Conditioning Inspired Gases: The Search for Relevant Physiologic End Points

Conditioning inspired gases during mechanical ventilation by adding heat and humidity is a well accepted standard of care.[1] Failure to heat and humidify inspired gas in an intubated patient results in well described complications, including inspissation of secretions, deciliation, bronchospasm, airway obstruction, atelectasis, and endotracheal tube (ETT) occlusion.[2] Though these issues are uniformly accepted, there remain at least 2 important unanswered questions concerning humidification:

- What levels of heat and humidification should be provided?
- How do we monitor delivered humidity?

The first question is oversimplified, as it fails to consider the important variables associated with patient status and the type of ventilation. Clearly, the humidification for noninvasive ventilation is different from that required for invasive ventilation.[3-5] There is currently no literature that evaluates the humidification needs of febrile versus normothermic patients. Heat and humidification in neonates is clearly more important, owing to the unique aspects of newborn temperature control, compared to adults.[6] Humidification needs for short-term versus long-term ventilation and in patients with normal lungs versus those with pneumonia and excessive secretions are similarly ill defined.

Perhaps the more pressing question is related to the goal of humidification. Is the goal to prevent the adverse effects of inadequate humidity (eg, provide at least the minimum humidity required to prevent ETT occlusion)? This can be considered the minimum required humidity. Is the goal to make the inspired gas a temperature and humidity equivalent to normal spontaneous breathing? Perhaps that could be termed physiologic humidity. Or is the goal to provide optimal humidity to maximize mucociliary clearance: the so-called "optimum humidity" described by Williams et al.[7]

Sottiaux argued that gas entering the trachea should be, at a minimum, equivalent to physiologic conditions (32–34°C and 100% relative humidity).[8] That recommendation is similar to one Chatburn and I made in the Journal some time ago.[9] Multiple factors undoubtedly determine the success or failure of any humidification strategy. The literature provides evidence of success with only a heat-and-moisture exchanger (HME),[10] and with a heated humidifier providing a supraphysiologic gas temperature.[11] This may well simply represent the resiliency of the respiratory tract; across a wide range of temperature and humidity values, the performance of the respiratory tract is unaltered.

Clearly there are situations in which the humidity is insufficient and ETT occlusion may result. An absolute humidity < 30 mg H_2O/L appears to be associated with a higher risk of ETT occlusion (Fig. 1).[12] Using a meta-analysis, Hess found that an HME is associated with a risk of ETT obstruction nearly 4 times that of heated humidification.[13] HME use in intensive care has substantially increased. Clinicians should recognize that, when using low tidal volume (to prevent lung injury), an HME with a large dead space may compromise a substantial portion of the tidal volume, which can cause hypercarbia and/or increase the minute ventilation requirement. Several authors have found benefits from removing the HME from patients with acute respiratory distress syndrome.[14-17] The choice of humidification device should be based not just on moisture output, but on effects on gas exchange. The new paradigm of low-tidal-volume ventilation should alter the use of HMEs in these patients.

As to the question of what temperature and humidity are required, there remains no evidence to suggest that absolute humidity > 30 mg H_2O/L improves outcomes. There is also no evidence that the temperature and humidity levels suggested by some[9,11] are harmful. The range for physiologic and optimum humidity may seem large, but the effect on the patient outcomes seems small.

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In this issue of the Journal, Solomita et al describe a method of measuring humidifier output, and they rediscovered that measurement of temperature alone is insufficient to determine adequate humidification.[18] About 15 years ago, Gilmour and colleagues found that the temperature at the patient end of the circuit cannot predict adequate moisture delivery.[19] Gilmour et al studied the effects of altering the increase in airway temperature as the gas traverses a heated-wire circuit, and found that the temperature increase from the humidification chamber to the Y-piece decreased the relative humidity to below 75%.[19] Based

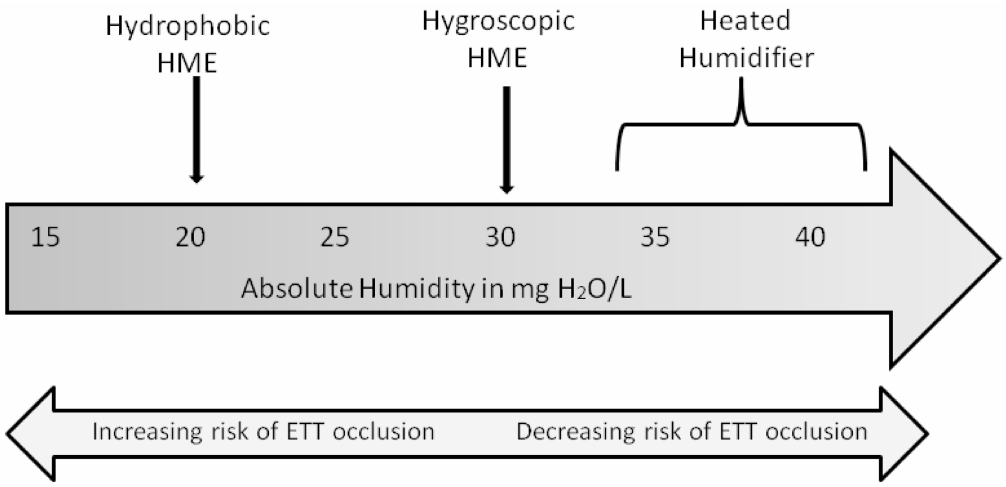

Fig. 1. Level of humidity provided by different humidification devices and the levels of absolute humidity associated with endotracheal tube occlusion.

on our current technology, heated humidifiers deliver the desired gas temperature at the airway, at the expense of humidity.

Solomita et al[18] describe a laboratory method of measuring water vapor delivery with a condenser. Their experiments are elegant, but limited. There is a systematic error in the system, which is probably related to its inability to capture all the moisture from the circuit. In their Figure 3, the water vapor recovered is always several milliliters less than that provided by the pump. And, though their system is elegant, it can only make measurements in the laboratory. The system might help determine device performance under laboratory conditions.

Perhaps more importantly, the paper[18] begs us to consider another question: how do we monitor delivered humidity? Despite the known limitations of monitoring temperature alone, temperature remains the standard, despite the fact that many of us have witnessed a dry humidifier delivering gas at 34–37°C at modest minute-ventilation conditions. Humidity sensors remain too fragile, too expensive, and too prone to saturation for routine clinical use.[20]

Technological limitations then lead us to clinical observation and common sense. Assessing the adequacy of humidification by secretion consistency and volume provides some data, but secretion consistency and volume can be affected by a host of confounding variables.[21] Water condensation in the tubing between the Y-piece and the ETT suggests gas saturation (ie, relative humidity of 100%). This has been validated clinically.[22] Until humidity measurements are routinely available, clinical observation by the respiratory therapist will remain the arbiter of humidification adequacy.

Humidification goals and humidification devices must change with evolving evidence. More research regarding the effects of various temperature and humidity levels across a wide range of patient populations seems in order. I believe that the use of low tidal volume requires that we reassess the use of HMEs when device dead space exceeds 20% of the set tidal volume. And while we await the introduction of reliable, robust humidity sensors, clinical assessment is our most reliable tool.

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The author declares no conflict of interest.

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