High-frequency percussive ventilation.

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Letters

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High-Frequency Percussive Ventilation

In the June 2010 issue of the Journal, Allan concluded from an in vitro study of high-frequency percussive ventilation (HFPV) that this ventilation mode may deliver injurious tidal volumes (V_T).[1] Allan claimed that the results from his experiments showed that with a commercially available Fleisch-type pneumotachograph (3700, Hans Rudolph, Shawnee, Kansas) he was able to measure the mean V_T of the high-frequency breaths and the low-frequency breaths delivered by HFPV. Allan's objective was to validate a pneumotachograph for HFPV and then exploit data to describe the behavior of both the high and low frequency breaths.

Lucangelo et al, in 2004, conducted a similar study of HFPV and published their data on mean airway pressure measurements, peak inspiratory pressure, positive peak flow, negative peak flow, and V_T in a lung model, using different levels of compliance and resistance.[2] In Lucangelo's experiment, V_T measurements were obtained by digitally integrating the flow signals of the high and low frequency breaths. Inspiratory positive and negative peak flows were measured during pulse breaths to determine the highest and lowest positive and negative values for the pulse flows characteristic of HFPV. A lung simulator (Medishield, Harlow Essex, United Kingdom) was used and set at 15 different elastic and resistive load combinations. Flow was measured with a number 2 Fleisch-like pneumotachograph connected to a differential pressure transducer positioned between the HFPV device and the lung simulator. The high-frequency rate was 500 breaths/min, the phase (conventional) rate was 10 breaths/min, and the inspiratory-expiratory ratio was 1:1.25. Using different combinations of compliance and resistance settings, measurements of peak airway pressure, mean airway pressure, inspiratory positive peak flow, inspiratory negative peak flow, and V_T were collected and fed to an analog-to-digital converter. Measured V_T ranged between 115 mL and 465 mL, using up to 45.43 cm H_2O of peak pressure, with various combinations of resistance and compliance settings on the test lung. The smallest V_T measured was associated with the highest impedance values of compliance and resistance. Lucangelo et al concluded that under similar clinical conditions, since V_T delivered decreased with a decrease in lung compliance, this could be interpreted as a lung-protective mechanism in HFPV.

As pointed out by Lucangelo et al, the gas flow characteristics of HFPV are unique in that the small percussive sub-tidal volumes have both positive and negative "minibursts," which cause gas wash-out that is thought to account for the smaller V_T actually delivered to the patient connection. The venturi system within the HFPV device portion of the ventilator circuit will reduce entrainment of gases as intrapulmonary pressures increase, which was demonstrated by the fall in inspiratory flow in that experiment.

Lucangelo et al concluded that HFPV creates a minimum amount of generated volume, even under maximum implied load conditions. The absolute values of the positive and negative peak flows with the delivered high-frequency breaths net a flow that nearly equals zero. Although the high-frequency breaths are useful for gas exchange, Lucangelo et al identified that they do not add significantly to the V_T delivered to the patient. Lucangelo differentiated between V_T that the lung actually experiences with HFPV by subtracting measured actual volume expired from actual V_T delivered with an inspiratory breath.

Allardet-Servent et al, in 2008, tested HFPV in a prospective randomized controlled in vivo animal study.[3] Forty-three rabbits were subjected to lung injury by intratracheal instillation of human gastric juices, and randomized to HFPV, low-V_T (6 mL/kg) mechanical ventilation, high-frequency-oscillation ventilation (HFOV), or high-V_T (12 mL/kg) mechanical ventilation. The group found that, like HFOV and low-V_T ventilation, HFPV provided lung protection while improving oxygenation and ventilation. The animals treated with HFPV showed dramatically less damage to lung tissue macroscopically and via microphotography, similar to the HFOV and low-V_T ventilated treatment groups. The lung-injury scores in the HFPV group were similar to those in the HFOV and low-V_T groups. Allardet-Servent postulated that with HFPV, like HFOV, there is a considerable drop in airway pressure between the trachea and the alveoli, and HFPV is therefore considered an effective lung-protective strategy.

Bougatef et al in, November 2007, reported on 15 years of experience using HFPV in premature infants, and described the successful management of respiratory distress syndrome with HFPV in a retrospective study that compared HFPV to conventional ventilation.[4] The infants included in that study were initially managed with an average peak inspiratory pressure of 25 cm H_2O, PEEP of 6 cm H_2O, a conventional rate of 40 breaths/min, and high-frequency rate of 600–900 breaths/min. There were lower incidences of chronic lung disease, pulmonary air leak, and elevated intracranial pressure in the HFPV group than in the conventionally ventilated group. Bougatef et al have been using HFPV in their neonatal intensive care unit since 1985, and they consider HFPV a lung-protective strategy for the smallest babies treated at their institution.

Mlcak et al, in 2002, compared HFPV and conventional ventilation in 86 pediatric patients with inhalation injury.[5] Forty-three patients were randomized to conventional ventilation and 43 to HFPV. Mlcak et al concluded that HFPV can significantly reduce delivered peak pressure, making HFPV an ideal protective ventilation strategy, especially in lungs that have been damaged by inhalation injury.

Reyburn et al, in 2008, compared noninvasive HFPV via nasal cannula to pressure-control intermittent mandatory ventilation (PC-IMV) in preterm lambs. Both groups were managed for 3 days.[6] Reyburn et al found that oxygenation and ventilation targets were sustained with lower airway pressure in the HFPV group than in the PC-IMV group. Notably, the HFPV group had less thinning of alveolar walls and more apoptosis and less proliferation among mesenchymal cells than the PC-IMV group. They concluded that HFPV via nasal cannula preserved the balance between mesenchymal cell apoptosis and proliferation in the distal air-spaces, so that alveolarization progresses in a natural fashion. Lung growth appeared to proceed during the 3-day ob-

servation in the HFPV group but not in the IMV group.

Chung et al (Allan was a co-author) recently published the first randomized controlled trial comparing HFPV and a low-V_T strategy in severely burned adult patients.[7] Over a 3-year period, 62 patients were randomized to either HFPV or a low-V_T strategy, to determine whether HFPV improved clinical outcomes in comparison to a low-V_T strategy. There was no difference in ventilator-free days at 28 days (the primary end point), but the number of patients who needed a rescue ventilation mode and the number of patients with barotrauma were significantly higher with the low-V_T strategy than with HFPV. Both the need for rescue ventilation and barotrauma were secondary end points in that randomized controlled trial.

Chung et al concluded that there was no significant difference in lung protection between the low-V_T group and the HFPV group, and that HFPV was at least no more harmful than their low-V_T strategy. In fact, HFPV may be more protective, since the incidence of barotrauma in the low-V_T group was higher. For burn patients with and without inhalation injury, the low-V_T strategy was inadequate in obtaining the oxygenation and ventilation goals.

The aforementioned papers describe delivered V_T and peak pressure during HFPV that is in stark disagreement with the data described in the Allan study.[1] Many more scientific papers exist that dispute the results that Allan described in his in vitro model and lead him to conclude that typical HFPV settings could be injurious to the lungs.

If Allan's objective was to validate the pneumotachograph, as stated in the abstract, then his paper lacked an explanation of how he accomplished that objective in that in vitro study. Since that pneumotachograph is the only one of its kind and not readily available to others, a control using volume ventilation with a conventional ventilator may have helped convince readers that his results were accurate. According to the manufacturer of the pneumotachograph, during the experiment conducted by Allan, there was "not a way to measure the high-frequency volumes correctly" (Hans Rudolph, Shawnee, Kansas) with the pneumotachograph used in the experiment.

Allan had the notion that he if he calculated the V_T of the high-frequency breaths and the low-frequency breaths separately and used an equation he could extrapolate the actual V_T delivered to the patient interface in the circuit. That notion convinces us that Allan does not fully understand the mechanism of breath delivery and gas-flow characteristics of HFPV if he tried to separate these measurements.

The V_T values derived in Allan's experiment were actually the sum of the measured high-frequency breaths and the low-frequency breaths, which would grossly inflate the size of the actual V_T delivered at the patient connection. What Allan did not account for was the gas-flow delivery characteristics of HFPV that Lucangelo so eloquently describes in his experiments.[2,8] If Allan had accounted for the wash-out flow of the high-frequency breaths by measuring the inspiratory positive peak flow and the inspiratory negative peak flow, he would not have obtained the high V_T measurements that he reported. The high-frequency breaths delivered by HFPV are not cumulative delivered tidal volumes. Overlooking that important fact about HFPV(that high-frequency breaths are not cumulative in relation to V_T) was an oversight by Allan. His claim that his in vitro study is the first to "provide an accurate and precise measurement of 2 separate HFPV gas-flow patterns" suggests that he overlooked the elegant work by Lucangelo et al. Allan should have remarked on the differences between his in vitro study and the work of his predecessors.

Very seldom are high-frequency rates less than 400–450 breaths/min used clinically. The typical clinical rate range, as indicated in previous scientific papers describing HFPV, are 500–800 breaths/min for adults, with an average of 600 breaths/min. For smaller children and infants, 600–900 breaths/min is typically used to provide optimal oxygenation and ventilation. Allan measured the V_T of high-frequency rates less than 400 breaths/min and considered those rates as "typical" HFPV settings.

Although Allan's study was an honest attempt to measure the V_T created by HFPV, the study design was flawed and therefore the conclusions were inaccurate. If it were true that HFPV delivers a mean V_T of 1,337 mL, it would be impossible to deliver HFPV in neonatal and pediatric intensive care units. How could a ventilator that generates such high V_T, as reported by Allan, be used in burn intensive care units for decades without reports of dangerous levels of volume and pressure being generated by HFPV?

In a recent article in *Chest*, a group recommended HFPV as an alternative ventilation mode for severe hypoxemic respiratory failure.[9] HFPV is listed along with HFOV, airway pressure-release ventilation, and extracorporeal membrane oxygenation as alternative ventilation strategies for refractory hypoxemia, and HFPV has a benefit over those other modes in that it mobilizes secretions from the lung periphery to the larger airways. HFPV, along with those other modes, are proposed to be used in an algorithmic approach to managing patients with severe hypoxemia. If this is the recommendation from those esteemed authors, we would not expect them to recommend a mode that could cause lung injury.

The concept of HFPV technology employs pneumatic switches and a series of engineering logic to recruit, stabilize, and ventilate diseased airway structures. Breath delivery to the patient is via an interface that consists of a unique venturi system designed to maintain low V_T even under conditions of low lung compliance. By delivering what are referred to as "percussive high-frequency sub-tidal volumes" in a stepwise fashion, HFPV recruits alveoli without airway hyperinflation. Precise engineering and appropriate clinical application make HFPV a lung-protective strategy that has saved many lives in the past and will continue to do so in the future.

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The author responds:

Short and colleagues dispute the findings of my recent pneumotachograph validation study published in RESPIRATORY CARE.[1] Their letter questions the validity of the study and its potential clinical implications, primarily that HFPV may not be synonymous with low-tidal-volume (low-V_T) ventilation. Citing clinical experience and animal-model studies, Short et al also remark on the lung-protective effects of HFPV. I also have had extensive clinical experience with HFPV and wish to expand upon the respiratory care community's comprehension of HFPV. My investigations have drawn a balance between noting HFPV's merits and deficits in the hope of improving the understanding of its application.

A pressure-differential pneumotachograph is one of the most accurate means of measuring high-frequency and low-frequency V_T. The data reported by Hager et al revealed that a pneumotachograph from the same manufacturer as the one explored in my study[1] was as accurate and precise, if not more so, than ultrasonic, heated-wire, and Pitot-tube flow sensors when gauging the volumes administered by HFOV.[2] Much of our knowledge regarding high-frequency gas flow is derived from studies that used pneumotachographs and plethysmographic models that have then been extensively validated in both animal models and clinical study.[2-8] If Short et al require further corroboration, I recommend they consult the cited studies. It would be erroneous to contend that pneumotachography is the sine qua non for high-frequency-V_T assessment (as I noted in my paper[1]), but the validity of pneumotachography is well founded.

Short et al questioned my pneumotachograph methodology by citing the 2004 study by Lucangelo et al.[9] That comparison is inappropriate for 3 reasons. First, the Lucangelo et al study failed to provide validated physical measurements and was thus seriously flawed. Second, that 2004 study used ventilator settings that are not relevant to the clinical management of adult acute lung injury or acute respiratory distress syndrome (ALI/ARDS). Third, Lucangelo et al used a pressure pneumotachograph that employed flow-measurement principles similar to the pneumotachograph I used, yet Short et al criticize such pneumotachograph measurements. I will further discuss the first 2 points.

Validation of sensors is indispensable. Simply interfacing a flow sensor with a test lung provides no assurance that the data obtained is either accurate or precise. A method of ensuring that a flow-sensor's results are valid is to compare the flow-sensor data to that derived from a reference standard device such as a plethysmograph-equivalent glass or metal chamber. With this information an investigator can then report, with confidence, not only that a sensor is accurate and precise, but how error-prone it might be in specific circumstances. My methodology was founded on this time-tested and repeatedly proven validation principle. All flow sensors have faults. What were the problems with the sensor used in the 2004 paper[9] by Lucangelo et al? To clarify, the 2004 study was done without an a priori validation of the sensor against an accepted standard. It is thus not possible to know whether those measurements were accurate or precise. Again, is validation important? It depends on how much measurement error one may find acceptable: less than 5%, 20%, or more than 50%? Regrettably, Short et al accepted Lucangelo's 2004 study as "elegant" and beyond doubt.

Short et al further state that "HFPV creates a minimum amount of generated volume, even under maximum implied load conditions." The following fundamental principle is certain: HFPV is a *pressure-limited mode of mechanical ventilation*, and just like any other such mode, it will decrease V_T in response to an increase in respiratory-system impedance. If one follows the logic of Short et al, that such a response is lung-protective, then by default any pressure-limited mode must also be lung-protective. Is this regardless of the administered V_T? Short et al assumed that, based on the unvalidated data from the 2004 paper,[9] which reported the largest low-frequency V_T as 465 mL, the greater than 1 L low-frequency V_T observed in my pneumotachograph study could not be correct. Did they note that the 2004 study's volumes decreased from 465 mL, whereas my measured volumes increased? A simple explanation follows: Lucangelo's group set the ventilator in such a way that it attained a low mean airway pressure of around 7–8 cm H_2O, whereas I *started* at 10 cm H_2O and *increased* the pressure from there. Furthermore, Lucangelo et al didn't change their ventilator settings, but instead either tightened the screws on the test lung to decrease compliance (up to 4-fold higher than mine) or narrowed the airway (up to 40-fold higher than mine). Lucangelo's volumes, at unchanged ventilator settings but at increasing impedance, had no other option but to start small and then fall, thus meeting Short's expectation of diminished volumes. Did Short et al examine the mean airway pressures quoted in my RESPIRATORY CARE paper[1] (ie, 10, 20, and 30 cm H_2O)? These pressures were explored in tandem with increasing inspiratory intervals. Therefore, the volumes that I measured could only have

increased from the Lucangelo et al starting point. Short et al have mistakenly cited Lucangelo's study to criticize mine, without recognizing that the 2 studies' results are actually in congruity. However, the 2 studies go on to explore HFPV's responses in different manners. The test lung model used in my study had fixed ventilation variables such as lung compliance and resistance. The latter variables were set at levels commonly seen in adult patient with ALI/ARDS. I also contend that the ventilator settings I used closely replicated clinically relevant settings. Velhamos et al (mean airway pressure 29.5 cm H_2O), Salim et al (mean ± SD airway pressure 23.6 ± 6.8 cm H_2O), Eastman et al (mean ± SD airway pressure 35.0 ± 8.0 cm H_2O), Chung et al (mean ± SD airway pressure 20 ± 10 cm H_2O), and others would appear to agree.[10-13]

Further, Short et al quoted peak inspiratory pressures. As with any other pressure-limited mode, peak inspiratory pressure reflects a combination of the effects of respiratory-system resistance and compliance on ventilator flow, whereas mean airway pressure is, in certain settings (eg, in the absence of V_T or gas-flow velocity data), a more reasonable reflection of lung compliance and the extent of lung inflation. It is also important to note that peak inspiratory pressure may not be strictly coupled with high-frequency V_T magnitude.[14] It is for these reasons that I selected mean—and not peak—airway pressure to gauge low and high V_T responses.

The 2004 Lucangelo et al study[9] *did not* provide high-frequency V_T measurements; they reported on high-frequency flow, which could possibly have been integrated to quantify high-frequency V_T. In contrast to my study, in which I quantified 2 separate volumes simultaneously, volume data were not reported in the 2004 piece.

Short et al expressed indignation at the large low-frequency volumes I found with HFPV. The following will serve as an illustration of what volumes HFPV can deliver. Airway pressure-release ventilation (APRV) is another example of a pressure-limited mode of mechanical ventilation. APRV, depending on the dynamic impedance of the circuit-airway-lung complex, has been known to administer V_T in excess of 4–8 mL/kg.[15,16] These large volumes may be mechanically or spontaneously driven and have raised a note of caution in condoning the application of APRV to all ALI/ARDS patients in the absence of favorable large-scale comparative studies. Surely Short et al have encountered such volumes while applying APRV at the bedside or during test-lung teaching opportunities. Importantly, one of their colleagues published the finding that HFPV "*mimics APRV*" but they did not provide V_T results.[17] Reflect on the fact that the observation of large V_T arising from a combination of prolonged inspiratory time with or without high airway pressure really isn't novel and occurs daily in every intensive care unit that chooses to either not govern V_T magnitude or, in the case of HFPV, fails to appreciate the scale of the administered V_T. Short et al make inappropriate assumptions regarding the high frequency range used by many HFPV investigators (discussed below), which suggests that they are unaware of reports such as the one by Eastman et al, who utilized HFPV with a 6-second inspiratory time at a mean airway pressure in excess of 30 cm H_2O.[12] These intervals and pressures are encountered in patients with severe ALI/ARDS maintained on APRV and, as already discussed in regards to mean airway pressure, with HFPV as well.

Short et al state that "The concept of HFPV technology employs pneumatic switches and a series of engineering logic to recruit, stabilize, and ventilate diseased airway structures." Such comments, by and large excerpts from the manufacturer's manual, have contributed to the inaccessibility of HFPV. I believe this discussion should have lent affirmation to many that HFPV best be considered as a high-frequency variant of a pressure-limited, time-cycled form of mechanical ventilation.

Short et al describe what the pneumotachograph I used measures when they state that HFPV functions "by delivering what is referred to as 'percussive high-frequency sub-V_T' in a step-wise fashion." The pneumotachograph I used did indeed measure that same "step-wise" high-frequency breath to which they attribute HFPV's efficacy. At risk of oversimplification, every upward positive "inspiratory" pressure has a downward "expiratory" pressure. For our purposes we can substitute flow or volume in the place of pressure. The pneumotachograph software sampled (at 500 Hz [ie, 500 cycles/s]) each and every high-frequency deflection and took the difference between the upward and downward phases over the time interval of a low-frequency breath to derive a net low-frequency V_T. Each low-frequency V_T was assessed starting from the first high-frequency breath that initiated a lung inflation, and ending with the high-frequency breath that preceded passive deflation. Contrary to the interpretation of Short et al, the separate high-frequency V_T were not subsequently *added* to the low-frequency volume. I then further *validated* the flow sensor methodology specifically to HFPV by using an established process (a glass-bottle plethysmograph and a mechanical lung) and showed that the sensor-measured high and low frequency volumes were accurate to within 2%, as is consistent with standard Bland-Altman analysis. Clinicians and investigators may differ with respect to interpretation of HFPV gas-flow patterns, but such exchanges could benefit from a validated flow sensor measurement.

Short et al state that, "Very seldom are high-frequency rates less than 400–450 breaths/min used clinically." Salim et al (as low as 350 cycles/min),[11] Hall et al (who used a *starting* frequency of 450 cycles/min and used frequency-reduction as an option to address hypercarbia),[18] Hurst et al (who *started* at 250 cycles/min),[19] Cioffi et al (who used a frequency as low as 200 cycles/min),[20] Eastman et al (who noted a lower-limit frequency capability of 200 cycles/min),[12] and other HFPV investigators[21] might disagree. Short et al may be unacquainted with the published research proposals made by other HFPV investigators, which include an HFPV frequency-based study arm with a frequency of 3 Hz (180 cycles/min).[18] I would caution Short et al against generalizing their practice to all HFPV-experienced clinicians. For instance, in adults, HFOV settings as low as 3 Hz have been utilized with no associated increase in untoward effects relative to higher-frequency approaches.[22] Furthermore, some experts are concerned that higher frequencies (up to 12 Hz) may also incur a form of ventilator-induced lung injury known as frequency-induced stretch.[23] It seems prudent to gather more flow-sensor-derived bedside data to fully understand the impact of HFPV on patients (as has been done with HFOV) before embarking on a potentially myopic frequency mandate.

My RESPIRATORY CARE paper's conclusions were criticized by noting that neonatal and pediatric patients have tolerated HFPV treatment; yet I used an *adult* model, with a respiratory-system impedance equivalent to that of an *adult,* in conjunction with mainly *adult-size* endotracheal tubes. To extrapolate my adult-model data to pediatric—let

alone neonatal—applications is inappropriate. A neonatal or pediatric test-lung exploration of HFPV with an appropriate-size endotracheal tube and ventilator circuit is needed. Indeed, the study by Lucangelo et al[9] appears more representative of a neonatal scenario.

In reference to clinical studies, Short et al state that those studies "describe delivered V_T and peak pressure during HFPV…." That is incorrect. There are no *clinical* studies. Only test-lung models have been used to measure and describe HFPV-delivered high and low frequency V_T.

Short et al discussed HFPV's clinical and animal-model derived attributes with the connotation that HFPV be deemed comparable to low-V_T ventilation. I recently reviewed[24] the evidence in support of HFPV, and none of the evidence is from large-scale randomized prospective trials.[24] This is not a denunciation of HFPV but a concession. Like many other ventilator or adjunctive modalities that had early clinical success (eg, HFOV, inhaled nitric oxide, etc), additional research is necessary before HFPV can be considered a standard of care for ALI/ARDS management. I am not alone in this belief.[21,25] I too have accessed HFPV's "rescue" role but emphasize that that does not yet equate to HFPV being a standard of care for ALI/ARDS. Low-V_T ventilation should play an integral part of any lung-protective strategy, and once its HFPV-based delivery is assured, could potentially promote the positive effects of HFPV on ALI/ARDS-associated outcomes.

Short et al criticized a recently published clinical trial by Chung et al,[13] a study in which I was a participant. The fundamental reason for developing a pneumotachograph was to ensure that low-V_T was being given to ALI/ARDS patients. Clearly, it is this population in which mortality has improved most from low-V_T strategies. The aforementioned burn-center study was neither powered to determine the "lung-protective" effects of HFPV in ALI/ARDS nor to ascertain a difference in mortality relative to that of low-V_T algorithms. Inhalation injury should not be equated with the more common causes of ALI/ARDS, as many severe-burn patients were excluded from the benchmark low-V_T studies.[26] The Chung et al study[13] lent support to the important role HFPV may play in the management of inhalation injury. Confirmatory studies are warranted. From an educational perspective, I think it also important to bring attention to the minimal cuff deflation that was used in the burn-center study. Endotracheal tube cuff deflation may reduce both high and low frequency V_T magnitude and thus may decrease the risk of volutrauma (Allan et al[14,24] and unpublished observations).

Was the use of a pneumotachograph reasonable? Were there any other methods available to ascertain the precision and accuracy of the sensor? Is further study warranted? The answer to all of these questions is an emphatic "yes" and I have elaborated on these points. From this perspective, I performed both an in-depth flow-sensor validation and a subsequent descriptive analysis of HFPV waveforms. The overarching goal of my RESPIRATORY CARE paper[1] was to help consolidate the role of HFPV in the broad scheme of low-V_T ventilation which, as required by the medical community as a whole, is a prerequisite to lung-protective ventilation. My work was apparently misinterpreted as a condemnation of HFPV, when in fact it was an effort to objectively contribute to our understanding of this useful ventilation mode. Rather than accepting the paper as is, the study was criticized by comparison with unvalidated data obtained at clinically irrelevant ventilator settings for the adult ALI/ARDS patient, misconceptions, and reference to non-applicable patient cohorts. Rather than a collegial exchange, emotions appear to have been interjected. This dialogue demonstrates that our collective awareness of the mechanistic and physiologic effects of HFPV is relatively limited, but, reassuringly, the accumulating evidence in support of both the beneficence and efficacy of HFPV continues to invigorate exploration.

I am neither an advocate for, nor endorse, any particular pneumotachograph make or manufacturer. However, the manufacturer of the flow sensor in question stands by the integrity of its measurement capacity and derived data (Gilbert Snedden and Aaron Dirks, personal communication, Hans-Rudolph, Shawnee, Kansas). The flow sensor, software, and test lung I used are commercially available.

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The author has disclosed no conflicts of interest.

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In Vitro Fluid Leak Around the Endotracheal Tube Cuff Is Easily Remedied

In the August 2010 issue of RESPIRATORY CARE, Deem and Treggiari reviewed the current evidence regarding endotracheal tube (ETT) design and the incidence of ventilator-associated pneumonia.[1] This letter is in regards to polyurethane-cuffed ETTs and seepage of secretions into the tracheobronchial tree. The theoretical advantage of polyurethane and silicone cuffs, compared to high-volume low-pressure polyvinyl chloride (PVC) cuffs, is the absence of creases on the inflated cuff surface, which can allow secretions to pass into the tracheobronchial tree, thereby increasing the incidence of ventilator-associated pneumonia. Deem and Treggiari gave an overview of the few clinical studies that have been performed, and pointed out some inadequacies in the current body of knowledge. I agree with them that more clinical studies are needed to better assess the efficacy and cost-effectiveness of polyurethane and silicone cuffs.

Included in Deem and Treggiari's paper were 2 photographs from an in vitro study, depicting the theoretical advantage of polyurethane cuffs. Dye was instilled on the top of a cuff inflated inside a plastic tube. After 15 minutes all of the dye had leaked past the PVC cuff, whereas none had leaked past the polyurethane cuff. They say a picture is worth a thousand words, and these pictures certainly are remarkable and I would expect them to be very influential on clinicians. It is obvious from the text that Deem and Treggiari did not use these photographs in an attempt to convince the reader that polyurethane cuffs should replace PVC cuffs; however, these photographs do exaggerate the superior in vitro performance of polyurethane cuffs, given that leakage around a PVC cuff in vitro is easily remedied.

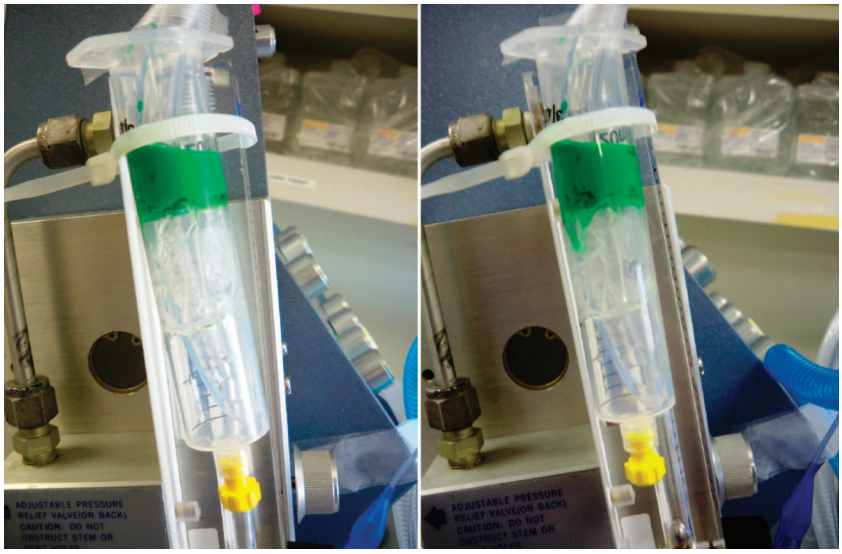

Fig. 1. Left: Mallinckrodt 7.5-mm inner-diameter endotracheal tube with a lubricated PVC cuff, 2 hours after 3 mL of colored whole milk was instilled above the cuff and the model was mechanically ventilated at 5 cm H_2O PEEP and 5 cm H_2O inspiratory pressure (ie, pressure below the cuff). Immediately after taking this picture, I agitated the cuff by moving the endotracheal tube up and down and side to side. Right: Three hours later (2 hours after agitating the cuff plus 1 hour after disconnecting the ventilator and thus removing the positive pressure below the cuff).

Dullenkopf et al[2] performed an in vitro study like the one described above, and found that after 10 min nearly all the fluid had passed by the PVC cuff, whereas none had leaked past the polyurethane cuff. However, when they treated the PVC cuff with a lubricating gel, leakage was substantially reduced. Lucangelo and colleagues[3] also performed a similar in vitro experiment, and found that PEEP of 5 cm H_2O essentially eliminated seepage around the PVC cuff, both in vitro and in vivo.

I performed a similar in vitro experiment (unpublished) with a couple of other modifications. I used a Mallinckrodt 7.5-mm inner-diameter ETT with a PVC cuff, inserted into a 30-mL syringe that has the same diameter (20 mm) as the average human adult trachea.[4] I lubricated the PVC cuff with a water-soluble jelly prior to insertion. Given that oropharyngeal secretions have a higher viscosity than water, I used colored whole milk, which at 70°F has a viscosity 2–3 times higher