 (-6 To 18)
Patients meeting only oxygen saturation criteria* for RD	1 (2)	3 (8)	6 (-16 To 4)
Patients meeting only ETCO ₂ criteria* for RD	14 (32)	14 (38)	7 (-28 To 14)

*Criteria for respiratory depression: Oxygen saturation <90%, ETCO₂ >50 mm Hg, an absolute ETCO₂ change from baseline of >10 mm Hg, or loss of the ETCO₂ waveform.

Table 4. Patients with changes in ETCO₂.

ETCO ₂ Criteria	Supplemental Oxygen (n=19)*	Room Air (n=16)*
ETCO ₂ >50 mm Hg	12	8
Absolute ETCO ₂ change of >10% from baseline	12	9
Loss of the ETCO ₂ waveform	7	7

*Some patients met more than 1 ETCO₂ criterion.

administered, the depth of sedation achieved, or the time to return to baseline alertness (Table 2).

Six patients in the supplemental oxygen group and 5 patients in the room air group experienced oxygen saturations less than 90% ($P=0.97$; effect size 0%; 95% confidence interval -15% to 15%) (Table 3).

Of the patients that met 1 or more criteria for respiratory depression, there were no differences between those patients receiving supplemental oxygen and those receiving room air. Thirty-five percent of all study patients met 1 or more ETCO₂ criteria for respiratory depression but did not experience hypoxia (Table 3). Tables 4 and 5 provide a detailed summary of ETCO₂ data.

Physicians identified respiratory depression in 8 of 11 patients who experienced hypoxia, including 3 of 6 patients in the supplemental oxygen group and 5 of 5 patients in the room air group (Table 6). Physicians failed to recognize respiratory depression in any patient who did not become hypoxic.

When oxygen saturation decreased below 90%, the median duration of hypoxia was 60 seconds in both groups (supplemental oxygen group, range 1 to 120 seconds; room air group, range 30 to 240 seconds). In contrast, in patients who met only ETCO₂ criteria for respiratory depression, the median duration of respiratory depression was longer in the patients receiving supplemental oxygen (417 seconds; range 3 to 600 seconds) compared with that of patients breathing room air (180 seconds; range 60 to 540 seconds).

No patients in either group experienced hypotension, bradycardia, or vomiting or required assisted ventilation, intubation, or admission. There were no other adverse events noted.

Table 5. Summary of ETCO₂ values >10% from baseline.

Patient	Baseline ETCO ₂ (mm Hg)	Highest or Lowest ETCO ₂ Value (mm Hg)
Supplemental oxygen group		
2	21	43
4	27	45
13*	22	24, 61
30*	39	9, 53
32	44	58
47	38	54
49	37	47
51*	45	36, 56
61	35	55
71	38	56
79*	34	21, 51
80	34	47
Room air group		
1	38	53
17	39	49
18	35	50
22	29	44
23	42	53
34	23	43
39	25	44
40	37	53
81	36	52

*These patients experienced a >10% change in ETCO₂ above and below their baseline value.

LIMITATIONS

We administered oxygen at 2 L per minute to the subjects in the treatment group. Although the addition of higher flow rates may have reduced the incidence of hypoxia in patients receiving supplemental oxygen, we believe this flow rate is consistent with common practice.

The results of our study indicated that the use of supplemental oxygen did not reduce the incidence of hypoxia in patients undergoing ED procedural sedation and analgesia with fentanyl and midazolam. The study was powered to detect a 20% difference in the incidence of hypoxic events. This decision was based on our collective experience and resulting opinion that 20% represents a clinically important difference. To prepare an abstract for the American College of Emergency Physicians *Research Forum*, we performed an analysis of the data

Table 6. Physician recognition of patients experiencing respiratory depression.*

Patients Identified	Supplemental Oxygen (n=20)	Room Air (n=19)
Number of patients identified with RD [†] /total number of patients who met 1 or more criteria* for RD	3/20	5/19
Number of patients identified with RD [†] /total number of patients who met oxygen saturation and ETCO ₂ criteria* for RD	2/5	2/2
Number of patients identified with RD [†] /total number of patients who met only oxygen saturation criteria* for RD	1/1	3/3
Number of patients identified with RD [†] /total number of patients who met only ETCO ₂ criteria* for RD	0/14	0/14

*Criteria for respiratory depression: Oxygen saturation <90%, ETCO₂ >50 mm Hg, an absolute ETCO₂ change from baseline of >10 mm Hg, or loss of the ETCO₂ waveform.

[†]The treatment team was given credit for identifying respiratory depression if any member of the team verbalized that the patient was experiencing respiratory depression or provided an intervention to assist breathing, including verbal or physical stimulation, airway realignment, use of additional oxygen or airway adjuncts, assisted ventilation, or intubation.

after enrolling 80 patients. This interim analysis revealed that only 13.9% of subjects had developed hypoxia. At this rate (ie, 13.9%), a reduction in the incidence of hypoxia of 20% (which was the basis for our initial power calculation) was impossible. Thus, the study was ended at 80 patients. Had we enrolled the 16 additional patients required by our power calculation, it is possible that the incidence of hypoxia could have reached 25%, making a reduction of 20% possible. However, this would have required that more than half of the 16 patients develop hypoxia, which we thought was highly unlikely. Thus, it is possible that a difference of less than 20% exists but that our study was not powered to identify this difference.

Members of the clinical staff were blinded to the type of gas being administered and to the ETCO₂ data, and they were unaware that their ability to recognize respiratory depression was being evaluated. Nevertheless, it is possible that knowing the study was evaluating the use of supplemental oxygen heightened their awareness for identifying respiratory depression. However, the potential for heightened awareness would be the same whether the patient was receiving supplemental oxygen or room air and therefore would not be expected to affect the results.

The research associates were not blinded to the purpose of this study, which could have resulted in bias during data collection. However, the research associates made no patient care decisions; they were present only to ensure protocol adherence and accurate data collection. In addition, all research associates participating in this study were physicians who received training specifically directed at identifying a physician intervention for respiratory depression.

Compared with adults, young children have a higher basal metabolic rate, increased oxygen consumption, and lower functional residual capacity, which results in less tolerance to hypoxia. We enrolled 12 patients younger than 18 years, but only 2 were younger than 12 years (Table 2). This number is insufficient to draw any conclusion about the use of supplemental oxygen during procedural sedation and analgesia in young children.

DISCUSSION

Supplemental oxygen (2 L/minute by nasal cannula) did not reduce (or trend toward reducing) the incidence of hypoxia in patients moderately sedated with midazolam and fentanyl. However, our lower-than-expected rate of hypoxia limits the power of this comparison. This is the first study specifically designed to evaluate the potential benefits or hazards of supplemental oxygen. Previous studies have provided some insight about the use of supplemental oxygen. However, none have been specifically designed to determine whether supplemental oxygen prevents hypoxia during ED procedural sedation and analgesia.

Miner et al^{9,16,17} compared adverse events in procedural sedation and analgesia patients with and without supplemental oxygen in 3 nonrandomized studies and noted conflicting results. In a study evaluating the usefulness of ETCO₂ monitoring during ED procedural sedation and analgesia, 5 of 47 patients (10.6%) receiving supplemental oxygen experienced hypoxia compared with 6 of 27 patients (22.2%) breathing room air.⁹ In another study of bispectral encephalographic analysis during ED procedural sedation and analgesia using a similar drug regimen, hypoxia was observed in 13 of 87 patients (14.9%) receiving supplemental oxygen compared with only 1 of 21 patients (4.8%) breathing room air.¹⁶ In a third study comparing propofol and methohexital for fracture and dislocation reduction, hypoxia was noted in 5 of 59 patients (8.5%) receiving supplemental oxygen compared with 6 of 44 patients (13.6%) breathing room air.¹⁷ These studies demonstrate conflicting results about the use of supplemental oxygen during ED procedural sedation and analgesia. However, they were neither blinded nor randomized, and none were specifically designed to evaluate the use of supplemental oxygen.

Respiratory depression during ED procedural sedation and analgesia may be associated with the depth of sedation rather than the specific agent(s) used.¹⁶ If this is true, our data suggests that supplemental oxygen might be unnecessary with any procedural sedation and analgesia regimen that provides moderate sedation. Nevertheless, we believe it would be prudent to wait for supporting data from additional studies before extrapolating the results of this study to other procedural sedation and analgesia regimens.

We found that physicians identified the presence of respiratory depression in 8 of 11 patients (73%) who experienced hypoxia (whether or not they also met ETCO₂ criteria for respiratory depression) compared with none of the 28 patients who met only ETCO₂ criteria (ie, never became

hypoxic). This result suggests that monitoring oxygen saturation is helpful in recognizing respiratory depression in patients undergoing ED procedural sedation and analgesia. There were no adverse events in any of the patients who became hypoxic. Whether this was the result of physician intervention (8 of 11) or simply a lack of adverse events associated with transient hypoxia (3 of 11) is unknown and a question our study was not designed to answer.

It is unclear why hypoxia was not recognized in 3 of the 6 patients in the supplemental oxygen group whose oxygen saturation decreased below 90%. The duration of hypoxia in all 3 patients was brief (<60 seconds), and the severity was mild (lowest oxygen saturation was >87%). It is possible that the ED staff simply missed these brief periods of hypoxia, or perhaps the physicians managing these cases decided to initially observe rather than treat such mild rapidly resolving hypoxia. The design of this study does not allow us to definitively answer this question.

Physicians in our study did not recognize respiratory depression unless the patient became hypoxic. Previous studies of procedural sedation have suggested that capnography may allow earlier (ie, before hypoxia occurs) recognition of respiratory depression.^{13,18,19} Our study was not designed to determine whether monitoring ETCO_2 results in earlier recognition of respiratory depression during ED procedural sedation and analgesia, but our results suggest the need for just such a study.

In patients with unobstructed airways, hypoventilation causes ETCO_2 levels to increase,⁹ whereas hypoventilation in the presence of a developing airway obstruction produces a decrease in ETCO_2 or loss of the ETCO_2 waveform.²⁰ It has been suggested that an absolute ETCO_2 change from baseline of greater than 10 mm Hg or loss of the ETCO_2 waveform may identify patients at risk for developing clinically significant respiratory depression,¹⁷ signaling clinicians to intervene by stimulating breathing (for increasing ETCO_2), repositioning the airway (for decreasing ETCO_2 or loss of the ETCO_2 waveform), or withholding additional sedatives.⁸ In our study, 28 patients experienced ETCO_2 changes consistent with respiratory depression, yet despite a lack of physician intervention, none developed hypoxia or other adverse events. According to our results, further studies are needed to better understand the implications and clinical significance of ETCO_2 changes during ED procedural sedation and analgesia.

We did note that in the 28 patients meeting only ETCO_2 criteria for respiratory depression, the ETCO_2 changes lasted significantly longer in patients receiving supplemental oxygen compared with those breathing room air. Although the reasons for this are not clear, we speculate that patients who experienced changes in ETCO_2 without hypoxia may have had a decrease in oxygen saturation, though not below 90%. This decrease in oxygen saturation may have been delayed in patients breathing supplemental oxygen, resulting in a longer period of respiratory depression. According to our data collection methodology, we were not able to definitively explain this finding. We plan to

more closely follow oxygen saturations during periods of subclinical respiratory depression.

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Author contributions: KD and CRC conceived the study and designed the trial. KD, CRC, and PD supervised the conduct of the trial and data collection. KD, CRC, and PD managed the data, including quality control. PD provided statistical advice on study design and analyzed the data. KD drafted the manuscript, and all authors contributed substantially to its revision. KD takes responsibility for the paper as a whole.

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