

- 1 Feel free to interrupt me for clarification, but I will probably postpone any discussion and especially disagreement for the completion of my presentation. I will do my best to make sure that there is not enough time at the end for disagreement.
- 2 ACEP Statement: The college leads off their expired CO₂ monitoring policy statement with "The American College of Emergency Physicians believes that quality emergency care should be available to all who seek it." The statements following this meaningful pronouncement are designed to be as vague as possible, but the tone seems to indicate that CO₂ monitoring, at least in verifying ETT placement, is good.
- 3 Controversy: In 1994, The Annals published a piece written by a lawyer who asserted that based on new ASA Guidelines, capnometry is now the standard of care for ETT placement confirmation, and the article was published alongside a full-page advertisement for the Nellcor colorimetric PetCO₂ detector. Mayhem ensued, prompting the Annals' editor to investigate how Nellcor knew about the article prepublication; it was discovered that the author was on Nellcor's bankroll.
 - 3.1 Continuous capnography is available in all three adult McGill hospital emergency departments, but no one I have spoken to has seen it used.
- 4 Carbon dioxide is an odorless, colorless gas whose concentration in air is .03% – effectively zero.
 - 4.1 CO₂ is continuously produced by cell metabolism and is transported in the blood in three forms: 65% as bicarbonate following conversion in the RBC by carbonic anhydrase, 25% bound to blood proteins (hemoglobin, for example), and 10% in plasma solution. The PaCO₂ reflects this last form of CO₂.
 - 4.2 CO₂ normally diffuses across its concentration gradient at the capillary-alveolar interface in less than half a second; the PaCO₂ therefore serves first and foremost as a measure of ventilation.
 - 4.3 It is useful to think of each alveolus as existing on the continuum from dead space (very high ventilation to perfusion = very low alveolar CO₂ content) to shunt (very low ventilation to perfusion = high alveolar CO₂ content).
- 5 Capnometry is measuring CO₂ during the respiratory cycle, capnography is the graphic display of this measurement over time.
- 6 Note that the pulse oximeter graph is *not* a display of SaO₂ over time, it is a display of the strength of the signal.
 - 6.1 In trend mode, the speed of the capnograph tracing is slowed, compressing the parts together and allowing the reader to see trends over time.
- 7 The CO₂ concentration at the end of expiration – a single moment in the respiratory cycle – is called the end-tidal CO₂ [PetCO₂]. This is a particularly relevant value because it most closely approximates PaCO₂ and is the numeric value displayed by the capnometer.
- 8 PetCO₂ vs. ScvO₂: Note that measuring CO₂ concentrations in either expired air or in the blood – the topic of this presentation – is completely different than measuring central venous oxygen saturation. The confusion stems from the use of ScvO₂ in EGDT for sepsis, and a new device, the conveniently-named Edwards catheter, manufactured by Edwards Lifesciences, which measures this parameter in realtime and produces a graphical display.
- 9 CO₂ concentrations can be measured by several mechanisms, but most sensors applicable to the ED use an infrared mechanism, similar to the pulse oximeter. CO₂ absorbs light at a particular wavelength, and when such a light is passed through a chamber, the degree to which the light is absorbed is compared to a reference to determine the CO₂ concentration.
- 10 ETCO₂ can be measured by one of two sampling techniques: mainstream and sidestream.
 - 10.1 Mainstream monitors straddle the airway; the measuring cell is placed in between the ETT or breathing mask and the respiratory circuit. They require a heating element to deal with condensation, are much bulkier, and are generally reserved for intubated patients.
 - 10.2 Sidestream monitors draw a continuous sample of the gas from the circuit into the measuring cell; they comprise the majority of capnograph mechanisms available for ED use.
- 11 There are a number of ways to monitor PetCO₂ on a spontaneously breathing patient with a sidestream capnograph.
- 12 These two pictures show how to rig a nasal cannula into a sidestream capnometer.
 - 12.1 Because the sample has to be brought to the sensor, there is a lag time, usually 2–3 seconds. This takes a bit of getting used to, as we are accustomed to the realtime ECG tracing.
- 13 The capnograph has four phases.
 - 13.1 Expiration starts at point A.
 - 13.2 AB represents exhalation of CO₂-free gas in dead space. (Phase I)
 - 13.3 BC represents exhalation of mixed dead space and alveolar gas. (Phase II)
 - 13.4 CD is the alveolar plateau, which represents exhalation of mostly alveolar gas. Constant or slowly uprising. (Phase III)
 - 13.5 D is the PetCO₂, and is the number displayed by the machine.
 - 13.6 DE represents the inhalation of CO₂-free gas. (Phase IV) The value at the end of inspiration represents the [CO₂] of the inspire.

- 14 We're going to go over some CO₂ physiology, but it's not necessary to review your med school textbooks to use capnography in the ED. You'll see when we get to the ED applications that most of them require very little background to apply to patients.
 - 14.1 Normally the PetCO₂ value is 38 mmHg, 3–5 torr below PaCO₂.
 - 14.2 The PetCO₂ is dependent on three variables: CO₂ production, delivery of blood to the lungs, and alveolar ventilation. That three crucial physiologic variables determine PetCO₂ is both its strength and weakness.
- 15 Increased PetCO₂
 - 15.1 Decreased CO₂ clearance = decreased ventilation (decreased central drive, muscle weakness/paralysis, diffusion problems/resp distress)
 - 15.2 Increased CO₂ production (e.g. fever, burns, hyperthyroidism, seizure, bicarbonate therapy, transient return of spontaneous circulation during cardiac arrest, release of tourniquets or reperfusion of ischemic areas)
- 16 Decreased PetCO₂
 - 16.1 Increased CO₂ clearance = increased ventilation, decreased CO₂ production (hypothermia, sedation, paralysis)
 - 16.2 Decreased delivery to the lungs (e.g. decreased cardiac output).
 - 16.3 V:Q mismatch: ventilating nonperfused lung units results in the expiration of CO₂-poor air, lowering PetCO₂ in, for example, pulmonary embolism.
- 17 In this simplified diagram, ventilating nonperfused lung lowers PetCO₂ while PaCO₂ remains stable.
- 18 V:Q mismatch: perfusing nonventilated lung, e.g. atelectasis. Looking at the difference between arterial PCO₂ and PetCO₂, you can optimize PEEP. This often won't elevate the PetCO₂, but will elevate the PaCO₂ and therefore widen the PetCO₂:PaCO₂ gap.
- 19 The difference between alveolar and arterial CO₂ is the A-a gradient for CO₂ or a-ADCO₂.
 - 19.1 It's normally between 3–5 mmHg, but can be widened in various disease states.
- 20 I'm going to quickly present some general things about the capnograph.
 - 20.1 If no CO₂, two causes must be rapidly excluded: No ventilation and no circulation. Mechanical failure is entertained when these two problems have been assessed.
 - 20.2 Low PetCO₂
 - 20.2.1 Failure to ventilate the lungs.
- 21 This often corresponds to a sudden decrease of PetCO₂ to zero or near zero with no waveform.
 - 21.1 Esophageal intubation
 - 21.2 Inadvertent tracheal extubation
 - 21.3 Apnea
 - 21.4 Breathing circuit obstruction (ETT, usually)
 - 21.5 Breathing circuit disconnection
- 22 Circulatory collapse / cardiac arrest (5 H's 5 T's).
- 23 This often corresponds to an exponential decrease in PetCO₂ with a discernible waveform.
 - 23.1 Most often this is due to a major cardiopulmonary event that generates a sudden increase in dead space, resulting in a widening of the alveolar-arterial CO₂ gradient (a-A DCO₂). Cardiac arrest does not drop the PetCO₂ to zero immediately, because the remaining CO₂ in the lungs must be washed out.
 - 23.2 As cardiac output falls, PetCO₂ drops and may level off at a new equilibrium (assuming minute ventilation is constant).
- 24 Decreased slope of phase II: slow expiration.
- 25 Kinking of the ETT. Note the loss of the alveolar plateau.
- 26 Obstructive lung disease. Note the marked change in the shape of the curve. This graph demonstrates not only a decreased slope of phase II, but, more impressively, an increased slope of phase III.
 - 26.1 In a patient with obstructed pulmonary disease, the alveolar plateau develops a slope. This is because gas exchange units that are less obstructed have higher V:Q ratios and empty first during exhalation, so that expired CO₂ is lower at the beginning of expiration than at the end.
 - 26.2 Another cause of upsloping phase III is that CO₂ moves between the pulmonary capillaries and alveoli at a constant rate, and ventilation decreases at the end of a breath, so CO₂ levels slowly rise over expiration even in normal lungs. This can be demonstrated with a forced expiration capnograph.
- 27 A dip in the expiratory plateau is called a curare cleft, it represents the patient breathing over the ventilator. This is the result of either inadequate anesthesia (paralysis or sedation), or inadequate oxygenation/ventilation.
- 28 Cardiogenic oscillations result from the heart beating against the lung, usually in a bradypneic patient. The inspiratory downstroke should be almost vertical; this graph depicts an endotracheal cuff leak.
- 29 Okay, now onto the ED applications.
 - 29.1 The use of capnometry in the confirmation of ETT placement is currently considered the standard of care for ED intubations. Everyone should be using capnometry of some sort. Colorimetric capnometers use chemically-impregnated pH-sensitive paper that changes color when exposed to CO₂. It has been engineered to avoid the red-green spectrum because 8% of the population is red-green colorblind. A purple color corresponds to 0–4 mm Hg, tan 4–15, yellow >15. These devices are considered reliable for approximately two hours. Adult devices are not suitable for kids under 15 kg.

- 29.2 If mouth-to-mouth ventilation occurred before intubation, or bag-valve-mask ventilation occurred before intubation, or the patient consumed carbonated beverages prior to intubation, a false-positive PetCO₂ may occur in the event of esophageal intubation. However, the PetCO₂ in this circumstance should decrease to zero within six breaths, as very little CO₂ is present. Since most of our ED intubations will fall into at least one of these three groups, it makes sense to routinely verify yellow color for six breaths if using a colorimetric device.
- 30 The presence of vinegar in the stomach may also cause an irreversibly yellow coloring of the device.
- 31 A rare confounder to PetCO₂ confirmation of tube placement occurs in the setting of a supraglottic ETT tip in a spontaneously breathing patient, which would produce lasting colorimetric changes and a potentially normal waveform. There have been several published cases of this, all in the context of nasal intubations. Anecdotal evidence also suggests that an oral ETT can migrate above the glottis during patient transfer or movement to create a similar false-positive reading.
- 31.1 In states of very low pulmonary perfusion (cardiac arrest), PetCO₂ may not be detected if adequate chest compressions are not taking place. Even with adequate chest compressions, there are a number of reasons that capnometry will fail with false-negative results, and the accuracy of capnometry is probably overestimated in studies, especially in the setting of cardiac arrest, with an estimate of one in ten misreads (false positive or false negative) in a metaanalysis.
- 31.2 Therefore, capnometry alone should not be considered a gold standard for ETT placement confirmation. Although it is the best single test, given the danger of a false negative and especially a false positive, multiple tests must be used simultaneously. There are many other options.
- 31.3 The bottom line for EPs is twofold: First, do not rely solely on capnometry for confirmation of tube placement, especially in cardiac arrest or prearrest patients. Secondly, if using a colorimetric device, get in the habit of observing a proper cycle of yellow-purple-yellow for six breaths postintubation.
- 32 Continuous capnography provides a reliable respiratory rate, and a reliable trend of the respiratory rate. I find RR to be the crucial vital sign, the crucially miscalculated and ignored vital sign.
- 32.1 An early study asserts that physicians don't care about RR, so nurses have stopped caring. The authors support this with an impressive array of experiments and chart reviews that demonstrate the discordance of recorded respiratory rates and "carefully measured" respiratory rates. In an audit of records from a Nashville VA hospital, they found that of 13,000 recorded respiratory rates, 94% were between 18 and 22 breaths/min. Note that the normal RR is 14-18. The authors thus decide that the RR should be discarded as a vital sign, and estimate that each year 3,500,000 hours of labor are wasted measuring respiratory rates, and, assuming an average salary of \$1.50 per hour, cost taxpayers over five and a half million dollars. They conclude that the elimination of the RR as a "Pan-hospital" routine would improve medical practice and patient care.
- 33 In 1980, the respiratory rate was resuscitated as a vital sign by this paper, which showed that "Respiratory rates, if carefully measured, are a sensitive and reasonably specific marker of acute respiratory dysfunction."
- 34 Though we now accept RR as an important physiologic parameter, contemporary studies show that it is still measured inaccurately. "We found that independent measures of respiratory rate may differ by more than 35%, which implies that a measured respiratory rate of 16 breaths/ min may actually represent a rate anywhere between 10 and 22 breaths/min."
- 35 Last year Dr. Lovett called the RR "The Vexatious Vital."
- 36 And my favorite, this paper published in the Annals. We see here a screenshot showing data from patients in the waiting room. "The figure displays an under-recognized clinical phenomenon for which we are proposing the term 'synypnea.' Synypnea is seen across the country and is defined as when all emergency department waiting room patients have the same respiratory rate. We think it is pathophysiologically linked to menstrual synchrony. There is little scientific exploration on this topic, however, which represents fertile grounds for original research."
- 37 Imagine a world with a reliable RR trend. I believe that experienced docs use the RR, perhaps subconsciously, as key measure of wellness, much more than the BP and HR. We don't talk about it much because it's never accurately measured, because no one wants to stand there for 60 seconds or even 30 seconds to count a RR, but I believe that having access to a reliable RR trend would greatly facilitate evaluation and reassessment by emergency physicians.
- 37.1 Modern continuous ECG monitors have been modified to detect RR by changes in transthoracic impedance, but this method is very sensitive to patient movements and is only reliable in paralyzed or sedated patients.
- 38 The pyroelectric polymer (PEP) device can be incorporated into an oxygen mask.
- 39 The precordial stethoscope offers a continuous auscultation of the heart and lungs. Low-tech genius.
- 40 There has been a lot of interest recently in a device that attaches to the earlobe and measures both oxygen saturation and CO₂ levels transcutaneously.
- 41 The use of PetCO₂ in CPR is well-studied. Recall that the major determinants of PetCO₂ are CO₂ production, alveolar ventilation, and pulmonary perfusion. Therefore if ventilation is held constant, and CO₂ production is assumed to be fairly constant, the PetCO₂ correlates linearly with pulmonary blood flow (cardiac output).
- 42 Numerous studies have confirmed that at low flow, the major determinant of PetCO₂ is cardiac output, and at physiologic cardiac output levels, the major determinant of PetCO₂ is ventilation.

- 43 In CPR, if there is an abrupt increase in cardiac output, as occurs with ROSC, ventilation becomes the major determinant after tissue CO₂ washout occurs.
- 44 Since our goal in the provision of CPR is to increase coronary perfusion pressure and cerebral perfusion pressure, both of which are primarily driven by cardiac output, PetCO₂ reflects the adequacy of CPR.
 - 44.1 The absence of CO₂ during CPR is most often due to either a failure to intubate the trachea or inadequate compressions. Long downtimes do *not* result in the absence of CO₂ on the initiation of CPR because although cellular metabolic processes may have ceased, much CO₂ remains in the lungs and tissues until it diffuses out of the body, a process that takes at least hours. It is not known how long it takes to washout CO₂ from an ametabolic corpse during CPR, and it may be that a twenty minute ambulance ride is sufficient, but if you don't get a PetCO₂ on the initiation of CPR, exclude the other two causes first. To restate this point: if a patient suffers a cardiac arrest in the ED and the initial PetCO₂ reading is zero, this is certainly the result of either esophageal intubation or inadequate compressions. If a patient died at midnight in her bed, and CPR is begun at eight in the morning and continues for 45 minutes en route to the hospital, it is possible that PetCO₂ levels are in fact zero, as all CO₂ has been removed from the lungs and no CO₂ is being generated to replace it, but this is a diagnosis of exclusion.
 - 44.2 Obstructive causes of cardiac arrest, such as tension pneumothorax, cardiac tamponade, and massive PE have the potential to cause zero or near-zero PetCO₂ readings as they cause a complete interruption of cardiac output.
- 45 If PetCO₂ monitoring is performed, chest compressions do not need to be halted during resuscitation to palpate a pulse. This takes on new significance with the recent ACLS guideline revisions, which stipulate that CPR should resume immediately after defibrillation. ROSC occurs in this diagram at point A and pulses become palpable at point B.
- 46 PetCO₂ can differentiate between PEA and pseudo-PEA, which probably accounts for the majority of our PEA ROSCs, especially in cold or overweight people, where pulse palpation carries a high false-negative rate. Ultrasound is probably a more useful modality in this application, however. In this graphic, the patient has no palpable pulses but a persistent PetCO₂ of 20, without CPR (stopped at point A). CPR is restarted at point B, which doesn't do much. At point C, dopamine is infused. At point D, a pulse is palpated and cardiac compressions are halted.
- 47 Boluses of bicarbonate, which is a CO₂-generating buffer, will result in a significant but transient rise in PetCO₂. This occurs within one minute of administration and lasts less than two minutes. High dose epi, which increases coronary perfusion pressure at the expense of cardiac output, decreases PetCO₂ by increasing afterload. We don't use high dose epi anymore. 1 mg boluses of epinephrine have variable effects on PetCO₂ – however, increases in PetCO₂ associated with an epinephrine bolus may suggest efficacy of the treatment, and a subsequent fall in PetCO₂ may suggest timing of additional boluses.
- 48 The optimum pressure applied to the chest, as well as CPR provider fatigue, can be assessed using PetCO₂. Declining efficacy of chest compressor #1 at point A, with a new chest compressor at point B.
- 49 Use of PetCO₂ to guide the termination of efforts.
- 50 This has received a fair amount of attention in the EM ultrasound literature and a related study in the Aug 3 issue of the New England Journal of Medicine. In the early part of the 20th century, Rudolph Eisenmenger performed animal experiments with a mechanical CPR device called The Biomotor, and analyzed expiratory CO₂ using a device called The Carboskop. He wrote, in 1939, "If during a resuscitation attempt the analysis of the expired air, performed about twice per hour, still shows plenty of carbon dioxide, then continuation of artificial respiration [and circulation] would be indicated. If, however, a sharply declining carbon dioxide elimination in expired air is found, or even more a cessation of [carbon dioxide elimination], then further attempts are futile."
- 51 The most often cited study on this topic demonstrated that a PetCO₂ of less than or equal to 10 after twenty minutes of resuscitation predicted an inability to bring about ROSC with 100% sensitivity. Other studies have shown 90% sensitivity for no ROSC if the initial PetCO₂ is <15 mm Hg. Of interest, in the Levine study, although PetCO₂ ≤10 was 100% sensitive at predicting nonsurvivors, so was the absence of a pulse at 20 minutes.
- 52 Summary: If, during the course of cardiac arrest, PetCO₂ is less than ten, resuscitation parameters must be modified by either increasing the rate/depth of compressions or optimizing pharmacologic therapy, or performing additional interventions directed at reversible causes of cardiac arrest. If PetCO₂ remains below ten despite optimization measures, termination of efforts should be strongly considered in a normothermic patient.
- 53 In hemodynamically stable patients without rapidly progressive lung pathology, PetCO₂ is an accurate reflection of PaCO₂; this has been confirmed in ED studies of patients who present with a wide variety of disorders. The gradient between PaCO₂ and PetCO₂ varies among patients and diseases, but that gradient remains stable in any given stable patient. Therefore, if an initial PaCO₂ is obtained and the gradient for a patient is established, PetCO₂ and pulse oximetry can be used to manage ventilator settings without blood gas analysis. In general, again, PetCO₂ is 3–5 mmHg lower than PaCO₂.
- 54 An ED study established the strong correlation between PetCO₂ and PaCO₂ in 39 patients with severe asthma. If using PetCO₂ to predict PaCO₂, note that high PetCO₂ almost always indicates high PaCO₂ whereas low PetCO₂ could be associated with low, normal, or even high PaCO₂. The best approach, if following PaCO₂ is important, is to correlate an initial PetCO₂ reading with a PaCO₂ by blood gas analysis.

- 55 Sudden changes in either ventilator apparatus or patient status will be picked up much more quickly with PetCO₂ than pulse oximetry. This applies to any patient, intubated or not, where ventilation is a concern – we want to know about hypoventilation before it leads to hypoxia, especially if they are receiving supplemental oxygen. The patient who just received systemic analgesia, the head injury or heroine OD who doesn't need require intubation now but is at risk for deterioration, the postictal patient, or the patient who presents with active respiratory disease – all may benefit from continuous capnography as a surrogate for PaCO₂.
- 56 This leads to the use of ETOC₂ as an adjunct in procedural sedation. Clinically monitoring ventilation is hard. An anesthesiologist I spoke with said "No specialist is half as good at predicting ventilation by chest rise as an anesthesiologist, and no anesthesiologist would make decisions based on chest rise." The limitations of the physical exam in the assessment of respiratory status in the sedated or ill patient are well-documented.
- 57 We can use the pulse oximeter to monitor ventilation if we withhold supplemental oxygen, but no one wants to withhold supplemental oxygen. It has been argued to me that the ventilation status is not important as long as there is no hypoxia, but I believe this is simplistic and dangerous thinking. According to a different anesthesiologist, anesthesia is like steering a large boat. Good anesthesiologists sense when the boat is moving off track and can nudge it back in the right direction, whereas lesser anesthesiologists don't notice they're on the wrong course until late, when the situation is much more dangerous. Note that this has nothing to do with hypercapnia, which is of little concern; we would like to address oversedation long before the patient needs to be bagged, which is a dangerous maneuver. Furthermore, your patient can have a respiratory arrest with a great saturation if supplemental oxygen is being delivered, and at that point, the saturation and, shortly thereafter, heart rate will fall quickly and precipitously. What if, in that situation, you can't bag the patient? To quote a comment published in the EM literature from 2005: "...respiratory complications continue to occur commonly during procedural intravenous sedation, despite a veritable industry of regulations and policies designed to prevent such critical incidents. It seems that the practical message missing from all the guidelines and policies is that *breathing is the only thing that counts.*"
- 58 PetCO₂ is often used as the gold standard in studies that look at ventilation, and has been studied in setting of emergency department procedural sedation in both children and adults, and it reliably and not surprisingly picks up hypoventilation/apnea faster than pulse oximetry. It is particularly useful in the post-procedure period, which is notoriously dangerous as stimulation ceases and staff walk away.
- 59 I assert that the use of chest rise as a sole marker of ventilation is inadequate, and doubly inadequate if the person responsible for ABCs is also the person doing the procedure. In either case my feeling is that another mode of ventilatory monitoring is required and that can be pulse oximetry if the patient is breathing room air, capnography, or one of the novel devices I spoke of earlier. Capnography offers the advantage over pulse oximetry of characterizing the quality of respirations.
- 60 Bronchospasm increases the slope of the expiratory plateau, because alveoli supplied with oxygen by normal low-resistance airways are relatively hyperventilated and have lower CO₂ concentrations than lung units that are less ventilated the result of obstruction. The less obstructed units empty first, so CO₂ concentration is lower at the beginning of expiration than the end. The slope can of course be measured and is represented by the term dCO₂/dt, the rise over the run. The slope has been proven to correlate to FEV₁, but is independent of effort, age, sex, and height. In a study of asthma patients and normal controls, the slope of the alveolar plateau was predictive of disease and well-correlated with peak flows. It's easy to imagine one day making decisions based on the evolution of the slope of the expiratory plateau.
- 61 PE obstructs blood flow to alveoli, producing lung units with very high ratios of ventilation to perfusion. Stated differently, PE produces dead space, and PetCO₂ is great for measuring dead space.
 - 61.1 The use of PetCO₂ is much more accurate in the diagnosis of PE than the A-a gradient because the things that widen the A-a gradient are in the ddx for PE, whereas very few things other than PE lower PetCO₂ in the nontachypneic, hemodynamically stable patient.
 - 61.2 There are a number of ways that EtCO₂ has been used in the assessment of PE.
- 62 An alveolar dead space fraction (V_d/V_t) >.4 has a sensitivity and specificity of 100% and 94%, respectively, in the diagnosis of PE. This is calculated by taking a blood gas and plugging it into an equation. This value is normally between .2 and .3. Top patient has pneumonia, bottom patient has PE.
- 63 Jeff Kline at Carolinas Medical Center, who has a stranglehold on emergency department PE research, demonstrated that simply calculating the area under the capnogram produced a sensitivity for PE of 100% at a cutoff of ≥50 mmHg-seconds, and a +LR of 12.3 for PE at a cutoff of ≤25.
- 64 Most recently, Dr. Kline investigated a new parameter, the expired pCO₂:pO₂ ratio, and found that he could stratify results into segments that, when taken in conjunction with clinical pretest probability, produce post-test probabilities of >90% and <2%.
 - 64.1 I had never heard of any of this, and I asked Dr. Kline why breath measurements have received so little attention. His response: "Reasons for slow adoption include limited data, lack of infrastructure to use the technique, and poor reimbursement. More generally, this simply reflects the fact that medicine is the slowest adopting sector (known to Wall Street). Every VC knows this. We are just slow. There is a famous book on the subject called the Diffusion of Innovation that will help anyone understand why we are slow. In contrast to software (for comparison) Physicians are way more skeptical of early innovation, because the consequences are life and death, not a computer program that does not work correctly. Physicians demonstrate a very

lemming way of adoption, and 95% like to be in the middle of the crowd with only 2.5% being very rapid or very slow to adopt."

- 65 Lastly: cardiac output. The Fick equation determines cardiac output by measuring the rate that CO₂ is cleared from the blood, and requires sampling of arterial and mixed venous blood. A permutation of the Fick equation can be applied and CO measured by forcing the patient to rebreathe CO₂, which can be done by adding dead space to the circuit. The process can of course be automated to cycle every few minutes and the resulting cardiac output value is highly accurate. This may seem more applicable to intensive care than emergency care, but it's very straightforward to calculate CO with PetCO₂, it's mostly just getting the right software on board. if we started using more PetCO₂, perhaps we would be talk a lot more about cardiac output.
- 66 In conclusion, I hope that you feel more familiar with carbon dioxide physiology, the capnograph, and the potential uses of capnography in the emergency department.
- 67
- 68 Reuben Strayer
- 69 McGill Emergency Medicine
- 70 August 2006
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