Advances in the Use of Capnography for Nonintubated Patients

Krauss, Baruch, MD, EdM[a]

[a] *Department of Emergency Medicine, Children's Hospital, Longwood and Department of Pediatrics Harvard Medical School, Boston MA, USA*

Abstract

This article reviews recent advances in the use of capnography as a diagnostic monitoring modality for nonintubated patients. These include assessing the response to treatment in patients in acute respiratory distress, determining the adequacy of ventilation in patients with altered mental status (including drug-induced alterations in consciousness during procedural sedation and analgesia and patient-controlled analgesia), assessing the ventilatory status of actively seizing patients, and detecting metabolic acidosis in patients with diabetes and gastroenteritis.

MeSH Words: Capnography, end-tidal CO_2, nonintubated

Introduction

Although the standard of care in anesthesia practice is well established for intubated patients, there has been little emphasis on the use of capnography in nonintubated patients outside of the operating room. Capnography has many clinical applications in both intubated and nonintubated patients and has been found valuable by emergency medicine physicians and Emergency Medical Services (EMS) in the prehospital as well as in-hospital setting. In addition to confirming the placement of the endotracheal tube and monitoring the tube position during transport, capnography can provide qualitative and quantitative assessments of cardiac output, gauge the effectiveness of cardiopulmonary resuscitation during cardiac arrest, determine prognosis in cases of cardiopulmonary resuscitation and trauma, titrate end-tidal carbon dioxide ($EtCO_2$) levels in patients with suspected increases in intracranial pressure, assess response to treatment in patients in acute respiratory distress, determine the adequacy of ventilation in patients with altered mental status (including drug-induced alterations in consciousness during non-operating-room anesthesia), assess the ventilatory status of actively seizing patients, and detect metabolic acidosis in patients with diabetes and gastroenteritis. The purpose of this article was to review the uses of capnography in nonintubated patients in emergency medicine and to discuss recent advances in its application as a diagnostic monitoring modality.

Indications for Capnography in Nonintubated Patients

The use of capnography in nonintubated patients has been studied in a variety of conditions (Table 1)

Table 1.

Clinical applications of capnography in nonintubated patients
Rapid assessment of airway, breathing, and circulation in critically ill or injured patients
Assessment of ventilatory status of actively seizing patients
Assessment and triage of victims of chemical terrorism
Assessing response to treatment for patients in acute respiratory distress
Determining adequacy of ventilation in patients with altered mental status
Detection of diabetic ketoacidosis
Detection of metabolic acidosis in gastroenteritis

Assessment of Critically Ill or Injured Patients

Airway integrity, breathing, and circulation can be rapidly assessed in critically ill or injured patients using the capnogram and $EtCO_2$ values (Fig. 1). The presence of a normal waveform denotes a patent airway and spontaneous breathing [1,2]. Normal $EtCO_2$ levels signify adequate perfusion [2,3]. Because the CO_2 measurement is airway-based and not muscle-based, capnography does not misinterpret motion artifacts and provides reliable readings in low-perfusion states.

Assessment of Ventilatory Status in Seizing Patients

Capnography is the only accurate and reliable modality available for monitoring actively seizing patients [4,5]. On the basis of capnographic data (capnogram, $EtCO_2$, respiratory rate), physicians and paramedics can distinguish among actively seizing patients with central apnea (flatline waveform, no $EtCO_2$ readings, no chest wall movement), actively seizing patients with ineffective ventilation and low tidal volume breathing (small waveform, low $EtCO_2$ values), and actively seizing patients with effective ventilation (normal waveform, normal $EtCO_2$ values).

Assessment of Victims of Chemical Terrorism

EMS systems have intensively focused on training in the effective management of mass casualty chemical terrorism events. Capnography provides a noninvasive assessment of the airway, breathing, and circulation and can facilitate the rapid identification of the common life-threatening complications of nerve gas exposure (Table 2) [4,6,7]. It can rapidly detect adverse events in the airway, respiratory, and central nervous system associated with nerve agents, including apnea, upper airway obstruction, laryngospasm, bronchospasm, and respiratory failure.

Assessment of Response to Treatment in Patients in Acute Respiratory Distress

Capnography may serve as a dynamic tool for monitoring the ventilatory status of patients in acute respiratory distress due to asthma, bronchiolitis, chronic obstructive pulmonary disease, congestive heart failure, croup, and cystic fibrosis. By measuring respiratory rate and $EtCO_2$ with each breath, capnography provides the clinician with instantaneous feedback.

Because the respiratory rate is measured directly from the airway via a nasal-oral cannula, the readings are more reliable than with impedance respiratory monitoring. In patients with obstructive apnea, impedence monitoring may interpret chest wall movements as a valid breath and display a respiratory rate even though the patient is not ventilating. By contrast, the capnogram will show a flatline waveform.

In tachypneic patients, $EtCO_2$ trends can be rapidly assessed with capnography. A patient with a respiratory rate of 30 breaths per minute will produce 150 $EtCO_2$ readings in 5 minutes. This provides sufficient information for the physician or paramedic to determine the vector of the patient's ventilatory status: worsening despite treatment (increasing $EtCO_2$ from baseline); stabilizing (stable $EtCO_2$); or improving (decreasing $EtCO_2$ from baseline). In some patients, it may be helpful to determine if there is a difference between the $EtCO_2$ and the partial pressure of carbon dioxide (pCO_2) and to quantify the gradient [8,9]. With correction for the gradient, the $EtCO_2$ trends can then be used as a substitute for serial pCO_2 measurements.

Figure 1
Capnographic Assessment of Airway, Breathing, and Circulation

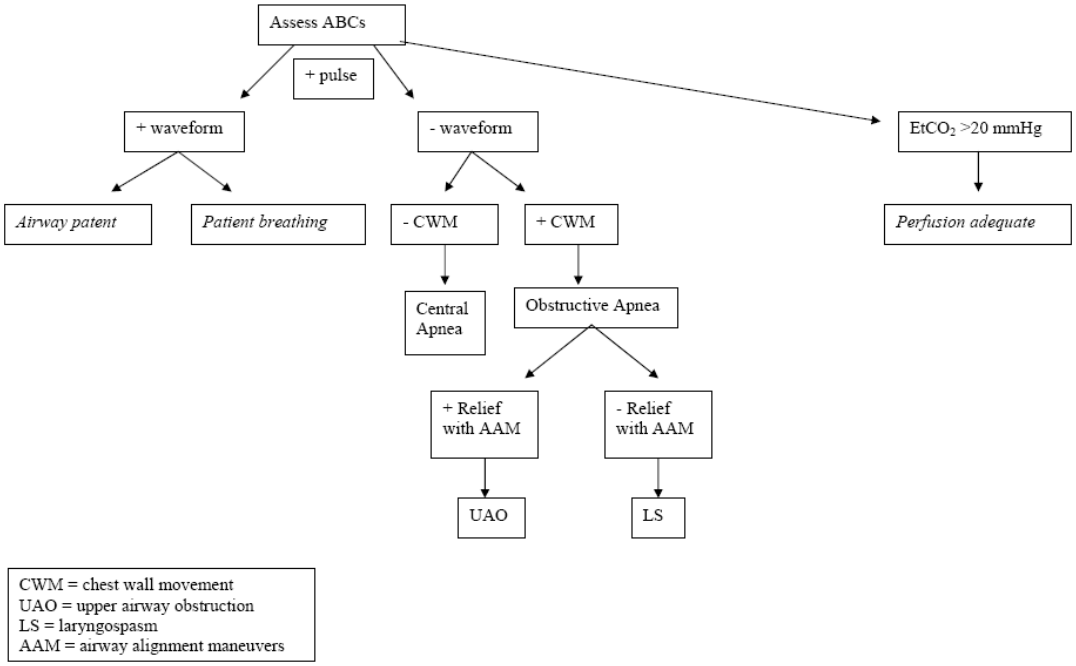


Table 2. Capnography identification of potential life-threatening complications of chemical agents.

Agent	Effects	Capnography
Nerve gas • Tabun • Sarin • Soman • VX	Seizures, diaphragmatic weakening or paralysis, hypoventilation, respiratory depression, apnea, loss of consciousness/coma	• Accurate readings during seizure activity (RR, $EtCO_2$, waveform) • Earliest indicator of respiratory compromise
Vesicants • Mustard gas • Lewisite	Airway edema, upper airway obstruction, bronchospasm	• Rapid identification of upper airway obstruction • Rapid identification of bronchospasm
Choking agents • Chlorine • Phosgene • Diphosgene • Chloropicrin • Ricin	Rapid, progressive, non-cardiogenic pulmonary edema and acute lung injury, bronchospasm, laryngospasm	• Earliest indicator of respiratory compromise • Rapid identification of bronchospasm • Rapid identification of laryngospasm
Cyanide	Sudden loss of consciousness/coma, seizures, metabolic acidosis with tachypnea, apnea	• Accurate readings during seizure activity • Earliest indicator of respiratory compromise • Non-invasive identification of metabolic acidosis
Incapacitating agents • Lacrimators (Mace) • Capsaicin	Laryngospasm, bronchospasm, respiratory failure	• Rapid identification of laryngospasm • Rapid identification of bronchospasm • Earliest indicator of respiratory compromise

Assessment of Obstructive Lung Disease

Patients with normal lung function, irrespective of age, have a characteristic rectangular or trapezoid-shaped capnogram and a narrow $EtCO_2$-pCO_2 gradient (0-5 mmHg), with the $EtCO_2$ accurately reflecting the pCO_2 [10]. The capnograms of patients with restrictive lung disease are similar to normal capnograms in six quantitative parameters: $EtCO_2$ value, respiration rate, take-off angle of the initial expiratory rise, elevation angle for the slope of the alveolar plateau, inspiratory time, and expiratory time (Fig. 2) [11]. However, in patients with abnormal lung function from ventilation-perfusion mismatch, the gradient will widen, depending on the severity of the lung disease, and the $EtCO_2$ will be useful for trending the ventilatory status, but not as a spot check which may or may not correlate with the concentration of carbon dioxide in arterial blood ($PaCO_2$) [8,9]. The capnograms of patients with obstructive lung disease are characterized by a more rounded ascending appearance during the initial phase of exhalation and an upward slope during the alveolar plateau (Fig. 2) [11]. This shape correlates with the changes in forced expiratory volume in one second (FEV_1) and peak expiratory flow rate [10-14]. The marked differences from the normal capnogram, particular in the angles of the initial expiratory rise and in the alveolar plateau, are progressive, and their magnitude increases with increasing severity of the respiratory impairment (as documented by a decreasing FEV_1) (Figs. 3,4). The differences from the normal capnogram are sufficiently large to suggest that capnography may be used as a non-effort-dependent method for distinguishing patients with obstructive lung disease from patients with normal lung function [11].

The capnogram is also useful as an objective measure of ventilatory status in asthmatic patients who are unwilling or unable to cooperate with spirometry (e.g., young children, patients on mechanical ventilation, and patients in acute respiratory distress) [12,13]. With further study, the capnogram may also prove beneficial as a screening tool to identify subjects who have underlying obstructive lung disease or patients who may be at risk of acquiring lower airway obstruction during procedural sedation and analgesia.

Assessment of the Adequacy of Ventilation in Patients with Altered Mental Status

Obtunded or unconscious patients, including those with alcohol intoxication or intentional or unintentional drug overdose and postictal patients (especially those treated with benzodiazepines), may have impaired ventilation. Capnography can differentiate patients with effective and ineffective ventilation as well as provide continuous monitoring of ventilatory trends over time so patients at risk of respiratory depression and respiratory failure can be identified.

Procedural Sedation and Analgesia

Capnography can detect the common adverse airway and respiratory events associated with procedural sedation and analgesia [15]. It is the earliest indicator of airway or respiratory compromise, which manifests as an abnormally high or low $EtCO_2$ before pulse oximetry detects a falling oxyhemoglobin saturation, especially in patients receiving supplemental oxygen.

Capnography can also rapidly detect both central and obstructive apnea during sedation. Loss of the waveform in conjunction with an absence of chest wall movement and absence of breath sounds on auscultation confirms the diagnosis of central apnea. Complete obstructive apnea is also characterized by loss of the waveform and absence of breath sounds, but chest wall movement is present. Response to airway alignment maneuvers can further distinguish upper airway obstruction from laryngospasm (Table 3).

Capnography may be more sensitive for the detection of apnea during sedation than the clinical assessment of ventilation. In a recent study, 10/39 (26%) patients experienced 20-second periods of apnea during procedural sedation and analgesia. All 10 episodes were detected by capnography but not by the anesthesia providers [16]. Further studies have confirmed the difficulty with which clinicians recognize prehypoxic respiratory depression without the help of capnography [17-21].

The amplitude of the capnogram is determined by the $EtCO_2$ and its width is determined by the expiratory time. Therefore, changes in these

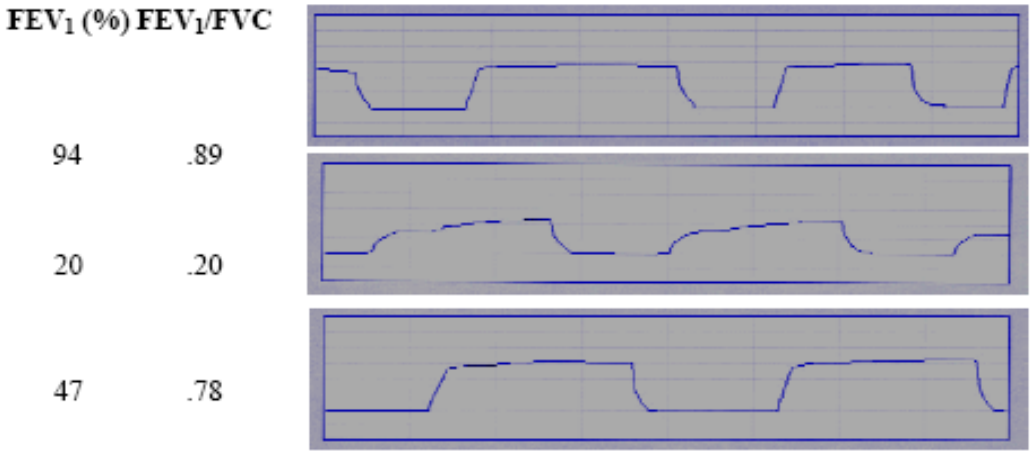


Figure 2. Capnogram shape in normal subjects and patients with obstructive and restrictive lung disease.

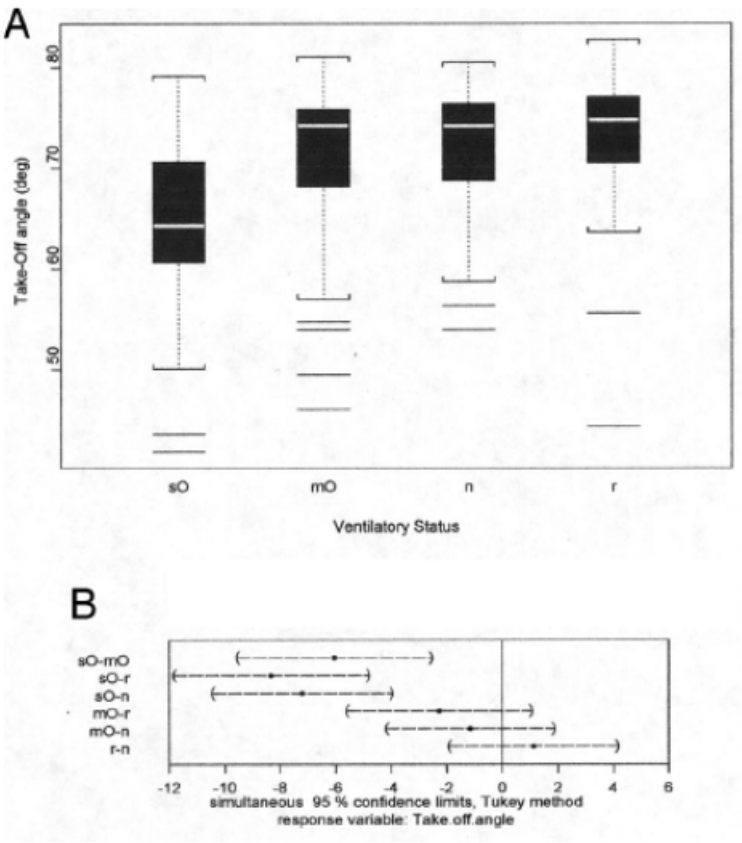


Figure 3. Take-off angle for each ventilatory condition with confidence intervals. sO = severe OD, mO = moderate OD, n = normal, r = restrictive lung disease.

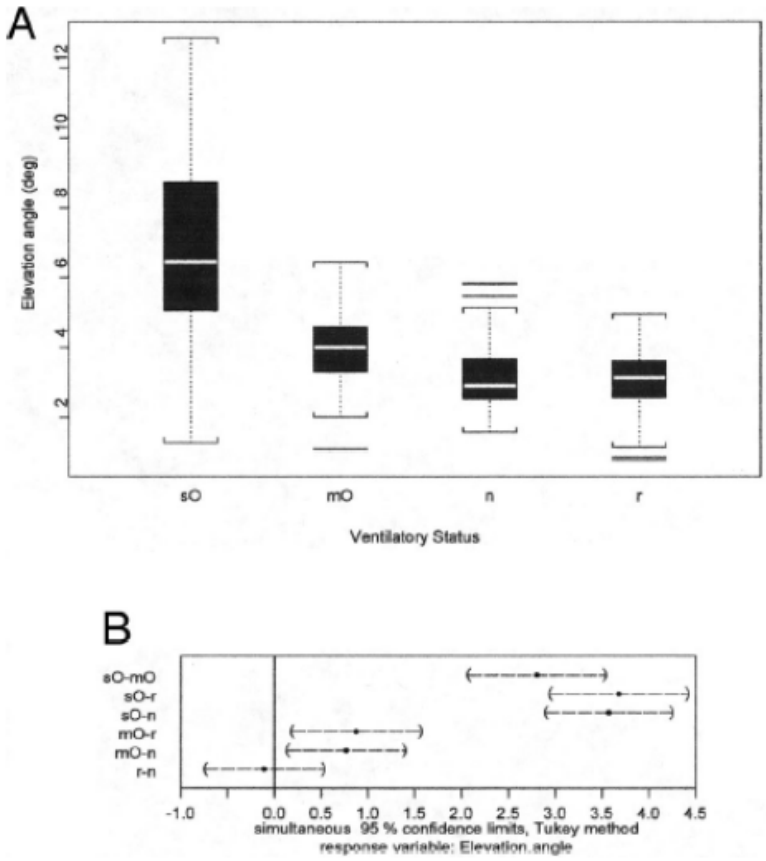


Figure 4. Elevation angle for each ventilatory condition with confidence intervals. sO = severe OD, mO = moderate OD, n = normal, r = restrictive lung disease.

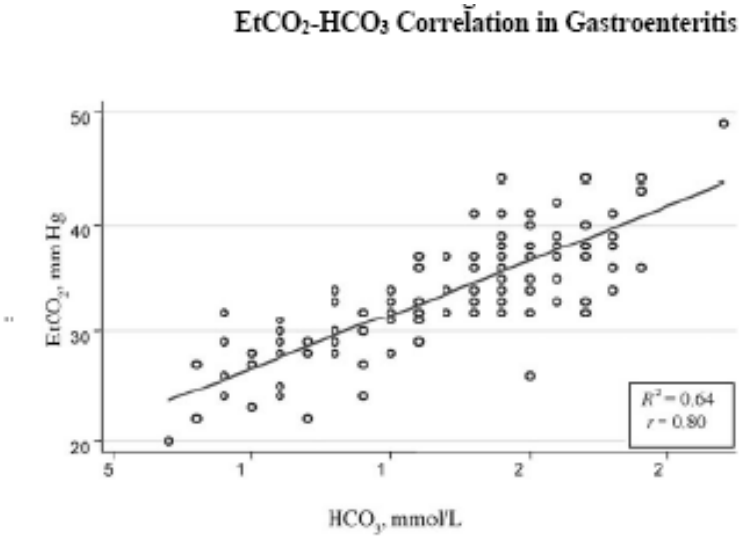


Figure 5. $EtCO_2$-HCO_3 Correlation in Gastroenteritis.

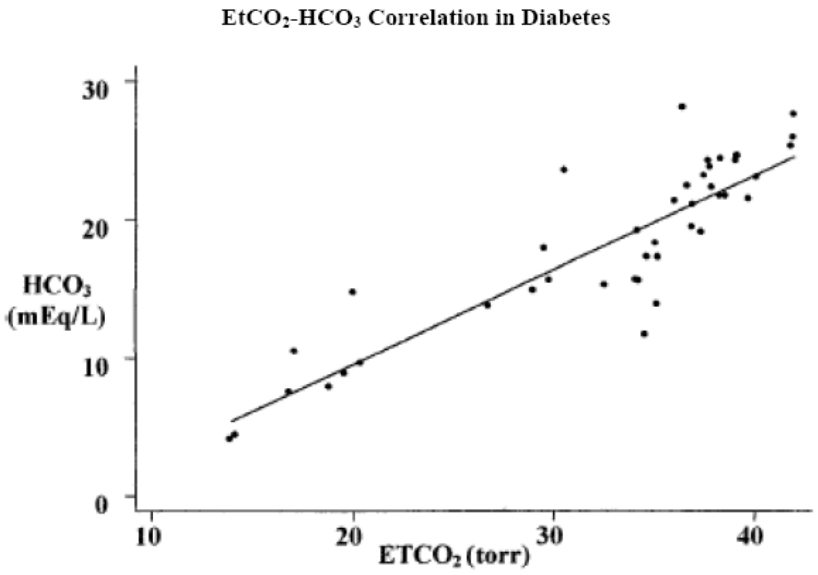


Figure 6. $EtCO_2$-HCO_3 Correlation in Diabetes.

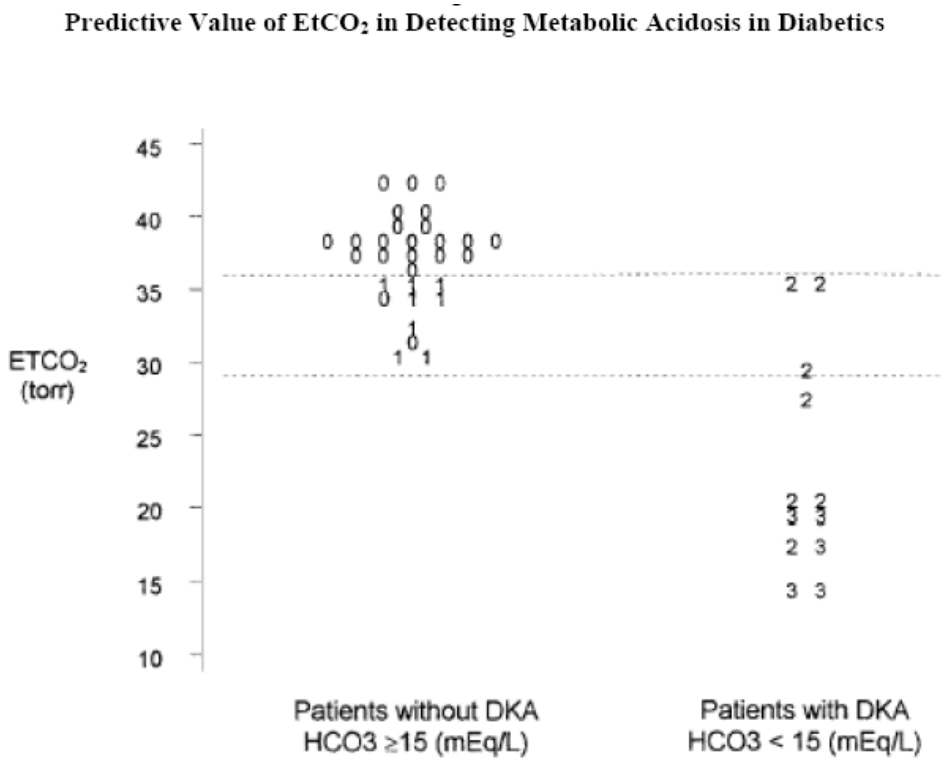


Figure 7. Predictive Value of $EtCO_2$ in Detecting Metabolic Acidosis in Diabetes.

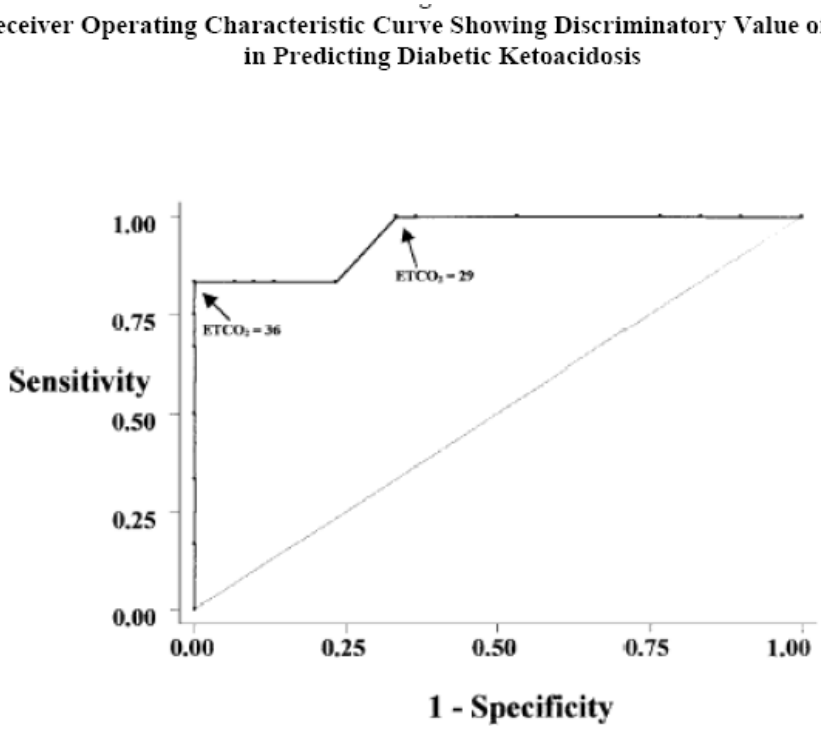


Figure 8. Receiver Operating Characteristic Curve Showing Discriminatory Value of $EtCO_2$ in Predicting Diabetic Ketoacidosis.

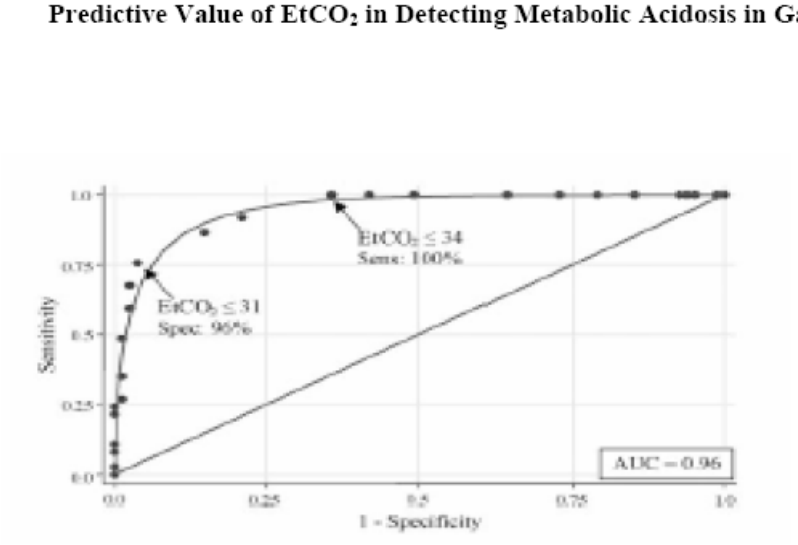


Figure 9. Predictive Value of EtCO2 in Detecting Metabolic Acidosis in Gastroenteritis.

parameters affect the capnogram shape. Hyperventilation results in a low amplitude and narrow capnogram. Of the two types of drug-induced hypoventilation that may occur during nonoperating room anesthesia, bradypneic hypoventilation (type 1), commonly seen with opioids, is characterized by an increase in expiratory time, $EtCO_2$, and $PaCO_2$. The depression in respiratory rate is proportionally than the depression in tidal volume. This process is represented graphically by a high-amplitude and wide capnogram (Table 3). By contrast, hypopneic hypoventilation (type 2), commonly seen with sedative-hypnotic drugs, is characterized by no change or a decrease in $EtCO_2$ and an increase $PaCO_2$, as the airway dead space remains constant and tidal volume decreases. The depression in tidal volume is proportionally greater than the depression in respiratory rate. This results in low tidal volume breathing which leads to an increase in the airway dead space fraction (dead space volume/tidal volume) and, in turn, an increase in the $EtCO_2$-$PaCO_2$ gradient. This process is represented by a low-amplitude capnogram (Table 3).

Bradypneic hypoventilation follows a predictable course, with $EtCO_2$ increasing progressively in a linear fashion until respiratory failure and apnea occur. Hypopneic hypoventilation, however, follows a variable, unpredictable, course: the $EtCO_2$ could remain stable, with resolution of the low tidal volume breathing over time as central nervous system drug levels decrease and redistribution to the periphery occurs; or periodic breathing with intermittent apneic pauses, which may either resolve spontaneously or progress to central apnea.

The low tidal volume breathing that characterizes hypopneic hypoventilation increases dead space ventilation when normal compensatory mechanisms are inhibited by drug effects. Minute ventilation, which normally increases to compensate for an increase in dead space, does not change or may decrease. As minute ventilation decreases, arterial oxygenation decreases. However, $EtCO_2$ may initially be high (bradypneic hypoventilation) or low (hypopneic hypoventilation) without a significant change in oxygenation, particularly if supplemental oxygen is given. Therefore, a drug-induced increase or decrease in $EtCO_2$ does not necessarily lead to oxygen desaturation and may not require intervention.

Pain Management

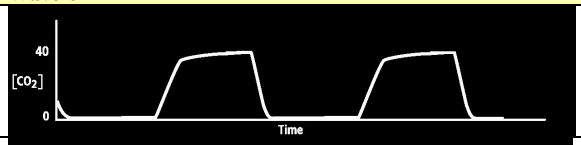
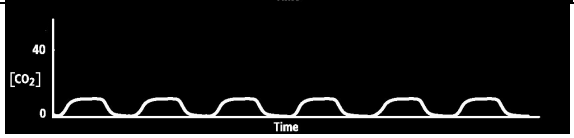
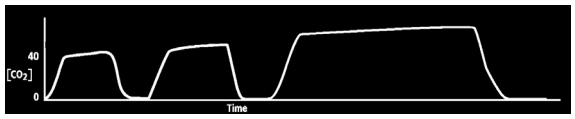
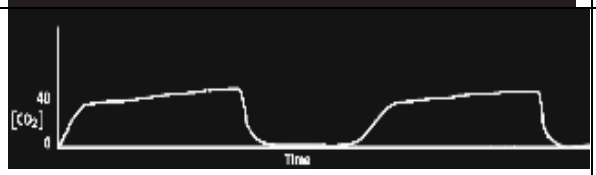
Patient-controlled analgesia (PCA) is currently monitored by pulse oximetry in the general medical or general pediatric wards and the Emergency Department. Capnography is not routinely used. Does monitoring ventilatory status during PCA have added value? In a study of 178 postoperative patients in the surgical ward using a PCA delivery system who were monitored with continuous pulse oximetry and capnography, investigators noted that 12% had desaturation to <90% for 3 minutes or more and 41% had respiratory depression (respiratory rate <10 breaths/minute) for 3 minutes or more [21]. These findings indicate that the integration of continuous oxygenation and ventilation monitoring in PCA delivery systems may allow for the future development of safety algorithms based on physiological parameters instead of lock-out times and absolute drug doses [21-23].

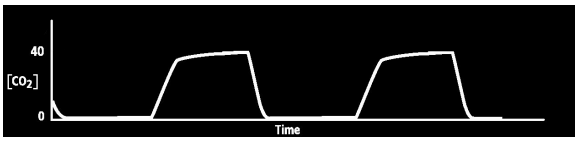
Detection of Metabolic Acidosis

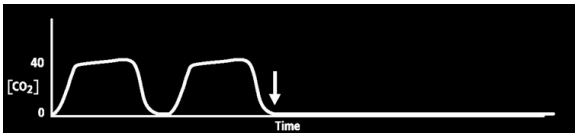
In addition to its established uses for the assessment of ventilation and perfusion, capnography is a valuable tool for assessing metabolic status. Specifically, it provides accurate information on how effectively CO_2 is being produced by the cellular metabolism.

Recent studies have shown that $EtCO_2$ and serum bicarbonate (HCO_3) are linearly correlated in patients with diabetes or gastroenteritis and that $EtCO_2$ can be used as an indicator of metabolic acidosis in these patients (Figures 5,6) [24-26]. As patients become acidotic, HCO_3 decreases and a compensatory respiratory alkalosis develops, with an increase in minute ventilation and a resultant decrease in $EtCO_2$. The increasing minute ventilation makes it possible to lower arterial carbon dioxide tension and, thereby, correct the underlying acidemia. The more acidotic the patient, the lower the HCO_3, and the higher the respiratory rate and lower the $EtCO_2$. $EtCO_2$ can therefore serve as a marker to distinguish diabetic patients who have ketoacidosis (metabolic acidosis, compensatory tachypnea, low $EtCO_2$) from diabetic patients who do not (nonacidotic, normal respiratory rate, normal $EtCO_2$) (Figures 7,8).

Table 3. Capnographic Airway Assessment in Non-operating Room Anesthesia.

Diagnosis	Waveform	Features		Intervention
Normal		SpO_2 $EtCO_2$ Waveform RR	Normal Normal Normal Normal	No intervention required Continue sedation
Hyperventilation		SpO_2 $EtCO_2$ Waveform RR	Normal ↓ decreased amplitude and width ↑	
Bradypneic hypoventilation (type 1)		SpO_2 $EtCO_2$ Waveform RR	Normal ↑ Increased amplitude and width ↓↓↓	Reassess patient and continue sedation
		SpO_2 $EtCO_2$ Waveform RR	↓ ↑ Increased amplitude and width ↓↓↓	Reassess patient Assess for airway obstruction Supplemental oxygen Cease drug administration or reduce dosing
Hypopneic hypoventilation (type 2)		SpO_2 $EtCO_2$ Waveform RR	Normal ↑ Decreased amplitude ↓↓↓	Reassess patient Continue sedation
		SpO_2 $EtCO_2$ Waveform RR	↓ ↓ Decreased amplitude ↓	Reassess patient Assess for airway obstruction Supplemental oxygen Cease drug administration or reduce dosing
Hypopneic hypoventilation with periodic breathing		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓ ↓ Decreased amplitude ↓ apneic pauses	
Physiological variability		SpO_2 $EtCO_2$ Waveform RR	Normal Normal Varying* Normal	No intervention required Continue sedation
Bronchospasm		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓ Normal, ↑, or ↓** Curved Normal, ↑, or ↓** wheezing	Reassess patient Bronchodilator therapy Cease drug administration

<table>
<tr><td>Partial airway obstruction</td><td rowspan="2"> 
</td><td rowspan="2">SpO2

EtCO2
Waveform
RR
Other</td><td rowspan="2">Normal or ↓
Normal
Normal
Variable
Noisy breathing and/or inspiratory stridor</td><td>Full airway patency restored w/ airway alignment
Noisy breathing & stridor resolve</td><td rowspan="2">Reassess patient
Establish IV access
Supplemental oxygen (as needed)
Cesase drug administration</td></tr>
<tr><td>Partial laryngospasm</td><td>Airway not fully patent with airway alignment
Noisy breathing and stridor persist</td></tr>
<tr><td>Apnea</td><td rowspan="3"> 
</td><td>SpO2

EtCO2
Waveform
RR
Other</td><td>Normal or ↓***
Zero
Absent
Zero
No chest wall movement or breath sounds</td><td colspan="2">Reassess patient
Stimulation
Bag mask ventilation
Reversal agents (where appropriate)
Cease drug administration</td></tr>
<tr><td>Complete airway obstruction</td><td rowspan="2">SpO2

EtCO2
Waveform
RR
Other</td><td rowspan="2">Normal or ↓***
Zero
Absent
Zero
Chest wall movementt and breath sounds present</td><td colspan="2">Airway patency restored with airway alignment
Waveform present</td></tr>
<tr><td>Complete laryngospasm</td><td>Airway not patent with airway alignment
No waveform</td><td>Positive pressure ventilation</td></tr>
</table>

A study of diabetic children presenting to the Emergency Department showed that an $EtCO_2$ of <29 mmHg identified 95% of the patients with ketoacidosis with 83% sensitivity and 100% specificity [24]. Conversely, no ketoacidosis was detected in patients with an $EtCO_2$ >36 mmHg. Using these data, nursing triage protocols can be developed to facilitate the assessment and treatment of patients with diabetic ketoacidosis.

$EtCO_2$ is similarly correlated with and predicts HCO_3 level in children with gastroenteritis [26], with maximal sensitivity occurring at EtCO2 ≤34 mmHg (sensitivity 100%, specificity 60%), and optimal specificity without compromise of sensitivity occurring at EtCO2 ≤31 mmHg (sensitivity 76%, specificity 96%) (Fig. 9). As a potential triage tool for determining the need for oral vs. intravenous rehydration, $EtCO_2$ could help identify patients with a clinically significant acidosis. An $EtCO_2$ of ≤31 mmHg has a positive likelihood ratio of 20.4 for detecting HCO_3 of ≤15 mmol/L and a positive likelihood ratio of 14.1 for detecting HCO_3 ≤13 mmol/L. These $EtCO_2$ values are 14 or 20 times more likely to occur in an acidotic patient than in a patient with HCO_3 >13 or >15 mmol/L.

Limitation of Capnography

Significant technical problems in the older generation capnography systems limited their effective use in nonintubated patients and restricted their clinical applications. These included interference with the sensor by condensed water and patient secretions in both mainstream and high-flow sidestream devices, cross sensitivity with anesthetic gases in conventional CO_2 sensors, lack of ruggedness for intra- and interhospital transport, and power consumption problems related to portable battery operation time. All have been resolved in the newer generation capnography monitors [27]. However, problems with accuracy continue to

affect high-flow sidestream systems. When the tidal volume of the patient drops below the flow rate of the system (e.g., in neonates, infants, and hypoventilating patients with low tidal volume breathing), the monitor will entrain room air to compensate, falsely diluting the $EtCO_2$ and slurring the ascending phase of the waveform [28-30].

The early capnography airway interfaces provided sometimes inconsistent measurements in mouth-breathing patients and patients who alternated between mouth and nose breathing. This problem, too, has been corrected, and the newer nasal-oral airway interfaces can simultaneously sample CO_2 and deliver low-flow oxygen. This allows for preoxygenation and continuous oxygen delivery during procedural sedation and analgesia.

Capnography is most effective for assessing a pure ventilation, perfusion, or metabolism problem. The findings are more difficult to interpret in conditions of mixed ventilation, perfusion, or metabolism. In patients with a complex pathophysiology, a ventilation problem may elevate $EtCO_2$ while a perfusion problem may simultaneously lower $EtCO_2$.

Conclusion

Capnography is a versatile noninvasive diagnostic monitoring modality for use in both intubated and nonintubated patients. Clinical applications include assessing vital signs in critically ill or injured patients, assessing ventilatory status in actively seizing patients, tracking response to treatment in patients with acute respiratory distress, monitoring ventilation in unconscious or obtunded patients, and detecting the degree of metabolic acidosis in children with diabetes or acute gastroenteritis.

References

1. Smalhout B, Kalenda Z. An Atlas of Capnography. The Netherlands: Kerckebusch Zeist, 1975.
2. Swedlow DB. Capnometry and capnography: The anesthesia disaster early warning system. Semin Anesthesia, 1986; 3:194-205.
3. Weil MH, Bisera J, Trevino RP, Rackow EC. Cardiac output and end-tidal carbon dioxide. Crit Care Med 1985; 13:907-909.
4. Krauss, B. Capnography as a rapid assessment and triage tool for chemical terrorism. Pediatr Emerg Care, 2005; 21:493-497.
5. Abramo TJ, Wiebe RA, Scott S, Goto CS, McIntire DD. Noninvasive capnometry monitoring for respiratory status during pediatric seizures. Crit Care Med, 1997; 25(7):1242-1246.
6. Abraham, RB, Rudick V, Weinbroum AA. Practical guidelines for acute care of victims of bioterrorism: Conventional injuries and concomitant nerve agent intoxication. Anesthesiology, 2002; 97:989-1004.
7. Centers for Disease Control and Prevention. Biological and chemical terrorism: strategic plan for preparedness and response. Morb Mortal Wkly Rep, 2000; 49 (RR-4):1-14.
8. Hardman JG, Aitkenhead AR. Estimating alveolar dead space from the arterial to end-tidal CO_2 gradient: A modeling analysis. Anesth Analg, 2003; 97:1846-1851.
9. Yamanaka MK, Sue DY. Comparison of arterial-end-tidal Pco_2 difference and dead space/tidal volume ratio in respiratory failure. Chest, 1987; 92:832-835.
10. Hoffbrand BI. The expiratory capnogram: A measure of ventilation-perfusion inequalities. Thorax, 1966; 21:518-523.
11. Krauss B, Deykin A, Lam A, Ryoo JJ, Hampton DR, Schmitt PW, Falk JL. Capnogram shape in obstructive lung disease. Anesthes Analg, 2005; 100:884-888.
12. Berengo A, Cutillo A. Single-breath analysis of carbon dioxide concentration records. J Appl Physiol, 1961; 16:522-530.
13. You B, Peslin R, Duvivier C, Vu VD, Grilliat JP. Expiratory capnography in asthma: Evaluation of various shape indices. Eur Respir J, 1994; 7:318-323.
14. Yaron M, Padyk P, Hutsinpiller M, Cairns CB. Utility of the expiratory capnogram in the assessment of bronchospasm. Ann Emerg Med, 1996; 28:403-407.
15. Krauss B, Hess DR. Capnography for procedural sedation and analgesia in the Emergency Department. Ann Emerg Med, 2007; 50:172-181.
16. Soto RG, Fu ES, Vila H, Miguel RV. Capnography accurately detects apnea during monitored anesthesia care. Anesth Analg, 2004; 99:379–382.

17. Burton JH, Harrah JD, Germann CA, Dillon DC. Does end-tidal carbon dioxide monitoring detect respiratory events prior to current sedation monitoring practices? Acad Emerg Med, 2006; 13:500–504.
18. Lightdale JR, Goldmann DA, Feldman HA, Newburg AR, DiNardo JA, Fox VL. Microstream capnography improves patient monitoring during moderate sedation: A randomized, controlled trial. Pediatrics, 2006; 117:1170-1178.
19. Deitch K, Chudnofsky CR, Dominici P. The utility of supplemental oxygen during emergency department procedural sedation and analgesia with midazolam and fentanyl: A randomized, controlled trial. Ann Emerg Med, 2007; 49(1):1-8.
20. Anderson JL, Junkins E, Pribble C, Guenther E. Capnography and depth of sedation during propofol sedation in children. Ann Emerg Med, 2007; 49(1):31-36.
21. Overdyk FJ, Carter R, Maddox RR, Callura J, Herrin AE, Henriquez C. Continuous oximetry/capnometry monitoring reveals frequent desaturation and bradypnea during patient-controlled analgesia. Anesth Analg, 2007; 105:412-418.
22. Hutchison R. Capnography monitoring during opioid PCA administration. J Opioid Manag, 2006; 2:207-208.
23. Maddox RR, Williams CK, Oglesby H, Butler B, Colclasure B. Clinical experience with patient-controlled analgesia using continuous respiratory monitoring and a smart infusion system. Am J Health Syst Pharm, 2006; 63:157-164.
24. Fearon DM, Steele DW. End-tidal carbon dioxide predicts the presence and severity of acidosis in children with diabetes. Acad Emerg Med, 2002; 9:1373-1378.
25. Estevan G, Abramo, TJ, Okada P, Guzman DD, Reisch JS, Wiebe RA. Capnometry for noninvasive continuous monitoring of metabolic status in pediatric diabetic ketoacidosis. Crit Care Med, 2003; 31:2539-2543.
26. Nagler J, Wright RO, Krauss B. End-tidal carbon dioxide as a measure of acidosis in children with gastroenteritis. Pediatrics, 2006; 117:260-267.
27. Colman Y, Krauss B. Microstream capnography technology: A new approach to an old problem. J Clin Monit, 1999; 15:403-409.
28. Sasse FJ. Can we trust end-tidal carbon dioxide measurements in infants? J Clin Monit ,1985; 1:147-148.
29. Gravenstein N. Capnometry in infants should not be done at lower sampling flow rates. J Clin Monit, 1989; 5:63-64.
30. Friesen RH, Alswang M. End-tidal PCO_2 monitoring via nasal cannulae in pediatric patients: accuracy and sources of error. J Clin Monit, 1996; 12(2):155-159.

Competing Interests: Baruch Krauss is a consultant for Oridian Medical, a capnography company, and holds two patents in the area of capnography

Funding: None declared

This manuscript has been peer reviewed

Correspondence:

Baruch Krauss MD EdM
Division of Emergency Medicine
Children's Hospital
300 Longwood Ave
Boston MA 02115
USA
Tel: (617) 355-4049 ext. 617
Fax: (617) 730-0335
Email: baruch_krauss@childrens.harvard.edu