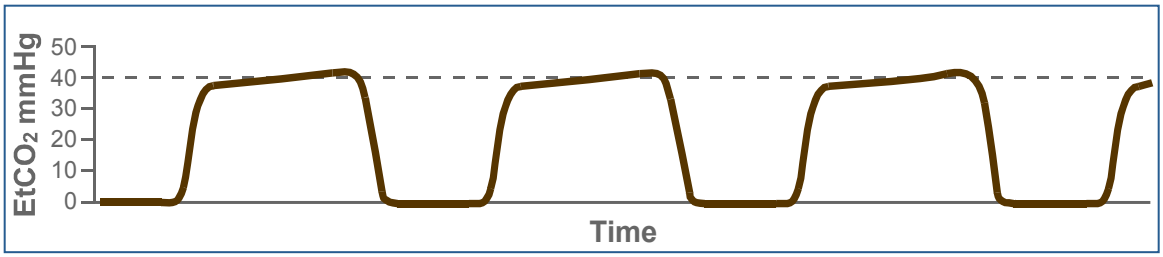
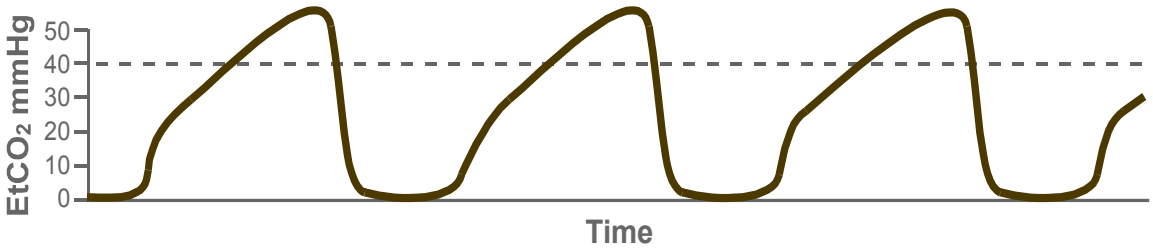
Slap the Cap

The Role of Capnography in EMS





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Slap the Cap! The Role of Capnography in EMS

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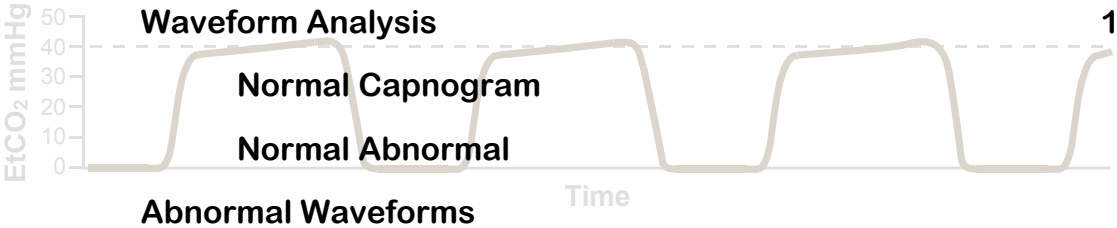


Introduction:

You are participating in one of the first nationally presented courses on capnography in emergency medicine. This comprehensive course is designed to supply you with the knowledge background necessary to understand the full spectrum use of capnography as a diagnostic tool. Just as the 12 Lead ECG is a diagnostic tool for acute coronary syndromes, capnography is a diagnostic tool for ventilation and perfusion. It is an objective, fast, and accurate way to triage, assess and monitor the ABC's in almost all aspects of the emergency medicine. This handout, while informative alone, is designed as a supplement to Bob Page's Course: Slap the Cap, which offers far more extensive practice, case presentations and explanations.

OBJECTIVES

By the end of this session, you will be able to:

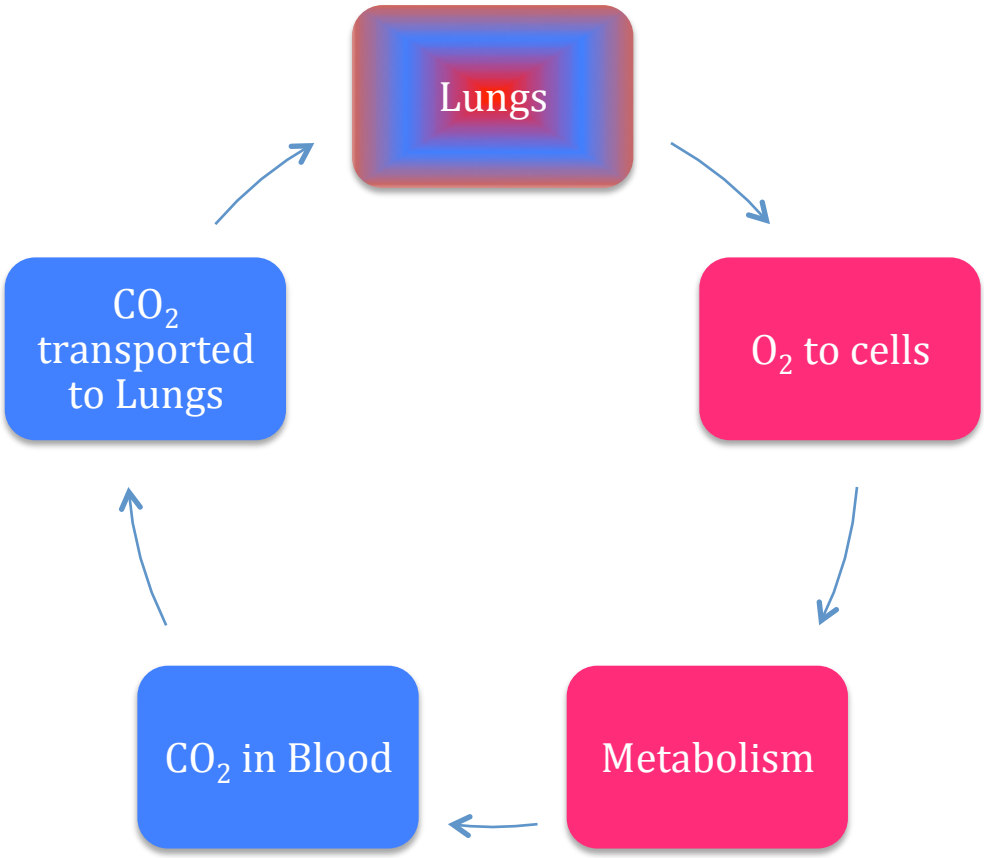
- **Describe the structure and function of the upper and lower airways.**
- **Describe the mechanics and science of ventilation and respiration.**
- **Describe the basic physiology of perfusion.**
- **Describe the relationship between ventilation and perfusion.**
- **Describe the principles behind CO_2 measurement.**
- **Describe the various methods of $EtCO_2$ measurement including quantitative and qualitative capnometry and capnography.**
- **Describe the technology of $EtCO_2$ measurement including mainstream, sidestream and microstream sampling.**
- **Identify the components of a normal capnogram waveform.**
- **Identify abnormal capnogram waveforms as related to various airway, breathing and circulation problems.**
- **Discuss the various clinical applications of capnography.**
- **Given various cases, discuss the role of capnography in identifying the problem and in the management of the patient.**

Anatomy and Physiology Review

This course is based on the performance of individuals that take capnography courses. While it would be easy to show what to look for on a capnogram, not knowing how or what it is telling you can be a double-edged sword.

The Circle of Life

In the popular Children's movie and play, "The Lion King" the hit song and theme is about the great circle of life. In a way, our body also has a circle of life. We are put here on earth and consume the oxygen that is given to us, in return we must give back CO_2. The human circle of life can be visualized with this graphic.



As we breathe in oxygen, it enters in our lungs and through the alveoli it diffuses into the blood stream. Oxygen is then carried, bound to hemoglobin molecule (on-loads) on the Red Blood Cells to the body cells that can use them for metabolism. The oxygen dissociates (offloads) from the hemoglobin molecule and diffuses into the cells.

Once the cells use oxygen for metabolism, the net product is energy (ATP) and waste product CO_2. CO_2 is then diffused into the blood stream and carried back to the lungs as bicarbonate Ion. Once in the lungs, the CO_2 is released out of the body as you exhale. In effect, air goes in and out and blood must go round and round to put it quite simply.

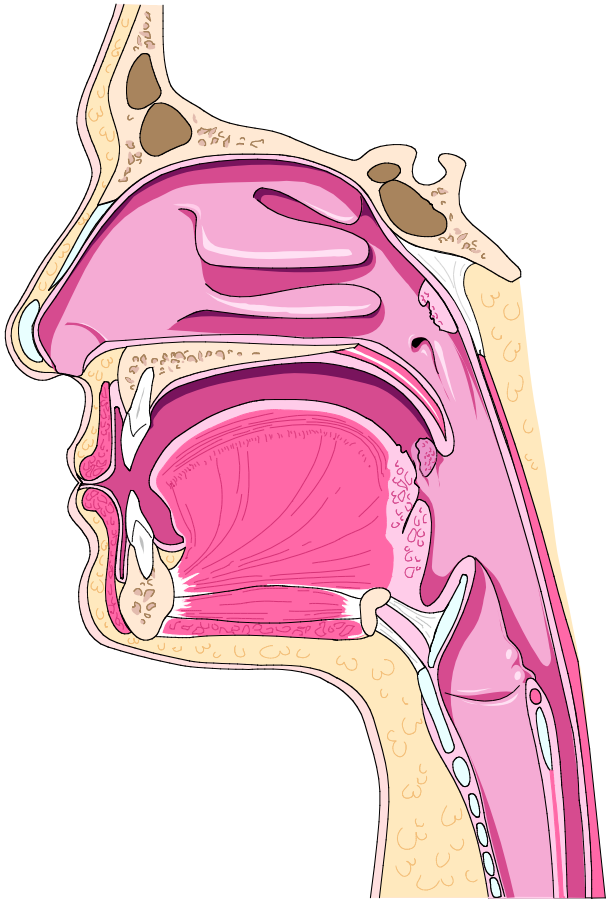
With normal physiology, and circulation is adequate, a prescribed amount of CO_2 should be exhaled. This is a *fundamental life process. Capnography is the only non-invasive tool that can measure this fundamental life process*. This is the main reason why capnography is such a valuable assessment tool.

Upper Airway

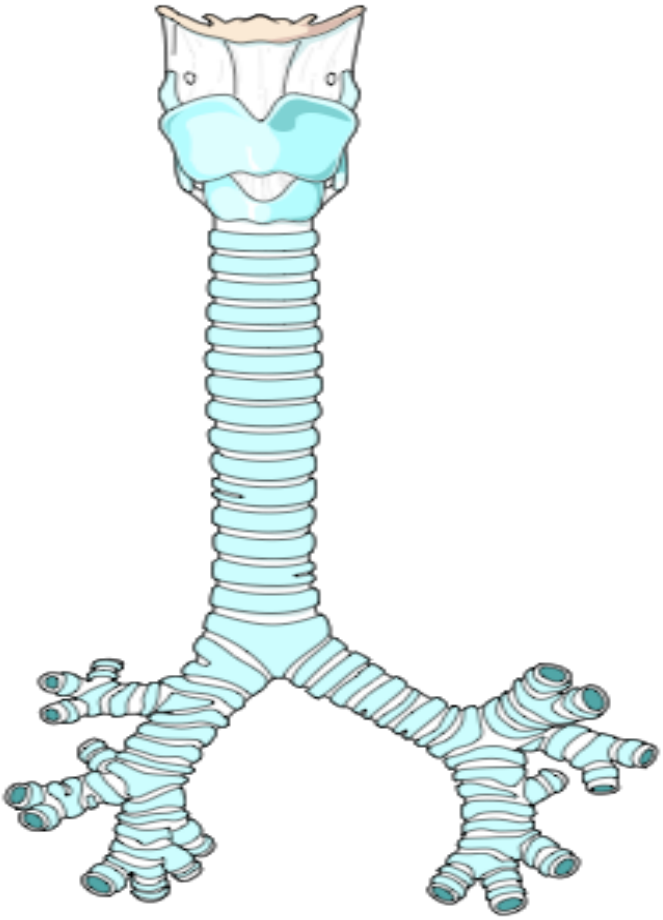
The physiology of the upper airway structures is to warm, filter and humidifies the air that you breathe in.

The various structures also cause a resistance to airflow on exhalation that offers a positive end expiratory pressure or "PEEP". PEEP is one of the three mechanisms you have to prevent collapse of the alveoli or *atelectasis*. The alveoli are the only place in the body you can exchange gas with the environment.

People with pathologies such as emphysema often need to "purse" their lips to provide PEEP because the disease destroys two of the three mechanisms. What would happen to PEEP if this patient were to be intubated?



Lower Airway



Larynx

Trachea

Carina

Left and Right Main stem Bronchi

25 divisions of the bronchial tree

Bronchioles

This area from the nose to the bronchioles is known as DEAD SPACE AIR. By definition this is air not available for gas exchange. This particular type is called *anatomical* dead space because there are not alveoli until you get to the end of the airway.

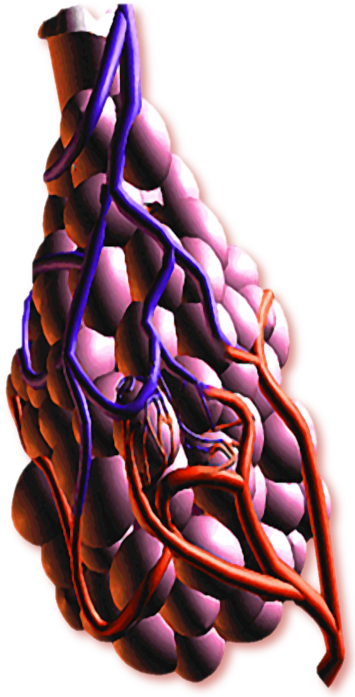
When the dead space is *alveolar*, this occurs when you have pathology such as air but no blood, blood but no air, or air and blood but the exchange surface is compromised. Understanding this pathology is the key to uncovering the cause and to reverse the situation.

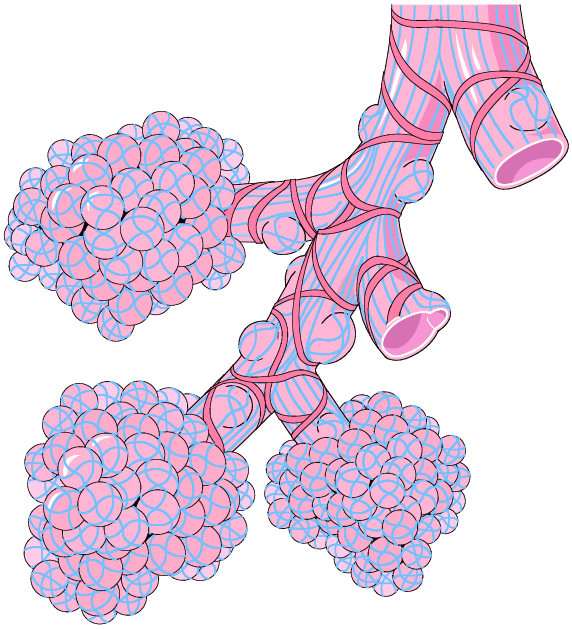
DEFINITION REVIEW:

Dead Space Air: Air that is not available for gas exchange.

Anatomical Dead Space: Air that occupies the space between the nose and bronchioles that never exchanges gas. (about 150ml in the average adult)

Alveolar Dead Space: When gas exchange doesn't occur because air is present but no blood is available to exchange gas. Or there is blood but no air. It could also be because the exchange surface is compromised by pulmonary edema, pulmonary effusion, or swollen membranes.

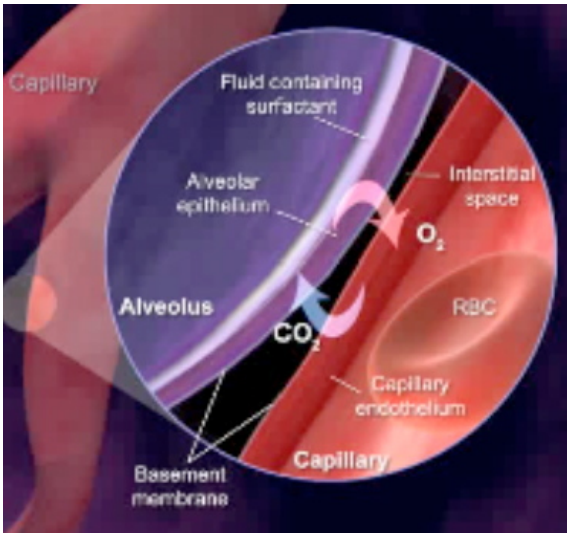




Bronchioles and Capillaries

This is where the gas exchange occurs in the lungs. It is the only place in the body where you exchange gas with the environment. This juncture is where most of the pathology we encounter occurs. The alveoli have elastin fibers around them to allow them to stretch and reform their shape. The inside of the alveolar membrane has surfactant to allow re-inflation and to prevent atelectasis.

Between the capillary and alveolar membrane there is a thin layer of fluid that O_2 and CO_2 travel across to exchange gas. In some circumstances, pathology and leaking capillaries into the interstitial space increase the fluid. This makes it harder to exchange gas and can results in atelectasis. CPAP is a useful tool for this pathology as it splints open the alveoli and allows higher pressure for gas exchange and can even prevent fluid intrusion.



RULE of LIFE # 1: Air Must Go In and Out

The Physiology of Breathing

Why do you breathe? Although you need oxygen to live, you breathe to get rid of CO_2. CO_2 is produced as a by-product of cellular metabolism. The chemoreceptors in you brain sense the levels of CO_2 and then report to the medulla. The medulla triggers the ventilator effort. So, as a result of eliminating the CO_2, you get oxygen in return. This is the circle of life as the plants take in the CO_2 and give us back oxygen.

Some people with pathology may breathe on a hypoxic drive; that is they breathe when oxygen levels are low. We were all told to watch out giving oxygen to a hypoxic breather because we could shut them down.

What we are talking about is depressing their stimulus to breathe. If a person loses their stimulus to live, we say they have clinical depression. To diagnose that, a trained screener that asks a series of questions will interview the patient. If one loses their stimulus to breathe that is called respiratory depression. So we screen for that by asking, "Are you too sad to breathe?" Well not really, but I think you can see something here.

Breathing is a Chemical Thing!

So if we want to know if breathing is adequate, then we ask the question: WWMD? What Would Medulla Do?

Since the medulla triggers the effort of breathing, just what happens? The diaphragm contracts and moves down. The intercostal muscles contract and pull up. This makes the chest wall expand and creates a negative pressure inside of your lungs, so air comes in. So we are all negative pressure breathers. In order for us to breathe, we have to create a negative pressure on the environment. This requires an intact thoracic bellows, and a lot of energy to make our muscles do the work of breathing.

That being said, this leads to one of the signs of adequate breathing, (or so we are taught) to be chest rise and fall. Well not so fast. In a flail chest or a open chest wound, the bellows is not intact so when the chest expands, the flail segment or hole in the chest will negate the pressure change, so we have chest rise and fall without air movement. That is not at all adequate. That is why you are taught to seal open chest wounds and secure a flail chest.

One more interesting note: To take a normal tidal volume breath at rest the chest need only create a 2mm/hg pressure difference to make air

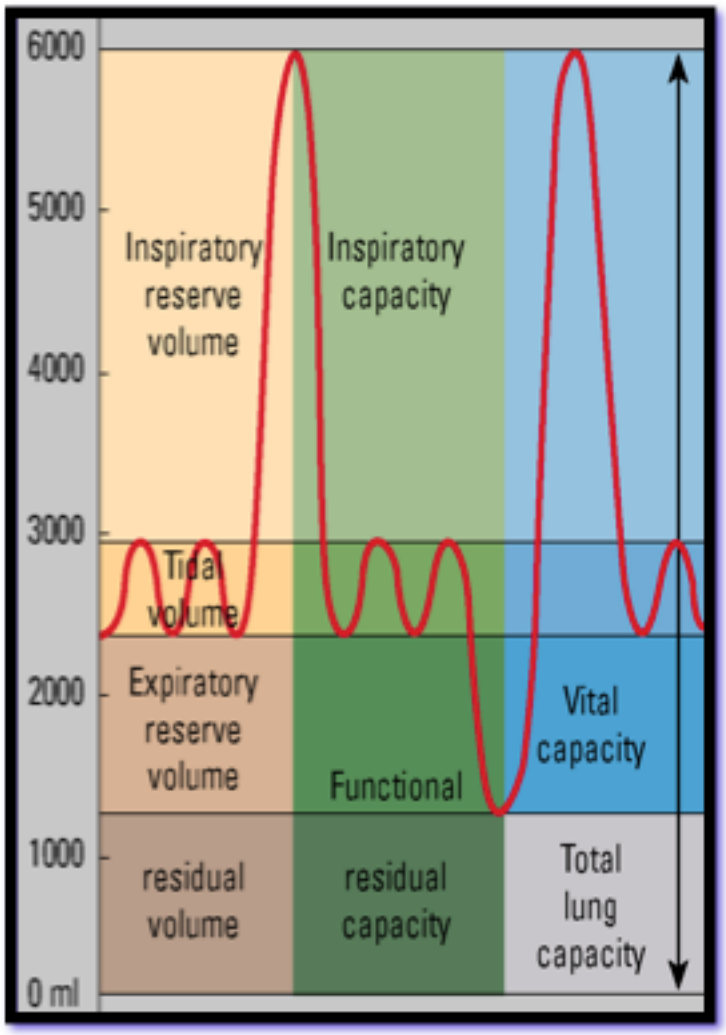
move into the chest. In most people this will only require a 2cm excursion of the chest wall. Hardly noticeable to count.

A Question of Volume

500ml is normal Tidal Volume for adult at rest

150ml of that is anatomical dead space

350 alveolar volume for gas exchange



Total Lung Capacity is about 6000ml

Vital capacity is the maximum amount you can exhale in a forceful exhalation, about 4800ml

This leaves about 1200ml residual volume.

The larger the volume the more alveoli that are recruited and the more gas exchange occurs.

Definitions:

Hyperventilation: Blowing off more CO_2 that you are making. This creates a deficit and in unchecked, can lead to cerebral vasoconstriction and a left shift of the oxyhemoglobin dissociation curve, not allowing O_2 to be released at the cellular level. The patient then becomes hypoxic, with a pulse ox reading of 100%. This is similar to the effects of CO poisoning.

Hypoventilation. Making more CO_2 than you can exhale. As CO_2 rises in hypoventilation, this can lead to acidosis and a right shift of the curve not allowing O_2 to bind easily to hemoglobin, so it cannot be carried to the cells to be used. As a result the cells become hypoxic. Pulse oximetry in acidosis will drop off after a few minutes.

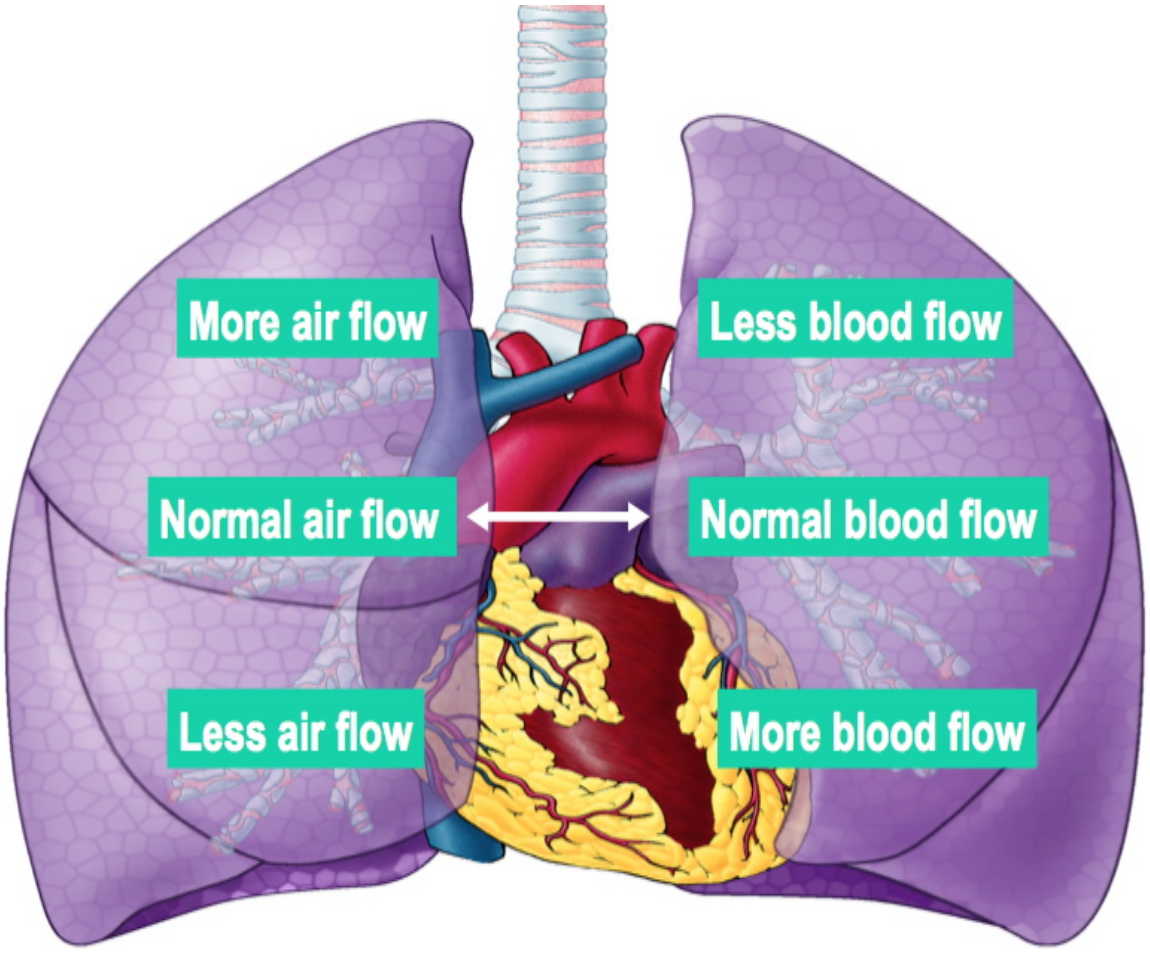
So the true measure of adequate oxygenation should include the measurement of expired CO_2. To oxygenate adequately, one must have normal CO_2 output. This is because oxygenation is dependent upon the RBC's ability to on load and offload oxygen. Hyper or hypoventilation will hinder this ability.

The FICK principle of oxygen transport shed further light on this when it mentioned that RBC's must be able to on load and offload O_2 as a condition necessary for perfusion to occur.

Other conditions mentioned by Fick including having enough oxygen, having enough RBC's and having the pressure to transport them around the body. That is commonly referred to as rule #2 of life. "Blood must go round and round."

The Balance

Optimal gas exchange occurs when there are equal ratios of blood and gas available. This is called the V/Q ratio; that is ventilation to perfusion ratio. There is a physiologic balance inside of your body. It is based on normal anatomy and physiology.



Earlier we discussed anatomical dead space. That referred to airway structures that carry air down to the alveoli. Once there it can be exchanged as ventilation is accomplished and respiration. The physiological balance exists as follows.

In the upper lobes of the lungs, V>Q so there is limited amount of gas that can be exposed because there is not sufficient blood supply there available for gas exchange.

In the middle lobes, V=Q so this is where most gas exchange occurs on normal tidal volume breaths.

In the lower lobes of the lungs, V<Q: that is there is more blood available to exchange gas, but not much air gets down there in normal breathing. The potential is there, but unused.

We have all heard of trying to assess respiratory adequacy with such subjective criteria as respiratory rate and chest rise and fall. With this balance so variable it can be next to impossible to know based on those criteria.

All things considered look at the following example.

2 Patients, Different rate and volume, who is exchanging the most gas?

A.	**B.**	
500ml V_T	**300ml V_T**	**(tidal volume)**
12 RR	**20 RR**	**(respiratory rate)**
6000ml V_M	**6000ml V_M**	**(minute volume)**

Although different they are still moving the same amount of air per minute, so what do you think, who is exchanging the most in a minute? Are they then same?

What are we leaving out here? Dead Space Volume (150ml per breath.)

A: Dead space volume of 1800ml B: Dead space volume of 3000ml

Alveolar volume for A is 4200, and B is 3000ml, so A is exchanging more!

BOTTOM LINE. Do you really think the medulla is measuring the rate and depth of breathing? NO!! It is measuring CO_2 for the adequacy of breathing and so should you whether the patient is breathing on his or her own or you doing it for them!

SECTION 2: The Technology of Capnography

It goes by many names but I have to clear the air with the correct names so we all can have a conversation about capnography;

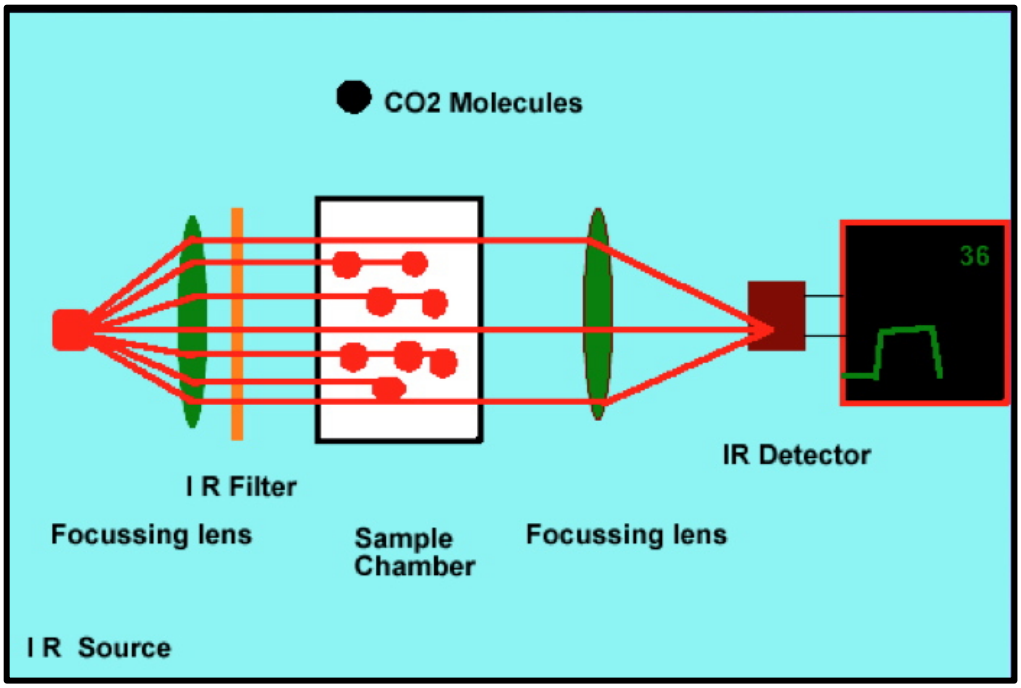
Qualitative Capnometry: Measure the CO_2 by the quality of the color change between purple and yellow. Make no mistake, this is simply detection of CO_2, it is not a measurement therefore it has very limited use in emergency medicine. Even its accuracy in confirming ET tube placement is questionable, so much in fact that it is recommended to have another device BESIDES that one.

Quantitative Capnometry: Measure the CO_2 and give us a value. Values are useful for triage, assessing the severity of a condition and trending therapy and response to therapy.

Quantitive Capnography: Measure the CO_2 and express it with a waveform and a value. This gives you an numeric value and a waveform.

Measuring $ETCO_2$

The technology by which CO_2 is measured is called infrared spectroscopy. That's a fancy way of saying take a sample of air and send IR light through it and measure the results. All devices measure CO_2 this way, some use broad spectrum beams, while other use more specific beams only for CO_2, Machine that use broad spectrum beams must be compensated for when giving nitrous oxide or O_2 concentration>40%



Definitions of Measure: ETCO2 values.

Two terms that are synonymous are $PetCo_2$ and $ETCO_2$. "Pet" is "Peak End Tidal." $ETCO_2$ is a slightly shortened version. The Peak End Tidal is the highest level the CO_2 obtains.

The normal range for CO2 in the body is 35 – 45mm/hg. The $ETCO_2$ in cases of normal perfusion have a very close correspondence with Blood Gas CO_2 ($PaCO_2$). The mean difference is 2 and the average difference is up to 5mm/hg difference. In some studied it even closer than that. So, In cases or normal perfusion, capnography can be used as a triage tool to determine the CO_2 levels in the body via exhaled CO_2 readings.

35-45 mm/hg	**Normal**
Less than 35mm/hg	**HYPOCAPNIA**
More than 45mm/hg	**HYPERCAPNIA**

Hypocapnia: $ETCO_2$ less than 35mm/hg

Clinical reasons for HYPOCAPNIA:

1. **Hyperventilation syndrome. The patient, for whatever reason is blowing off more CO_2 than they are making. Or what is worse, we could the one doing the hyperventilation. This can be due to many reasons including compensation for a metabolic condition, i.e, DKA or involuntary reflex such as in Cushing's response to a closed injury and ICP. REMEMBER capnography does not tell you why or how they are hyperventilating, it simply measures the CO_2. You can take the reading in the context of the physical exam and that could be very helpful.**

2. **Hypoperfusion: Shock. If blood doesn't get back to the lungs to exchange gas, the amount of CO_2 coming out will be diminished. The following conditions will cause hypocapnia: Shock, hypotension, pulmonary embolism, doing CPR are just a few of the hypoperfusion reasons for hypocapnia.**

3. **Hypothermia: Since CO_2 is produced as a normal byproduct of metabolism, in a hypothermic state, the body's metabolism is slowed down, so less CO_2 is produced soles is blown off. By the same mechanism, fever (increased metabolism) will produces spikes in CO_2. For the most part, healthy people will increase their breathing to compensate for this. This is called effortless tachypnea, which is increased respiratory rate, without dyspnea. Most are not even aware they are breathing fast.**

Hypercapnia: ETCO2 more than 45mm/hg

Clinical reasons for HYPERCAPNIA:

1. **Ventilatory failure. The purpose of ventilation is to normalize the CO_2 in the body. In a hypercapnic state the CO_2 being exhaled is the same as the CO_2 in the body indicating hypoventilation and failure of the ventilatory system to auto-correct the CO_2 levels. This condition, if allowed to exist, can lead to acidosis, which can lead to hypoxic cells. Another important note to make about hypercapnia is that if there is higher than normal to high CO_2 exhaled then perfusion to the lungs has to be adequate. In other words, a patient cannot be hypercapnic and hypotensive.**

2. **CO_2 retention. It is well known that some end-stage COPD patients retain CO_2. Over time, CO_2 levels increase slowly and the increase is usually overlooked by the medulla. In end-stage COPD, the stimulus to breathe can become the hypoxic drive (low O_2) because the medulla is numb to the CO_2's signals. For this reason, the patient with COPD can retain CO_2, but still maintain normal acid-based balance by compensating with the kidneys. When the stimulus to breathe is the hypoxic drive, increased amounts of oxygen given supplementary could cause respiratory depression by suppressing the low O_2 stimulus. This is why we are told to be careful administering oxygen to late COPD. The way you can tell that the CO_2 retainer is a hypoxic breather is after oxygen administration the CO_2 levels will increase above their normal baseline. This is why it is so important to measure $ETCO_2$ before oxygen administration.**

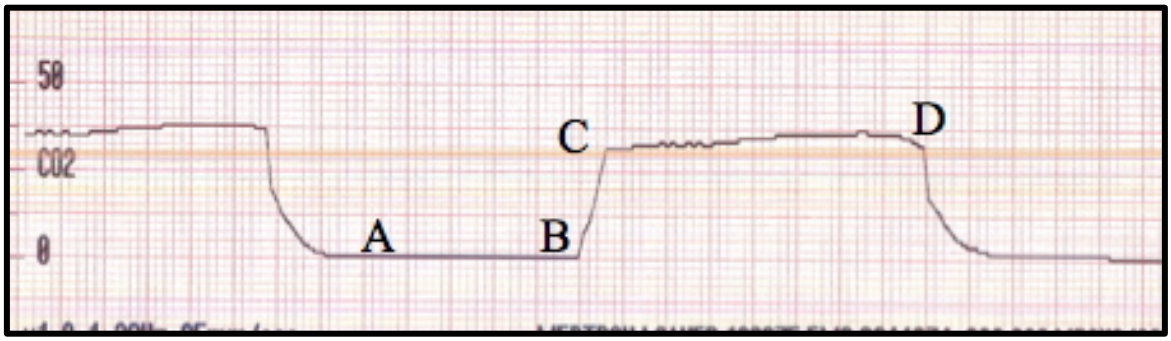
3. **Acidosis: Acidosis occurs when the blood Ph drops below 7.35. Respiratory acidosis is present when there is high $ETCO_2$ and low Ph. Acidosis causes a right shift of the oxyhemoglobin dissociation curve causing oxygen to have difficulty binding to hemoglobin where it can be carried to the cells. This causes reduced oxygenation to the cells.**

Reading the Wave forms (Riding the Waves)

"An $ETCO_2$ value without a wave form is like a heart rate without an EKG." (B. Page, 1998)

Imagine caring for a patient with extreme tachycardia without any way of determining the rhythm you're about to treat. Although we will treat tachycardia, the selection of medications can be lethal if the wrong one is chosen. That is why emergency care providers spend hours mastering EKG interpretation skills. Although the rate is why we treat, the ECG is what we treat. The good news is, there are very few capnograms to understand. Let's look at a waveform analysis.

In reading a waveform, it's important to realize that capnography measures CO_2 flow, not airflow.



Phase I is called respiratory baseline (A-B). This is at the start of exhalation and is dead space air without CO_2. As the alveolar CO_2 makes its way out of the body, you'll see a vertical upstroke called Phase II.

Phase II is called respiratory upstroke (B-C). During respiratory upstroke the CO_2 from the alveoli rapidly rises and is measured upon exhalation. For most patients this should be nearly vertical. It then makes an abrupt 90-degree turn to Phase II, the expiratory plateau.

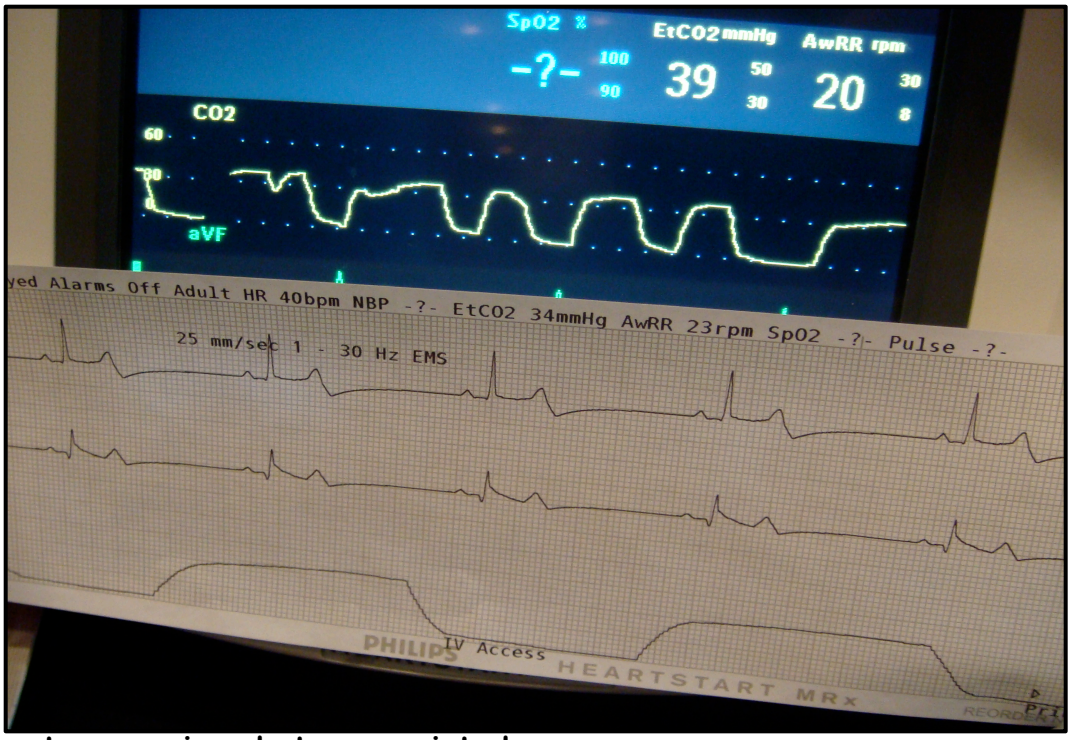
Phase III is called the expiratory plateau (C-D). The expiratory plateau should be flat, like a plateau. During this phase, smaller alveoli gradually increase the CO_2 until it reaches peak level. At the peak level is where the $ETCO_2$ is actually measured and a value is given.

Phase IV is inspiratory downslope. During this phase, inhalation occurs and the CO_2 is rapidly purged from the airways and the alveoli as pressure is brought in. This concludes the capnogram and makes the square waveform that you see pictured.

So the normal capnogram will be square with a flat plateau and an $ETCO_2$ between 35 and 45. What can we say about this? A square waveform means there is no obstruction to CO_2 flow. A flat plateau means they're exhaling their CO_2 to peak level, and a flat baseline means there's no re-breathing.

The normal capnogram.

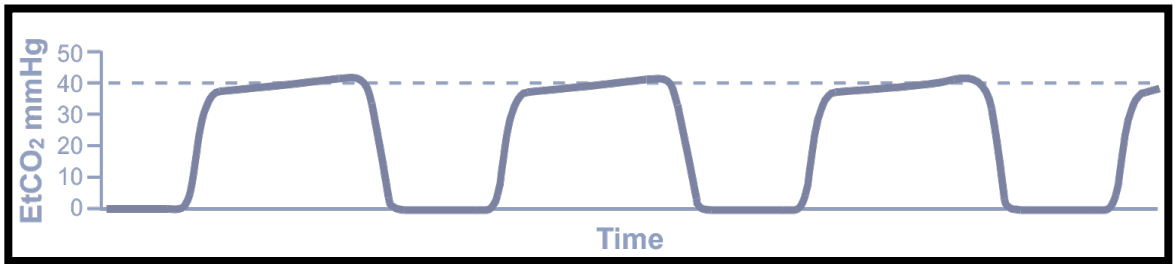
When you first discover capnography you'll notice a difference between the on-screen wave form and the printed waveform. This is a result of a technology trade-off so that a waveform can be seen on the screen. Remember that the normal ECG speed is 25 mm per second. That means that the normal monitor screen can display just three seconds at a time. If a patient is breathing once every five seconds, then it would be impossible to see a wave form on the screen. Therefore, the manufacturers, through software, have increased the speed of the CO_2 channel (user definable) to ten times its normal rate. This allows the user to see waveforms across the screen in real time. These waveforms, however, are not diagnostic except for the presence of a square waveform or a flat-line. In other words, to accurately diagnose a waveform, the user should print the waveform real time on the paper. It is best practice to have a dedicated channel on your multi-parameter monitor, preset to capnography. Never try to interpret a waveform, other than a square or a flat line, on a monitor scale. Always print one out.



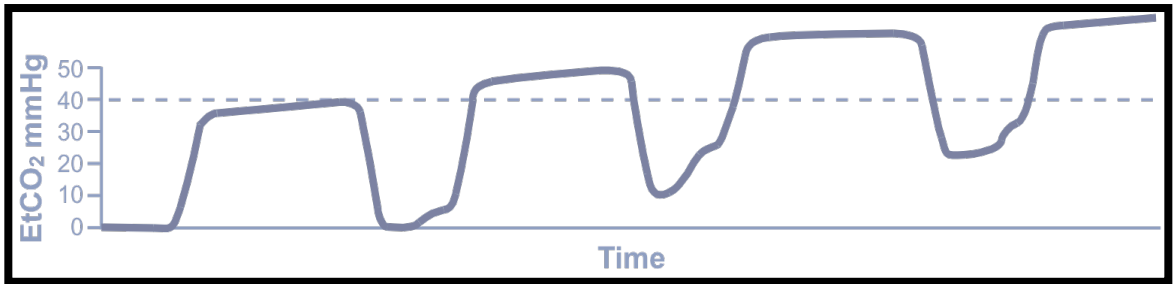
Direct comparison between printed versus onscreen capnogram.

Steps to Capnogram Interpretation.

Step I. Is there CO_2 present? (Do you have a waveform? Any wave form?)

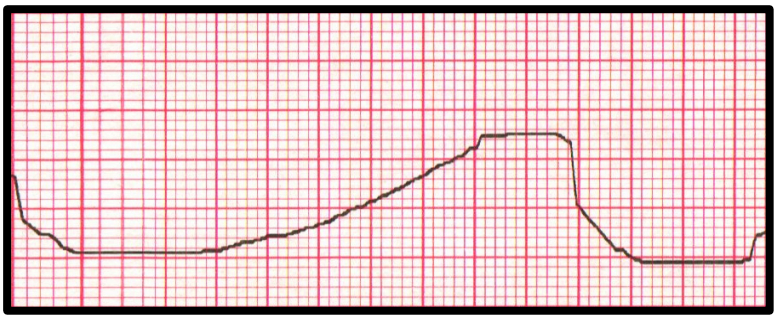


Step 2. Look for re-breathing. With re-breathing comes a breath-to-breath increase in the respiratory baseline. Rebreathing patterns such as below have been recorded in Morbid obesity, late term pregnancy, and on "low end, preset ventilators" that the exhalation valves did not open.



Step 3. Respiratory upstroke. It should be a square. However, observe for sloping or prolongation.

Step 4. Look at the alveolar expiratory plateau. It should be flat. If the expiratory upstroke is curved over that affects the plateau, this is known as alveolar emptying. An uneven alveolar emptying occurs secondary to a bronchospasm. This is different from standard inflammatory response. During inflammation, most small airways are narrowed, however they are narrowed evenly throughout. Therefore CO_2 is eliminated at the same rate, causing a square waveform. These patients may wheeze chronically because of this narrowing. However, this wheeze is characterized by a single tone, also known as a monophonic wheeze. The waveform will be square in most inflammatory processes. However, when a bronchospasm occurs, the lower airways are narrowed at different diameters causing an uneven alveolar emptying. This causes the capnogram to slope towards the plateau instead of forming a square waveform. This pattern, sometimes called a shark's fin, is truly diagnostic of a bronchospasm.

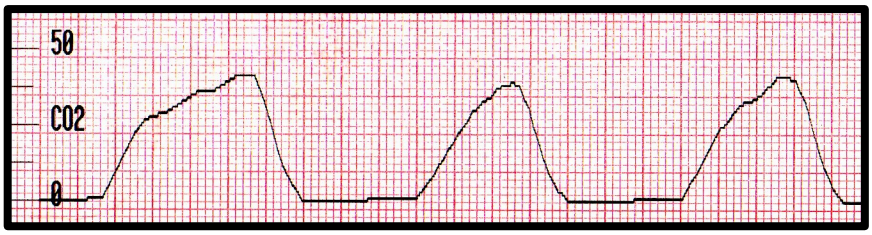


Uneven Alveolar Emptying

Bronchospasm (Not a Shark fin)

Most commonly seen in asthma

Not all bronchospasms will have a shark's fin. A true shark's fin exists when you lose the alveolar plateau altogether. This is a serious condition, which means that the CO_2 is higher than it shows and the patient is probably in autopeep. What can be said is that a square wave form all but rules out a bronchospasm because the alveoli are emptying equally. This is an objective finding. Remember, it must be assessed on the actual print out and not on the monitor scope.

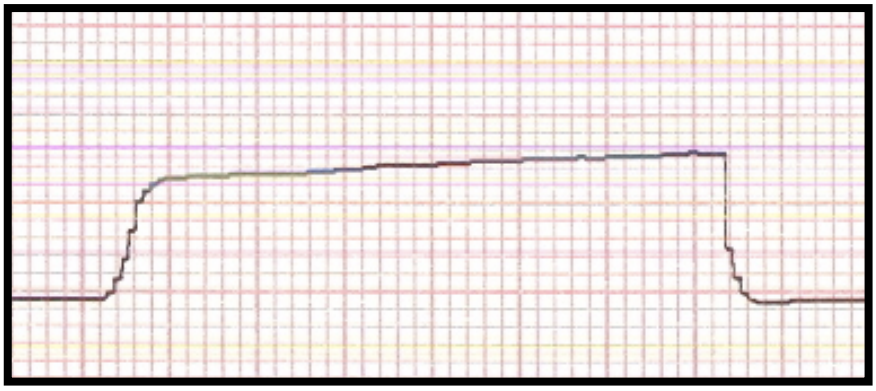


True "shark's Fin" waveform

Loss of Plateau

Step 5. Finally, read the CO_2. The end title CO_2 value is a valuable triage and trending tool. The normal mean CO_2 is 40, with the range being 35-45 mm mercury. Having a normal CO_2 and a square waveform indicates the following:

1. There is no obstruction to CO_2 emptying.
2. Their breathing is adequate. (A CO_2 of 40 means the perfusion is normal, therefore the CO_2 reading should equal the blood gas CO_2, so ventilation is normal.)
3. Their perfusion is adequate because you cannot get a normal or high CO_2 reading while hypoperfusing. In other words, the ABCs are intact.



$ETCO_2$ = 40

The Normal Capnogram

This tool can give you an objective

measurement of the patient's airway patency, breathing adequacy, and circulatory proficiency with every breath they take. It is the ultimate measure of a fundamental life process called diffusion.

So the benefits of capnography are, you get a value and a waveform. It gives you rapid, objective and reliable assessment data of the ABCs. This data is measurable and trendable. In the old days, we used to triage patients with a "quick look". This allowed us to assess the dead for shockable rhythms so that time to therapy can be reduced. Today, we have capnography for the living (or the dead), and so we say "slap the cap" as our quick look for the living. With objective evidence of the ABCs it can help us rapidly identify immediate life threats.

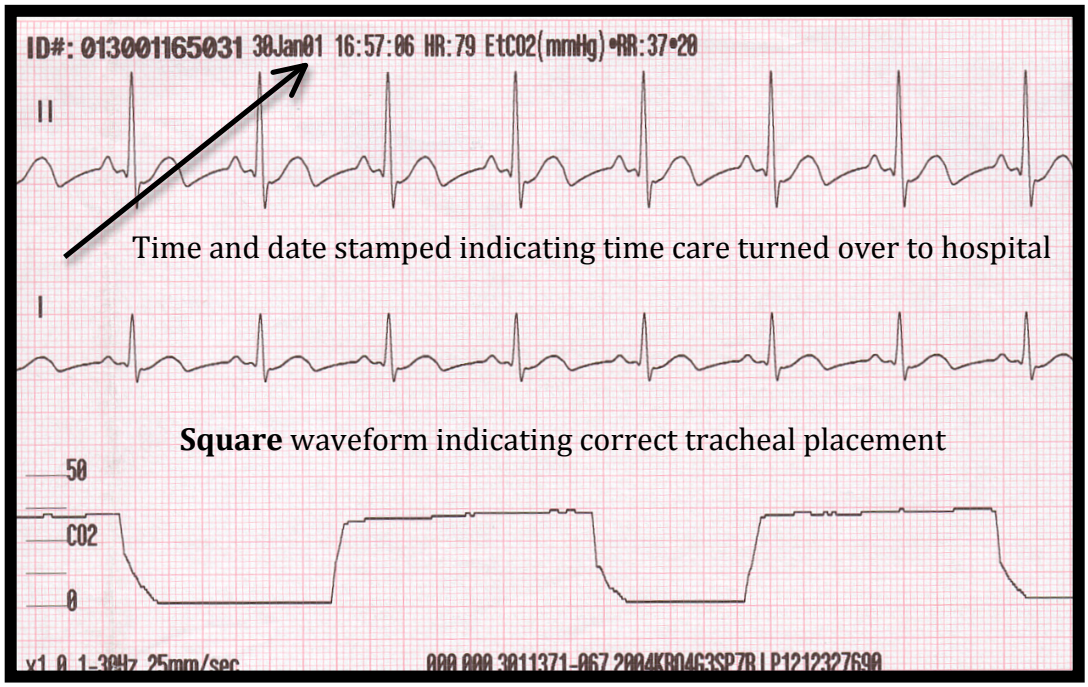
Section 3: Clinical Applications

The intubated patient.

Part I. To confirm tube placement.
This is probably the most common use of capnography, yet limiting oneself to this use only is a huge waste. In the beginning, color change devices would detect CO_2 levels. This is widely believed to be able to accurately predict when the endotracheal tube is misplaced in the esophagus. Theoretically, there should be no CO_2 exhaled from the esophagus, on the trachea. However, in low perfusion states, this is not a very accurate reading and the manufacturer even suggests using another confirming device besides this one.

Since modern capnography can measure CO_2 all the way down to one (NOTE: This is an objective measurement, not a color change), the numbers however, cannot confirm correct tube placement because low CO_2 readings can also come out of the esophagus or will also show when the tube is in the hypopharynx. For this reason, there is "no value of CO_2 that would confirm endotracheal placement".

This leaves the waveform that is the gold standard for endotracheal tube confirmation. Tube confirmation is confirmed with a SQUARE waveform. With a square waveform, the tube cannot be in the esophagus, or the hypopharynx. It must be in the trachea, regardless of the value of the return of CO_2.



Right mainstem intubation. A square waveform can occur with a right mainstem intubation because the tube is still in the main airway. Therefore, auscultation in the fifth intercostal space midaxillary, bilaterally, is necessary to rule out right mainstem intubation.

Not only can capnography square waveform confirm correct endotracheal tube placement, but it can also confirm most BIADs (blind insertion airway devices).

Q&A: Can capnography confirm ET tube placement?
YES with SQUARES
Can capnography detect esophageal intubation?
YES no Squares!
Can capnography detect hypopharyngeal intubation?
Yes, no Squares
Can capnography detect right mainstem intubation?
NO, Still must listen to both sides, even with squares

TUBE and CAPNOGRAPHY PEARLS

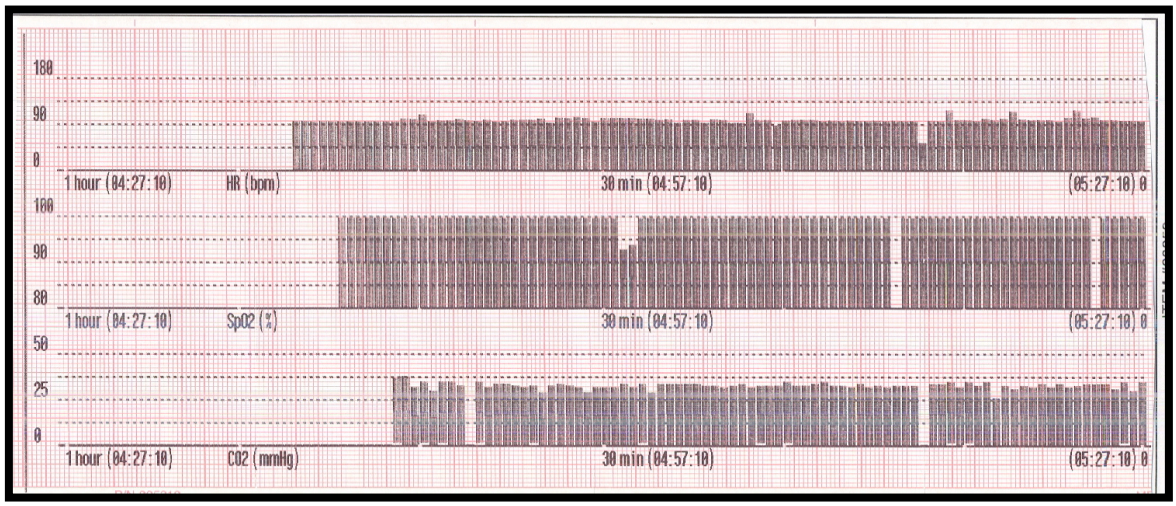
- **Measured ETCO2 alone cannot confirm ET Tube placement, the square waveform must be printed to confirm.**
- **Low perfusion states will produce a smaller "square" but you can still confirm the tube is in the trachea.**
- **In cardiac arrest, CPR must be performed, and ventilation must occur in order for capnography to even show anything on the capnogram.**

- **In some cases of pediatric or infant intubation, it may be necessary for a cuffed tube be used with a small amount of air in the cuff to get a waveform as leaking air around the tube will prevent from coming up the tube to be measured.**

Setup for confirmation. Now this a brand specific task, so check with your own monitor for setup instructions. There ARE some basic premises you need to follow that all devices will need to address.

1. **Make sure the device has capnography waveform set up default on one of the channels. Turn on the machine as you check equipment.**
2. **Make sure the capnography filter line is already plugged in and sampling, while you are preparing the equipment. This will eliminate the delay that can happen with sensor warm-up times and auto zero.**
3. **Place the airway CO_2 sensor on the ET Tube or have it ready for use ON THE FIRST BREATH to check correct placement!**
4. **PRINT the waveform immediately as you confirm. Leave capnography unit attached to patient for tube vigilance, trending, and ventilation check.**

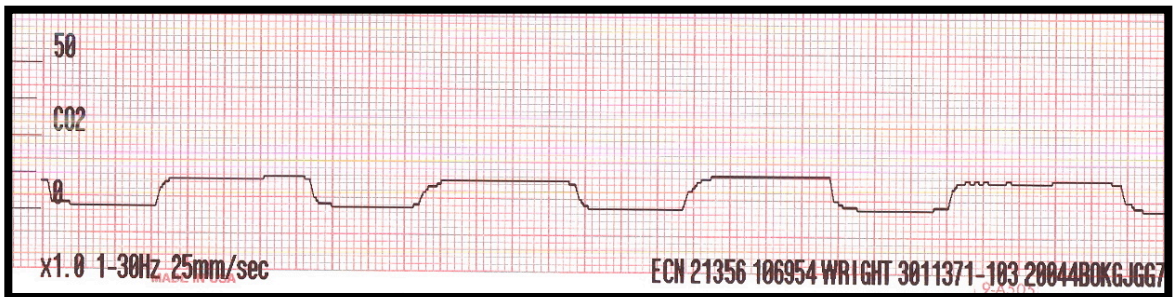
TRENDING after intubation, or the entire call.
One of the huge advantages of these monitors is that most manufacturers offer some sort of trending options for user defined parameters. Below is a trend of $ETCO_2$ Heart Rate and SPO_2 before during and after an RSI procedure. This type of documentation is a great defense tool.



Note that the objective proof shows that the patient did not desaturate during the procedure and that the heart did not increase or decrease (no catecholamine response) and the CO_2 remained stable (adequate ventilation and tube did not come out)

Part II. Adequate Ventilation after intubation

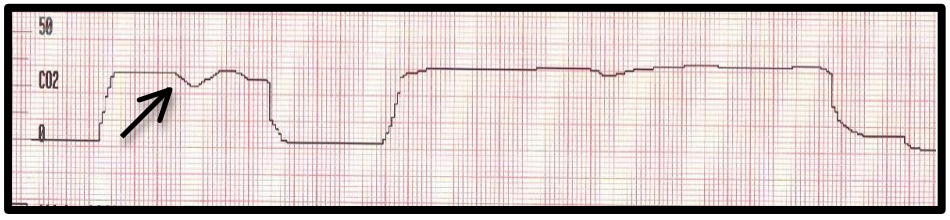
While it is imperative that we conform endotracheal tube placement in the trachea, more important is how the patient is ventilated after intubation. A tube in the trachea/airway eliminates the dead space cause by a facemask and resistance of upper airway structures, but it also make it incredible easy to over ventilate (hyperventilate) the patient. The following capnogram illustrates a patient intubated and confirmed but is being too aggressively ventilated, resulting in an $ETCO_2$ of less than 20.



This is unacceptable and causes harm to the patient. However, how would someone even know how much to squeeze the bag anyway? Subjective "criteria" such as breaths per minute and a certain "quality of chest rise" is NOT reliable. The body measures CO_2. Why don't we all? With an objective, reliable tool such as capnography, it is easy to accomplish this safely and effectively.

Part III. Early warning time for intubated patient waking up.

While RSI may be necessary in some cases to facilitate intubation, it can also create problems for airway management when the paralytic wears off. As a neuromuscular blockage begins to wear off, the diaphragm will be the first to try to move. This cannot be seen by observation but is easily detected by a capnogram. This is called a curare cleft.



Curare cleft

This little indention tells you that you have maybe 3 minutes to sedate the patient before they begin to waken or start to fight the tube.

PART 3: CLINICAL APPLICATIONS

The Non Intubated Patient

Although many systems and protocols mandate the use of Capnography for intubated patient, another area that is commonly overlooked in in the non intubated patient.

This has the potential to be used on nearly every patient encounter as a triage tool. Refer to my website, www.multileadmedics.com to download the actual cases from the capnography page. This also has extensive bibliography of many of the resources used in the preparation of this project. Bob will also show clear cut examples in the Slap the Cap course.

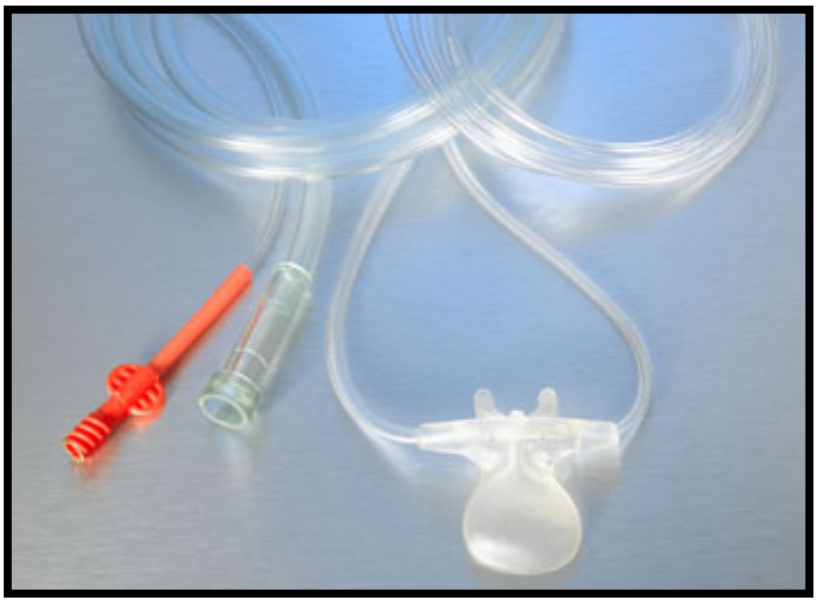
Below however, is a synopsis of clinical uses for capnography in the non intubated patient.

Part 1 How do you do this?

All manufacturers of capnography products make adapters for the intubated and non-intubated patient. The non intubated patients has several options:

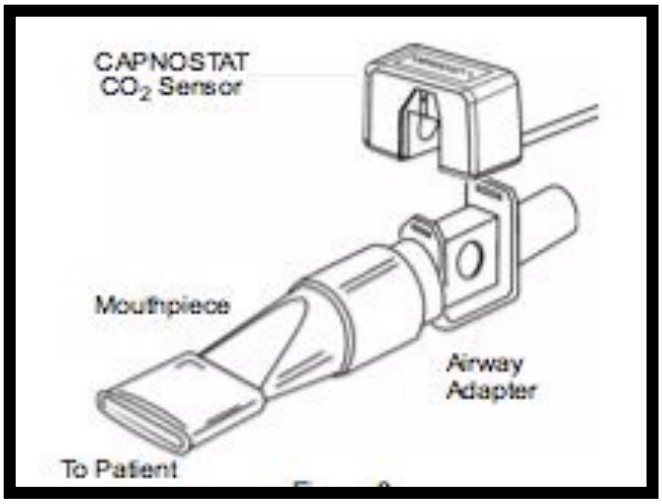
Nasal cannula

Oral/Nasal Cannula: The can also be use to administer O_2 saving extra money spent on separate cannula.



"Puffer" mouth piece over an intubation adapter

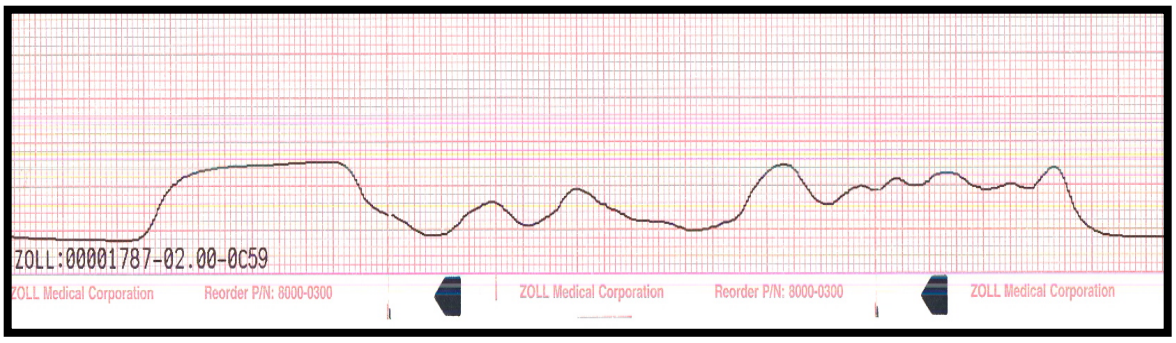
This device works well if you do not have a continuous nasal cannula. You can do a quick triage by having them puff into the mouthpiece and get a spot check, then perform the therapy and check again after therapy. No manufacturer recommends giving updraft treatments directly through capnography filter lines although it can be done.



It has also been demonstrated that the nasal cannula devices can be used under CPAP masks. (see the March 2012 supplement on Capnography for Oridion/JEMS magazine on my website) The readings are reliable, just about 10mm/hg (CO_2 washout) lower than baseline without CPAP but the waveform is not altered.

Oral/Nasal cannulas have also been used successfully under a non rebreather mask as well.

Depending on the manufacturer, the device plugs into the main machine and the reading is set up there. Again, check which one you have to find out how to do it. PRACTICE OFTEN. Learn how the device works.



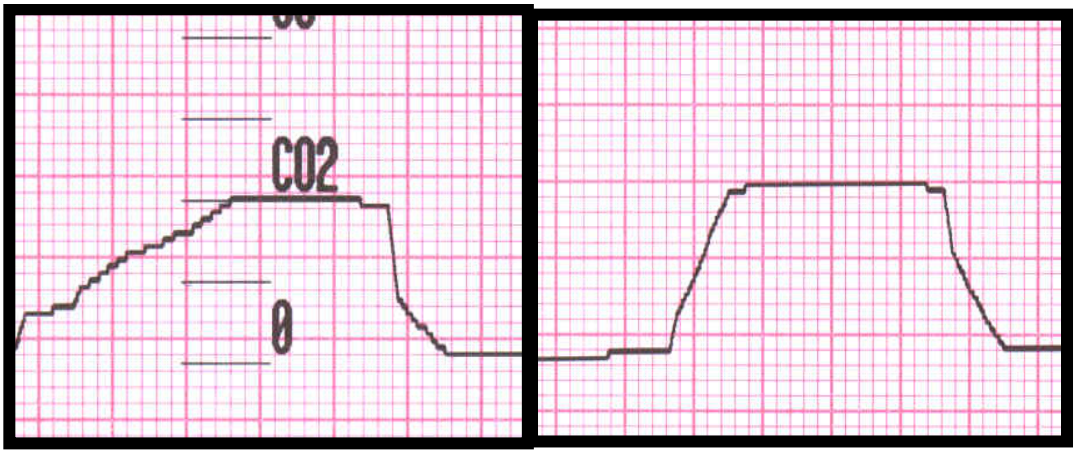
Patient Talking Artifact!

Part 2: Some Specific Uses

RESPIRATORY

Asthma: Capnography can be used a triage tool for asthma attacks. It can assess the severity and can give an accurate measure of Arterial blood gas thus helping the caregiver to triage the severity of the attack. It can also be used to trend and document therapy success or not.

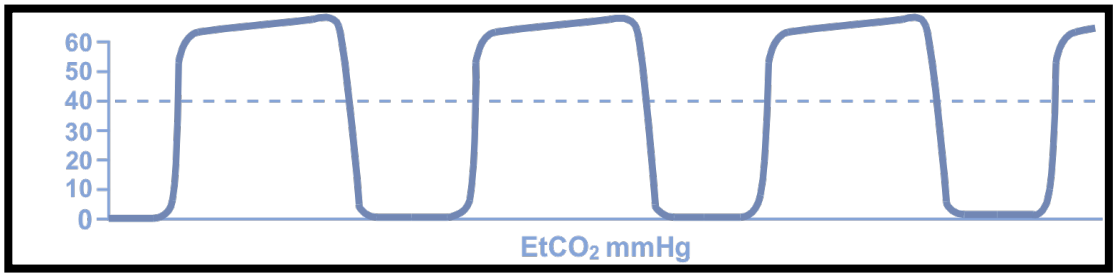
Same patient before albuterol (left) and after albuterol (right) This is objective proof this was a bronchospasm because the drug corrected the bronchospasm. This response came within three minutes of therapy time.



A patient with a square waveform does not have a bronchospasm, so the benefit of a bronchodilator must be carefully examined and weighed against the risk of tachycardia.

COPD: capnography can be used to determine if bronchodilator therapy is necessary, and also can assess and trend possible CO_2 retainers.

CHF with Pulmonary Edema: This is perhaps the most difficult diagnosis to make anywhere (CHF versus COPD) It has been reported that as much as 50% of patients with CHF were given unnecessary bronchodilator therapy. The capnography can triage the severity an also prevent this mistake:



This capnogram shows a high $ETCO_2$ (ventilator failure), but there is no objective evidence of bronchospasm, avoiding unnecessary and possibly harmful therapy.

Can we really use this device to triage a respiratory patient? Does the blood gas CO_2 correspond with the $ETCO_2$? Below is an abstract from a 2005 article that was published in the Annuals of Emergency Medicine. There are others, see my website and the articles, or Google this article and it will lead you to others.

Concordance between capnography and arterial blood gas measurements of carbon dioxide in acute asthma.

Corbo J, Bijur P, Lahn M, Gallagher EJ. Ann Emerg Med. 2005 Oct;46(4):323-7

In this study, 39 Patients, 37 Classified as "Severe Asthma," received simultaneous measurements of arterial carbon dioxide and end-tidal carbon dioxide. The mean difference between Pa02 and PetCO2 was 1.0 mm Hg. The median Difference was 0 mm Hg. Only 2 patients were outside the 5 mg HG agreement (1-6, 1-12).

"In patients with acute, severe asthma exacerbations, we conclude that concordance between PetCo2 obtained by capnography and PaCo2 measured by arterial gas is high."

Bottom Line for EMS: End tidal CO2 gives a good indication of the arterial blood gas level and can help a paramedic evaluate the severity of an asthmatic's condition.

So as you can see, for the respiratory patient, the evidence to date supports the value of capnography as a triage tool. Furthermore, the waveform is the only objective reliable tool for assessing bronchospasm.

Not only can capnography be used to triage, it can also be used to trend therapy effectiveness and monitor for relapses.

- **Sedation: Anybody sedated for any reason must be monitored for respiratory depression. Download the standards (from my website) from all organizations and see where your profession falls in place on the use of capnography Drugs such as recreational ones, legal ones, narcotics, benzo's, depressants can all cause respiratory depression. Recent and well-done studies have proven the benefits of capnography in enhanced patient safety: How does capnography show respiratory depression? The $_{ETCO2}$ will trend up with a normal baseline indicating hypoventilation, and hypercapnia.**

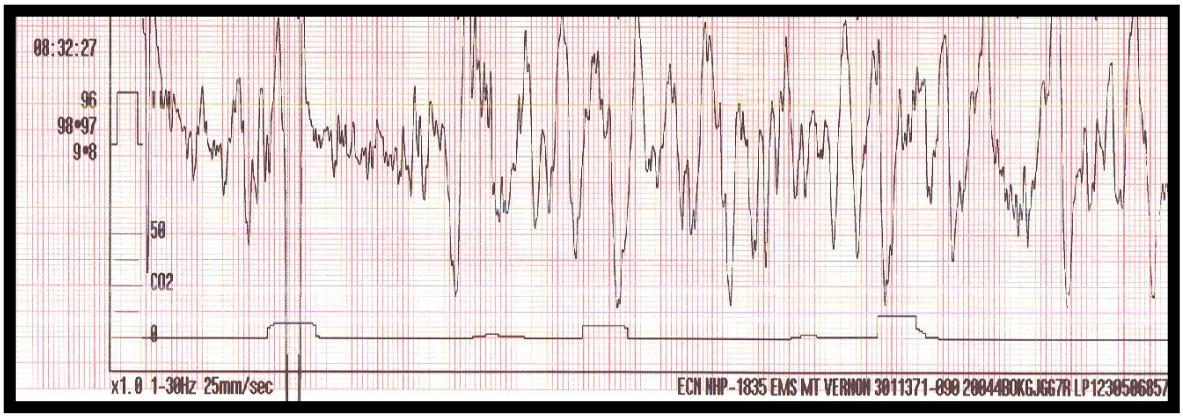
[Ann Emerg Med. 2010;55:265-267.]

In this issue, Deitch et al[1] report a randomized controlled trial of emergency department (ED) procedural sedation and analgesia with and without continuous capnography. They found a significant decrease in hypoxemia when this ventilatory monitoring modality was added, thus providing the first objective evidence in our setting that this technology can enhance sedation safety. This important finding will spur many

What is this? A PCA Pump with Capnography override

- Supports current ASA and Joint Commission standards mandating CO_2 monitoring for all anesthesized patients (intubated and non-intubated)
- Provides an additional safety net at the bedside to continuously monitor patient respiratory response to infusion therapy

Seizures: When someone has a Generalized Seizure, such as a tonic/ clonic, it affects both hemispheres of the brain and the medulla. When the medulla is involved, the patient does not breath during seizure activity. This is easily determined by capnography during the seizure, (see below capnogram or refer to in class cases I present.



The capnogram shows small ineffective breaths (small waveforms) this is noisemaking (involuntary) as thoracic muscle contract during the seizure. This is proof that the seizure is on going. When the patient goes post-ictal, the breathing will resume. If it does not, the patient needs to be ventilated to normal $ETCO_2$ and anti-seizure meds need to be given.

Any generalized seizure will cause apnea during the seizure, though most are short in duration (2-10 seconds), not breathing is usually not noticed.

Metabolic Uses: DKA

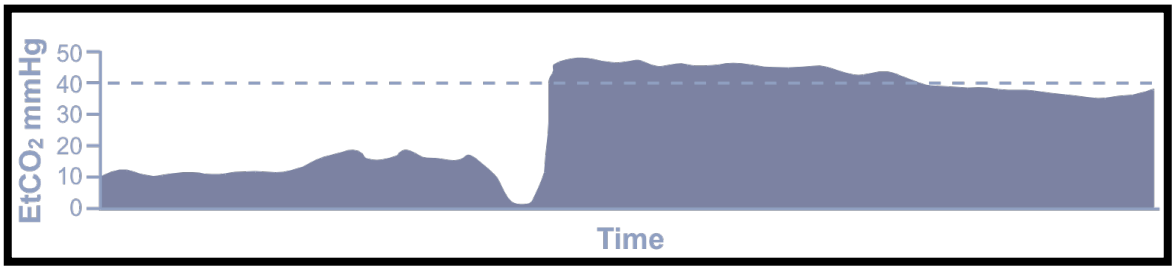
Since CO_2 is carried in the blood stream and bicarbonate Ion, it has a direct clinical relationship to serum bicarb levels. Therefore, if the patient has a high Blood glucose, measure their $ETCO_2$. If it is less than 29, then the patient has DKA. The blood gas bicarb will show a very low level as well, indication metabolic acidosis.

Pulmonary Embolism:

This is easy. The combination of $ETCO_2$ and an ABG CO_2 can easily call a V/Q mismatch. All you need then is a CT scan to figure out where it is and they are on their way. A high blood gas CO_2 and a low $ETCO_2$ tells us the CO_2 is not getting to the lungs to be exhaled.

Perfusion?

The 2010 AHA criteria recommend capnography for monitoring the effectiveness of CPR, and for detecting the presence of ROSC (return of spontaneous circulation. Here is a trend that shows ROSC after 4 min of CPR and a defibrillation.



ROSC will show a sharp increase in CO_2 levels. When this happens, stop pushing and start squeezing the BVM to return the CO_2 to normal. Most cases that have ROSC has $ETCO_2$ that go into the 70-90's!

Trauma?

In Tension Pneumothorax, pressure in the chest collapses a lung and then presses on the right side of the heart making it hard to fill with blood. It only takes about 7mm/hg pressure to stop the blood flow into the right atria. The first and must reliable sign of a TENSION pneumothorax is the sudden drop in perfusion that is picked up immediately on a capnogram. By the same token, when the chest is successfully decompressed, it is not a rush of air but a sudden increase in $ETCO_2$ that confirms decompression success. Furthermore, the capnogram can be used to keep watch in case it develops again.

The same is true for Pericardial Tamponade and cardiocentesis. In each of these obstructive forms of hypoperfusion, the capnogram will remain square because it is a perfusion problem, not an airway problem, but you knew that, right?

Closed Head Injury. ITLS and the Brain Trauma Foundation have taken the lead in recommending capnography as the way titrate CO_2 ventilations in the patient with a closed head injury. If the patient has a GCS of less than 9 and they are posturing, have unequal pupils, or dropped two in front of you, then they should be selectively ventilated to an $ETCO_2$ between 30-35mm/hg. If the patient does not the signs (above) of deterioration, then ventilate the patient to levels, 35-45. Never ever bag them to lower than 25mm/hg. It causes cerebral vasoconstriction and creates an alkalosis not allowing O_2 to dissociate from hemoglobin, make the brain injury worse.

In Conclusion:

There are many more cases to be seen, through my seminars and publications. I hope you enjoyed the seminar! I really get into it because I believe this will help you do your job better and since we are health care, it is our patients that will benefit from this in the end.

It is easy to say no to a new idea, Throughout 2011 I surveyed 3800 of students in my capnography class. About 30% had had a formal capnography course before, and of those, the average time in class was 1 hour. There is no way to gain adequate knowledge and experience doing this. Since most systems require capnography for one purpose, tube confirmation, so that is all they ever pursue knowledge on. Ignorance (the lack of knowledge thereof) is the biggest factor in the slow adoption of this tool. And this lack of understanding and knowledge goes to all levels of Emergency services.

It is my hope and prayer that this class has touched you personally and sparked a fire that will help carry this idea to all levels of medicine. It takes great courage to take a new idea and run with it. Please let me know if there is anything I can do to help you.

For more information on your instructor or for information on these or other classes I present, or to get to your area for a class, visit the web site at

www.multileadmedics.com

or e-mail me at lead2noclue@mac.com

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