A Comparison of Demand-valve and Bag–Valve Ventilations in a Swine Pneumothorax Model

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Abstract. **Objective:** Two means of delivering artificial ventilation readily available to out-of-hospital personnel are the bag–valve (BV) and the O_2-powered demand-valve (OPDV). However, use of the OPDV has been limited because of concerns that it may worsen an underlying pneumothorax. This study compared the changes in size of pneumothorax in swine ventilated with the 2 devices. **Methods:** Three swine were anesthetized, intubated, and instrumented with a femoral arterial line and a pediatric Swan-Ganz catheter. A chest tube was placed, the chest was opened, and the lung parenchyma was visualized. The lung was disrupted by a single stab with a #10 scalpel; the chest was then sealed; and a pneumothorax was created by injecting 30 mL of air through the chest tube. The animals were ventilated by 12 emergency medical technicians using either BV or OPDV. After 10 minutes of ventilation, the pneumothorax volume was measured. **Results:** When comparing final pneumothorax volumes after 10 minutes of ventilation with the 2 devices, there was no significant difference (mean ± SD = 40.8 ± 28.2 mL vs 52.3 ± 23.1 mL, p = 0.286). **Conclusion:** There is no difference in final pneumothorax volumes after OPDV or BV ventilation. **Key words:** ventilation; pneumothorax; assisted ventilation; bag–valve; demand valve; emergency medical services. ACADEMIC EMERGENCY MEDICINE 1998; 5:977–981

MANAGEMENT of the critically injured trauma patient may include endotracheal intubation and artificial ventilation.[1] Two devices are readily available to the out-of-hospital provider for administering artificial ventilations: the bag–valve (BV) and the O_2-powered demand-valve (OPDV). Historically, the use of the OPDV in the trauma patient has been limited, primarily because of the perceived increased risk of barotrauma resulting in the worsening of an underlying pneumothorax,[2–5] and because of the inability to assess ventilatory compliance.[6]

Recent in-vitro studies have demonstrated that OPDV ventilation does not result in greater tidal volume, peak airway pressure, or maximum airway pressure when compared with BV ventilation,[5] and that OPDV may result in less gastric distention than BV in nonintubated patients.[4] Further, in an examination of the effects of both BV and OPDV on the size of a closed pneumothorax in an animal model, Murray et al. found no significant difference in pneumothorax size between the 2 ventilation methods. This study, however, had significant limitations: A relatively large initial pneumothorax was introduced (15–20 mL/kg); the size of the lung injury was small; and all of the ventilations were performed by one of the investigators.[7]

This study was designed to assess the effects of BV and OPDV ventilations by out-of-hospital providers on an intubated patient with an underlying lung injury and pneumothorax. We hypothesized that there would be no significant increase in the size of pneumothorax using either device.

METHODS

Study Design. Four female swine ranging from 26 to 40 kg were used as the animal model for this study. Each animal was sedated with IM xylazine (5 mg/kg) and telazol (0.5 mg/kg). An IV catheter was placed in an ear vein, and anesthesia was induced and maintained with sodium pentobarbital through this peripheral IV site. The animal was orally intubated using a 7.0 cuffed endotracheal tube (ETT). Tube placement was confirmed by auscultation of breath sounds, and the ETT was secured and connected to a large animal ventilator (Harvard Apparatus, South Natick, MA) on room air (FiO_2 0.21). A femoral arterial line was established for measurement of blood pressure (BP) and arterial blood gases (ABGs). A pediatric Swan-Ganz catheter with cardiac output (CO) capability was placed in the femoral vein and positioned by

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waveform analysis. CO was measured by thermodilution saline infusion. A right-sided chest tube was placed, using a 12-fr Salem sump tube (Sherwood Medical, St. Louis, MO) as a chest tube, through an approximately 2.5-cm incision in the right axillary line. The tube was directed anteriorly and superiorly and secured in place. The incision was then sealed with a purse string suture and covered with petroleum jelly. To induce right chest injury, an approximately 10-cm incision was made on the right anterior chest. Using a small pair of rib spreaders, direct visualization of the lung parenchyma was obtained. A #10 scalpel blade was inserted into the lung parenchyma in a single stabbing motion to the depth of the cutting surface. The chest wall incision was then sutured closed in multiple layers to ensure a good seal and covered with petroleum jelly. A 10-minute observation period took place after all instrumentation.

Population and Setting. For the artificial ventilation trials, 16 emergency medical technicians (EMTs) trained in ETT placement and ventilation were recruited from local emergency medical services (EMS) agencies. The EMTs were not informed of the purpose of the study or the presence of the pneumothorax, but rather were asked to ventilate the animal as one would ventilate a trauma patient suspected of having chest injuries necessitating artificial ventilation. The participants were allowed to ventilate the animal at whatever rate and volume they believed appropriate.

This study was approved by the institution's institutional review board and the animal use committee. The requirement for written informed consent was waived. The care and handling of all animals were in accord with NIH guidelines.

Study Protocol. Four EMTs were assigned to ventilate each animal. Each EMT ventilated the animal for two 10-minute trials; one trial with OPDV (Life Support Products, Irvine, CA), one trial with BV (Pulmonex BIRD, Dallas, TX). The order in which the devices were used was alternated.

Any air present in the chest cavity before each trial was aspirated using a 60-mL syringe. Then, to induce pneumothorax, 30 mL of air was introduced through the chest tube and the chest tube was sealed. The participants then ventilated the animal for 10 minutes. BP and ABGs were followed throughout the trials. After each 10-minute trial, all air in the pleural space was aspirated from the chest tube and measured. Following each 10-minute trial, the animal was rested on the ventilator at room air for 10 minutes. The process was then repeated: All air was again aspirated from the chest tube; 30 mL of air was instilled; the chest tube was resealed; and the next trial was initiated. The process was performed in the same manner for each trial. The model and the method of creating and subsequently measuring the pneumothorax were similar to those described by Murray et al.,[7] and are based on the work of Seaberg et al.[8] and Eger and Saidman.[9]

Data Analysis. Group values are reported as mean ± SD. The change in pneumothorax size in the BV and the OPDV trials was analyzed using paired t-test. Differences between final pneumothorax volumes for the BV and the OPDV trials were compared using Student's t-test. Analysis of variance (ANOVA) was used to determine differences in blood gas and hemodynamic variables for OPDV, BV, and control (ventilator) measurements. ANOVA was also used to detect differences in variables of interests between animals and between participants. All statistical analysis was performed using SAS Statistical Analysis Software (SAS Institute, Cary NC).

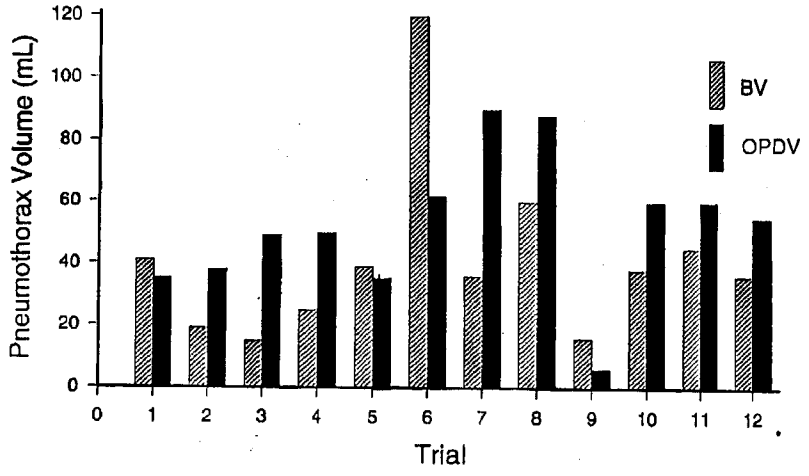

Figure 1. Actual final pneumothorax volume for each pair of trials. BV = bag–valve; OPDV = O_2-powered demand-valve.

This design was intended to result in 16 trials with each device, providing a power of 90% to detect a 50% (15-mL) change in pneumothorax size.

RESULTS

One of the animals suffered cardiac arrest and died during the experiment. A veterinarian examined this animal after death, and found it to have a pneumomediastinum with significant bullous lung disease bilaterally. All of the data for this animal were subsequently excluded. Thus, data for 12 trials with each device are included in the analysis.

After 10 minutes of ventilation with the BV, the mean increase in pneumothorax size was 10.8 ± 8.1 mL (95% CI: −7.06 to 28.72 mL, paired t-test, p = 0.230). After 10 minutes of ventilation with OPDV, the mean increase in pneumothorax size was 23.3 ± 14.7 mL (95% CI: 8.64 to 39.02 mL, paired t-test, p = 0.006). The mean final pneumothorax volume after 10 minutes of BV ventilation was 40.8 ± 28.2 mL (95% CI: 24.5 to 57.1 mL). The mean final pneumothorax volume after 10 minutes of ventilation with OPDV was 52.3 ± 23.1 mL (95% CI: 39.0 to 65.6 mL). There was no significant difference between the final pneumothorax volumes following ventilation with the 2 devices (Student's t-test, p = 0.286).

There was no difference in the size of pneumothorax created when comparing the different EMTs, the sequences of BV and OPDV use, or the orders of the participant (i.e., first participant compared with last participant). There was a significantly greater increase in pneumothorax size in one of the animals when compared with the other two (ANOVA, p = 0.021); however, this was consistent across all trials involving that animal. The actual final pneumothorax volumes for each trial are shown in Figure 1.

There was no significant difference in the hemodynamic profiles of the animals when comparing ventilation by OPDV, BV, and the ventilator (Fig. 2). For ABGs, both OPDV and BV resulted in higher PaO_2 concentrations than the control, and BV ventilation resulted in lower $PvCO_2$ than OPDV or the control. There was no difference in $PaCO_2$ or PvO_2 (Fig. 3). Both OPDV and BV resulted in higher peak inspiratory pressures than the control, but the peak inspiratory pressures created by BV and OPDV did not differ (Fig. 4).

DISCUSSION

Only recently has OPDV ventilation been subjected to scientific investigation. In 1994, Menezi and Winslow used a test lung to demonstrate that OPDV ventilation did not result in increased intrapleural pressures or tidal volumes compared

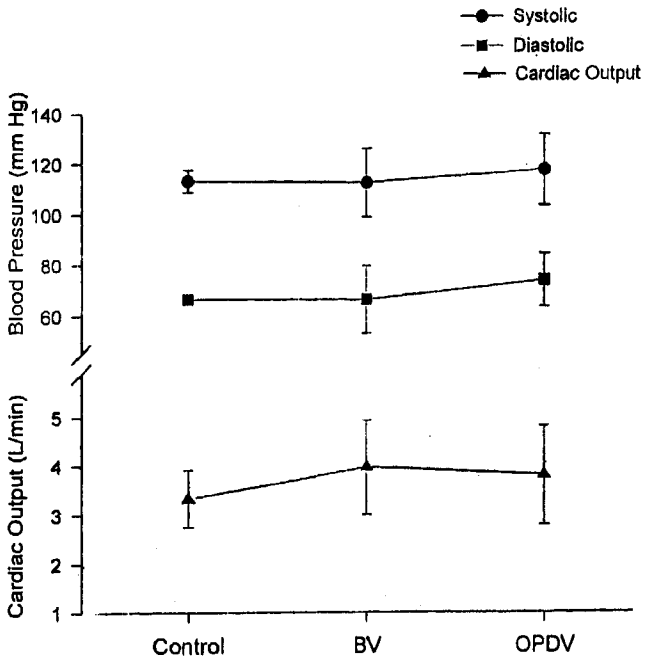

Figure 2. Hemodynamic parameters for each experimental condition. Values are mean ± SD. BV = bag–valve; OPDV = O_2-powered demand-valve.

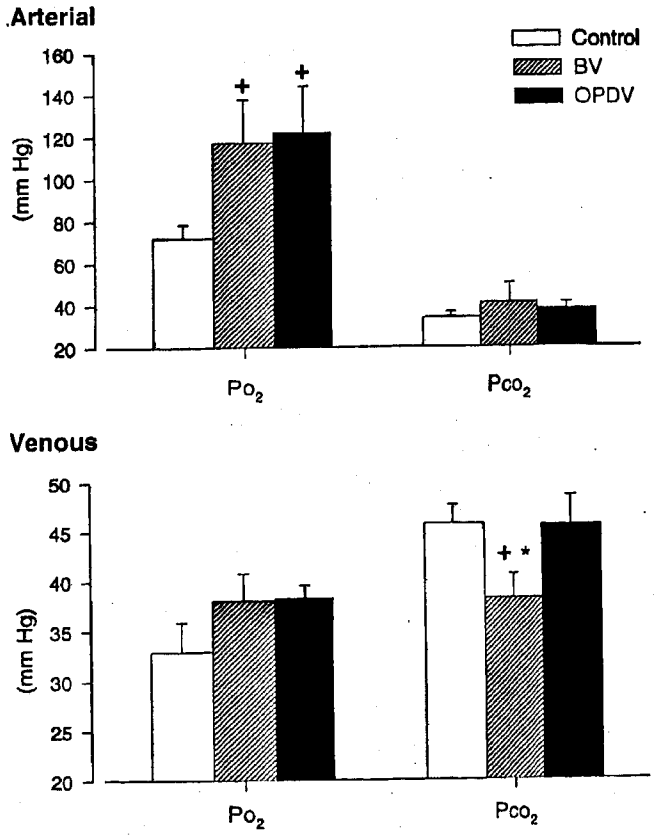

Figure 3. Blood gas analysis for each experimental condition. Values are mean ± SD. BV = bag–valve; OPDV = O_2-powered demand-valve. + Significantly different from controls (ventilation); * significantly different from OPVD.

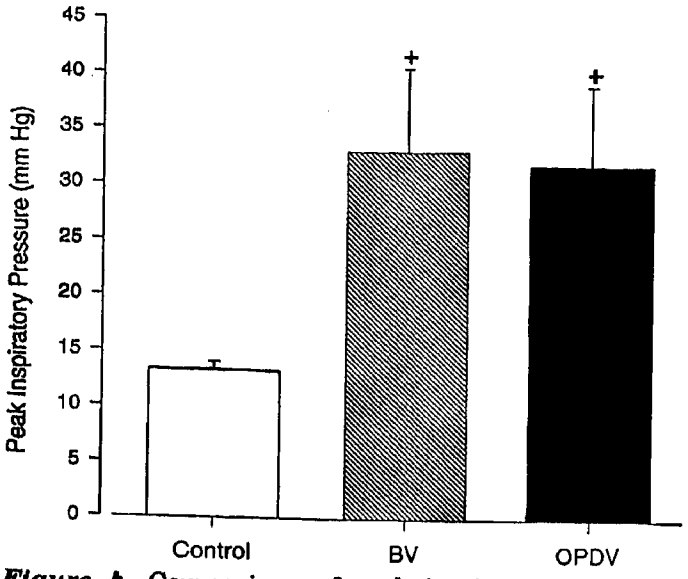

Figure 4. Comparison of peak inspiratory pressures during ventilation with bag–valve (BV), O_2-powered demand-valve (OPDV), and control (ventilator). * significantly different from control (ventilator).

with BV ventilation in nonintubated patients.[4] Mosesso et al. used the same test lung to demonstrate that tidal volumes and airway pressures are not significantly increased when the OPDV is used for intubated patients.[5] Building on this work, Murray et al. compared OPDV and BV ventilations in a swine pneumothorax model.[7] That work provided the framework for our investigation.

Although we found a significant increase in pneumothorax volume following OPDV ventilation, we did not find any difference in final pneumothorax volume when comparing BV and OPDV. These results are similar to the findings of Murray and colleagues, who found no difference in the sizes of pneumothorax when ventilations were performed with either BV or OPDV. That study was limited, though, by the introduction of a relatively large initial pneumothorax (approximately 15–20 mL/kg); a small lung injury (established with a 14-gauge needle); and the fact that all of the ventilations were performed by one of the investigators.

In our trial, we induced a smaller initial pneumothorax. Total lung volume in swine is estimated to be 55 mL/kg[10]: our initial pneumothorax affected approximately 2% of total lung volume. Also, we used out-of-hospital providers to perform the ventilations. We believe the OPDV is an out-of-hospital tool, and that EMS personnel are more appropriate study participants. We also believe that, by blinding them to the purpose of the study, we are more likely to have obtained objective data. Further, the EMTs were not given an exact rate or tidal volume at which to ventilate the animal (as in the study by Murray et al.), allowing the animals to be treated more like actual patients.

We found no significant change in hemodynamic measurements during ventilation with either device; thus, the clinical import of these changes in pneumothorax size remains unclear. However, this was an animal model using swine, and there is some question as to whether a swine model capable of consistently creating a tension pneumothorax exists.[7,10] Barton et al. has described a swine tension pneumothorax model using a balloon occlusion catheter, but that model required displacement of nearly 60% of total lung capacity before significant compressive effects were observed.[10]

Limitations and Future Questions

Limitations of this study include the use of the animal model. Swine do not usually develop tension pneumothorax because of their mediastinal anatomy. Also, although we kept the size of injury the same for all animals (the cutting surface of a #10 scalpel blade), the extent of injury depended more on the underlying structures (large vs small airways) that were damaged. We chose this injury in an attempt to better simulate the air leak in chest trauma. However, this type of injury is not well controlled. We would argue, though, that chest injuries among patients also differ for the same reasons.

Our model was also limited to a single injury: a parenchymal defect in the right lung. We would expect most trauma patients with chest injuries to have other injuries. It is impossible to predict what the additive effect of a worsening pneumothorax with other injuries might be, but the combination certainly could result in hemodynamic compromise. Another limitation of our single-injury model is that the injury could have been occluded by progressive clotting over the course of the study, which would have reduced or prevented air leak as the study progressed. The use of a large injury was intended to provide for more consistent air leak, which Murray et al. had discussed as an important factor. Further, we found no significant difference in the volume of pneumothorax when comparing the first trial for each animal with the last trial for each animal, or in any of the "sequence" comparisons. This suggests that the amount of air leak was consistent throughout the course of the trials.

The reliability of measuring pneumothorax volume by aspirating air through the chest tube may be questionable. This was the technique described by Murray and colleagues, and it was based on work by Seaberg et al.[8] examining the effects of nitrous oxide on pneumothorax size. We know of no other reasonable way to measure pneumothorax volume in a large animal study of this design.

We did not control for the training or experience

of the EMTs. Although all had been trained in intubation and artificial ventilation, and all had at least 2 years of EMS experience, many had not previously used the OPDV in actual patients.

Finally, only 3 animals were included in the analysis for this study. The fourth animal died halfway through one of the trials, and was found to have significant lung disease. All of the data for that animal were excluded from the analysis. It is unclear whether inclusion of those data, or the inclusion of more trials in general, would alter our results.

In future studies, attempts should be made to better control the parenchymal injury. The use of a stint or a catheter that is left in place might alleviate concerns about clotting at the injury site. Trials involving both larger and multiple injuries might also be worthwhile. Repeating this study in a model that reliably develops tension and the associated cardiopulmonary compromise would be important; however, we are unfamiliar with any such model. Ultimately, randomized, controlled clinical trials may be necessary to demonstrate that demand-valve ventilation is as safe as bag–valve ventilation.

CONCLUSION

There is no difference in the final volumes of pneumothorax when out-of-hospital personnel perform ventilations with an OPDV or a BV. Neither device caused clinically significant changes in our model.

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