Original Contributions

Peak Pressures During Manual Ventilation

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INTRODUCTION: Manual (bag) ventilation sometimes achieves better oxygenation than does a mechanical ventilator. We speculated that clinicians might generate very high airway pressure during manual ventilation (much higher than the pressure delivered by a mechanical ventilator), and that the high airway pressure causes alveolar recruitment and thus improves oxygenation. Such high pressure might injure alveoli in some patients. METHODS: We tested the hypothesis that manual ventilation may involve substantially higher pressure than is delivered by a mechanical ventilator. We asked experienced respiratory therapists to manually ventilate a lung model that was set to represent several typical clinical scenarios. RESULTS: We found that the peak airway pressure generated by the therapists was sometimes in excess of 100 cm H_2O. CONCLUSIONS: The high airway pressure during manual ventilation would be considered extreme in the context of conventional mechanical ventilation, which raises questions about whether manual ventilation causes barotrauma. *Key words: mechanical ventilation, lung mechanics, lung recruitment, resistance, elastance, barotrauma.* [Respir Care 2005;50(3):340–344. © 2005 Daedalus Enterprises]

Introduction

Manual (bag) ventilation is commonly administered to patients in intensive care units during cardiopulmonary resuscitation[1,2] or as a temporary measure while mechanical ventilation is being arranged. Manual ventilation is also occasionally used to achieve better oxygenation than is possible with conventional mechanical ventilation.[3,4] We speculated that this intriguing but poorly understood advantage of manual ventilation might arise because clinicians are relatively unconstrained in the ventilation patterns they can provide during manual ventilation. For example, when manually ventilating severely collapsed lungs, the clinician is free to deliver substantially higher

airway pressure than would be delivered by a mechanical ventilator. The higher pressure could increase lung recruitment and oxygenation, but it might also increase the risk of barotrauma. Elucidating the mechanical stresses applied to the lungs during manual ventilation may help us optimize manual ventilation and better ensure patient safety.

We hypothesized that respiratory therapists (RTs) sometimes generate very high airway pressure when manually ventilating severely obstructed or restricted lungs. We tested that hypothesis by having experienced RTs manually ventilate a lung model while we monitored peak airway pressure (P_{peak}), tidal volume (V_T), and ventilation frequency (f).

Methods

Measurements

We asked experienced RTs (8–35 years professional experience) at Fletcher Allen Health Care, Burlington, Vermont, to manually ventilate a lung model (training and test lung model 1601, Michigan Instruments, Grand Rapids, Michigan), which was set to specific values of resistance

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(R) and compliance (C). The RTs manually ventilated the lung model as they saw fit, using a standard plastic 1.8-L adult manual resuscitation bag (catalog number 5387, Hudson RCI, Temecula, California). To recruit the RTs, who were unaware of the aims of the study, we set up our apparatus in a room adjoining the intensive care unit and elicited participation from on-duty RTs. As the different components of the study took place on different days, different groups of RTs participated in each component. Informed consent was obtained from each RT, and the study was approved by the institutional review board of the University of Vermont.

Two resistance values were used: (1) a low value of 15 cm H_2O/L/s, to represent an intubated subject with little airway pathology, and (2) a high value of 50 cm H_2O/L/s, to represent a subject with severe obstructive lung disease. Two compliance values were used: (1) a high quasi-normal value of 0.033 L/cm H_2O, and (2) a low value of 0.012 L/cm H_2O, to represent restrictive lung disease. This produced 4 different load combinations:

Load rC: normal resistance with normal compliance
Load RC: high resistance with normal compliance
Load rc: normal resistance with low compliance
Load Rc: high resistance with low compliance

The RTs manually ventilated the lung model with each load combination (rC, RC, rc, and Rc) for 40 seconds. Pressure and flow were measured just proximal to the resistive element in the lung model (which corresponds to the airway opening of a patient) with a piezoresistance pressure transducer and pneumotachograph (model 3700B, Hans Rudolph, Kansas City, Missouri), amplified and low-pass filtered at 30 Hz (model SC-24, Scireq, Montreal, Canada), sampled at 200 Hz by a 12-bit analog-to-digital converter, and stored for later analysis in a personal computer. During manual ventilation the RTs were unable to see either the computer monitor or the lung-model pressure and V_T reading dials of the lung model. The RTs were also blinded to the results from other RTs.

Study Design

In the first part of the study the RTs ($n = 9$) were instructed to ventilate the lung model as if it were a 70-kg adult patient. No further information was provided to the RTs. To reduce the visual feedback the RTs might receive from movement of the ventilated compartment, the lung model was covered with a bed sheet. This permitted the RTs to gain some sense of movement during ventilation, similar to observing a patient's chest, but prevented precise evaluation of the tidal elevations in the model's ventilated compartment.

On a separate day, 3 different clinical scenarios were presented to the RTs. They were then asked to manually ventilate the lung model accordingly.

Clinical Scenario 1. Load RC, $n = 10$. "A 25-year-old man, 70 kg, with a history of bronchial asthma, was admitted to the intensive care unit last night because of acute severe asthma. The patient was started on intravenous methylprednisolone, continuous albuterol nebulizer, and helium-oxygen mixture. This morning the patient feels worse and is getting tired. His arterial blood gas values are pH 7.28, P_{aCO_2} 51 mm Hg, and P_{aO_2} 69 mm Hg. The patient is sedated and intubated but the ventilator is malfunctioning. You are asked to manually ventilate the patient until another mechanical ventilator is brought to the bedside."

Clinical Scenario 2. Load rc, $n = 10$. "A 23-year-old man was brought by his friend to the emergency department after he was found unresponsive in his dormitory room. The patient has overdosed on narcotics. His arterial blood gas values are pH 7.12, P_{aCO_2} 90 mm Hg, and P_{aO_2} 71 mm Hg. The patient was intubated. You are asked to manually ventilate the patient until a ventilator is set up."

Clinical Scenario 3. Load Rc, $n = 7$. "A 65-year-old man, 70 kg, with a history of chronic obstructive pulmonary disease, was admitted a week ago to the hospital because of sepsis. The patient was intubated when he developed respiratory distress secondary to acute respiratory distress syndrome. The patient is sedated and being ventilated with a low-tidal-volume strategy. He is receiving positive end-expiratory pressure of 10 cm H_2O. The fraction of inspired oxygen is 0.6. His blood oxygen saturation dropped to nearly 80% as he was turned in bed for nursing care, and the saturation did not improve when the fraction of inspired oxygen was increased to 1. You decide to manually ventilate the patient in an effort to improve his oxygenation."

Data Analysis

The 40-second recordings of pressure and flow were divided into individual breaths. Flow was numerically integrated to produce a volume signal for each breath, after subtraction of a small constant to correct for baseline drift. P_{peak}, V_T, and f were then determined for each breath and averaged. One-way analysis of variance with Bonferroni correction was used to test for the effects of load or clinical scenario on each of the measured variables. Differences were considered statistically significant when $p < 0.05$.

Results

Figure 1 shows the mean P_{peak}, V_T, and f obtained from each RT in the first part of the study, in which the lung model was randomly rotated through the loads rC, RC, rc, and Rc. Only P_{peak} varied significantly between the 4 loads. The differences in V_T and f between the different loads were nonsignificant. However, all the variables showed substantial differences between the different RTs at any
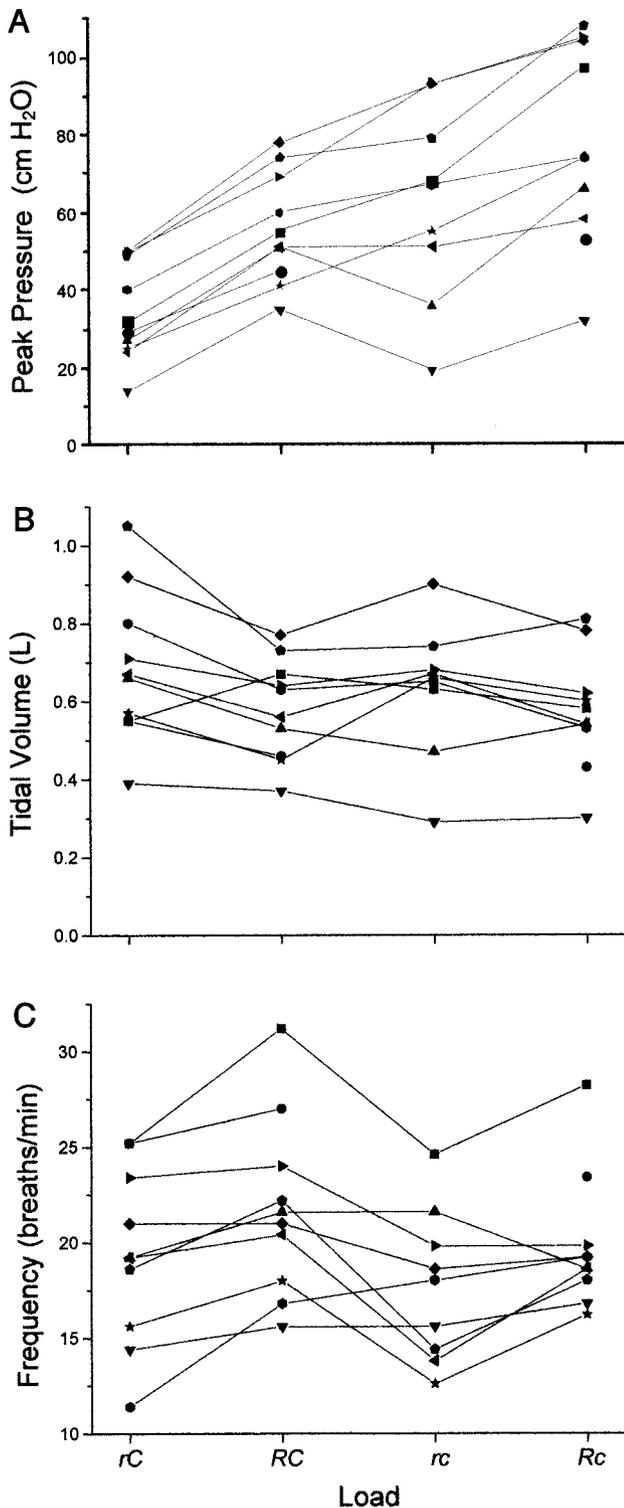
Fig. 1. Peak pressure, tidal volume, and respiratory frequency during manual ventilation of a lung model, with 4 different resistance-compliance load combinations: rC = normal resistance with normal compliance; RC = high resistance with normal compliance; rc = normal resistance with low compliance; Rc = high resistance with low compliance (see text). Each set of connected points represents the data from a single respiratory therapist.

particular load, despite that the RTs received identical patient information. In particular, some of the RTs generated P_{peak} of > 100 cm H_2O (see Fig. 1A). This difference in performance may be partly explained by differences in physical strength, as the male RTs produced higher P_{peak} than the female RTs. With load Rc the mean $\pm$ SD P_{peak} was 91 ± 20 cm H_2O among the 6 male RTs, versus 56 ± 18 cm H_2O among the 4 female RTs.

Figure 2 shows the P_{peak}, V_T, and f values obtained in the second part of the study, averaged across all the RTs, when the lung model was ventilated according to the 3 hypothetical clinical scenarios. Again, P_{peak} significantly depended on load, but V_T and f did not.

Discussion

This study was motivated by our clinical impression that manual ventilation is sometimes used as a supplement to conventional mechanical ventilation to transiently improve oxygenation of patients with lung injury—a notion that is supported by the literature.[3,4] We suspected that the greater efficacy of manual ventilation might be due to higher airway pressure. Our results suggest that might be the case. While manually ventilating various simulated lung pathologies the RTs maintained both V_T and f (see Figs. 1B, 1C, 2B, and 2C), despite a doubling of P_{peak} (see Figs. 1A and 2A). Thus, the RTs clearly targeted a particular minute ventilation, regardless of the mechanical load they achieved from manual bagging. In addition to the high P_{peak} this produced, the constant f may also be problematic in situations of high airway resistance, in which intrinsic positive end-expiratory pressure can develop, with its attendant adverse consequences for hemodynamics. In any case, some of the P_{peak} values we recorded were well over 100 cm H_2O (see Figs. 1A and 2A), which is much higher than the currently recommended limits for P_{peak} (40 cm H_2O) and plateau pressure (32 cm H_2O) for mechanically ventilated patients with acute lung injury/acute respiratory distress syndrome.[5,6] Alveolar recruitment maneuvers in the injured lung have had mixed success[7–10] and may require the application of pressure considerably greater than 40 cm H_2O.[9] This suggests that the extremely high airway pressure generated during short periods of manual ventilation may be responsible for improving oxygenation beyond that achieved by the ventilator.

Our results, however, raise another important issue. High airway pressure can cause lung overdistension that may be damaging if the pressure is transmitted through to the alveoli. A high airway resistance will protect against this, as with the load RC. However, when lung disease is predominately restrictive, the maintenance of a fixed V_T can lead to high pressure being applied to the delicate alveolar tissues, which increases the risk of alveolar injury. For example, with load Rc, the V_T ranged from 0.3 mL to 0.8
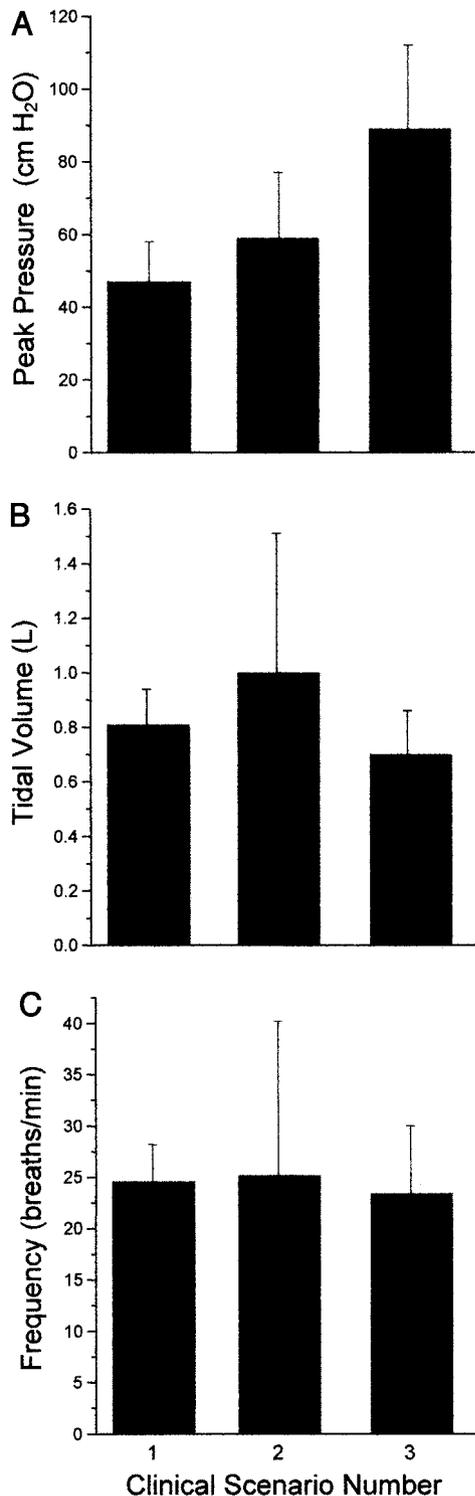
Fig. 2. Peak pressure, tidal volume, and respiratory frequency during manual ventilation of a lung model, with 3 resistance-compliance load combinations, corresponding to 3 different clinical scenarios (see text).

mL, which, with a compliance of 0.012 L/cm H_2O, produces peak alveolar pressure of 26–68 cm H_2O. Those pressures could be particularly dangerous when the lung is already injured. Indeed, the results of a recent large randomized clinical trial indicate that lung injury is reduced when overdistention is limited by the use of lower V_T,[6] though, admittedly, that study concerned longer-term mechanical ventilation, for which the phenomenon of ventilator-induced lung injury is well described.[11]

By contrast, little is known about the impact of recruitment maneuvers on the development or exacerbation of lung injury. The potential for manual ventilation to exacerbate lung injury is thus a subject for further investigation. Few other investigators appear to have considered this issue. Clarke et al[12] examined P_{peak} in sedated, ventilated patients and found P_{peak} of 37–74 cm H_2O, which is not as high as in the present study, but still higher than is considered acceptable for mechanical ventilation. A case of pneumoperitoneum associated with manual ventilation has also been reported.[13] The small number of other studies reported on manual ventilation in the adult population deal largely with issues of technique.[1–4,12,14]

We have based our discussion so far on the P_{peak} measured proximal to the resistive element in the lung model, which corresponds to the pressure at the airway opening of a patient, which is also readily obtainable. Of course, what really matters in terms of the risk of lung injury during ventilation is the peak transpulmonary pressure that is generated.[15] Peak transpulmonary pressure can be considerably less than P_{peak} when central airway resistance is high, so patients with severe airway obstruction may be relatively protected against the dangers of barotrauma. On the other hand, patients with restrictive disease or large time-constant differences throughout the lung could be at considerable risk. If the airway pressures commonly applied to the lungs during manual ventilation are injurious, then strategies can be implemented that limit the pressure delivered, such as providing the operator with a real-time measurement of the pressure he or she is generating,[16,17] or by replacing the operator with a controlled mechanical pump.[2] However, either strategy would probably reduce the purported advantages of manual ventilation by reducing its ability to open recalcitrant regions of closed lung.

Conclusion

Although our results indicate that high airway pressure during manual ventilation may be problematic, our study is only an initial examination of this issue under the contrived conditions of a lung model. It may be that experienced RTs modulate manual ventilation of real patients on the basis of subtle clinical indicators that we have not considered. Consequently, this investigation now needs to be taken to the bedside so that we can learn how to balance

the recruitment of closed lung units against the risk of barotrauma.

REFERENCES

1. Clayton TJ, Pittman JA, Gabbott DA. A comparison of two techniques for manual ventilation of the lungs by non-anaesthetists: the bag-valve-facemask and the cuffed oropharyngeal airway (COPA) apparatus. Anaesthesia 2001;56(8):756–759.

2. Terndrup TE, Cherry RA, McCabe JB. Comparison of ventilation performance: standard resuscitation bag and the resuscitation bag controller. J Emerg Med 1990;8(2):121–125.

3. Maxwell L, Ellis ER. The effects of three manual hyperinflation techniques on pattern of ventilation in a test lung model. Anaesth Intensive Care 2002;30(3):283–288.

4. Patman S, Jenkins S, Stiller K. Manual hyperinflation: effects on respiratory parameters. Physiother Res Int 2000;5(3):157–171.

5. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338(6):347–354.

6. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000;342(18):1301–1308.

7. Patroniti N, Foti G, Cortinovis B, Maggioni E, Bigatello LM, Cereda M, Pesenti A. Sigh improves gas exchange and lung volume in patients with acute respiratory distress syndrome undergoing pressure support ventilation. Anesthesiology 2002;96(4):788–794.

8. Richard JC, Maggiore SM, Jonson B, Mancebo J, Lemaire F, Brochard L. Influence of tidal volume on alveolar recruitment: respective role of PEEP and a recruitment maneuver. Am J Respir Crit Care Med 2001;163(7):1609–1613.

9. Villagra A, Ochagavia A, Vatua S, Murias G, Del Mar Fernandez M, Lopez Aguilar J, et al. Recruitment maneuvers during lung protective ventilation in acute respiratory distress syndrome. Am J Respir Crit Care Med 2002;165(2):165–170.

10. Brower RG, Morris A, MacIntyre N, Matthay MA, Hayden D, Thompson T, et al. Effects of recruitment maneuvers in patients with acute lung injury and acute respiratory distress syndrome ventilated with high positive end-expiratory pressure. Crit Care Med 2003;31(11):2592–2597.

11. Dreyfuss D, Saumon G. Ventilator-induced lung injury: lessons from experimental studies. Am J Respir Crit Care Med 1998;157(1):294–323.

12. Clarke RC, Kelly BE, Convery PN, Fee JP. Ventilatory characteristics in mechanically ventilated patients during manual hyperventilation for chest physiotherapy. Anaesthesia 1999;54(10):936–940.

13. Lopez Rodriguez A, Lopez Sanchez L, Julia J. Pneumoperitoneum associated with manual ventilation using a bag-valve device (letter). Acad Emerg Med 1995;2(10):944.

14. Patman S, Jenkins S, Smith K. Manual hyperinflation: consistency and modification of the technique by physiotherapists. Physiother Res Int 2001;6(2):106–117.

15. Ricard JD. Barotrauma during mechanical ventilation: why aren't we seeing any more? Intensive Care Med 2004;30(4):533–535.

16. Yetsko D, Damian C, Gentile M. Use of a pressure manometer during manual ventilation: a bench study. Respiratory abstracts. Toronto: Cardinal Health, 2002. Available at http://www.cardinal.com/mps/focus/respiratory. Accessed January 7, 2005.

17. Redfern J, Ellis E, Holmes W. The use of a pressure manometer enhances student physiotherapists' performance during manual hyperinflation. Aust J Physiother 2001;47(2):121–131.