Script for the educational video entitled "Identifying and achieving mechanical ventilation targets"

Slide 1

This educational video, entitled "Identifying and achieving mechanical ventilation targets", was composed by Bob Demers.

Slide 2

This module is a so-called "Interactive video". You will need to view it on a system which provides both audio and video. At various points during the presentation, a chime will sound. At that point, viewers are required to highlight the words or phrases in their handout which correspond to the words/phrases which display in yellow text in the video images. Before we proceed with the video, viewers should ensure that they have a hard copy of the handout, and a highlighter, in hand. If either of these is not available, pause the video at this time in order to secure them. In addition, at various points during the screening of the video, viewers will be directed to interrupt it, in order to undertake specific psychomotor tasks. The signal for pausing will coincide with this sound effect [auto skid sound effect]. If viewing the video by means of a computer, the video can be paused simply by hitting the spacebar on the computer keyboard. Resumption of play will be enabled by hitting the spacebar a second time. If using an alternative category of video player, the viewer should use the pause/resume control which is integral to that player for interruption and resumption of play.

Slide 3

At the conclusion of this module, viewers should be able to:

1) recite the Henderson Equation from memory;

2) stipulate the ratio between arterial carbon dioxide tension (p_aCO_2) and bicarbonate ion concentration ($[HCO_3^-]$) which is compatible with homeostatic values for arterial hydrogen ion concentration ($[H^+]$) and arterial pH (pH_a);

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3) describe a therapeutic strategy which we might employ in order to determine a target p_aCO_2 which will elicit a homeostatic $[H^+]$, as well as its' correlate, pH_a;

4) apply that strategy to a patient who presents with an episode of diabetic ketoacidosis;

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5) implement the strategy with a patient with pre-existing chronic obstructive pulmonary disease who presents with an episode of acute-on-chronic hypercapnia;

6) apply the strategy to a patient with a history of congestive heart failure who presents with acute hypercapnia secondary to an overdose of barbiturates;

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7) implement the strategy with a neonatal patient who presents with an episode of pneumonia complicating bronchopulmonary dysplasia; and

8) describe a method which could be used to decrease p_aCO_2 by fifty percent, given a value for tidal volume and respiratory rate.

Slide 7

I will begin this presentation by describing an actual scenario which took place years ago at a university hospital on the East Coast. A patient was transported to the Emergency Department late one night, and his clinical condition prompted the House Officer on duty to order an Arterial Blood Gas (ABG) analysis. Within minutes, the arterial carbon dioxide (p_aCO_2) reading was revealed to be 67 torr. The House Officer fixated on this admittedly elevated figure, and decided to intubate and mechanically ventilate the patient immediately, choosing a p_aCO_2 value of 40 torr as the subsequent target. About eleven hours later, the attending physician in the Intensive Care Unit to which the patient had been subsequently admitted (who also happened to be the Director of Pulmonary and Critical Care Medicine) was conducting morning rounds. When supplied with the details of the preceding intubation scenario, he was not pleased. This Director of Critical Care was higly critical of the care which had been dispensed in the Emergency Department. He suggested that this patient should have been managed more conservatively, implementing more aggressive steps, up to and including intubation and mechanical ventilation, only after more conservative measures were observed to have failed. Furthermore, he vocalized his view that the choice of 40 torr as a target was unrealistically low for a patient with long-standing COPD. As fate would have it, the patient was well known to the ICU team as a "frequent flyer" who had been diagnosed with moderate-to-severe chronic obstructive pulmonary disease years before. At that point in time, I worked for the Division of Pulmonary and Critical Care Medicine and served as an ICU Specialist. Part of my duties consisted of conducting periodic inservice sessions for the Respiratory Care Practitioners (RCPs) who practiced in that institution. I was dispatched to confer with the RCP who had assisted at the intubation the night before. In the ensuing conversation, this RCP, who was quite experienced, disclosed that he had not agreed with the course of therapy that had been implemented, but was disinclined to speak up. I counseled this RCP that members of the care team who are meek or reticent as a therapeutic strategy is being developed do their patients a disservice. RCPs, as well as nurses, are expected to be patient advocates and, to the extent that they might fail to be assertive, this inaction reflects adversely, both upon them and on their respective professions. In other words, the Acute Care Team is no place for "wallflowers".

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As mentioned earlier, it was this pulmonologist's practice to treat COPD patients conservatively. On those occasions when a patient clinically deteriorated, despite the team's best efforts, s/he would be intubated and mechanically ventilated, choosing a low-normal arterial pH as a target. This pH_a value would coincide with a baseline p_aCO_2 which was clearly higher than the middle of the homeostatic range for that parameter, and sometimes outside the homeostatic range entirely.

Slide 9

The pulmonologist's *modus operandi* is a perfectly logical and systematic approach, insofar as patients of this type, when left to their own devices, will adjust their level of ventilation in like manner. Implementing this strategy usually involves a fairly large time window, and was characterized by its' devotees as a method which was "more art than science". In this presentation, an alternative approach will be described, wherein a target p_aCO_2 will be identified and, once that is done, a systematic strategy to rapidly attain that target will be developed. Consequently, the strategy which will be described and developed during the balance of this presentation can be characterized as "more science than art".

Slide 10

Lawrence J. Henderson was an American physiologist who developed the equation which bears his name in 1908. The Henderson Equation, shown here, is remarkably simple and straightforward. Eight years later, Karl Albert Hasselbalch modified the Henderson Equation, which resulted in the formulation of the Henderson-Hasselbalch Equation. That expression has proven to be a stumbling block to generations of chemistry, physiology, medical, and allied health students, owing to its' complexity. We can use the Henderson Equation to calculate the homeostatic value for hydrogen ion concentration ($[H^+]$) simply by substituting the normal value for p_aCO_2 (40 torr) and $[HCO_3^-]$ (24 mEq/L) into the expression. Doing so reveals that 40 nM/L represents the middle of the homeostatic range for $[H^+]$. In point of fact, whenever the <u>ratio</u> of p_aCO_2 to $[HCO_3^-]$ is 40/24, a homeostatic $[H^+]$, as well as its' correlate, pH_a, will result.

Slide 11

The fraction 40/24 can be reduced to its' simplest form by dividing the numerator and denominator by eight, generating the fraction five thirds. If/when the Clinical Laboratory supplies us with p_aCO_2 /$[HCO_3^-]$ data pair which obeys this relationship, the $[H^+]$ and pH_a will be observed to be homeostatic. Armed with this information, we can incorporate it into a strategy for determining a target p_aCO_2 for any patient whose ABGs have strayed from the homeostatic range.

Slide 12

All we need do to ascertain a proper target p_aCO_2 is to: 1) observe the $[HCO_3^-]$ value incorporated in the ABG report; and 2) multiply that number by 5/3. This operation will yield a value for p_aCO_2 which, once imposed, will elicit homeostatic values for both $[H^+]$ and pH_a.

Slide 13

Consider this example: a patient with a history of poorly-controlled diabetes mellitus who presents with a pH_a of 7.23 units, a p_aCO_2 of 22 torr, and a $[HCO_3^-]$ of 9 mEq/L. This history and these lab values are compatible with a profound metabolic acidemia and a classic diabetic ketoacidosis. Because the primary component here is the metabolic component, a respiratory care practitioner (RCP) would not anticipate being involved in the therapeutic scheme. Nevertheless, we can calculate the level of hyperventilation which would succeed in restoring acid-base homeostasis

here. Pause the video now in order to calculate the p_aCO_2 value which would trigger the emergence of normal values for $[H^+]$ and pH_a. When you have completed that task, restart the video.

Slide 14

The reported $[HCO_3^-]$ was 9 mEq/L. Multiplying that number by 5/3 yields a p_aCO_2 figure of 15 torr. Enter this number onto your handout and highlight it. From this, we conclude that, if the prevailing carbon dioxide tension were 15 torr, a homeostatic $[H^+]$ and pH_a would supervene. We can quickly verify this by substituting those figures back into the Henderson Equation. Doing so confirms that our calculations were correct.

Slide 15

Consider this familiar example, a patient with a history of severe chronic obstructive pulmonary disease. It is common for these patients to be mildly acidemic, but this patient presents with an arterial pH which is appreciably lower than the lower limit of normal, which might necessitate an aggressive course of therapy. Initially, the pH_a is 7.29 units, the p_aCO_2 is 72 torr, and the $[HCO_3^-]$ is 34 mEq/L. Experienced RCPs will recognize this data set as being compatible with an acute-on-chronic hypercapnic episode, and a diagnosis of partially compensated respiratory acidemia. On the following slide, screen capture software has been used to illustrate how the web-accessible Acid-Base CLinIMApp can be used to confirm this diagnosis.

Slide 16

First, we key in the reported p_aCO_2 value of 72 torr. Then, the digits "34", corresponding to the reported $[HCO_3^-]$, are entered via the computer keyboard. The software proceeds to scribe lines corresponding to these values and applies the Henderson Equation to calculate the prevailing $[H^+]$, which is 50.82 nM/L. The app also applies the Henderson-Demers Equation to derive the value for pH_a, which is observed to be 7.29. Because this pH_a value agrees with the level which was reported by the lab, we confirm that the values are mutually compatible with the Henderson Equation and the Henderson-Hasselbalch Equation. Also note that the "Classification" listed is "partially compensated respiratory acidemia", and that the point of intersection of the scribed lines is located within the yellow region labelled accordingly.

Slide 17

This patient's clinically tenuous state prompts the Care Team to intubate and mechanically ventilate. We are asked to determine the target p_aCO_2 which would result in a homeostatic pH_a. Pause the video now to calculate that number and, after you have finished, restart the video.

Slide 18

As you'll recall, the patient's initial $[HCO_3^-]$ reading was 34 mEq/L. Multiplying that number by 5/3 yields a target p_aCO_2 value of 57 torr. Enter this number onto your handout and highlight it before proceeding. The accuracy of this result can be verified quickly and easily, as shown here. Remember, at the outset we stated that the target p_aCO_2 which results from the implementation of

this strategy will elicit a homeostatic $[H^+]$ and pH_a. In other words, if we impose a p_aCO_2 of 57 torr, the resultant $[H^+]$ will be 40 nM/L, and the corresponding pH_a will be 7.40.

Slide 19

Let's step back for a moment, and pose the following question: Can we imagine a scenario in which we might be <u>disinclined</u> to impose a homeostatic pH_a? Actually, on occasion, we might choose to readjust the pH_a to a level which is slightly different than the middle of the homeostatic range. Victims of COPD provide a perfect example of this category of patient. Because the progression of their disease from mild, to moderate, to severe causes them to spontaneously and progressively readjust their baseline p_aCO_2, $[HCO_3^-]$, $[H^+]$, and pH_a levels away from the middle of their respective homeostatic ranges, we would be well advised to choose values for those parameters which are aligned with the baseline values resulting from the escalation of COPD.

Slide 20

Having said that, let's reexamine our previously-computed target p_aCO_2, which, if we were to actually impose it on this patient, would bring him back to a homeostatic level which he hasn't experienced for years. It would be far wiser for us to impose a p_aCO_2 level more in keeping with the patient's chronically elevated state. After all, if we succeed in reversing the acute-on-chronic episode with which the patient initially presented, and he is able to eventually walk out the door, the respiratory center located in his medulla oblongata will have restored his baseline p_aCO_2 to that level. In other words, simply because we CAN restore this patient's p_aCO_2 to the textbook "normal" value, that doesn't imply that we SHOULD do so.

Slide 21

On the basis of the foregoing reasoning, let's assume that we select a target p_aCO_2 of 61 torr. We can use the Henderson Equation to determine what the resultant $[H^+]$ would be, as shown here. Notice that the subsequent $[H^+]$ is slightly higher, and the pH_a slightly lower, than the middle of their respective normal ranges, which is wholly in keeping with the typical baseline values for a patient with moderately severe COPD. To the extent that your physician/RCP/nursing colleagues solicit your input with respect to decisions of this type, you should be ready and eager to share this strategy with them.

Slide 22

Our next example consists of a 53-year-old female with a previous history of Congestive Heart Failure. She is transported to the Emergency Department following an overdose of barbiturates, at which time her pH_a is observed to be 7.11 units, her p_aCO_2 is 88 torr, and her $[HCO_3^-]$ is 27 mEq/L. Does this category of patient represent a situation wherein we'd be inclined to restore the $[H^+]$ and pH_a to the homeostatic level? And, if we're prompted to answer in the affirmative, how rapidly might we choose to do so?

Slide 23

The answer to those questions are: 1) yes!; and 2) immediately! We would be well advised to promptly restore acid-base homeostasis in this patient because her hypercapnia is acute. In such cases, rapid restoration is indicated. In a moment, you will be asked to pause the video in order to compute the target p_aCO_2. After you are finished doing so, restart the video.

Slide 24

The target p_aCO_2 is 45 torr, and is calculated as shown here. Enter this value onto your handout, and highlight it before proceeding. We should be aware that, although acute metabolic compensation for a primary respiratory acidemia tends to be very weak, it may not be completely absent. For that reason, the subsequent ABG analysis might reveal a prevailing $[HCO_3^-]$ value which is marginally lower than its' initial reading of 27 mEq/L.

Slide 25

This does not constitute a major problem. All we need do, if we observe that the $[HCO_3^-]$ falls slightly, is to recalculate the target p_aCO_2 accordingly. If this becomes necessary, be sure to inform your nontherapist colleagues about the mechanism which is operative here. This is a matter of simple courtesy to the other members of your team. In addition, it will serve to consolidate your role as the "go-to guy"/"go-to girl" with respect to ventilator adjustments.

Slide 26

The next scenario involves an infant who had spent the first three months of her life as an inhabitant of a neonatal nursery. Two weeks after NICU discharge, she presented to the Emergency Department with respiratory distress and an infiltrate on chest X-ray. While breathing air, an arterial specimen was procured, which yielded a pH_a of 7.31 units, a p_aCO_2 of 57 torr, a p_aO_2 of 71 torr, and a $[HCO_3^-]$ of 28 mEq/L. Initially, it was unclear whether or not this baby needed to be intubated. But, in order to be prepared for that step, it behooves us to calculate the target p_aCO_2 which should be chosen should it become necessary to ventilate her. Pause the video now in order to carry out that exercise and, after you've completed it, restart the video.

Slide 27

The application of the Henderson Equation, as shown here, reveals that a p_aCO_2 of 47 torr would elicit acid-base homeostasis. Write this value onto your handout and highlight it. It should be appreciated that BPD is a chronic disease which, in many respects, mirrors its' adult counterpart, COPD. Each of those conditions triggers an elevated baseline p_aCO_2. Consequently, the target p_aCO_2 for this BPD patient should be set marginally higher than 47 torr. Let's select 50 torr instead.

Slide 28

If we do that, we can easily predict the $[H^+]$ and pH_a values which would result. The results of those calculations are depicted here. Note that the baseline $[H^+]$ is within the normal range, as is the pH_a. This is wholly in keeping with the observed behavior of such patients, who, when they are

clinically stable, demonstrate a completely compensated respiratory acidemia. Happily, this patient responded well to the administration of intravenous antibiotics, and aggressive therapy was averted.

Slide 29

Up to this point, we've considered the selection of various target p_aCO_2 levels. In the remainder of this video, we will describe the logistical operations which we might implement in order to succeed in reaching a given target. The first step in predicting a p_aCO_2 level consists of determining the tidal volume (V_T) and the respiratory rate (RR) exhibited by our patient. As we all know, the algebraic product of these two parameters will determine the prevailing minute ventilation (MV) figure. Furthermore, we were taught long ago that minute ventilation and p_aCO_2 are inversely related. As MV rises, p_aCO_2 will fall, and vice versa.

Slide 30

If the ABG findings reveal that p_aCO_2 is supernormal, we are well advised to increase MV by increasing the respiratory rate. Conversely, the detection of a subnormal p_aCO_2 on the ABG report would prompt us to reduce MV by decreasing the RR. Although these statements are physiologically sound, they are only qualitative. If we are inclined to either increase or decrease the RR, by what degree should it be raised or lowered, we might ask. The answer to that question is: by implementing the simple formula shown here. The mathematical derivation of this equation is provided in the verbatim script which is downloadable at the same site from which this video was accessed.

Slide 31

Consider this example, wherein we recall the initial p_aCO_2 of the patient described in Example 3. We were told that her p_aCO_2 was 88 torr, but we weren't supplied with the additional information that, at the time the ABG specimen was procured, her V_T was 420 mLs and her RR was 10/min. If you'll recall, the target p_aCO_2 which we calculated for this patient was 45 torr. By applying the formula supplied in the previous slide, we conclude that the respiratory rate should be increased from 10 to 20 breaths/minute.

Slide 32

The initial minute ventilation figure is simply the product of V_T and RR, or 4,200 mLs. We weren't told this at the time, but this patient's body weight was 60 kilograms, or 132 pounds. This translates to a deadspace figure of approximately 132 mLs. With this datum in hand, we can proceed to compute the tidal alveolar ventilation figure, 288 mLs. Initially, then, this patient's minute alveolar ventilation (or "MAV") was 2,880 mLs. We've decided that we'e going to increase her RR to 20/min and, after we've done so, her final MAV will be 5,760 mLs. Finally, let's calculate the percentage increase in MAV, which is the final MAV minus the initial MAV, that quantity divided by the initial MAV. As we can see, this represents a doubling of the MAV, which should elicit a commensurate reduction in p_aCO_2. When the subsequent ABG is performed, we might be obliged to make "fine-tuning" adjustments to the ventilator, but the resultant p_aCO_2 will be very close to the predicted value.

Slide 33

Of course, we are all aware that we could have chosen to increase the MAV by imposing an elevation in tidal volume. Wouldn't this be as precise as choosing to change RR? Actually, such is not the case. A change in tidal volume will NOT elicit a proportionate change in MAV, for reasons to be explained shortly.

Slide 34

Before delving into the physiologic reasons for the statement that's just been made, it is useful to determine if it is ever advisable, or even acceptable, to alter tidal volume in lieu of changing the prevailing respiratory rate. It is both advisable and acceptable to do this on occasion, and, in fact, it might even be preferable to make adjustments in tidal volume instead of RR on purely clinical grounds. For example, if a patient's spontaneous RR is already elevated, we might be reluctant to impose a new RR which is even higher. That being said, we must realize that the p_aCO_2 which results from a variation in tidal volume will not be as predictable as the target p_aCO_2 elicited as a consequence of altering the RR.

Slide 35

The reason for this lack of precision resides in the non-varying nature of the patient's deadspace volume figure. The loss of precision related to alterations in tidal volume does not constitute a "fatal flaw", as long as clinicians are aware of it, and its' physiologic basis.

Slide 36

In order to demonstrate this, let's reconsider the patient described on Slide 32. The first three bullet points shown here have been carried over from that slide. In this exercise, we will choose to double the tidal volume, in lieu of doubling the respiratory rate. Doing so would result in a V_T of 840 mLs. Because this patient's deadspace volume reading is 132 mLs, her TAV would be 708 mLs. This would result in a final MAV of 7,080 mLs, which would trigger a resultant p_aCO_2 which would be appreciably lower than our intended goal. Of course, because we are aware of this mechanism, we can easily choose to increase V_T by a smaller increment, and observe the resultant effect on p_aCO_2. The important point is that we understand the physiologic underpinnings of this approach. To the extent that we master this therapeutic strategy, our patients will be well served, and we will assume our rightful place in the caregiver hierarchy.

Slide 37

In summary, a logical and systematic approach to identifying the target respiratory rate to be imposed on the mechanically ventilated patient requires prior knowledge of the Henderson Equation. Multiplying the prevailing $[HCO_3^-]$ value by 5/3 will furnish us with a p_aCO_2 target which will, if imposed, reestablish acid-base homeostasis. If we choose to adjust the level of ventilation by means of an alteration in respiratory rate, the implementation of this formula will enable us to achieve whatever target p_aCO_2 that we select, both quickly and easily.

Slide 38

Alternatively, we might select to target a p_aCO_2 which does <u>not</u> coincide with perfect homeostasis, such as when we are confronted with a patient with pre-existing chronic hypercapnia. In instances such as these, we can employ the Henderson Equation to anticipate the hydrogen ion concentration, and its' correlate, arterial pH, which will result when that alternative target is attained. This approach will tend to minimize a series of trial-and-error adjustments that can sometimes be tedious and time-consuming. For various clinical reasons, the ICU team might opt to modify the tidal volume in lieu of the respiratory rate. This is perfectly acceptable, but practitioners must be prepared to accept subsequent p_aCO_2 values which are less predictable than those elicited by alterations in respiratory rate.

Slide 39

Many of the concepts to which I have referred in this presentation are explored in considerable detail in various other modules. These Universal Resource Locators ("URLs") are provided for those viewers who might seek to augment their skills inventories pertaining to the topics covered in those modules.

Slide 40

No narration.

Appendix:

In order to derive the equation which will enable us to ascertain the respiratory rate setting which will allow us to achieve a given p_aCO_2 target, we begin by calculating carbon dioxide excretion as follows:

$$CO_2 \text{ excretion} = (\text{mole fraction of } CO_2) \bullet (\text{minute alveolar ventilation})$$

The first term of this equation can be expressed as:

$$\text{mole fraction of } CO_2 = p_ACO_2 \ / \ P_B$$

Since $p_ACO_2 = p_aCO_2$, this reverts to:

$$\text{mole fraction of } CO_2 = p_aCO_2 \ / \ P_B,$$

where p_aCO_2 (arterial carbon dioxide tension) and P_B (barometric pressure) are both expressed in torr.

The second term of the equation can be expressed as:

$$\text{minute alveolar ventilation} = (V_T - V_D) \bullet RR$$

where V_T (tidal ventilation) and V_D (deadspace ventilation) are expressed in mLs, and RR (respiratory rate) is expressed in breaths per minute.

The equation now reverts to the following form:

$$CO_2 \text{ excretion} = (p_aCO_2 / P_B) \bullet [(V_T - V_D) \bullet RR].$$

For a given patient over the course of a discrete observation period, and for whom we will be altering respiratory rate in lieu of tidal volume, "P_B", "V_T", and "V_D" will all be constant. Combining those like terms yields the following expression:

$$CO_2 \text{ excretion} = [(V_T - V_D) / P_B] \bullet (p_aCO_2 \bullet RR).$$

Although CO_2 excretion can be subject to change over the long term, secondary to a substantial change in the patient's body temperature, for example, it will be stable over the short term. If we decide to impose a new (final) RR which differs from the prevailing (initial) RR, the following equality will apply:

$$[(V_T - V_D) / P_B] \bullet RR_{final} \bullet p_aCO_{2\ final} = [(V_T - V_D) / P_B] \bullet RR_{inital} \bullet p_aCO_{2\ initial}$$

Dividing both sides of the equation by the constant factors incorporated within the first term on either side of the equation leads to the simplified expression:

$$RR_{final} \bullet p_aCO_{2\ final} = RR_{inital} \bullet p_aCO_{2\ initial}$$

Finally, dividing both sides of the equation by $p_aCO_{2\ final}$ yields the final form of the equation:

$$RR_{final} = RR_{inital} \bullet [p_aCO_{2\ initial} / p_aCO_{2\ final}]$$

Notice that this formula is applicable whether the final respiratory rate is either higher or lower than the initial respiratory rate.