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Perioperative Ventilation Strategies

Key Points

- We use intraoperative mechanical ventilation very commonly, but inappropriate parameter-setting can expose patients to substantial anaesthetic harm
- Ventilation strategies should prioritise adequate gas exchange and mitigation of the risks associated with mechanical ventilation
- Physiological consequences of mechanical ventilation and risks of ventilator induced trauma are exacerbated by lung pathology
- Individualised ventilation strategies should adapt settings, within accepted limits, and be continuously titrated to avoid disease-specific risks
- Ventilator parameters (rate, volume, and time functions) are related directly, mechanically, and physiologically, so should be changed within an overall strategy

MCQs

1. Regarding ventilatory parameters:

a. $SpO_2 > 98\%$ and $EtCO_2 < 5.5$ should be achieved regardless of ventilator settings

b. Injudicious ventilator parameter selection may result in volu-, baro-, atelecta-, or bio-trauma

c. After parameters have been set at the beginning of the case, further titration is unnecessary

d. Mode and parameter setting should not exceed lung protective ventilation (LPV) limits, except in hypoxia or very poorly compliant lungs

e. Inspiratory time is limited by respiratory rate and I:E ratio

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2. True or False

a. Turning up respiratory rate results in more dead space ventilation relative to alveolar ventilation

b. Compliance does not vary across lung regions

c. a lung unit is an area of parenchyma with similar mechanical properties

d. the time constant (τ), is the time taken for alveolar and central airway pressures reach equilibrium

e. rapid cyclical inflation and deflation due to high respiratory rate predisposes to ventilator induced lung injury (VILI)

3. True or False

a. In Trendelenberg positioning and supine patients with pneumoperitoneum or central adiposity, PEEP must be set to zero

b. Auto PEEP describes the default PEEP value selected by the contemporary theatre ventilators

c. are unrelated to anaesthetic practice

d. ventilator induced lung injury only occurs in the presence of lung pathology

4. Mechanical ventilation in pre-existing pulmonary diseases may result in:

a. dynamic hyperinflation in airflow limitation

b. over-distended alveoli in restrictive lung disease

c. over-distended alveoli in emphysema

d. dynamic hyperinflation and regional overdistended alveoli in COPD

e. pneumothorax due to ruptured bullae in COPD

MCQ Answers 1. FTFTT, 2. TFTFT, 3. FFFF, 4. TTTTT

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Introduction

Mechanical ventilation (MV) is a cornerstone of intraoperative anaesthetic practice but is surprisingly poorly understood. It's clear that one size doesn't fit all, and simply accepting default settings is rarely appropriate. On the other hand, striving to achieve physiological ideals (derived in awake upright patients not undergoing intra-abdominal surgery) can result in volu-, baro-, atelecta-, and bio-trauma (to name a few) and exacerbates existing lung pathologies [1, 2].

Both under-ventilation and over-ventilation predispose to postoperative pulmonary complications (PPCs) and risk significant lung injury, so modern strategies target optimal lung mechanics and more modest parameters (physiological and biochemical) thereby seeking to reduce the anaesthetic contribution to risk of adverse perioperative events [see the 2014 BMJ meta-analysis in BMJ Open by Tao et al.].

Interactions between positive pressure ventilation, pulmonary physio-mechanics and pathological processes are complex. So it's absolutely essential that individual MV parameters are programmed (within accepted limits) as part of an individualised ventilation strategy and titrated against clinical parameters.

Setting Rate and Volume Parameters

Having read the following explanations I hope you'll appreciate that these parameters are interrelated and that changing one setting will usually have an impact on the others. These considerations are particularly important in the context of lung disease and surgical factors (site, patient position, CO_2 inflation etc.), so it's vital that ventilation strategies are individualised considering disease-specific factors.

As you know, minute ventilation ($\tilde{V}_{min}$) is gas flow per minute, calculated as the product of the inspiratory volume, or tidal volume (V_T) and the ventilatory rate. V_T and rate can be readily manipulated and is titrated against physiological and biochemical targets.

Programming the absolute duration of inspiratory and expiratory phases of and their (I:E) ratio is necessarily limited by ventilatory rate. Taking this further, setting inspiratory time in volume targeted modes may determine the magnitude of gas flow delivery, potentially result in excessive system pressures.

MV can be broadly classified as mandatory, assisted or a combination. Rate, volume, and time values must be selected in mandatory modes, although some of these may be automatically determined. In assisted modes, spontaneous inspiration is augmented to a target pressure or volume and a 'backup' rate can be set if additional ventilator-delivered breaths are required. The duration of inspiration and expiration may be programmed in both mandatory and assisted modes.

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Rate

In conventional mandatory ventilation, a rate of 10-20bpm usually provides a $\tilde{V}_{min}$ sufficient to create and maintain favourable trans-alveolar partial pressure gradients in adults. Higher rates may be indicated for some pathologies and are routine for non-adults. Rates in excess of three times the physiological range are used in High Frequency Ventilation and lower rates may be programmed for ventilator weaning and in individuals susceptible to gas-trapping (more below).

Provided that V_T remains constant, increasing ventilatory rate increases alveolar ventilation which may of course be beneficial. But increasing ventilatory rate reduces inspiratory and expiratory times, which may actually worsen gas exchange and predispose to complications in susceptible individuals (more below). And as if that's not reason enough, faster respiratory rates have an additive role in ventilator induced lung injury (VILI) [3], which we are increasingly realising is a consideration in even relatively short periods of MV.

Volume

Of the machine-delivered inspiratory volume (V_T), a proportion will always be unavailable for gas exchange. This 'dead-space' volume comprises physiological (conducting airways and alveoli at which no gas exchange occurs) and apparatus dead space. Whilst the former is relatively fixed, we should minimise the latter to improve efficiency of ventilation. Fresh gas flow ($\tilde{V}_A$) available for exchange in the alveolar compartment is calculated as the product of ventilatory rate and what remains of V_T once dead-space volume is accounted for:

$$\tilde{V}_A = r \cdot (V_T - (V_D + V_{APP}))$$

Equation 1. $\tilde{V}_A$: alveolar ventilation, r: rate, V_T: tidal volume, V_D: physiological dead space volume, V_{APP}: apparatus dead space volume.

Assuming that the distribution of alveolar perfusion approximates $\tilde{V}_A$ (and in the absence of parenchymal disease), O_2 uptake and CO_2 elimination may be improved by increasing alveolar ventilation relative to dead space ventilation. What is less well known is that simply dialling up the rate leads to a proportionally greater increase in dead-space ventilation relative to $\tilde{V}_A$. This is not a sensible strategy.

The most straightforward way to increase tidal volume in positive pressure ventilation (PPV) is to increase inspiratory pressure (paying attention to rate and time variables). But volume change is of course also determined by system compliance which must not be considered to be uniform throughout the lung fields. Beyond the normal physiological variation you know from reading West's Bible, distribution of gas flow and

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inspiratory volume will be asymmetrical in pathologies associated with differential regional compliance. This predisposes to dynamic hyperinflation, over-distension of alveoli in more compliant lung units and exaggeration of physiological gas flow: perfusion ($\tilde{V}_A$:Q) mismatching. See suggested strategies below for more on how to optimise ventilation.

Evidence of the potential harm caused by exposure to MV comes from studies of the pathogenesis of VILI and acute respiratory distress syndrome (ARDS). ARDS probably requires synergism of pulmonary insult and MV in predisposed individuals [4, 5], but VILI can develop in previously normal lungs in as few as 6 hours [4, 6, 7]. Risk factors include exposure to high end-inspiratory volumes (volutrauma) [8], shear stress resulting from cyclical inflation and deflation of differently compliant lung units (atelecta-trauma) and large transpulmonary pressure gradients [9]. So allow enough time for inflation and deflation!

Data (from ARDS trials) suggest that limiting V_T to 4-8ml/kg ideal body weight (IBW) [10, 11] and plateau pressure (a surrogate for transpulmonary pressure) to less than 30-35cmH_2O [10] decreases early morbidity and mortality. But because we know that high inflationary pressures and inspiratory volumes can be harmful over even pretty short periods [12], lung protective ventilation should be seen as the norm whenever you connect to a ventilator. It's now standard practice to target V_T 6-8ml/kg IBW [13, 14] and limit plateau (or peak) pressure to 30cmH_2O [12].

As you'll see in equation 1; limiting V_T and P_{PLAT} (to achieve lung protective targets) without a compensatory increase in rate may result in hypercapnoea in poorly compliant lungs. It may therefore be necessary to accept supra-physiological $PaCO_2$ (permissive hypercapnoea) or use another means of CO_2 removal. Other strategies include the manipulation of time, rate, and mode variables to improve flow distribution; titration of positive end expiratory pressure (PEEP) to limit atelecta-trauma [15] and shunt; patient repositioning; and pharmacological agents.

Time Variables

Inspiratory time, expiratory time and their (I:E) ratio can be individually programmed in most MV modes. These parameters are interdependent and predetermined to a certain extent by ventilatory rate. Spoiler alert - their manipulation may predispose to complications.

For the purposes of theoretical modelling, groups of alveoli and their terminal airways possessing similar mechanical characteristics are termed 'lung units'. The time required to fully inflate or deflate lung units is determined by both compliance and resistance to

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flow, the product of which, the time constant (τ), determines the speed with which alveolar and proximal airway pressures reach equilibrium.

Many disease processes affect lung tissue mechanics and thus inspiratory (τ_i) and expiratory (τ_e) time constants. But because these changes are rarely homogenous, regional flow pattern variations occur. Lung units with low τ_i tend to fill quickly, with subsequent distribution of flow to units with greater resistance (which may more distensible). PPV therefore causes over-distension of compliant units alongside collapse of compressed units in a variety of pathologies, predisposing to complications and exaggerating $\tilde{V}_A$:Q mismatching.

Time variables may be controlled in many mandatory and assisted modes of ventilation; but it's imperative to ensure synchrony with any spontaneous effort to reduce distress, work of breathing, and breath stacking.

Inspiratory Time

Inspiratory time (t_i) is the duration over which pressure is applied in PPV to deliver V_T and is typically set at 1 second when respiratory rate is 20bpm. Any increase in t_i occurs at the expense of expiratory time (t_e) unless rate is reduced to compensate.

Increasing t_i may improve oxygenation by permitting equilibration of pressure between differently compliant lung regions, increasing mean airway pressures, and avoiding atelectasis. Reducing t_i predisposes to the complications of increased flow and system pressures in volume controlled modes [8].

Expiratory Time

Expiratory time (t_e) is typically set at 2 seconds when rate is 20bpm and, opposite to t_i, can only be maintained or increased at the expense of t_i without change in rate.

Pressures generated in PPV may be sufficient to overcome increased airway resistance and distend alveoli, but exhalation is passive and diminished if tissue elasticity is reduced. So conditions in which highly compliant alveoli empty into bronchioles with greater resistance to flow, such as COPD, require longer t_e to permit lung unit emptying. If t_e is in too low to permit sufficient emptying before inspiratory cycling begins again, gas trapping and the generation of intrinsic PEEP ($PEEP_i$) may occur, with both respiratory and haemodynamic consequences.

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I:E ratio

This is usually set at 1:2, but ratios of 1:2 to 1:4 may be required to prevent dynamic hyperinflation in severe airflow limitation. Awake patients may be more comfortable with shorter inspiratory times and high inspiratory flow rates.

Inverse ratio ventilation (IRV), in which t_i exceeds t_e, may be used if adequate oxygenation cannot be achieved by other methods including increased PEEP. Greater mean airway pressures and improved filling of lung units with high τ_i are likely to be key here. But caution should be exercised in using IRV in patients with airflow limitation to avoid generation of $PEEP_i$.

Proposed benefits of IRV include:

1) reduced shunt due to prevention and resolution of atelectasis
2) increased efficiency of CO_2 elimination due to delayed lung unit emptying
3) lower V_T and transpulmonary pressures due to reduced $\tilde{V}_{min}$.

Suggested Strategy

Following a thorough clinical assessment, consider first whether lung physio-mechanics are likely to be normal.

1) Targets in the absence of overt pulmonary pathology:

Rate	10-20bpm
V_T	6-8ml/kg IBW
I:E	1:2
P_{PLAT}	<30cmH_2O

Adjust parameters in response to clinical examination, blood gas analysis and monitoring. Recruitment manoeuvres may be required, particularly if a period of hypoventilation precedes starting MV.

PaO_2 may be optimised by adjusting $\tilde{V}_{min}$, I:E ratio, PEEP and FiO_2. Normocapnoea should not be pursued at the expense of exceeding pressure limits, unless there is urgent indication to do so. Development of a respiratory acidosis (pH < 7.25) should prompt reconsideration of pathology, mode of ventilation and alternative methods of CO_2 removal.

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2) Targets in pathophysiological states

Modify the above parameters considering predominant manifestations of pulmonary disease:

Airflow limitation

Narrowing of medium and small-calibre airways significantly increases resistance to gas flow, disrupting regional and global pressure-flow relationships and variably increasing time constants. Injudicious PPV programming in such individuals may result in volume redistribution, alveolar over-distension and ventilator trauma.

Dynamic hyperinflation due to insufficient t_e and unfavourable I:E ratios exacerbates $\tilde{V}_A/Q$ mismatching and further increases risk of trauma. $PEEP_i$ estimation may be necessary to avoid acute deterioration.

Restrictive lung disease

In the absence of significant airflow limitation, low compliance results in reduced τ. Affected lung units fill quickly, with rapid gas flow redistribution, $\tilde{V}_A/Q$ mismatching and over-distension of relatively unaffected units. Attention to volume and pressure settings may mitigate risk of trauma in such individuals.

Emphysema

The characteristic destruction of lung parenchyma and diminishment of elastic recoil results in markedly increased distensibility of affected tissue. Because tissue damage is patchy, with changes characteristic of causative agents and disease processes, affected units are slow to open and prone to over-distension. A low rate with long t_i and t_e should be set to permit filling and emptying of slower units and P_{PLAT} limited to prevent trauma.

COPD

Because emphysematous changes coexist with airflow limitation in COPD, both τ_i and τ_e may be significantly prolonged. A low rate and prolonged expiratory phase (I:E ratio 1:2.5 or 3.0) may allow for slow changes in airway flows and prevent gas trapping. Airway pressures must be limited to prevent over-distention and the potential for rupture of bullae.

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ARDS

ARDS is characterised by absolute volume loss with decreased total system compliance [16] but pathological changes are not evenly distributed and even targeting tidal volumes of 6-8ml/kg may over-distend aerated lung units and exacerbate shunt through compression atelectasis.

Optimal ventilatory strategies in ALI and ARDS remain the subject of debate, with some advocating bedside quantification of injured and healthy lung volumes in order to permit individualisation of V_T targets and the limitation of regional distending pressures [17]. The weight of evidence supports limitation of V_T to 4-8ml/kg IBW [10, 11] and plateau pressure to less than 30-35cmH_2O [10] as the least deleterious strategy.

Further Reading

The trainee-led ALPINE study by the fantastic PLAN should soon be published in the BJA – keep your eyes peeled!

For a sensible review of perioperative FiO2 titration, check out the RCT by Meyhoff et al in 2012 in Anesthesia & Analgesia.

The classification of PPCs (Post-operative Pulmonary Complications) has been a hot topic over recent years. Check out the great work by Tom Abbott (BJA 2018:120) to upskill yourself fast!!

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References

A 1. Vincent, J.-L., et al., Thirty years of critical care medicine. Critical Care, 2010. 14(3).

2. Soni, N. and P. Williams, Positive pressure ventilation: what is the real cost? British Journal of Anaesthesia, 2008. 101(4): p. 446-457.
3. Hotchkiss, J.R., et al., Effects of decreased respiratory frequency on ventilator-induced lung injury. American Journal of Respiratory and Critical Care Medicine, 2000. 161(2): p. 463-468.
4. Gajic, O., et al., Ventilator-associated lung injury in patients without acute lung injury at the onset of mechanical ventilation. Critical Care Medicine, 2004. 32(9): p. 1817-1824.
5. Howell, D.C.J. and G.J. Bellingan, Acute Lung Injury and Acute Respiratory Distress Syndrome (ALI/ARDS), in Respiratory Disease and Its Management, A. McLuckie, Editor. 2009, Springer-Verlag London Ltd: Godalming. p. 1-17.
6. Determann, R.M., et al., Ventilation with lower tidal volumes as compared with conventional tidal volumes for patients without acute lung injury: a preventive randomized controlled trial. Critical Care, 2010. 14(1).
7. Dries, D.J., et al., Time course of physiologic variables in response to ventilator-induced lung injury. Respiratory Care, 2007. 52(1): p. 31-37.
8. Dreyfuss, D., et al., HIGH INFLATION PRESSURE PULMONARY-EDEMA - RESPECTIVE EFFECTS OF HIGH AIRWAY PRESSURE, HIGH TIDAL VOLUME, AND POSITIVE END-EXPIRATORY PRESSURE. American Review of Respiratory Disease, 1988. 137(5): p. 1159-1164.
9. Pavone, L.A., et al., Injurious mechanical ventilation in the normal lung causes a progressive pathologic change in dynamic alveolar mechanics. Critical Care, 2007. 11(3).
10. 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. The New England journal of medicine, 2000. 342(18): p. 1301-8.
11. Moran, J.L., A.D. Bersten, and P.J. Solomon, Meta-analysis of controlled trials of ventilator therapy in acute lung injury and acute respiratory distress syndrome: an alternative perspective. Intensive Care Medicine, 2005. 31(2): p. 227-235.

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12. Lipes, J., A. Bojmehrani, and F. Lellouche, Low Tidal Volume Ventilation in Patients without Acute Respiratory Distress Syndrome: A Paradigm Shift in Mechanical Ventilation. Critical care research and practice, 2012. 2012: p. 416862-416862.
13. Hubmayr, R.D., Point: Is Low Tidal Volume Mechanical Ventilation Preferred for All Patients on Ventilation? Yes. Chest, 2011. 140(1): p. 9-11.
14. Dennis, D., W. Jacob, and P.V. Van Heerden, Ventilation - how often are we correct? Anaesthesia and Intensive Care, 2012. 40(4): p. 638-642.
15. Mols, G., H.J. Priebe, and J. Guttmann, Alveolar recruitment in acute lung injuiry. British Journal of Anaesthesia, 2006. 96(2): p. 156-166.
16. Gattinoni, L. and A. Pesenti, The concept of "baby lung". Intensive Care Med, 2005. 31(6): p. 776-84.
17. Mattingley, J.S., et al., Sizing the lung of mechanically ventilated patients. Critical Care, 2011. 15(1).