Clinical Management Strategies for Airway Pressure Release Ventilation: A Survey of Clinical Practice

Andrew G Miller RRT-ACCS RRT-NPS, Michael A Gentile RRT FAARC, John D Davies MA RRT FAARC, and Neil R MacIntyre MD FAARC

BACKGROUND: Airway pressure release ventilation (APRV) is a commonly used mode of ventilation designed to increase mean airway pressure and thus oxygenation. Different strategies for clinical management have been described in the literature but are largely based on physiologic concepts, animal data, and small clinical trials. The purpose of this study was to determine how APRV is currently managed by surveying practicing respiratory therapists with experience using APRV. METHODS: A 15-item survey was developed by the authors and posted on the AARConnect online media platform in January 2016 after being declared exempt by our institution's institutional review board. Survey questions were derived from a literature review of recommended APRV settings. Responses were limited to one per institution. RESULTS: The survey was completed by 60 respondents who used APRV. Of the 4 key initial APRV settings (P high, P low, T high, and T low), there was good agreement among survey responders and published guidelines for setting initial T high (4–6 s) and initial P low (0 cm H_2O). There was some disagreement regarding initial P high, with 48% of responders matching P high to conventional ventilation plateau pressures but another 31% using conventional ventilation mean airway pressure plus 2–5 cm H_2O. The most disagreement was with the T low setting, with only 47% of survey responders agreeing with published guidelines about using the expiratory flow signal to set T low. There was good agreement among survey responders and published guidelines for what changes to make when gas exchange was outside of the targeted range. A substantial number of respondents accepted P high and APRV release volumes that may exceed lung-protective limits. CONCLUSIONS: There is only limited consensus among practitioners for initial APRV settings, probably reflecting the paucity of good clinical outcome data and confusion surrounding the physiology of this mode. *Key words: APRV, airway-pressure release ventilation* [Respir Care 2017;62(10):1264–1268. © 2017 Daedalus Enterprises]

Introduction

Airway pressure release ventilation (APRV) is a mode of mechanical ventilation originally described as CPAP with an intermittent release phase configured with inspiratory time greater than expiratory time. It can also be described as pressure control intermittent mandatory ventilation with inverse ratio of inspiratory time to expiratory time (I:E) and unrestricted spontaneous breathing.[1,2] Conceptually, APRV is a form of open lung ventilation designed to increase mean airway pressure without increasing set PEEP, set driving pressure, or tidal volume in patients with acute lung injury, ARDS, postoperative at-

Mr Miller is affiliated with the Respiratory Therapy Department, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania. Mr Gentile and Dr MacIntyre are affiliated with Duke University Medical Center, Durham, North Carolina. Mr Davies is affiliated with Duke University School of Medicine, Durham, North Carolina.

Supplementary material related to this paper is available at http://www.rcjournal.com.

This work was supported by departmental funding. Dr MacIntyre discloses relationships with InspiRx Pharmaceuticals, Breathe Technologies, Ventec Life Support, Alana Healthcare, and Pulmonx. Mr Davies discloses a relationship with ResMed. Mr Gentile discloses relationships with Medical Dynamics and Draeger. Mr Miller has no conflicts to disclose.

Correspondence: Neil R MacIntyre MD FAARC, Duke University Medical Center, Respiratory Care Services, PO Box 3911, 2301 Erwin Road, Durham NC 27710. E-mail: neil.macintyre@duke.edu.

DOI: 10.4187/respcare.05494

electasis, or increased chest wall elastance. To date, some reports,[2-8] but not all,[9] have found conventional mechanical ventilation and APRV to have similar safety profiles. When compared with conventional strategies, small randomized trials and a larger propensity-matched study indicate that APRV may improve oxygenation (as measured by P_{aO_2}/F_{IO_2}) but appears to have no effect on mortality or other patient-oriented outcomes.[3-8] Despite the lack of high-level evidence that APRV improves important patient oriented outcomes, APRV is used in many ICUs around the world.[3]

Although APRV settings are similar in concept to those used during conventional mechanical ventilation, there are important technical differences. The 4 key APRV settings are: (1) lung inflation pressure or P high; (2) lung inflation time or T high; (3) lung deflation pressure or P low; and (4) lung deflation time or T low. Importantly, APRV is usually set with a long T high/short T low to create an inverse I:E. Also, during T high, spontaneous breathing (with or without pressure support) can occur.

Specific management strategies to set these 4 parameters have been described in the literature.[10,11] However, these strategies are largely based on theoretical considerations, animal studies, and small clinical trials rather than large randomized trials. Moreover, how APRV is actually being managed at the bedside is generally unknown. The purpose of this study was to examine APRV usage and clinical management strategies through the use of a survey instrument administered to practicing respiratory therapists.

QUICK LOOK

Current knowledge

Airway pressure release ventilation (APRV) is a commonly used mode of ventilation designed to increase mean airway pressure and thus oxygenation. Different strategies for clinical management have been described in the literature but are largely based on physiologic concepts, animal data, and small observational trials. How APRV is being managed at the bedside in different institutions is largely unknown.

What this paper contributes to our knowledge

This survey highlights the lack of consensus among practitioners regarding initial APRV settings, probably reflecting the paucity of good clinical outcome data and confusion surrounding the physiology of this mode. In addition, some respondents reported APRV settings outside of current lung-protective standards, reflecting an underappreciation of the potential harm that can result from APRV.

Methods

A survey instrument relating to APRV clinical management strategies was developed by the authors and declared exempt by our institution's institutional review board. The survey was developed after reviewing the published literature and included clinical management choices included in recommendations from Habashi[10] and Modrykamien et al.[11] The initial survey instrument was tested on 144 personal contacts to refine the questions and clarify ambiguities. After receiving approval from the AARC Board of Directors, the final survey was posted on the AARConnect listserv on January 4, 2016. Reminder posts were posted weekly for 4 weeks. Participants were limited to one per institution to eliminate institutional bias.

The survey consisted of 15 questions (see the supplementary materials at http://www.rcjournal.com), which related to hospital demographics, mechanical ventilation protocol use, rescue modes used, and APRV management strategies. CIs were calculated using the modified Wald method to underscore that the results lack precision.[12]

Results

There were a total of 60 responses from hospitals that used APRV. Respondent and hospital demographic data are summarized in Table 1. In these hospitals, proning capabilities existed in 71%, high-frequency oscillators were available in 34%, and extracorporeal membrane oxygenation was available in 49%. Nevertheless, 74% of these hospitals used APRV as the initial rescue strategy for patients failing conventional ventilation. APRV was managed via institutional protocol in 48% of centers, and 44% used pressure support during APRV.

Respondents' initial approaches for APRV management are summarized in Table 2. To put these into perspective, the 4 key APRV settings (P high, T high, P low, T low) in our survey response are compared with 2 commonly recommended published strategies from the Cleveland Clinic[11]

Table 1. Respondent and Hospital Demographic Data

Respondent/Hospital	Results	95% CI
Respondent role, *n* (%)		
Director/manager	17 (28)	0.18–0.39
Staff therapist	18 (30)	0.20–0.43
Educator	12 (20)	0.12–0.32
Supervisor	10 (16)	0.09–0.28
Other	3 (4)	0.01–0.14
Demographics		
Number of beds, mean ± SD	461 ± 278	389–533
Number of ICU beds, mean ± SD	63 ± 60	48–79
Trauma center, *n* (%)	29 (48)	0.36–0.61

N = 60.

Table 2. Airway Pressure Release Ventilation Settings

Setting	*n* (%)	95% CI
Initial P high ($n = 48$)		
Equal to plateau pressure on conventional mechanical ventilation	23 (48)	0.34–0.62
2–5 cm H_2O above $\bar{P}_{aw}$ on conventional mechanical ventilation	15 (31)	0.20–0.45
Equal to $\bar{P}_{aw}$ on conventional mechanical ventilation	6 (13)	0.05–0.25
Goal V_T of 6 mL/kg/PBW	2 (4)	0.004–0.15
25 cm H_2O	2 (4)	0.004–0.15
Initial P low ($n = 50$)		
0 cm H_2O	39 (78)	0.65–0.87
2–5 cm H_2O	6 (12)	0.05–0.24
Variable depending on oxygenation	3 (6)	0.01–0.17
Match PEEP on conventional mechanical ventilation	2 (4)	0.003–0.14
T high ($n = 49$)		
4–6 s	32 (65)	0.51–0.77
Desired $\dot{V}_E$ and breathing frequency	5 (10)	0.04–0.22
Desired I:E	5 (10)	0.04–0.22
2–3 s	4 (8)	0.03–0.20
6–8 s	3 (6)	0.01–0.17
T low ($n = 49$)		
Set time (0.2–0.8 s)	19 (39)	0.26–0.53
When expiratory flow equals 56–75% peak expiratory flow	18 (37)	0.24–0.51
When expiratory flow equals 41–55% peak expiratory flow	5 (10)	0.04–0.22
Per desired I:E	5 (10)	0.04–0.22
When expiratory flow equals 25–40% peak expiratory flow	2 (4)	0.004–0.14
During release phase, what is the targeted V_T? ($n = 53$)		
6–8 mL/kg/PBW	20 (38)	0.26–0.51
No limit	19 (36)	0.24–0.49
4–6 mL/kg/PBW	10 (19)	0.10–0.32
8–10 mL/kg/PBW	3 (6)	0.01–0.16
10 mL/kg/PBW	1 (2)	< 0.001–0.11
What is the maximum allowed P high? ($n = 47$)		
35 cm H_2O	21 (45)	0.31–0.59
30 cm H_2O	9 (19)	0.11–0.33
40 cm H_2O	9 (19)	0.11–0.33
No limit	8 (17)	0.09–0.30

$\bar{P}_{aw}$ = mean airway pressure
V_T = tidal volume
PBW = predicted body weight
$\dot{V}_E$ = minute volume
I:E = ratio of inspiratory time to expiratory time

and the University of Maryland Shock Trauma Unit[10] in Table 3. For P high, both published strategies recommend matching the plateau pressure (P_{plat}) from the baseline conventional ventilation settings, but only 48% of our survey responders agreed with that. Another 31% used an initial P high 2–5 cm H_2O above the mean pressure on baseline conventional mechanical ventilation. There was more agreement with P low. Both published strategies recommend 0 cm H_2O, and 78% of the survey responders agreed. There was also substantial agreement with initial settings for T high. Both published strategies recommended 4 or 4–6 s, and 65% of the survey responders agreed. Of note is that 20% of the survey responders felt that minute ventilation and I:E should also be taken into account for setting T high. Substantial variability in our results was seen in the approach to T low. Both published strategies recommend setting T low using the expiratory flow signal to ensure that substantial auto-PEEP is generated, one recommending setting T low to end when 40% of the peak expiratory flow is reached and the other recommending setting T low to end when 50–75% of the peak expiratory flow is reached. In contrast, only 51% of our survey responders used expiratory flow criteria to set T low (37% using 56–75% of peak expiratory flow), whereas 39% used a fixed T low (0.2–0.8 s) when using APRV.

For changes made when gas exchange is below targets for oxygenation or ventilation, our survey responders were remarkably consistent and in general agreement with published recommendations.[10,11] To improve ventilation, P high would be increased to increase tidal volume by 98%

Table 3. Initial Recommendations Versus Survey Results

Setting	Cleveland Clinic[11]	University of Maryland Shock Trauma[10]	Current Survey
Initial P high	P_{plat} on conventional mechanical ventilation	P_{plat} on conventional mechanical ventilation	P_{plat} on conventional mechanical ventilation (48%); 2–5 cm > $\bar{P}_{aw}$ on conventional mechanical ventilation (31%)
Initial P low	0 cm H_2O	0 cm H_2O	0 cm H_2O (78%); 2–5 cm H_2O (12%)
Initial T high	4 s	4–6 s	4–6 s (65%); $\dot{V}_E$ or I:E (20%)
Initial T low	40% of peak flow	50–75% of peak flow	Set time without regard to peak flow (39%); 56–75% of peak flow (37%)

P_{plat} = plateau pressure
$\bar{P}_{aw}$ = mean airway pressure
$\dot{V}_E$ = minute volume
I:E ratio = ratio of inspiratory time to expiratory time

of our survey responders, and T high would be shortened to increase breathing rate by 90% of our survey responders. Interestingly, 51% of our survey responders would also add pressure support to improve ventilation. To improve oxygenation, again P high would be increased by 94% of our survey responders, whereas increasing the I:E (increase T high/decrease T low) would be done by 78% of our survey responders. One interesting departure from published recommendations[10,11]is that 52% of our survey responders would increase P low to improve oxygenation.

Our survey responders did not always seem concerned about limiting maximal and tidal lung stretch to minimize ventilator-induced lung injury. Thirty-six percent of our survey responders would accept APRV P high settings > 35 cm H_2O, and 44% would accept APRV release volumes > 8 mL/kg (ideal body weight).

Discussion

Our survey results showed that APRV was the first choice for most respondents for patients failing conventional mechanical ventilation. However, there was only modest consensus for initial settings used during APRV among those surveyed. In addition, respondents' initial management strategies often deviated from published guidelines.[10,11]

APRV offers some interesting physiologic features that may have utility in the management of patients with acute respiratory failure. However, APRV is not an on-off switch; it requires manipulation of 4 key variables. At the present time, the optimal way to configure these variables for various clinical scenarios is largely based on physiologic concepts, small trials, and clinical experience rather than on results from large randomized trials. As a consequence, interpreting trials and making recommendations about APRV is problematic.

The most marked variability in survey responses and deviation from APRV published guidelines was in the choice of T low. Both published guidelines[10,11] recommend using expiratory flow analysis to ensure that the set T low creates significant auto-PEEP. However, 39% of our respondents used an arbitrary set time instead. The use of auto-PEEP rather than set PEEP to maintain alveolar recruitment is controversial. Whereas set PEEP distributes to all alveolar units, auto-PEEP is most prominent in alveolar units with slow emptying (high-compliance/high-resistance units) and is least prominent in alveolar units with fast emptying (low-compliance/low-resistance units).[13] Conceptually, this would seem opposite of the clinical goals for PEEP in patients with alveolar inflammation, flooding, or collapse. Clinical trials are clearly needed to sort this out.

A high percentage of respondents appeared to expose patients to potentially harmful end-inspiratory and tidal lung distention. Current standards for lung-protective conventional ventilation are to limit end-inspiratory P_{plat} to < 30 cm H_2O and tidal volume to < 8 mL/kg.[14,15] The 2 published APRV guidelines[10,11] do not specifically address these parameters. Instead, the only recommendation is to limit P high to < 30–35 cm H_2O, a limit that 36% of our respondents were willing to exceed. Importantly, limiting P high is not the same as limiting P_{plat}, and the harm from exceeding P high may be worse than that from exceeding P_{plat}. Specifically, P_{plat}, by definition, is measured in the absence of patient effort and under no-flow conditions. In the setting of nearly normal chest wall mechanics, P_{plat} is an approximation of the end-inspiratory transpulmonary pressure, the actual stretching pressure on lung tissue. In contrast, APRV encourages patient effort and patient-generated flow (often augmented with pressure support) that can take the end-inspiratory trans-pulmonary pressure well above the set P high. Targeting a P high of < 30–35 cm H_2O may thus underestimate the risk of overdistention during APRV. We believe that this important point is often underappreciated by clinicians managing patients receiving APRV.

The published APRV guidelines[10,11] pay little attention to monitoring tidal volumes, a parameter increasingly appreciated as an independent risk factor for ventilator-in-

duced lung injury.[14,15] Reasonable data support generally limiting tidal volumes to < 8 mL/kg (ideal body weight).[14,15] However, 44% of our respondents were willing to exceed this value for the release volume. Importantly, the effective tidal distention of the lung during APRV is not only the release volume but also whatever volume is added during spontaneous breathing (with or without pressure support).

Although we did not specifically address driving pressure in our survey, this is another parameter that could reflect regional tidal alveolar overdistention during APRV.[14] The driving pressure during conventional ventilation is the P_{plat} minus the measured PEEP. However, this is not equivalent to P high minus P low in APRV. This is because of the potential differences in set P high versus a true P_{plat} noted above and because the actual total PEEP in the lungs is the set P low plus the unevenly distributed intentional auto-PEEP. Thus, determining a driving pressure during APRV is clearly problematic and in need of considerable further study before being incorporated into APRV management algorithms.

Our survey has important limitations, and thus results should be interpreted cautiously. The sample size of 60 is small, and the sampling method used carries self-selection bias because only members of the AARConnect Adult Acute Care Section were surveyed. The small sample size also prevented any analysis of regional differences in clinical practice. However, respondents were probably well-versed in APRV, had a special interest in APRV, and were motivated to contribute to our understanding of APRV. Our survey results therefore would seem to provide exploratory data opening a window into the clinical management of APRV.

Another important limitation of our study is simply the nature of a voluntary survey. Questions must be short and few in number to obtain a reasonable response level. Details of the clinical scenario surrounding a question are thus limited, and nuances cannot be explored. Finally, the terminology surrounding APRV is confusing, a situation worsened by manufacturers insisting on proprietary names and non-standardized ways to display settings and monitors.

Conclusions

There is only limited consensus among practitioners for initial APRV settings, probably reflecting the paucity of good clinical outcome data and confusion surrounding the physiology of this mode. In addition, there may be an underappreciation by some practitioners of the potential harm that can result from APRV.

REFERENCES

1. Daoud EG. Airway pressure release ventilation. Ann Thorac Med 2007;2(4):176-179.
2. Daoud EG, Farag HL, Chatburn RL. Airway pressure release ventilation: what do we know? Respir Care 2012;57(2):282-292.
3. González M, Arroliga AC, Frutos-Vivar F, Raymondos K, Esteban A, Putensen C, et al. Airway pressure release ventilation versus assist-control ventilation: a comparative propensity score and international cohort study. Intensive Care Med 2010;36(5):817-827.
4. Maxwell RA, Green JM, Waldrop J, Dart BW, Smith PW, Brooks D, et al. A randomized prospective trial of airway pressure release ventilation and low tidal volume ventilation in adult trauma patients with acute respiratory failure. J Trauma 2010;69(3):501-510; discussion 511.
5. Putensen C, Zech S, Wrigge H, Zinserling J, Stüber F, Von Spiegel T, Mutz N. Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury. Am J Respir Crit Care Med 2001;164(1):43-49.
6. Sydow M, Burchardi H, Ephraim E, Zielmann S, Crozier TA. Long-term effects of two different ventilatory modes on oxygenation in acute lung injury: comparison of airway pressure release ventilation and volume-controlled inverse ratio ventilation. Am J Respir Crit Care Med 1994;149(6):1550-1556.
7. Varpula T, Jousela I, Niemi R, Takkunen O, Pettilä V. Combined effects of prone positioning and airway pressure release ventilation on gas exchange in patients with acute lung injury. Acta Anaesthesiol Scand 2003;47(5):516-524.
8. Varpula T, Valta P, Niemi R, Takkunen O, Hynynen M, Pettilä VV. Airway pressure release ventilation as a primary ventilatory mode in acute respiratory distress syndrome. Acta Anaesthesiol Scand 2004; 48(6):722-731.
9. Sasidhar M, Chatburn RL. Tidal volume variability during airway pressure release ventilation: case summary and theoretical analysis. Respir Care 2012;57(8):1325-1333.
10. Habashi NM. Other approaches to open-lung ventilation: airway pressure release ventilation. Crit Care Med 2005;33(3 Suppl):S228-S240.
11. Modrykamien A, Chatburn RL, Ashton RW. Airway pressure release ventilation: an alternative mode of mechanical ventilation in acute respiratory distress syndrome. Cleve Clin J Med 2011;78(2):101-110.
12. Motulsky H. Intuitive biostatistics : a nonmathematical guide to statistical thinking. New York: Oxford University Press; 2010:32-33.
13. Kacmarek RM, Kirmse M, Nishimura M, Mang H, Kimball WR. The effects of applied vs auto-PEEP on local lung unit pressure and volume in a four-unit lung model. Chest 1995;108(4):1073-1079.
14. Amato MB, Meade MO, Slutsky AS, Brochard L, Costa EL, Schoenfeld DA, et al. Driving pressure and survival in the acute respiratory distress syndrome. N Engl J Med 2015;372(8):747-755.
15. Neto AS, Simonis FD, Barbas CS, Biehl M, Determann RM, Elmer J, et al. Lung-protective ventilation with low tidal volumes and the occurrence of pulmonary complications in patients without acute respiratory distress syndrome: a systematic review and individual patient data analysis. Crit Care Med 2015;43(10):2155-2163.