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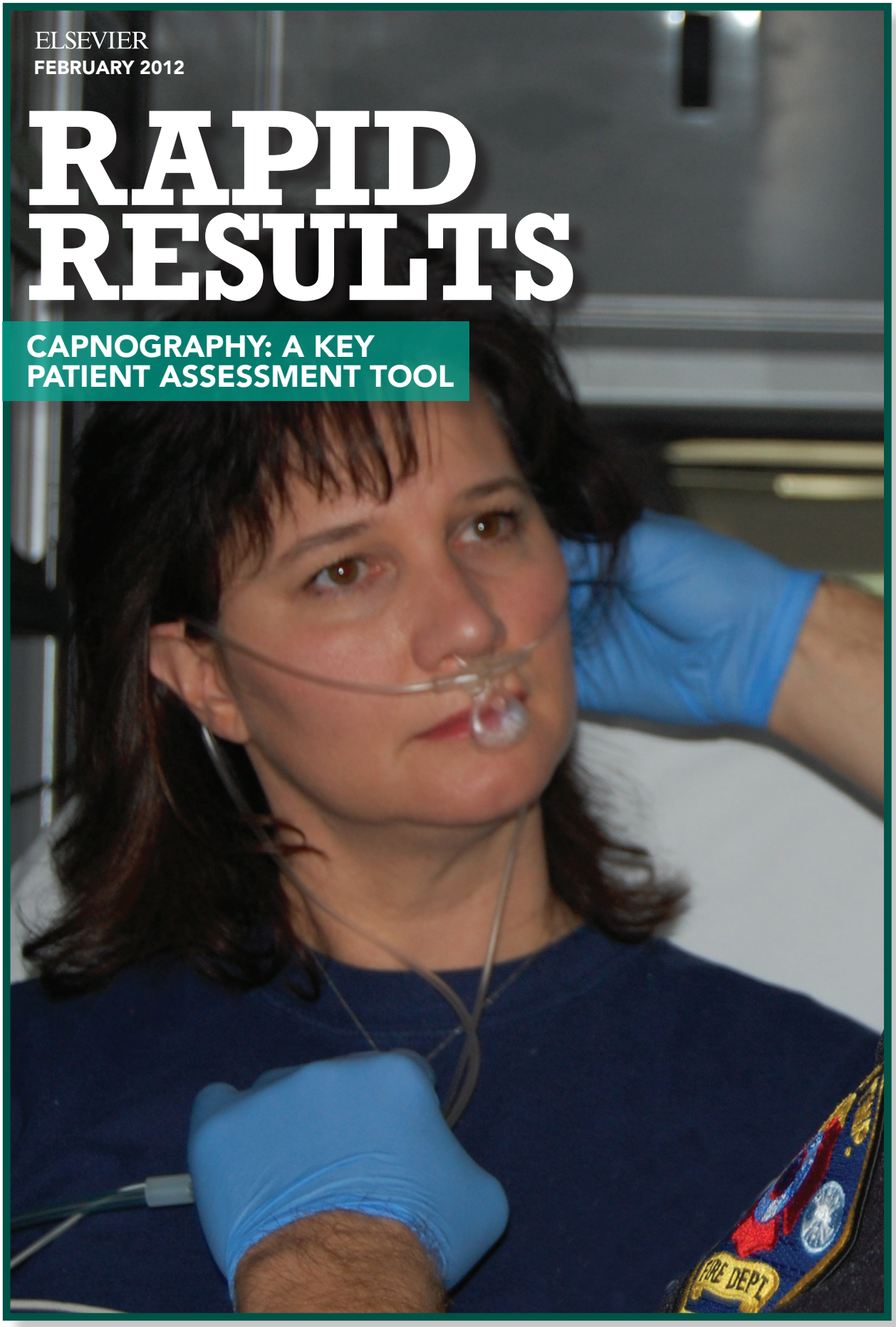
CAPNOGRAPHY: A KEY PATIENT ASSESSMENT TOOL



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Introduction

By A.J. Heightman, MPA, EMT-P

This supplement focuses on how waveform capnography has become a solid standard of care treatment modality. Its use is being expanded daily to offer optimal assessment and care of conscious and unconscious patients.

In "Basic Capnography," Jeffrey M. Goodloe, MD, NREMT-P, FACEP, discusses how waveform capnography greatly expands the assessment, diagnostic and management capabilities of EMS systems and points out that it can, and should, be used by ALS *and* BLS providers. Medical directors and EMS administrators are realizing that significant care benefits can be realized for a multitude of additional EMS patients when waveform capnography is placed in the critical-thinking minds and skilled hands of BLS professionals.

Congestive heart failure (CHF) is a significant medical condition that dominates the respiratory responses of many EMS systems. In "Pump Problems," Keith Wesley, MD, FACEP, presents capnography's important role in the assessment and monitoring of patients with CHF, emphasizing that 50% of CHF patients are readmitted to the hospital within six months of their last hospitalization and have an overall mortality of about 10%. He explains the current treatment approaches for CHF and the need for EMS personnel to be able to evaluate the status of the ventilatory and circulatory system of patients via capnography.

In "The Smoke of Metabolism," Troy Valente, BA, NREMT-P, explains how capnography can serve as an ideal assessment tool in the field, rapidly and accurately alerting clinicians to perfusion and/or metabolic problems.

Asthma is a common inflammatory disease that involves periodic episodes of severe but reversible bronchial obstruction. In "Severe Asthma,"Bob Page, BAS, NREMT-P, CCEMT-P, NCE, shows how capnography can provide a good indication of your patient's arterial blood gas level, therefore giving you an objective, reliable and accurate tool for assessing the severity of an asthma attack.

Page refers to capnography as one of the most important clinical upgrades he's seen in more than 30 years because it's the only noninvasive measure of a fundamental life process and can be used as a triage tool for the patient with a wide variety of complaints. It gives EMS providers an a patient's airway, breathing and circulation assessment in as little as three breaths.

The team of authors contributing to this supplement do a masterful job of explaining how you can use capnography as an important assessment and monitoring tool and, more importantly, to optimize the care you render to your patients.

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Basic Capnography

Advancing BLS patient assessment & management

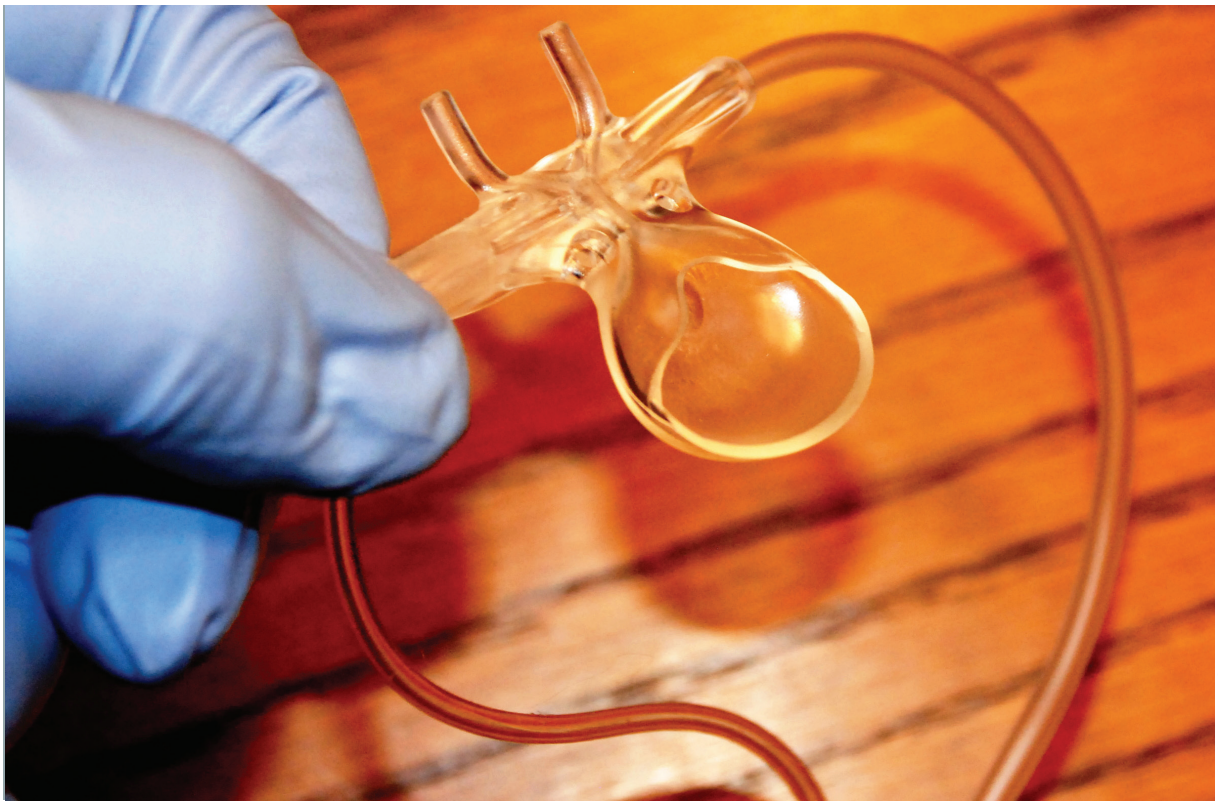


PHOTO A.J. HEIGHTMAN

Many regional and state EMS protocols now allow the use of capnography by ALS and BLS personnel.

By Jeffrey M. Goodloe, MD, NREMT-P, FACEP

Your 72-year-old male patient, Mr. Appleby, complains of progressive shortness of breath during your assessment. He repeatedly says, "Can't breathe," between difficult breaths. Chief complaint established. Now what? Through a rapid, focused exam, you note labored respirations at 22 per minute and diminished bilateral breath sounds with crackles. Or are those faint rales?

His concerned wife tells you he has a history of congestive heart failure (CHF). A quick downward scan shows you pedal edema. You announce your diagnostic achievement, "Your CHF is worsening, sir." He gives you a blank stare. As your EMT partner applies a nonrebreather oxygen mask with 100% oxygen concentration, his wife asks you, "Will that make his COPD (chronic obstructive pulmonary disease) worse?" So much for a straightforward call.

Many EMS professionals measure their career progress by the number and type of psychomotor skills they perform. Counting airways placed, bags squeezed, chests compressed and deformed limbs splinted gives us a natural method of measuring at least a part of our EMS experience. But are these examples or other treatments rendered the most important part of our experience? Are they even the most important part of our patient care? Likely, they are not.

As the practice of EMS medicine continues to mature at an impressively fast pace, one skill becomes the foundation that all others rest on; it's a skill heavy on "psycho" and light on "motor"—the skill of critical thinking.

How does this apply to our elderly male with CHF? Or does he have COPD? Could he have

both? Or maybe his dyspnea isn't due to either one. What else could be going on? Now we're critically thinking and not just impulsively jumping to a quick conclusion, which is easy for us but often wrong for our patients.

We need more information about our patient. His blood pressure is 140/90, pulse is 100, and pulse oximetry reads 96% on the non-rebreather mask with high-flow oxygen (it was 92% on room air). Other medical history includes hypertension, diabetes and a heart attack. He takes multiple daily medications, including a diuretic, an angiotensin-converting enzyme inhibitor, an oral hypoglycemic, a beta-agonist inhaler and a steroid inhaler. He isn't improving on the non-rebreather mask oxygen. So what's the problem? CHF, COPD, both or neither?

OK critical thinking masters, you want some waveform capnography information to rapidly establish a more accurate diagnosis and related treatment? But wait, you're on a BLS unit. Why do you want that waveform capnography information? Can you do anything with it? Assessment capabilities are clinically relevant only when your patient management can and will change based on the revealed information. Besides, waveform capnography isn't even in the national scope of practice for EMTs. That's one of those "paramedic things," right? No, wrong!

Waveforms

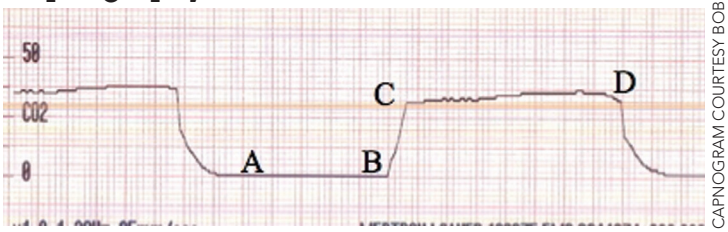
It sure is a good thing we don't all wait on national scopes of practice to define local standards of care. How would a national scope of practice get established in the first place unless someone dared to expand the "status quo?" So let's do that for BLS professionals, at least in discussion, through a brief primer on capnography physiology, normal capnography waveform analysis and interpretation of at least one abnormal capnography waveform that could prove particularly relevant to the clinical case at hand.

Capnography is the measurement of exhaled carbon dioxide (CO_2) in graphic waveform format. Technically, the isolated numerical value of the level of exhaled CO_2 is so often interlinked with the waveform produced at the same time that we're typically referring to both when using the term "capnography." It's believed to be particularly valid while interpreting exhaled CO_2 to narrow a differential diagnosis of dyspnea.

Let's look at an example of a normal physiology capnography waveform (see Figure 1, at right). Moving left to right, we find the interval represented from Point A to Point B to be without measured CO_2. This is the period without exhaled air, representing the period just prior to exhalation. Once exhalation starts, we would expect to see a rapid detection of CO_2, as well as a rapid elevation in the CO_2 levels. This is exactly what's seen in the waveform from Point B to Point C.

Although local protocols may vary, most indicate $EtCO_2$ values in the 30–35 mmHg range for acute head injury with increased ICP concerns.

Figure 1: Normal Physiology Capnography Waveform



CAPNOGRAM COURTESY BOB PAGE

As exhalation continues, CO_2 from the lowest airways, including alveoli, is sensed by an airway circuit detector. During this phase of respiration, represented from Point C to Point D, levels gradually approach the highest amount of CO_2 expected before inhalation.

The pinnacle of respiratory cycle CO_2 is at Point D, referred to as the end-tidal carbon dioxide ($EtCO_2$). Perhaps an easier way to think of this concept is to expand the term to "end of the tidal volume of exhaled air" level of CO_2. In normal human physiology, we can expect an $EtCO_2$ level to be in the 35–45 mmHg range.

What happens to the measured CO_2 level when we inhale? We should expect it would drop and typically, drop rapidly. That's also what we see when looking at the waveform moving from point D (the end-tidal value) back to the baseline Point A.

The process (and waveform) repeats itself with every respiratory cycle of exhalation and inhalation. Reference texts exist to guide you through normal capnography waveform analysis on a far more detailed level, but the preceding will serve BLS personnel just fine in applying this assessment tool to some very sick patients.[1,2]

With normal waveform appearance and $EtCO_2$ values in mind, let's look at capnography waveform showing abnormal physiology (see Figure 2, p. 6).

What's the major difference in this example? The segment from point C to point D, which we just established is the segment that represents exhalation, is different. If the waveform's rate of rise vs. time (also known as the "slope" of the waveform) is less than normal, then something must be slowing down the release of CO_2.[3]

What kinds of things will slow exhalation of air? Bronchospasm and mucus are the first two things

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most EMS professionals correctly identify as exhalation inhibitors. Over time, as metabolism continues to produce waste products that change into CO_2, levels of CO_2 may rise unless exhalation becomes easier. Thus, although the slope from Point C to Point D on the capnography waveform may be slower, Point D may be much higher than normal, reflecting overall CO_2 retention, also known as hypercarbia.

In this example, let's say that Point D on Figure 2 is no longer 45 mmHg; it's 70 mmHg. That sounds like a real problem. In fact, it sounds like it could be a real COPD-exacerbation-kind-of problem.

Let's go back to our clinical case now that we've expanded your scope of practice—at least for Mr. Appleby's treatment. You place a nasal cannula device with a sensor built in to measure exhaled CO_2 (see Figure 3, below).

Figure 2: Waveform with "Shark Fin" Physiology Morphology

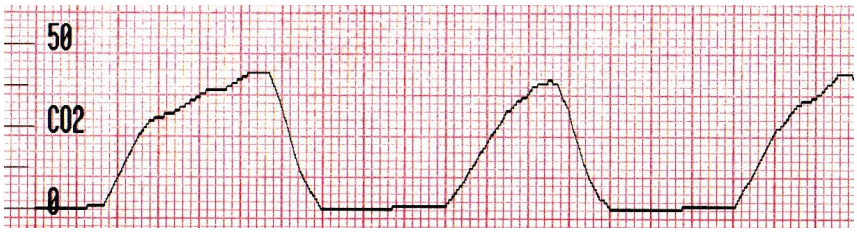
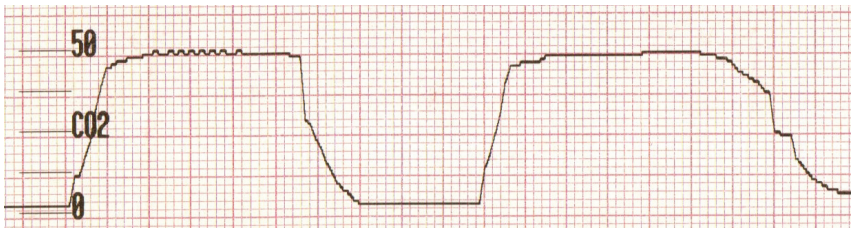


Figure 3: Nasal Cannula with Capnography Sensor



CAPNOGRAMS COURTESY BOB PAGE

Your monitor shows capnography waveforms that look like Figure 2. Critical thinking says, "I heard a history of CHF and see pedal edema, but respiratory assessment with the additional data of waveform capnography now tells me COPD (CO_2 retention) is likely the bigger problem here."

What is your treatment plan now? Can you do something as an EMT to lower retained CO_2 in a dyspnea patient? Your partner removes the nonrebreather mask as you prepare to do two things. You need to 1) assist the patient with his prescribed beta-agonist inhaler to open the lower airways; and 2) assist his ventilations with a bag-valve mask with supplemental oxygen. You purposefully hyperventilate the patient and see the capnography waveforms get shorter over time.

Consistent with shorter waveforms, $EtCO_2$ levels fall to 55 mmHg. Mr. Appleby is breathing easier and is more responsive. We shouldn't be surprised by his alertness because hypercarbia can be a common cause of altered mental status. Good job, and you did all that without a paramedic in sight.

OK fellow paramedics, let's not get stingy with good assessment capabilities, right? Let's see how BLS use of capnography can help us if we change just one vital parameter and one finding after examining Mr. Appleby.

An ALS-BLS crew arrives first to the same location. No history can be obtained from Mr. Appleby because he's unresponsive. His $EtCO_2$ value exceeds 100 mmHg. Although local ALS protocols may vary, most indicate endotracheal intubation (ETI) in this setting for two reasons—protection of the airway and little likelihood that bag-valve-mask ventilation will rapidly improve such profound hypercarbia.

A paramedic performs ETI and carefully confirms correct tracheal placement. Can waveform capnography help in that regard? Sure, because the stomach doesn't rhythmically "exhale" CO_2 up the esophagus. The lungs obviously do that very thing via the trachea; thus, waveform capnography is essential in advanced airway placement confirmation.[4] Rhythmic rise and fall with exhalation and inhalation respectively gives all of us on scene great comfort that the endotracheal tube is correctly positioned. Therefore, waveform-capnography-educated EMTs can become expert airway placement lookouts.

Further, in most EMS systems with BLS and ALS response crews, it's the EMTs who perform the majority of bag-valve-mask or bag-valve-airway ventilations. In those situations, who better to gauge ventilation effects than the ventilator? Waveform capnography is a great breath-by-breath (or ventilation-by-ventilation) feedback tool to guide ventilation rates and volumes.

Feedback Tool

Let's put this last concept into practice taking care of a 22-year-old female victim of a motor-vehicle collision. She was ejected and sustained an obvious head injury. She's confused with a Glasgow Coma Scale score of 12, which seems to worsen in short order. What could be happening? One possibility is increased intracranial pressure from the head injury. You use waveform capnography and see such waveforms as those in Figure 1 with $EtCO_2$ levels of 45 mmHg.

Because normal $EtCO_2$ is 35–45 mmHg, you might think that everything is fine ventilation-wise.

Actually, it's probably not. You need to intervene and work to lower suspected increased intracranial pressure (ICP).

One thing you can do to reduce ICP is to prevent cerebral vasodilation by reducing circulating CO_2. How can you do that? What did you do for your COPD patient earlier? Hyperventilation will also work wonders for your head-injury patient.

As with all good things, excessive good efforts may produce bad results, specifically in this instance of hypocarbia-related cerebral vasoconstriction compromising cerebral perfusion. Waveform capnography allows you to more precisely gauge ventilation rate and volume effects.

Again, although local protocols may vary, most indicate to achieve $EtCO_2$ values in the 30–35 mmHg range for acute head injury with increased ICP concerns. Some well-performed studies indicate some concerns that present methods of $EtCO_2$ determination may not be accurate enough in certain trauma settings.[5–7]

Clinical adjustments that may be warranted from the findings in these studies are still unclear, although your medical director may be contemplating target $EtCO_2$ values lower than historically recommended in light of these studies and hopefully further related investigations.

In many local EMS protocols, use of waveform capnography isn't limited to the severely dyspneic or traumatized patient, and it isn't limited to paramedic use. Here's an example of one such protocol (see Figure 4, above).

Conclusion

This article introduces the concept of waveform capnography being appropriately included in the BLS scope of practice. Waveform capnography use substantially expands the assessment, diagnostic and management capabilities of EMS systems and professionals. These significant care benefits can be realized for a multitude of additional EMS patients when waveform capnography is also placed in the critical-thinking minds and important treating hands of BLS professionals.

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Disclosure: The author has reported receiving no honoraria and/or research support, either directly or indirectly, from the sponsor of this supplement.

Figure 4: Acute Dyspnea Asthma Protocol

EMT-B

GENERAL SUPPORTIVE CARE
OBTAIN VITAL SIGNS
O2 VIA NC, NRB, OR BVM AS APPROPRIATE
APPLY CARDIAC MONITOR (when available)
ASSIST PT WITH PT'S OWN ALBUTEROL INHALER/NEBULIZER (when applicable)
MEASURE END-TIDAL CO2 & MONITOR CAPNOGRAPH (when available)
ADULT: APPLY CPAP IF INDICATED

To view the entirre protocol, visit jems.com/supplements.

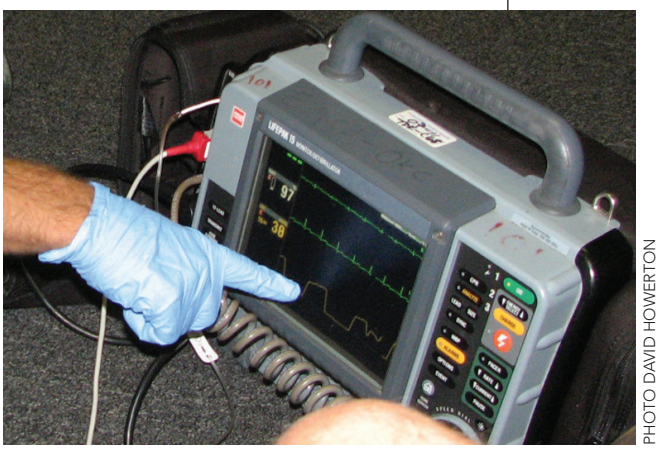

PHOTO DAVID HOWERTON

In many local EMS protocols, use of waveform capnography isn't limited to paramedic use.

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Pump Problems

Understanding capnography's role in CHF patients

PHOTO RICK BRADY

Capnography is one of the most sensitive vital signs that should be monitored while treating a CHF patient.

By Keith Wesley, MD, FACEP

Congestive heart failure (CHF) is the most common underlying condition that results in 9-1-1 calls for shortness of breath. It's a significant medical condition that EMS sees frequently, with new onset and acute exacerbation of chronic CHF results in more than 300,000 hospital admissions yearly. In fact, 50% of CHF patients will be readmitted to the hospital within six months of their last hospitalization—20% within one month. And the overall mortality from CHF is 8%.[1]

The reasons for this high recurrence of CHF are multi-factorial (see Figure 1, p. 9). Regardless of the cause, having a proper appreciation for this syndrome, understanding its pathophysiology and aggressively treating it in both the hospital and prehospital arena has been shown to significantly reduce this high mortality rate.

Pathophysiology

CHF is a syndrome defined by a constellation of specific signs and symptoms relating to any mechanism that impairs the efficiency of the heart to eject blood. Traditional teachings regarding CHF have focused on total body volume, incorrectly believing that the patient suffering from CHF must be volume overloaded and that simple diuresis was the key to therapy.

We now recognize that CHF results in cardiovascular compromise when, for whatever reason, the heart's cardiac output is diminished. This can occur if the heart develops a dysrhythmia, such as atrial fibrillation, that reduces the amount of blood ejected with each compression (i.e., stroke volume) by as much as 25%.[2]

It can occur following a myocardial infarction

when the myocardium's ability to contract is impaired because of a loss of the infarcted muscle mass.

CHF most often occurs due to chronic hypertension where the myocardium must increase in size (hypertrophy) to have sufficient force to eject blood out of the ventricle against the significantly elevated arterial pressure. This hypertrophy impairs stroke volume by decreasing the size of the ventricle. Also, the hypertrophy muscle doesn't relax normally, and this further decreases stroke volume by not allowing the ventricle to fill normally during diastole.

The human body responds to this insult on the heart's function by engaging several autonomic compensatory reflexes (see Figure 2, at right). Key to this reflexive response is the kidney, which is highly responsive to changes in blood volume and views the decreased cardiac output as a sign of hypovolemia. In response, the kidney releases the hormones renin, aldosterone and angiotensin.

Renin and aldosterone increase sodium absorption in the urine to boost circulating blood volume, while angiotensin increases peripheral vascular resistance. Increased peripheral vascular resistance occurs through vasoconstriction of the arteries and veins.

In EMS, we refer to increased arterial resistance as "afterload," because it's the pressure against which the heart must work "after" it leaves the heart. Venous vasoconstriction increases the volume of blood that's returned to the right side of the heart and is referred to as "preload" because it loads the heart with blood "before" ventricular contraction.

Although this cycle of dysfunction is occurring, the ability of the left ventricle to pump the blood it's receiving from the right ventricle is impaired, and the pressure in the pulmonary veins increases, causing fluid to move from the pulmonary capillaries into the tissue surrounding the alveoli, the interstitial space. This fluid impairs the ability of the lung to exchange carbon dioxide and oxygen.

If the pressure continues to rise, the interstitial fluid can move into the alveoli completely, blocking their ability to function. This is the process that causes the patient to become short of breath. The body responds to this pulmonary insult by releasing adrenalin to increase both the heart rate and respiratory rate. The patient must expend more and more energy to breathe, which is referred to as work of breathing (WOB). If the WOB becomes too great, then the patient begins to hypoventilate, and eventually, stops breathing altogether.

The problem with this compensatory reflex is that unless the heart can handle the increased workload and the pulmonary edema can be tolerated, the body continues to pump more adrenalin, angiotensin and aldosterone into the system. This requires the heart to work harder, leading to myocardial ischemia, decreased myocardial contraction and lowered cardiac output, which only perpetuates the cycle of reflexive response, pulmonary congestion and decreased cardiac output. This process continues until the patient quits breathing or experiences cardiogenic shock (see Figure 3, p. 10).

Figure 1: Causes of CHF Re-admission

Diet/Medication Non-Compliance	24%
Acute Coronary Syndrome	40%
Infection (often respiratory)	25%
Inability to Get Follow-Up	19%
Inappropriate Prescription	16%
New Dysrhythmia	4%

Source: Vinson JM, Rich MW, Sperry JC, et al. Early readmission of elderly patients with congestive heart failure. *J Am Geriatr Soc.* 1990;38(12):1290–1295.

Figure 2: Compensatory Reflexes to CHF

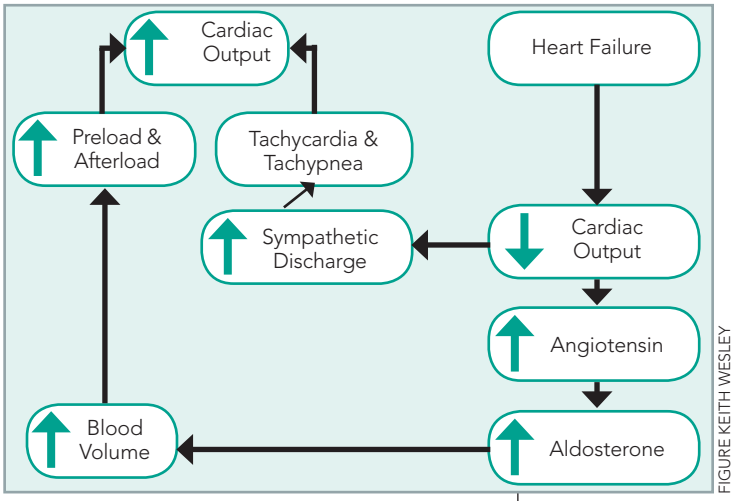

FIGURE KEITH WESLEY

Treatment Approach for CHF

With our new understanding of the pathophysiology of CHF, the goal of therapy is two-fold: 1) interrupt the cycle of autonomic reflexes and 2) decrease the work of breathing.

The most powerful therapy of interrupting the autonomic cycle is to reduce the preload and afterload with the administration of nitroglycerin. This can be accomplished by sublingual administration of one spray or tablet every five minutes, which equates to 80 micrograms/minute. IV nitroglycerin is more efficient and more easily titrated to effect.

Administration of an ACE inhibitor (e.g., 25 mg of captopril sublingually) interrupts the effect of the elevated angiotensin levels, reducing myocardial workload. The result is improved myocardial contraction and relaxation, which promote increased cardiac output.

Reducing the work of breathing is also

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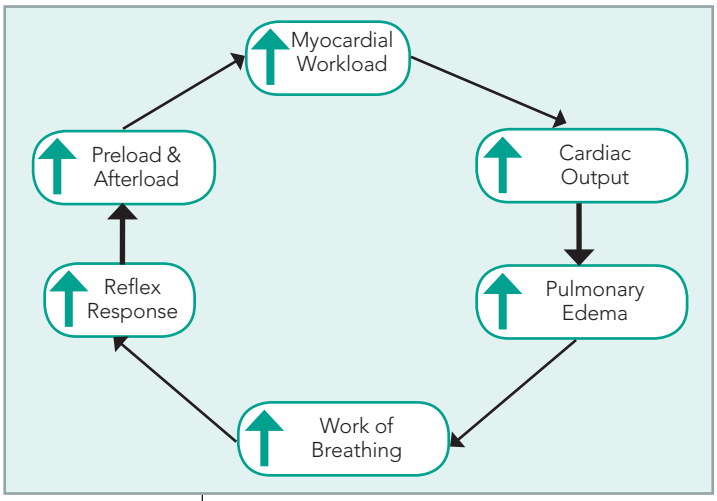
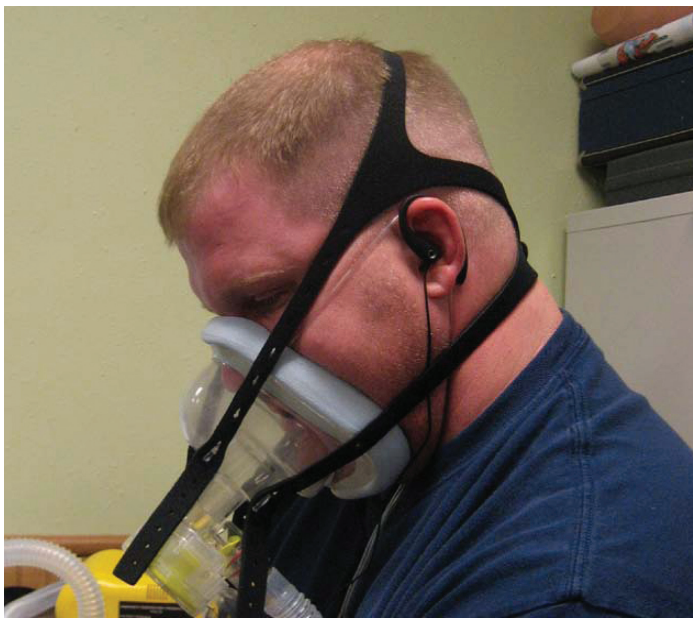
Figure 3: Cycle of CHF & WOB



accomplished with ventilatory support with continuous positive airway pressure (CPAP). CPAP increases the pressure in the alveoli, promoting the movement of the interstitial fluid back into the blood stream. Reduction in pulmonary edema reduces elevation in adrenalin and further decreases the work of breathing.

Traditional therapy for CHF used to consist of high doses of diuretics, such as furosemide. However, recent studies have shown that CHF patients are rarely in volume overload.[3] Instead, they suffer from inappropriate volume distribution. That is, there's an increased amount of fluid in the interstitial spaces in the lung and peripheral tissues; not an overall circulating volume. Therefore, the role of diuretics has become secondary and reserved only after the cycle of autonomic reflexes has been interrupted.

Figure 4: Oridion Capnoline under CPAP mask.



Until the myocardial workload is reduced with nitroglycerin and the pulmonary edema relieved with CPAP, any excess fluid can only be dealt with after it's back in the circulatory system. In fact, several studies indicate that early, high-dose furosemide therapy causes the kidneys to misinterpret the diuresis as further dehydration and results in the release of anti-diuretic hormone (ADH), which promotes water reabsorption and the occurrence of a second episode of CHF (rebound) after the patient is admitted.[4]

CHF Complications

As can be predicted from understanding the pathophysiology of CHF, the two most significant complications include respiratory failure as a result of the energy required to breathe and cardiogenic shock once the heart is no longer able to compensate for the increased myocardial workload.

Respiratory failure from CHF is similar to any other cause that increases the work of breathing beyond the body's ability to compensate. Elevated adrenalin levels precipitate the rise in respiratory rate and volume. However, the supply of adrenalin is limited, and once it's depleted, the respiratory rate begins to drop.

Movement of CO_2 is affected first because its elimination from the lungs is totally dependent on the rate and depth of ventilations. As the ventilatory rate and depth decrease, carbon dioxide (CO_2) levels increase. Elevated CO_2 levels have a narcotic-like effect on the brain, causing a decreased level of consciousness that further reduces ventilatory drive, resulting finally in coma and respiratory arrest.

The ability of the heart to compensate for the elevated adrenergic tone and autonomic reflex hormones is finite. Left uncorrected, the strain on the heart becomes too great and the cardiac output plummets, resulting in shock. Because this shock state is a primary result of heart failure, it's termed cardiogenic shock. The patient's blood pressure drops, resulting in systemic hypoperfusion, which eventually leads to cardiac arrest.

Capnography & CHF

Capnography has traditionally been viewed as a means to monitor the ventilatory status of a patient. Although this is true, it isn't the only process that capnography monitors. For CO_2 to be released from the pulmonary vasculature, it must be delivered to the lungs by the circulatory system. To accomplish this, the cardiac output must be sufficient to perfuse the lungs. Therefore, when interpreting capnography in the CHF patient, EMS providers must evaluate the status of both the ventilatory and circulatory system.

Ventilatory status: The capnography of a patient with an increased WOB will reveal low, normal

or elevated CO_2 levels. Low CO_2 levels (hypocapnea) indicates hyperventilation; elevated CO_2 levels (hypercapnea) indicate hypoventilation. Normal values may mean the patient is breathing normally, but those values could also indicate that the patient is transitioning from hypo to hypercapnia or vice versa. This is why continuous capnography is vital to determine whether the patient's condition is changing.

Circulatory status: As long as the patient's blood pressure is normal or elevated, as it most often is during a CHF exacerbation, the level of CO_2 being delivered to the lungs will be consistent with adequate tissue perfusion. Therefore, in the absence of any ventilatory compromise, the CO_2 levels will be normal or slightly elevated, which can indicate a higher than normal level of metabolic activity (myocardial workload).

If cardiogenic shock occurs, the perfusion to the lung is compromised, and the amount of CO_2 delivered is significantly reduced. In the absence of ventilatory compromise, the CO_2 level is a valuable indicator of cardiac output and tissue perfusion.

Ventilation and circulatory status: However, in the CHF patient there's the potential for ventilatory and/or circulatory compromise, which can complicate the interpretation of the capnography values. The following guide outlines the best approach to this interpretation:

1. The patient's blood pressure is normal or elevated. High CO_2 levels indicate respiratory hypercapnea/hypoventilation and worsening WOB. Normal CO_2 levels require close monitoring for possible trend. Low CO_2 levels indicate respiratory hypocapnea/hyperventilation compensating to increased WOB.
2. The patient's blood pressure is low (signs of shock). Low CO_2 levels indicate respiratory hypercapnea/hypoventilation as result of impending respiratory failure. Normal CO_2 levels require close monitoring for possible trend. Low CO_2 levels indicate hypoperfusion and impending cardiovascular collapse.

Changes in the ventilatory and circulatory status of the CHF patient will affect CO_2 levels sooner than changes in the oxygen saturation. Changes in CO_2 levels more accurately indicate the changes in the patient cardiorespiratory status; therefore, capnography should be a vital component in the monitoring of the CHF patient.

Capnography & CPAP

As discussed earlier, CPAP is a vital component in the treatment of respiratory distress in CHF. The questions are, how is capnography performed with CPAP, and does CPAP affect the accuracy of capnography?

HealthEast Medical Transportation in St. Paul, Minn., conducted a study to determine the most accurate and reliable means of performing capnography while administering CPAP. It examined the three most common devices at the time: Port-O-Vent, Respironics Whisperflow and Boussignac. The package insert for all three devices indicates that capnography could be accomplished by attaching a CO_2 sampling line at various points in the CPAP circuit: the exhalation port, a nipple in the T-piece and a nipple on the mask.

This was compared with capnography obtained from the Oridion Capnoline, which is a combined nasal/oral sampling cannula worn under the CPAP mask (see Figure 4, at right).

The Oridion Capnoline was found to be the most reliable and accurate means of monitoring end-tidal CO_2. There was no noticeable leak at the mask-face seal with any of the three devices studied. Additionally, the Oridion Capnoline was the only method that provided a physiologic waveform, a factor that's crucial when monitoring the patient with bronchospasm.

Conclusion

CHF is a common cause of respiratory distress in EMS patients. New theories of its pathophysiology indicate that aggressive therapy with nitrates and CPAP significantly reduces its mortality.

Capnography is one of the most sensitive vital signs that should be monitored while treating the CHF patient. Changes in CO_2 level indicate the quality of ventilation and the perfusion status of the patient.

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The Smoke of Metabolism

How capnography optimizes your perfusion & metabolic assessment

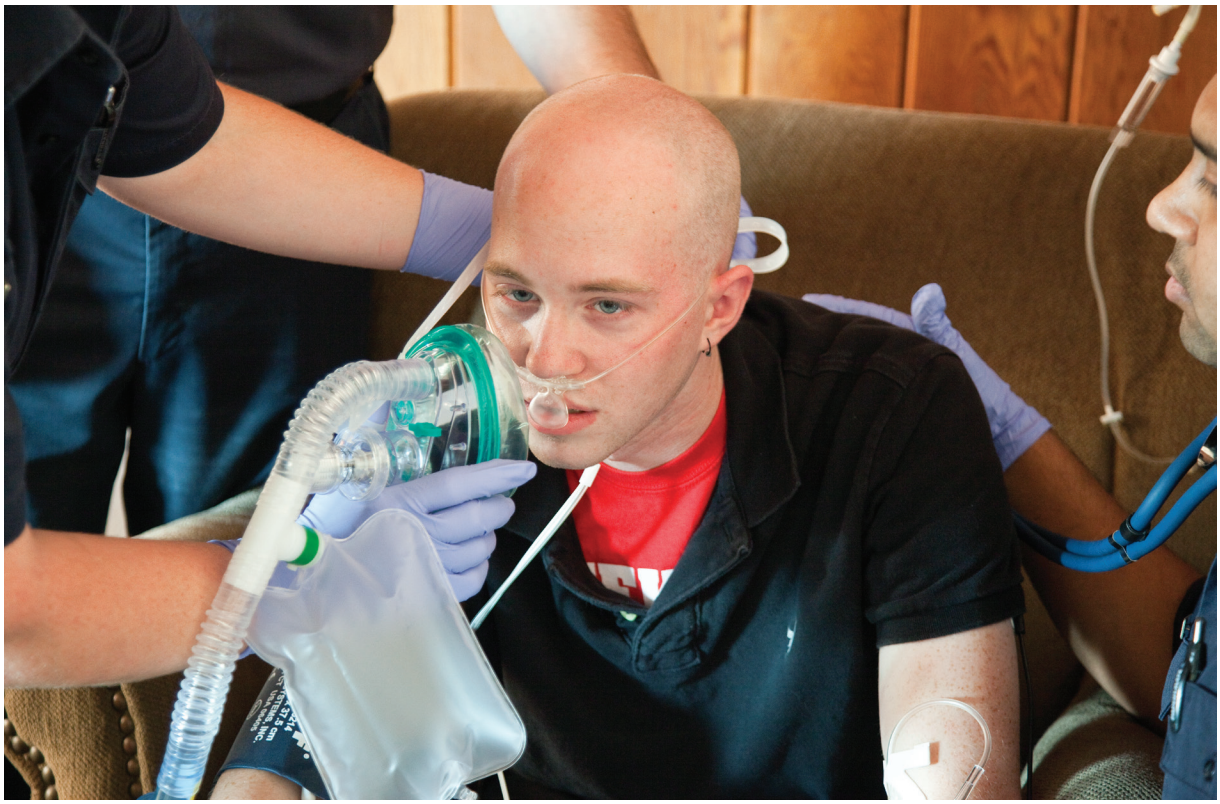


PHOTO RICK BRADY

Anything affecting the metabolic rate affects CO_2 production and, consequently, corresponding $EtCO_2$ levels.

By Troy Valente, BA, NREMT-P

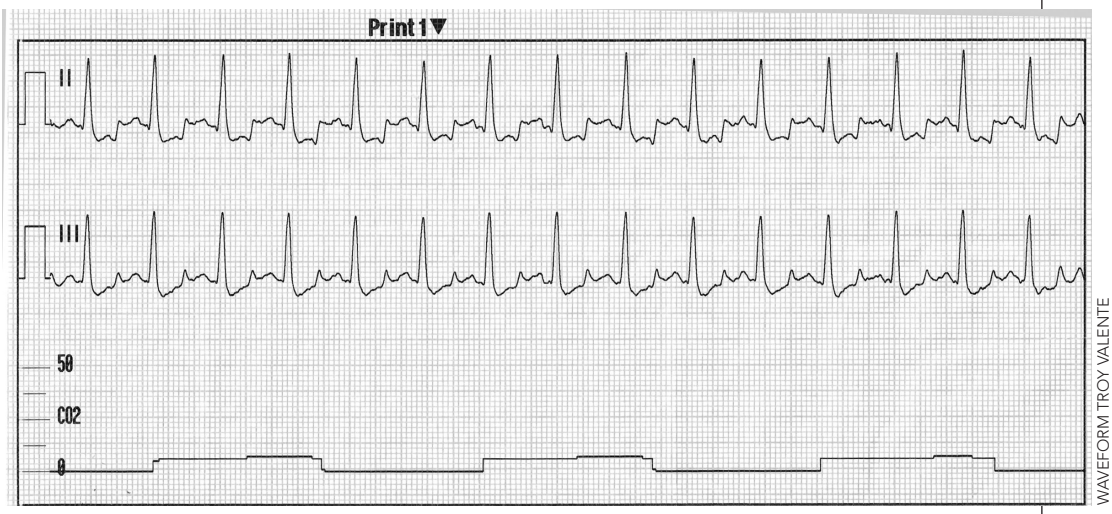
When EMS providers hear the word, "capnography," most think of such phrases as "ventilation vital sign," "airway management tool," "shark fin" and "tube placement." Although we shouldn't minimize these important uses of capnography, the additional, powerful assessment capabilities capnography offers are far greater. Capnography can rapidly and accurately alert clinicians to perfusion and/or metabolic problems and provide deeper insight than traditional assessment tools. So let's broaden the capnography perspective. Yes, it's a device we place at the patient's airway; but capnography is measuring a by-product that goes much deeper than that.

The Greeks first believed that the body had a central combustion center and that this system had a by-product. They named this by-product *capnos*, which is Greek for "smoke." Today, we call this combustion center "metabolism" and *capnos* "carbon dioxide" (CO_2). Simply put, CO_2 is the "smoke" of metabolism.[1] More specifically, CO_2 is created after the acidic by-products of metabolism are buffered by bicarbonate:

Acid ($2H^+$) + Bicarbonate (HCO_3^-) <=> CO_2 + H_2O

Because CO_2 is a by-product of metabolism, anything affecting the metabolic rate affects CO_2 production and, consequently, corresponding end-tidal carbon dioxide ($EtCO_2$) levels. Metabolic pathways must be supplied with essential nutrients, such as sugar, water and oxygen for a normal metabolic rate to occur. So if perfusion is compromised, so is the delivery of these components necessary for metabolism. The result is a decreased metabolic rate, resulting in decreased

Figure 1: Diabetic Ketoacidosis



WAVEFORM TROY VALENTE

Patients with diabetic ketoacidosis will have low $EtCO_2$ levels, secondary to depleted bicarbonate and deep and rapid respirations.

CO_2 production. Or, if a patient is in metabolic acidosis (clinically defined as an acidotic pH with depleted bicarbonate), less CO_2 will be created, resulting in lower $EtCO_2$ levels.

Anaerobic metabolism increases during poor perfusion states, which increases acidic byproducts. However, simply increasing acid in the system doesn't equate to an increase in CO_2 production.

The reason is quite complex. However, an oversimplified explanation is that bicarbonate availability and catalyst concentrations are the rate-limiting factors to CO_2 production. It isn't like a chemistry lab in which you're putting drops in a flask waiting for the water to turn a pink hue.

In the human body, bicarbonate levels are strictly regulated by the kidneys and are slow to respond to pH changes. Thus, you can have all the acid you want building up in the system, but without a proportional increase in bicarbonate available for buffering, you won't see the increase in CO_2 production that may seem intuitive.

Capnography Limitations

Two important limitations to capnography relate to circulatory and metabolic assessment. Although it's a diagnostic tool, capnography isn't specific. Capnography provides a reliable and accurate $EtCO_2$ level, but there's no printout or voice from the machine that tells you *what* is causing the $EtCO_2$ to change. Underlying causes must be identified by diving into your patient's clinical picture and coupling the capnography reading with your traditional assessments.

Another limitation occurs when a patient with compromised perfusion and/or metabolic status also has an excessive respiratory rate (RR) or tidal volume (V_T); these will also lower the patient's $EtCO_2$ levels. Capnography can't differentiate when more than one etiology is affecting $EtCO_2$. For example, in patients with diabetic ketoacidosis (DKA) and Kussmal's respirations, the $EtCO_2$ will be very low secondary to depleted bicarbonate and deep and rapid respirations.

Capnography can't tell you how much of the low $EtCO_2$ is from depleted bicarbonate vs. Kussmal's respirations. However, through experience, you can begin to develop your own "anecdotal $EtCO_2$ thresholds" that will help you distinguish between a low $EtCO_2$ attributed to just an increased respiratory rate vs. an *excessively* low $EtCO_2$ that might have a compounding circulatory and/or metabolic issue in play.

Let me illustrate these concepts by discussing six relevant cases.

Case One

You respond to an 86-year-old female patient needing an interfacility transfer for suspected pneumonia. The patient is sitting in a wheelchair in her bedroom. She appears alert and orientated, and slightly pale with mild labored breathing. She has a pulse of 112 bpm, blood pressure of 102/66, a RR of 24 with no excessive V_T and no fever. Are you suspicious of compensated shock? Good.

You don't need capnography to suspect compensated shock. What capnography provides is a more in-depth perfusion assessment on compensated vs. decompensated shock, as well as the severity of shock. Research has shown that an $EtCO_2$ greater than 20 indicates perfusion is adequate.[1]

If this patient's $EtCO_2$ were 30, you should consider dehydration or other "mild" circulatory issues. If it were 26 with all other vitals the same,

CONTINUED ON PAGE 14

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your suspicions of compensated shock would be confirmed.

If the $EtCO_2$ were in the low 20s, you could now be positive the patient was in compensated shock and should be concerned about the imminent "transition" to decompensated shock (again, assuming no excessive RR or V_T).

Case Two

EMS providers can use capnography to trend the success, or failure, of fluid resuscitation based on the change of $EtCO_2$ values.[2] Optimally, we'd like to see $EtCO_2$ trend upward, indicating an improvement in the patient's circulatory status. For example, you're treating a hypotensive, tachycardic trauma patient with an $EtCO_2$ of 18 (no excessive RR or V_T). After fluid treatment, you trend the patient's $EtCO_2$ in a positive direction to a level of 22, and the patient's tachycardia and hypotension are less severe as well. Although the patient's blood pressure and pulse have improved, they're ambiguous as to the perfusion status of the patient.

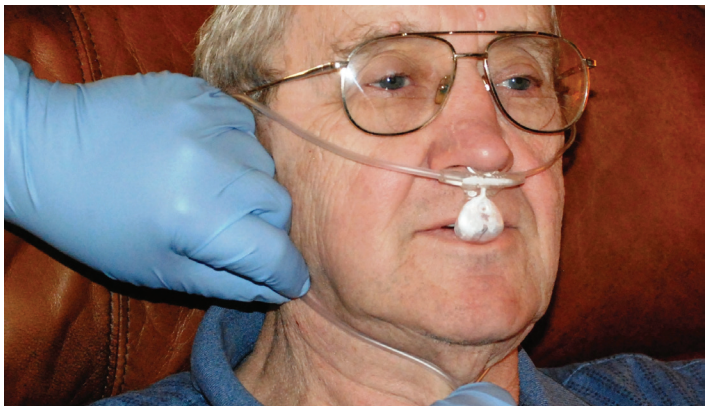

PHOTO A.J. HEIGHTMAN

The patient doesn't have to be an obvious trauma to use the perfusion trending capabilities of capnography.

However, an $EtCO_2$ of 22 is a diagnostic indicator that your treatments have significantly improved perfusion. Remember, blood pressure doesn't necessarily equal perfusion, just like ventilation doesn't equal oxygenation. Thus, knowing the $EtCO_2$ level is crucial to more specifically assessing perfusion status. However, trending blood pressure is still necessary to avoid displacing clots that the body has created to slow bleeding.

Case One Revisited

The patient doesn't have to be an obvious trauma patient to use the perfusion trending capabilities of capnography. Let's go back and reconsider the patient from the first case. If her $EtCO_2$ had been 22, we could have still treated her with judicial fluid resuscitation and seen whether her $EtCO_2$ trended upward.

If her $EtCO_2$ had improved to 24–26, you would have just decreased the severity of her compensated shock. This is clinically significant. Without capnography, you may not have known how close the patient was to decompensated shock or the true effect of your treatment.

Case Three

Research on pediatric diabetics has found that a threshold $EtCO_2$ of 30 can be used to differentiate a diabetic suffering from hyperglycemia with DKA (DKA is less than 30 $EtCO_2$), from a diabetic with hyperglycemia who isn't in DKA ($EtCO_2$ greater than 30).[1]

If a patient is hyperglycemic without an excessive RR or V_T (no Kussmal's ventilations), assess their $EtCO_2$. If their $EtCO_2$ is greater than 30, they probably still have adequate bicarbonate. If their $EtCO_2$ is less than 30, bicarbonate is now becoming depleted and we'd expect their blood gases to show metabolic acidosis.

Without capnography, we often assume that patients are in DKA if they have high blood sugar. Kussmal's would be another obvious sign of DKA, but patients can be DKA for a period of time prior to the onset of Kussmal's.

Kussmal's is a frank indication of metabolic acidosis and is initiated later by the hypothalamus after other compensatory measures have failed.

By using capnography to become more specific on your assessment of hyperglycemia, you can gain deeper insight into your patient's metabolic status, as well as provide a more detailed hand-off report to the hospital. Also, it should guide how aggressive you need to be with fluid resuscitation, as well as insulin administration if you work in a system that allows that treatment regime.

Case Four

The American Heart Association Guidelines for CPR and ECC algorithms on tachycardia and bradycardia often have an early fork in the road of treatment based on a simple, yet fundamental question: stable or unstable? Now that we know the circulatory assessment capabilities of capnography, it can become the perfusion tool of choice to answer this proverbial question.

For example, a patient in ventricular tachycardia with an $EtCO_2$ of 24 (no excessive RR or V_T) is stable from a perfusion perspective. If the same patient had an $EtCO_2$ of 17, they're below the $EtCO_2$ perfusion threshold of 20 and therefore, unstable. Or consider an unconscious patient with a bradycardic rate of 32. Although their mentation has deemed them as obviously unstable, capnography still has value in this case beyond airway and breathing monitoring in an unconscious patient. If you pace the patient, use $EtCO_2$ to assist with confirming mechanical capture and the effectiveness

of mechanical capture.

We'd expect this patient to have an $EtCO_2$ below 20. If you obtain mechanical capture, the $EtCO_2$ will rise. If the mechanical capture is sufficient enough to perfuse tissue, the $EtCO_2$ would need to increase above 20. Also, if $EtCO_2$ suddenly decreased and/or dropped below 20, you've at least lost the effectiveness of your mechanical capture, if not capture all together.

Case Five

You respond to a 61-year-old female patient with a general complaint of "just not feeling well." Her blood pressure is 108/72 with a regular pulse of 98, and she's afebrile. Capnography can triage patients with ambiguous complaints, such as dizziness, malaise and a general feeling of sickness. If a patient has a normal $EtCO_2$ level, a BLS track might be appropriate. If a patient has excessively low $EtCO_2$ levels that can't be explained by increased RR or V_T, further investigation is warranted, and EMS providers should give the patient a full ALS assessment. Further, if the providers choose an ALS track, they should capture a 12-lead ECG because it's a critical part of this patient's clinical picture.

Case Six

Cardiac tamponade and/or a tension pneumothorax with enough displacement to compress the heart and great vessels will cause low $EtCO_2$ levels secondary to compromised cardiac output. If you're lucky enough to have aggressive fluid resuscitation work for your cardiac tamponade patient, we'd expect to see a positive trend upward in the patient's $EtCO_2$. Likewise, after decompression of a tension pneumothorax that was compressing the mediastinum, we'd also expect to see $EtCO_2$ rise. However, if $EtCO_2$ stays low even after decompression of the chest, consider two things: 1) your decompression wasn't effective, and the heart and great vessels are still compressed or 2) there's another issue causing shock in the patient. Maybe the blunt/or penetrating trauma that caused the tension pneumothorax also caused internal bleeding. Again, capnography isn't specific to what's causing $EtCO_2$ changes and can't differentiate when more than one etiology is affecting $EtCO_2$.

Conclusion

By now, we've hopefully moved capnography into a new, expanded category deeper than the typical uses for airway management. Thoroughly understanding the origin of CO_2 and which factors, other than airway and breathing, can change $EtCO_2$ levels helps EMS providers use capnography for circulatory and metabolic assessment. Providers are able to move from the theoretical to the practical and use capnography to gain deeper insight into the circulatory and/or metabolic status of your patient.

The only missing piece of the puzzle is practice. If you want to be able to use capnography to its fullest potential, you should use capnography for every patient with altered mentation and/or ABCs. By doing this, EMS providers will begin to develop an intrinsic vocabulary on what "the norm" is for patients with various ailments. With this framework, a deviation from the norm becomes clearer. Just like with breath sounds, assess breath sounds on every patient to know what normal breath sounds are. Subsequently, when something is out of the ordinary, it becomes obvious.

Simply put, CO_2 is the 'smoke' of metabolism.[1]

The final step is then being able to incorporate abnormal findings into a more accurate and expanded differential diagnosis. Specifically for capnography, first rule out an excessive RR and/or V_T. Then, let experience guide you to identify *excessively* low $EtCO_2$ levels. Finally, put that in the context of your patient's circulatory and/or metabolic picture and see how that changes your clinical perspective. Update your differential diagnosis accordingly and give yourself a pat on the back as your capnography capabilities are in a whole new category of progression and competence.

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Severe Asthma

Capnography as a triage tool

PHOTO RICK BRADY

In trending the $EtCO_2$ through continuous monitoring, you can observe patient improvement or the direction you're headed.

By Bob Page, BAS, NREMT-P, CCEMT-P, NCEE

You're called to a residence for a 22-year-old male who's having trouble breathing. On arrival you locate the patient, who appears to be in acute respiratory distress. He's sitting in a chair and leaning forward in a tripod position. He has a hard time speaking and states he has a history of asthma and that this latest attack started about 25 minutes ago. He has used his inhaler twice without relief. He tells you he believes that his puffer is empty.

He's speaking in short but complete sentences. His skin is pink, warm and dry. Your partner places a combination nasal/oral capnography filter line cannula with oxygen (O_2) delivery capability on the patient and turns on the cardiac monitor with capnometer and pulse oximeter.

His initial capnogram shows a respiratory rate of 20 with a sloped leading edge to the waveform and an end-tidal carbon dioxide ($EtCO_2$) of 48. The pulse ox reading is 92%.

Realizing the severity of the condition, you hand the patient a small volume nebulizer with albuterol. After about three minutes of bronchodilator therapy, the patient appears less anxious. The capnogram shows a much different waveform this time because it has squared off, and the reading dropped to 40. The pulse ox increased to 94%. Auscultation of the chest reveals diffuse monophonic wheezes throughout the lateral vesicular fields.

After questioning the patient, he reveals that he's not up to date on all his asthma medications because he can't afford them. He's transported to the hospital where his $EtCO_2$ is trended and remains stable with a square waveform.

On arrival, you discuss the case with the attending physician noting the non-compliance with his asthma meds for financial reasons, as well as your assessment findings and the patient's response to therapy. The physician auscultates the patient's chest and, after noting that the patient still has some wheezing present, orders IV Solumedrol.

Discussion

Asthma is a common inflammatory disease that involves periodic episodes of severe but reversible bronchial obstruction. Frequent repeated attacks may lead to irreversible damage in the lungs and the development of chronic asthma.

Asthma affects millions of Americans and is responsible for thousands of deaths per year. It is more common in children and young adults, yet can occur any time in life.[1]

There are two basic types of asthma. The first one is called *extrinsic* asthma and involves acute episodes triggered by an allergic reaction to an inhaled irritant. Frequently, there's a family history of allergies, such as hayfever. The onset usually occurs during childhood or in young adults. About 80% of the time, it occurs before age 10.[2] Childhood asthma usually improves with age.

The second type of asthma is *intrinsic* asthma. In this disease, other types of stimuli initiate the acute attack. These stimuli include respiratory infection, exposure to cold air, exercise and exertion, stress, inhalation of irritants (e.g., cigarette smoke) and certain drugs, such as aspirin.

Many patients have a combination of the two types of asthma. Whatever the cause, it's an airway problem, plain and simple. It interferes with respiratory gas exchange. We understand that it can be bad, but how do we really know? How can we judge the severity of the asthma attack? How do we know that what we're doing is working?

Assessment Tools & Techniques

Traditional assessment of the patient with a respiratory complaint has been focused around subjective techniques, such as counting respiratory rates, estimating tidal and minute volumes by subjectively judging chest rise and fall to what "appear" adequate, along with the use of accessory muscles.

The accuracy of these rates can be debated forever, but in the end it must not be that important as respiratory rates are often estimated. And to think we're comparing what we get to an arbitrary number that we're tested over. The purpose of breathing is not to make the chest rise and fall; it's to ventilate and to eliminate the waste product of metabolism, CO_2.

I teach my students that noisy breathing is obstructed breathing, but not all obstructed breathing is noisy. Chest auscultation is another technique we're taught. The classic wheezing is the sound most closely associated with asthma.

The wheezing sounds, heard on exhalation first, are a result of a narrowed airway. The narrowing of the airway can be caused by chronic inflammation or acute inflammation due to a disease process or other mechanical wheezing.

To reiterate, the noise is made by a *narrowed* airway. The inflammatory process causes inflammation that's diffuse, so all airways are narrowed at the same width. The result is a single, toned wheeze called a monophonic wheeze. Imagine a group of flutes playing the same note.

An asthma attack occurs when bronchospasms occur, causing the airflow to be reduced from the alveoli at different diameters. This causes several different "pitched" wheezes to occur—polyphonic wheezes.

Imagine several different sizes of horns playing different notes. Sounds simple, right? The problem is the lack of sensitivity in auscultation due to inexperience of the clinician or misidentifying of the sounds.[3] Perhaps another reason is not being able to hear anything anyway because of ambient noise. It could be that the patient isn't taking deep enough breaths to make an audible wheeze. Also, stethoscope training widely varies with little consistency on how to do it correctly.[4] With practice and proper training, however, stethoscopy skills can be a valuable asset.[5]

Capnography's Role

I refer to capnography as a clinical upgrade. In my opinion, it's the most important clinical upgrade I've seen in the more than 30 years I've provided care. Because it's the only noninvasive measure of a fundamental life process, it can be used as a triage tool for the patient with a wide variety of complaints. In fact, capnography can give you your airway, breathing and circulation assessment in as little as three breaths.

EMS providers have questioned the accuracy of these non-invasive CO_2 monitors, specifically regarding their concordance with blood gas and partial pressure CO_2 ($PaCO_2$). In patients with severe asthma attacks, however, a study showed that the concordance of the $EtCO_2$ and the blood gas CO_2 was high.[6]

The meaning for EMS is clear. Capnography gives a good indication of the arterial blood gas level, thus providing EMS providers with an objective, reliable and accurate tool for assessing the severity of an asthma attack.

Using the $EtCO_2$ values to triage and to trend response to therapy is a leading reason for the use of capnography in the emergency setting. Patients early on in an asthma attack will tend to hyperventilate due to catacholamine release. This will lead to hypocapnia (low $EtCO_2$ reading below normal range, which is less than 35mmHg). These are considered to be *mild* asthma attacks. As the patient begins to tire, $EtCO_2$ may return to normal (35–45 mmHg). This patient is experiencing a *moderate* asthma attack.

The patient in the case mentioned earlier fell

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into this category. Finally, if the $EtCO_2$ rises above normal range (hypercapnia), then the patient is in *ventilatory failure* and hypoventilation. Aggressive therapy is warranted.[5]

Again, the value measures the adequacy of ventilation and to another extent, oxygenation. In recalling the Fick Principle of O_2 transport, one of the conditions of perfusion is that red blood cells must be able to offload their O_2 molecule into the blood stream to be used by the cells for metabolism. In a state of hyperventilation, it causes a left shift of the oxyhemoglobin curve. In English, that means it makes the O_2 bind more tightly to the red blood cells.

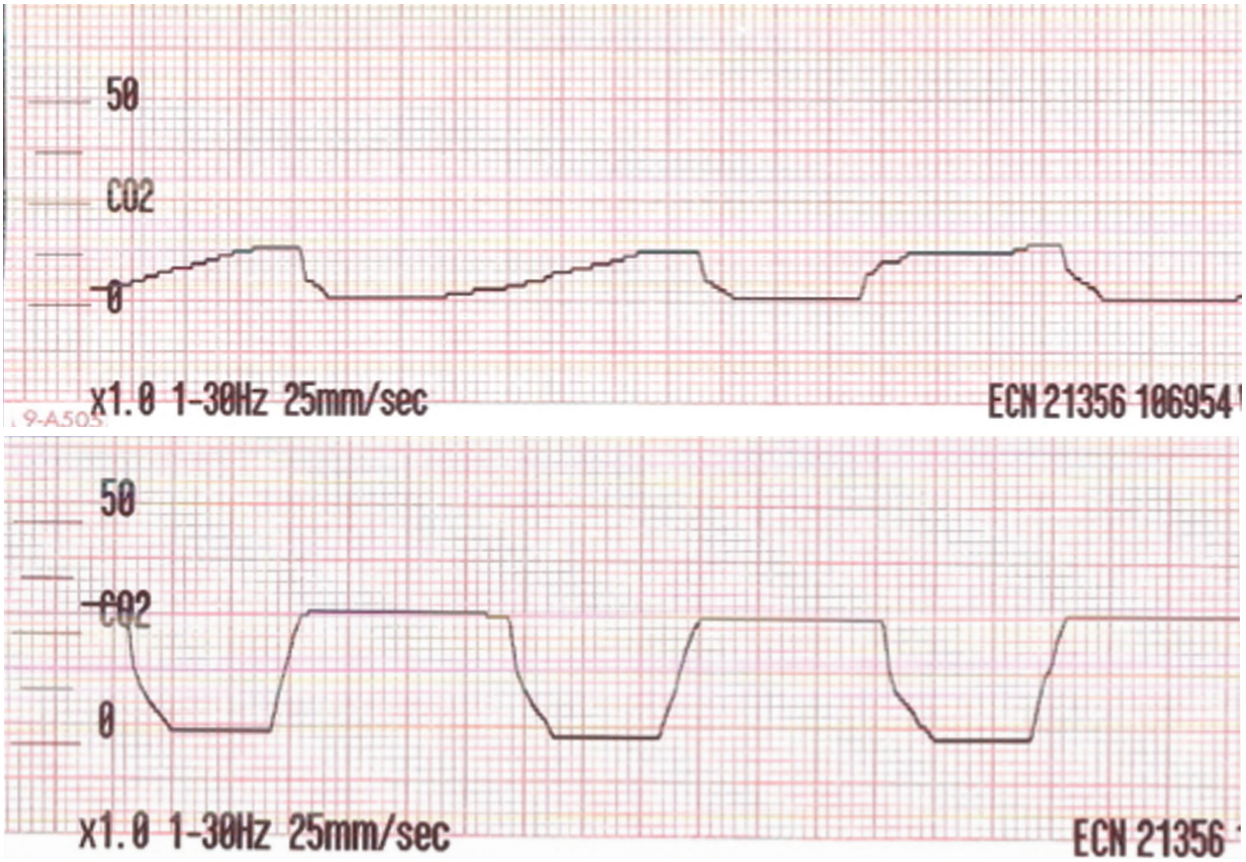
The result is hypoxic tissues with a pulse ox reading of 100%. Conversely, if the patient is exhibiting hypercapnia (hypoventilation), that will cause a right shift of the curve with acidosis and then the hemoglobin can't bind well. So the O_2 may not get to the cells.

Rapid descent of the saturation of peripheral oxygen (SPO_2) is evident in cases in which there's hypercapnia. So pulse oximetry and capnography both have a role in assessing the respiratory patient. An SPO_2 of 90 is the same as a PaO_2 of 60. A PaO_2 less than 60 would be considered hypoxemia. Therefore, the combination of hypoxemia and hypercapnia can instantly triage a patient to the ominous category of respiratory failure.

In trending the $EtCO_2$ through continuous monitoring, you can observe patient improvement or the direction you're headed. If the $EtCO_2$ trends upward (above 50), the patient is getting worse; if it trends downward (below 50), then the patient is improving. If the $EtCO_2$ remains the same, then the patient is remaining stable.

The patient is also triaged before and after therapy to see if they're improving. The value, however, can tell you their breathing adequacy. The actual waveform is used to assess the airway status.

Figure 1: Bronchospasm Before & After Therapy



WAVEFORMS BOB PAGE

These two capnograms, showing before and after therapy, demonstrate the triage and trending capability of capnography.

Technical Aspects

The waveform on the screen usually doesn't look like the printout because most cardiac monitors' screens scroll at a rate of 25mm per second and typically display about three seconds of activity at a time. This works well for heart rates. But when a patient takes 12 breaths per minute, you wouldn't see a waveform on the scope.

The screen section chosen to display the waveform is sped up about 10 times so the waveform is visible. But when the waveform is printed, it's printed real time on the paper and will be a lot

wider than what you see on the scope.

So I recommend that the paper capnogram be used to evaluate for airway issues, not the scrolling part on the screen. The scrolling part on the screen can be used to look at a square capnogram (for tube placement confirmation) or apnea (flat line). Everything else should be evaluated on the printout. It's the easiest and most accurate way to study the capnograph.

Capnography measures CO_2 flow and draws a picture of CO_2 flow over time. CO_2 comes from the alveoli. So if the capnogram is a square, then there's no obstruction preventing the CO_2 from moving in and out of the airway. Inflammatory process can cause a narrowing of the airway, but the alveoli still empty at the same rate and will display a square waveform.

Even though the patient may have wheezes, they don't have a bronchospasm because the waveform is square. What does this mean? Bronchodilators may not be necessary and probably wouldn't work on inflammation.

On the other hand, patients who have a bronchospasm (e.g., patients with reactive airway disease), have *uneven alveolar emptying*. This means some alveoli rapidly purge their CO_2; others may be more constricted, so it takes longer. This is what produces the severe angle to the upstroke and plateau on the waveform.

Bronchospasm responds well to bronchodilator therapy, such as albuterol. In this case, the patient had a bronchospasm. That rapid triage with capnography gave the EMS provider objective, reliable and measurable evidence of what the problem was, how severe it was and what to do about it.

The most severe bronchospasm will produce a waveform that truly looks like a "shark's fin." In other words, the leading edge of the capnogram curves or bends over and angles up until the inhalation phase. When this happens, the waveform loses the "plateau," or flat part. This is troubling and means that the $EtCO_2$ is much higher than it reads, and the patient can't empty the alveoli due to air trapping. It also means the patient's airway is so bad that they're at risk for dynamic hyperinflation syndrome. Another word for this is "auto peep."

Although this can occur in the spontaneous breathing patient, it also can easily occur with assisted ventilation, positive pressure ventilation and with continuous positive airway pressure when the patient is breathing too much. This occurs when the respiratory rate and depth doesn't allow enough time to fully exhale. As a consequence, the alveoli can't completely empty; with each breath, the alveoli trap more air. This can result in increased work of breathing to inhale and exhale, and even apnea. This condition will show a waveform without an alveolar plateau—a true shark's fin.

Conclusion

In the patient with an asthma attack, if the patient responds to bronchodilator therapy, the resulting objective waveform will show the bronchospasm has been relieved—with the waveform now being square. If it doesn't change, then it may be time for another medication.

In the patient with acute asthma attack, capnography is a valuable tool for triage of the severity of their condition. It also objectively trends the effectiveness of your treatment. Having the ability to provide reliable, objective and accurate assessment of the asthma patient is an important clinical upgrade that's ready for prime time.

BOB PAGE, BAS, NREMT-P, CCEMT-P, NCEE, is a nationally recognized expert in capnography presenting seminars nationally and inter-nationally for more than 13 years. His capnography courses are among the first comprehensive, nationally presented courses on capnography. Contact Bob at his website www.multileadmedics.com or via e-mail at lead2noclue@mac.com.

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Clinical Upgrade

Expanded use of capnography

PHOTO ANN MARIE NORDER

Capnography is a clinical upgrade that enhances your ability to assess, manage and maintain the patient's ABCs.

By Bob Page, BAS, NREMT-P, CCEMT-P, NCEE

You and your partner are called to a pediatrician's office for an emergency transfer to a pediatric intensive care center. On arrival at the office, you're met by the pediatrician who tells you the patient you're transporting has respiratory syncytial virus (RSV) and "is going to stop breathing." The pediatrician further states that the patient is in exam 2 with his mom and that you should load and get to the hospital right away. Aircraft are grounded by severe weather, and it's a 35-minute transport time.

The patient is a 42-day-old infant who's being held in his mother's arms. As you approach the infant, you perform a quick assessment from across the room. The baby's acting appropriately (as far as you know); his skin is normal, and there are no signs of distress, such as nasal flaring or retractions. The baby's young mom seems oblivious to what's going on.

The doctor returns to the room and asks why you haven't left yet. You respond, "We need to do a quick assessment to determine what we are dealing with." Your partner applies a pediatric/infant nasal cannula capnography filter line. After seeing the waveform (C-1) your partner says, "OK, we need to head to the hospital now and get set up to assist breathing. This kid is grunting; we're going to have to help him. His lungs sounds are hard to hear. I think he might have some slight wheezes, but not every breath."

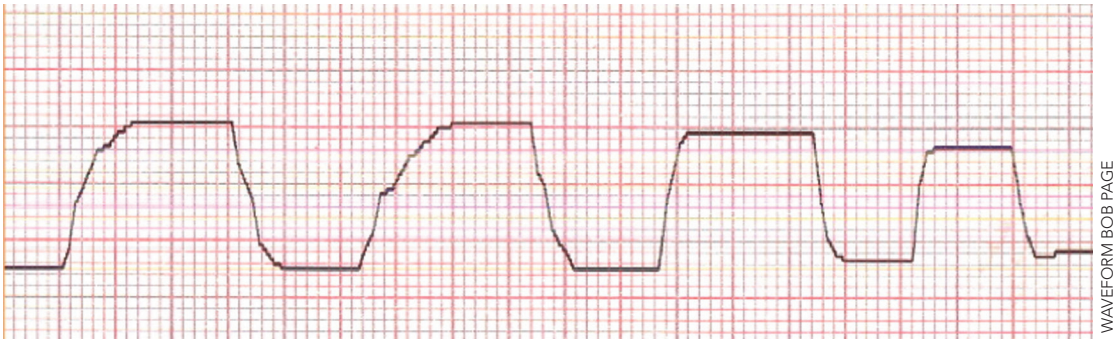
Seven minutes into transport the baby stops breathing. You get out the pediatric bag-valve mask (BVM) and ventilate the child while turning the positive end-expiratory pressure (PEEP) valve attachment to about 6. As you squeeze the bag, you notice the waveforms squared off with PEEP. You continue to ventilate the baby, carefully watching the end-tidal carbon dioxide ($EtCO_2$) reading to keep it at about 40 mmHg (see Figure 1, p. 24).

On arrival at the Pediatric Intensive Care Unit, the pediatric intensivist commends you on the care you rendered. Stat blood gases come back to normal, and the respiratory therapist gives you a smile and a thumbs up as you complete your run report. The team gathers around the infant and intubates him. They prepare to hook him up to a ventilator. There's some discussion about the PEEP settings.

The intensivist asks, "How much PEEP did you have on him?"

"6," you reply.

Figure 1: Grunting



WAVEFORM BOB PAGE

This capnogram shows grunting in a 42-day-old infant with RSV. The first two waveforms show the upstroke starting to angle toward airway obstruction. But when the baby grunted, the waveforms squared off. This helped them later determine the correct PEEP setting.

"Sounds good to me; let's start him there because it worked well for them." The team sets the same amount for the ventilator settings.

Discussion

What was going on in this case? RSV causes a narrowing of the smaller bronchioles because of an infection and inflammation. As these tiny airways narrow and the baby exhales, the venture effect collapses the alveoli on exhalation, causing atelectasis. The baby is grunting to keep the alveoli open.

In other words, he's "PEEPing" himself. Therefore, when the baby stopped breathing, the EMS providers had to take over breathing for the infant. But they also had to secure PEEP because the baby was grunting to keep his alveoli open. Had the medic not have known this or even not had a PEEP valve on his BVM, he couldn't have adequately ventilated.

What's amazing is that the initial triage in this case showed the grunting and the effects of the baby grunting. The first two waveforms show the obstruction to flow starting, but when the baby grunted, it squared off. That's objective, reliable and accurate. That's a clinical upgrade if there ever was one. There's no way the paramedic would have even known how much PEEP to set, so he used the waveforms as his guide. He set the minimum PEEP to keep the waveforms square.

Seizures or Seizure-Like Activity

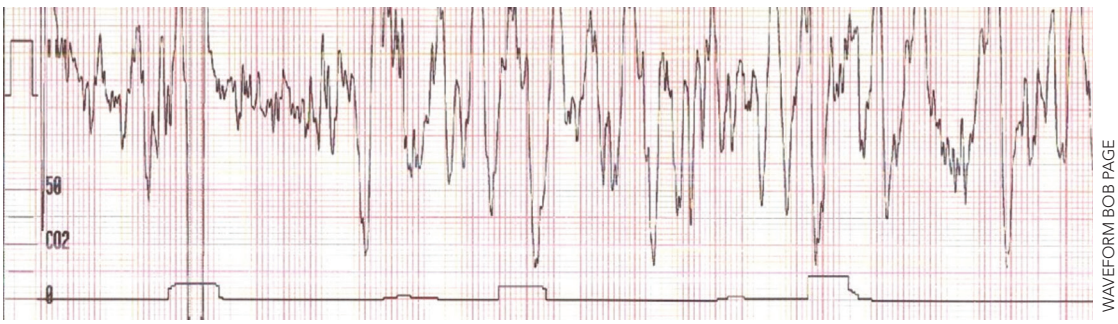
Generalized seizures, including tonic clinic and absence epilepsy affect both hemispheres of the brain and the medulla. So when a person has a seizure, they do not breathe. They make noise; this is involuntary and has been described as an "epileptic cry." This isn't effective ventilation, and it manifests itself as a small, erratic waveform with very low values on a capnogram (see Figure 2 below).

The period after the seizure is called the post-ictal phase. During this time, the skeletal muscle activity may have ceased, but the patient may still be seizing. The neuronal discharges in the brain continue, but the muscles aren't moving. The capnogram reads this as apnea. This is evidence that ventilatory support is needed and that anti-seizure medications may also be needed. Note that the post-ictal phase is defined by the fact that the patient is breathing with normal waveforms, and more importantly, with a normal CO_2 value (see Figure 3, p. 22)

This is where capnography is particularly useful. After the seizure, the EMS provider can readily

CONTINUED ON PAGE 22

Figure 2: Tonic-Clonic Seizure Capnogram

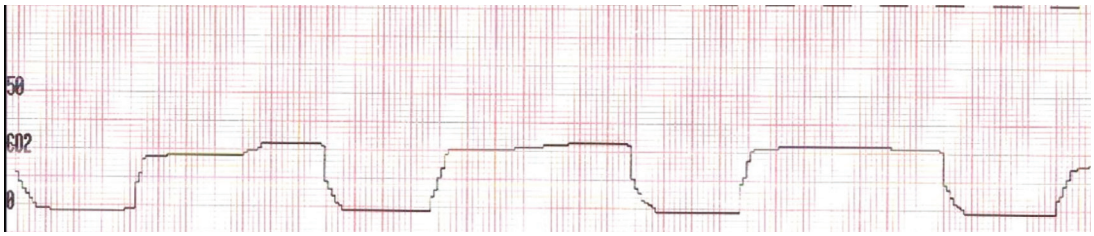


WAVEFORM BOB PAGE

This capnogram shows a man having an active tonic clonic seizure. Note the ECG leads aren't getting a good reading (obvious movement artifact), but the small waveforms means he isn't breathing, rather noisemaking. Generalized seizures affect both hemispheres of the brain and the medulla.

 CONTINUED FROM PAGE 21

Figure 3: Post-Ictal Capnogram



This capnogram shows the post-ictal phase. Notice the ECG leads haven fallen off; however, he's breathing normally post-seizure. This also means his seizure is over for now.

assess the adequacy of ventilation. This is important, especially for cases in which a benzodiazepine was used to control or break the seizure.

In the case of absence epilepsy, these seizures involve blank stares and maybe eye fluttering. Some patients even appear to be daydreaming or act like they're falling asleep. In such a case, capnography will detect the apnea during the event and can differentiate simple fainting from another condition.

The following capnogram was taken from the case a 6-year-old boy who "fell asleep in class," falling out of his chair and hitting his head on the desk next to him. The child was acting appropriately on scene and was transported as a "precaution." En route, while conversing with medics, the boy stopped talking mid-sentence and just stared motionless with a few eyelid flutters. The paramedic's noticed that the capnogram went flat line for 24 seconds. Then the child picked up his sentence on the exact word he left out (see Figure 4, below).

Then he just snapped out of it with no recollection of the event. At the hospital, the paramedics reported their findings and a neurology consult was ordered. The following day, an EEG revealed absence of epilepsy, previously undiagnosed. You may recall absence epilepsy is a generalized seizure (formerly called petit mal). Because it's generalized seizure, these patients won't breathe either. Most attacks are brief—less than 15 seconds—but could go longer. Capnography makes an excellent assessment for the patient who has seizures or seizure-like activity.

Pain Medicine

Any time a patient is sedated for any reason, you have to carefully monitor their respiratory status for signs of respiratory depression. Capnography has been proven in numerous prospective trials to be the most accurate and only objective measurement of breathing adequacy in the sedated patient.[1–4] In fact, hospitals are enhancing patient safety during procedures. When patients are on a patient-controlled analgesia (PCA) pump, they use capnography as a safety and as an early warning of respiratory depression or other adverse airway events. Many hospitals are purchasing PCA pumps with a capnography override to prevent inadvertent doses or overdose from being given.

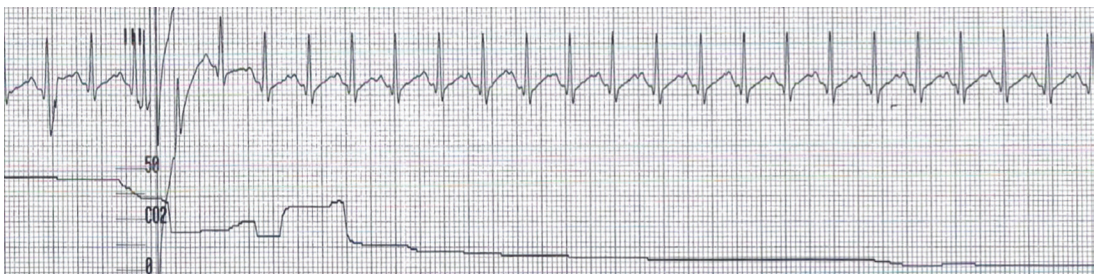
With a capnogram, detecting respiratory depression is simple. Place it on a patient for initial baseline and record. Then, using continuous monitoring, trend the $EtCO_2$. If it trends higher, then respiratory depression is occurring. However, a slowing rate and an increase in $EtCO_2$ can mean the same thing. Obviously if a patient becomes apneic, the waveform will disappear with the first breath they don't take.

How to Get Capnography

Remember the full clinical value of capnography can be appreciated only when devices are set up with the right capnography default settings. The waveform and the value should both be on the screen. This warm-up preset will allow for faster assessments. Providers should also get into the habit of printing the capnogram; you can't analyze it properly on the scope.

My students often cite ignorance or cost as the reason they don't use capnography for the non-intubated patient. As an educator, I can fix the ignorance part. Financially speaking, services should partner with others, including hospitals that will also be using them for in-hospital use to increase

Figure 4: Absence Epilepsy Capnogram



This 6-year-old boy fell out his chair and hit his head.

buying power to keep the costs as low as possible.

This technology should be budgeted in as the costs of doing business. Remember when you prioritize purchases, capnography is a tool with the ability to assess all patients. Get creative; many vendors sell the same filter lines. Let them compete for your business. Negotiate a price for buying a year or two-year's supply; lock in the price, and then purchase them as you need them.

Back to education. If you have a device now but aren't sure how to use it, get help. Seek out those who know. There's some excellent resource material that can help you study up on the subject. You'll need some hands-on practice to see what normal capnograms look like and the things that make them look abnormal. Bust open a case of oral/nasal cannulas and have your squad play with them. Wear them while you talk (talking capnogram) and while you cough, sneeze, sign and hiccup.

Breathe quickly, breathe slowly, give oxygen through it, put on continuous positive airway pressure and see what it does. Watch the effects of the waveform and the values. Do your own research. One group of volunteers discovered that the average respiratory rate for an adult at rest was 8. So much for 12–20, huh?

Another group uses a designated breather who wears capnography during scenario labs and breathes with the manikin to feel what it's like for a patient. The other students get to see what the capnogram looks like before they need to do it on a real patient. The goal is to get the students and EMS personnel as much experience as they can reading normal and "normal abnormal" waveforms. Comfort reading capnograms will lead to increased usage and better patient assessments.

Widespread Use

I teach about 60–70 classes a year all over the world on this subject, and I must admit it's more accepted today than it was in 1998 when I started teaching my course. I've noticed, however, some other issues. Through student sampling, I'm getting to the crux of the matter.

I think part of the reason for the slow adoption is the general lack of knowledge about how this technology really works and how it applies to the field practice. CO_2 detection (purple to yellow) was out front for at least a decade before the ability to measure CO_2 via a nasal cannula ever came out. This has led to a paradigm of using capnography only as a device to confirm tube placement. Some states have even required and endorsed its use for intubation only.

Add to this early sidestream devices required 300 mL of airflow per minute to get good reading; also, the technology had to be calibrated and compensated for gases other than CO_2. Today's nasal oral cannula devices can accomplish the task with specific CO_2 sensors and flow rates as little as 50mL per minute. Early nasal cannulas didn't work well with mouth breathers, but today's oral/nasal cannula filter lines can pick up from either.

Summary

Although the technology is much improved, there's still distrust among some. Some critics say it's, "just another fancy tool to make you treat it, not the patient." I've heard that many times before. Changing to a new way of doing things is never easy. We resist change. However, this is one of the most beneficial ways we can increase our "clinical quotient."

Bottom line: Capnography is a clinical upgrade that enhances your ability to assess, manage and maintain the patient's ABCs.

The time for capnography is now. I've been watching it grow in the EMS field for more than a decade. I've heard of some great uses all over the world. It takes courage and support to make this change for the intubated and non-intubated patient.

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