CLINICAL OXYGENATION AND RESPIRATORY FAILURE MADE SIMPLE --

Part II - MANAGEMENT OF RESPIRATORY FAILURE FOR THE EVERYDAY CLINICIAN

I. HYPOXEMIA

A. Knowing the WHY behind the clinical scenario

*If hypoxemia develops, it is far more important to the health of the patient to know why, than it is to correct the hypoxemia via the application of supplemental oxygen!*  
Do not allow the low SpO2 to distract you and divert your attention from finding the cause of the hypoxemia.

A good example of poor practice is highlighted in the recent revision of the Practice Guidelines For Sedation And Analgesia By Non-Anesthesiologists:

"If hypoxemia develops during sedation/analgesia, supplemental oxygen should be administered."

Such a concept is a recipe for respiratory arrest. It is a common occurrence in the ICU to review a chart of a newly intubated patient from the floor who has hypoxemia documented in their chart, followed by the application of oxygen, without sufficient (or any) investigation of the cause of the hypoxemia.

B. *In pts with lung disease, the A-a gradient is where the money is, NOT THE pO2!*

The A-a gradient is considered to be proportional to the severity of lung disease; it is used as a standard quantitative marker of the severity of the lung disease.  
*The pO2 or SpO2 mean nothing without a concomitant statement about the FiO2.*

C. What are the safe limits of hypoxemia?

**SHORT TERM MILD-MODERATE HYPOXEMIA IS, IN ITSELF, NOT DANGEROUS OR DAMAGING -- RATHER, IT IS THE CAUSE OF THE HYPOXEMIA THAT WILL HURT THE PATIENT.**

➢ Evidence

- 14 year old w severe hypoxic encephalopathy after choking. After terminal extubation, SpO2 25-40% for 18 hours. Normal HR and BP, good perfusion, normal ECG, no metabolic acidosis on ABG despite a pO2 of 24 mmHg (pH 7.30, PaCO2 59).
- Young, previously healthy patient had massively aspirated. His oxygen sats, verified by ABG’s, remained 30-40% for hours, despite high FiO2 and aggressive ventilatory maneuvers. He recovered completely without any neurologic deficits.
- Two male patients (35 and 58 years) over last few years with pneumonia/ARDS who had sats in the 70's for a week despite best efforts (no ECMO). No other organ failure. Both recovered well with no neurological or psychological deficit and are back to work.
- Young patient with pneumonia transferred in, sats varying form 50-85% most of the afternoon and evening. During one of our PEEP trials, the SpO2 fell to 25%, and it took almost 30 min to get back to > 50%. We tried everything: High PEEP, low PEEP, inverse ratio, different
modes, 30 x 30 recruitment, prone, and then 30 x 30 RM in prone. Kept him prone overnight. By next morning his SpO₂ crossed to > 90% and we started decreasing FiO₂.

- Saturation climbing Everest without oxygen is ≤ 40% (and that's exercising!).
- Low flying altitude (~ 14,000 ft, non pressurized) corresponds to an SaO₂ below 82%.
- At flying altitude, in pressurized aircraft - SpO₂ mid 80’s - mid 90’s.
- US Navy submarines maintain a constant FiO₂ of 18%.
- SpO₂ at rest in people living at altitude (SpO₂ at exercise even lower):
  - 5,000 ft -- SpO₂s in low 90s
  - 13,000 ft -- SpO₂s low-high 80’s.
  - Higher altitude -- SpO₂ down to 60’s?
- Patients after terminal extubation run low SpO₂s (40s-70s) for hours
- In research sleep studies, saturations commonly range 80-90%, often lower.
- Studies in thousands of pts showing that when anesthesiologists are not aware of the SpO₂, moderate levels of hypoxemia (ie. SpO₂s in the 80s) occur commonly, with no adverse effect to patients.
- Studies of nighttime SpO₂ of post-op patients receiving PCA opioids, breathing room air: About 20% desaturated into the 80’s. No harm to the pts.
- Evidence from the years previous to the development of the pulse oximeter would overwhelmingly suggest that there is NO evidence, even in patients with CAD, that mild-moderate hypoxemia, for the short term, is deleterious

II. RESPIRATORY FAILURE, VENTILATORY FAILURE, & CLINICAL MANAGEMENT

A. Distinction between respiration and ventilation

- **respiration** - technical definition: use of oxygen at the cellular level to produce energy (plants respire also).
  - clinical definition: Adequacy of oxygenation. Assessed by the A-a gradient (ie., SpO₂ at whatever FiO₂).
- **respiratory failure** = failure to oxygenate. An infrequent reason for mechanical ventilation.
- **ventilation** -- refers to breathing (mechanical process of chest going up and down, resulting in bulk transport of gas thru the tracheobronchial tree).

- Normal breathing is referred to as spontaneous ventilation.
- Patients are “ventilated”, and the machine is a mechanical ventilator.
- The management of the mechanical ventilator to supply artificial ventilation is ventilatory management. (There no such thing as artificial respiration, at least by today’s technology.)
- **ventilatory failure** = failure to ventilate (i.e. breathe) adequately, resulting in insufficient elimination of CO₂ -- i.e. the patient simply isn’t ‘breathing enough’ for one reason or another. By far, this is the most common reason for mechanical ventilation.

NB: Breathing goes hand in hand with a patent airway. There is no such thing as separation of the two.
B. There are FIVE, and only five indications for tracheal intubation. These are the 5 'p's:

- Positive pressure: This is ventilatory failure -- i.e. patient cannot physically breathe adequately on his own, so needs the air ‘blown into him’ -- admittedly a somewhat primitive concept, but effective nonetheless. This is by far the most common indication for mechanical ventilation.
- Protection: (airway protection – e.g. a pt with a stroke)
- Patency: (airway patency – e.g. a pt after head/neck trauma who has an ETT to assure airway patency)
- Pulmonary hygiene: (e.g. a pt with a stroke who cannot clear his secretions)
- pO\textsubscript{2} -- i.e. hypoxemia; this is the least common!

This may seem contrary to experience, especially in the ICU. However, it is almost always possible to oxygenate a patient if they are given a high enough FiO\textsubscript{2}, assuming their ventilatory capacity (i.e. their breathing effort) is adequate. Most often, a drop in the pO\textsubscript{2} is associated with or secondary to insufficient ventilatory capacity, i.e. ventilatory capacity is insufficient to maintain the pO\textsubscript{2}.

Regardless, it is actually the insufficient ventilation that causes the need for ventilatory support, altho certainly, many pts have both respiratory and ventilatory failure. Hypoxemia alone (without ventilatory insufficiency), is rarely, if ever, an indication for mechanical ventilation. (In my 25 years of practice, I have seen three adults who suffered from severe pure hypoxemia without ventilatory insufficiency-- two from PE, one from advanced congenital heart disease. None required intubation, and in fact, none of them looked bad clinically, either.)

C. Ventilatory fatigue and the rapid shallow breathing index (RSBI = f/V\textsubscript{T})

- “The inability to sustain spontaneous ventilation (i.e. ventilatory failure) is usually the consequence of an imbalance between ventilatory demand and the capacity of ventilatory muscles to maintain work of breathing (WOB).”

***I.E. VENTILATORY FAILURE = FAILURE OF WOB***

- The vast part of early management of "respiratory failure" is actually all about management of ventilatory failure -- i.e. failure of WOB. Failure of WOB is caused by respiratory muscle fatigue.
- Work of breathing has two components: 1) The respiratory rate and 2) the depth of each breath (i.e. the tidal volume).
- The physico-mechanics of muscular work indicate that of the two components of work of breathing, tidal volume requires more energy than respiratory rate. As a result, energy requirements of WOB are minimized by preferentially limiting the depth of each breath (ie. reducing tidal volume), as opposed to reducing the respiratory rate. So when the respiratory muscles fatigue, the body's (involuntary) response is to reduce tidal volume, which is manifest clinically as shallow breathing.
- The problem with that is that a drop in TV mandates an increase in the frequency of breathing (resp rate) because the body tries to maintain minute alveolar volume in order to maintain the pCO\textsubscript{2}.

Therefore, ventilatory fatigue and failure are commonly manifest by rapid shallow breathing -- i.e., the adaptation to fatigue is to decrease the tidal volume of breathing, which mandates an increase in the resp rate as noted above.

The take home message: Rapid shallow breathing is an adaptation to fatigue. Rapid shallow breathing is a manifestation of lack of ventilatory muscle power and
ventilatory endurance, which defines ventilatory failure, which is a prelude to respiratory failure and respiratory arrest, because:

- The rapid shallow breathing initially maintains minute ventilation ($V_e$), but unfortunately, the decrease in tidal volume increases the proportion of dead space ventilation, leading to decreased elimination of CO$_2$ -- i.e. a fall in ventilatory efficiency -- and a rise in the pCO$_2$.

  Dead space ventilation = wasted ventilation

  This obliges the tachypneic patient to increase overall minute ventilation to defend the pCO$_2$. But the patient is already fatigued and cannot increase $V_e$. Subsequently, gross ventilatory failure ensues as the patient is unable to maintain the ventilatory work necessary to bring the progressively increasing pCO$_2$ back to normal.

D. Clinical significance of rapid shallow breathing

- The Rapid-Shallow Breathing Index (RSBI) = frequency of breathing (i.e. resp rate) ÷ tidal volume ($V_t$). This is abbreviated $f/V_t$.
- Pathophysiologically, the RSBI is considered a measure of respiratory muscle endurance, and has been demonstrated to correlate extremely closely with measured work of breathing (and the lower the WOB, the greater the chance of successful weaning).
- An increase in the RSBI (mostly via a decrease in $V_t$ -- i.e., shallow breathing) is a precursor of ventilatory failure.
- Beware when this pattern develops post-extubation, especially in muscle depleted patients (e.g. the elderly, physically unfit, or nutritionally depleted).
- The clinical occurrence of rapid shallow breathing is evident by simply looking at the patient, and is often associated with paradoxical breathing. That is another reason why the clinical assessment from the foot of the bed is emphasized in the next section as the most important part of the basic assessment of the ventilatory status of spontaneously breathing patients.

E. Respiratory rate vs. tidal volume:

- High resp rates are ALWAYS associated with shallower tidal volume. This is a physiologic truism.
- Shallow tidal volume breathing is less efficient ventilation (because of higher dead space fraction).
- Narcotics promote decreased resp rate -- and decreased resp rates are frequently associated with larger tidal volumes (another physiologic truism). By definition, this is more efficient ventilation because the volume increase in each breath is all attributable to an increase in the alveolar portion of each breath, thereby increasing the ‘efficiency’ of each breath.
- Caveat: During spontaneous ventilation, very deep breathing (i.e. very large TVs) may lead to airway obstruction (as happens in patients with CO$_2$ narcosis, who typically breathe very slowly, with very deep breaths). This is because larger TVs mandate high inspiratory airflow velocity, which causes a sucking in of pharyngeal tissues, resulting in upper airway obstruction.

F. Clinical management of failure of WOB: Two choices:

1. Mechanical support of WOB - invasive mechanical ventilation (via tracheal intubation), or noninvasive mechanical support (BiPAP or CPAP).
2. Control of spontaneous WOB - Use of narcotics to control respiratory rate and pattern.
   a. Narcotics reduce WOB. How?
      - Decreased resp rate $\Rightarrow$ ↓ WOB  
        [target for spont RR is $\sim$ 16-20/min; as noted above, you don't want resp rate too low, bec that will cause airway obstruction.]
      - Increased TV associated with narcotics $\Rightarrow$ ↑ WOB (as noted above)
        But: the volume increase in each breath is all attributable to an increase in alveolar ventilation, thereby increasing the ‘efficiency’ of each breath.
        Combine that increased efficiency, with the decreased resp rate, and overall WOB is reduced! (despite the increase in TV).
   
   b. Characteristics of narcotic infusions:
      - rapidity of onset and offset
      - tolerance
      - histamine release - promotes edema formation
      - Morphine:
        - slow onset, slow offset
        - high tolerance develops because of specific morphine receptor properties
        - tolerance develops very rapidly
        - histamine release at high doses - promotes edema
      - Fentanyl:
        - rapid onset, rapid offset
        - medium tolerance, develops slower than with morphine
        - no histamine release
      ∴ Fentanyl is the mainstay of treatment when opiates are used in ICU for control of WOB.

III. BASIC PRINCIPLES OF CLINICAL ASSESSMENT

The important bedside clinical parameters involved in assessing ventilatory sufficiency are simple and easily assessed by even minimally trained persons. In addition to the SpO$_2$ (and FiO$_2$), there are three clinical parameters, and there are three ventilatory parameters read off the ventilator:

- Three clinical parameters:
  - How the pt looks, overall, from the foot of the bed.
  - The mental status (wakefulness and cooperativeness).
  - The spontaneous ventilatory pattern (i.e. good symmetrical expansion vs. paradoxical rocking chest/abdom movement; and use of accessory muscles. Respiratory rate is also a qualitative part of this; see notes regarding rapid shallow breathing, above.

The first two parameters above are the foremost basic principles of clinical assessment in any clinical situation faced by any practitioner in medicine, i.e. not unique to ventilatory management of pts or any particular aspect of the management of surgical or ICU patients.

These three clinical parameters can be summed up as ‘just looking at the pt from the foot of the bed.’ For example, a pt who is breathing well might be described as “he looks good, he’s awake and cooperative, and his ventilatory pattern is good.”

These parameters are more important than the ventilatory parameters below. Since the beginning of time, acute post-op patients in the operating room have been extubated based on these clinical parameters alone, without all the numbers, blood gasses, etc.
And three parameters read from the ventilator:

- total resp rate (≤ 24/minute)
- minute ventilation (VE)* (< 0.15 Liters/kg")
- tidal volume (≥ 5 cc/kg")

"based on lean body wt

*Notes re VE:

- Minute ventilation (VE) is, obviously, the product of resp rate x tidal volume, so why not just look at the RR and the TV separately?
- Increased VE is indicative of excessive work of breathing, a characteristic of pts who fail weaning. It’s therefore long been used as the classic ‘weaning’ parameter. Two of the reasons why it’s so useful are because of its simplicity, and because it’s so easy to measure - it’s read right off the ventilator. Thus, VE is additional information that often gives a handle on the respiratory and ventilatory status of the pt, more so than just the RR and TV alone, especially when the latter are equivocal.
- Unfortunately, a normal VE alone has conclusively been shown not to be able to differentiate pts who succeed vs those who fail extubation. Rather, it is primarily a high VE that is helpful, in predicting those not ready for extubation.
- The highest RSBI possible within the guidelines above (with a RR of 24, and a TV of, say, 300 cc for a 60 kg pt) is 80, which is close to the reported guidelines for RSBI (i.e. 100 as the cut off value).
- Women tend to breathe at a higher RSBI, due to a lower ventilatory muscle mass than in men. Accordingly, a 1996 study showed that among pts extubated with a RSBI > 100, men were more likely to require reintubation than women (i.e. consistent with women normally breathing at a higher RSBI than men).
- Ventilatory parameters that depend on pt cooperation (such as negative inspiratory pressure and vital capacity) are recognized to be unreliable, and therefore none are included in the above guidelines. Moreover, it has been suggested that maneuvers such as a NIF and VC are needlessly stressful to pts, and inappropriate in some settings (such as post-op open heart surgery) where it is sought to minimize cardiac workload and stress.
- In any extubation process, getting the pt’s spontaneous ventilatory rate into a regular pattern is the first priority to be addressed, vs. assuring tidal volume. During emergence from anesthesia, for example, once the pt’s breathing has evolved into a regular rate and pattern, a gradually increasing tidal volume will be usually be noted soon thereafter.

IV. CPAP and BiPAP -- a brief outline

A. Basics:
   - CPAP - to address the oxygenation component
   - BiPAP - adds positive pressure to CPAP in order to support the WOB component

B. Three classes of "respiratory" failure:
   - hypercarbic - e.g. COPD, asthma (i.e. ventilatory failure) → BiPAP is 80+% effective
   - hypoxemic-CHF → Start with CPAP, add BiPAP if WOB is escalating; efficacy 50+% (Note re differences betw pulmonary edema due to ARDS vs CHF: - In patients with comparable CXRs, the degree of hypoxemia is way worse with ARDS than with typical APE (acute pulmonary edema) due to CHF)
- One of the mechanisms of ventilatory failure in pulmonary edema, whether due to APE or ARDS, is that the lungs become wet and boggy. This reduced compliance increases WOB, and given comparable CXRs, is usually far worse in ARDS than in APE.
- APE responds much easier and faster to treatment than does ARDS bec of the above two factors -- i.e. given comparable CXRs, both the hypoxemia and the reduction in lung compliance are not as bad in APE as in ARDS.
- Given that the CXR cannot reliably distinguish APE vs ARDS, the ready response of APE to treatment can sometimes be used diagnostically to differentiate it from ARDS.

C. Airway obstruction - CPAP is the specific tx of choice
   - Steroids should also be considered in specific cases

V. SUMMARY:

THE RESPIRATORY SYSTEM HAS THREE COMPONENTS, NOT JUST TWO:

i. pO₂ (oxygenation)
ii. pCO₂ (ventilation)
iii. work of breathing (respiratory muscles)
   ★ Each one must be addressed individually!