New Approaches to Ventilation

Introduction

Of all fields in ICU, mechanical ventilation might be the one most steeped in myth. As is common in medicine, we have stereotyped and perhaps even romanticised a necessary but invasive and often harmful practice; we have also created an arcane and confusing terminology.

Many books and articles on mechanical ventilation start with a definition of "respiratory failure" and list this as the main (or sole) indication for mechanical ventilation. Respiratory failure is then usually defined by numbers, and broken down into two forms, commonly termed "Type I" and "Type II" - the former said to be characterised by arterial hypoxia without a raised partial pressure of carbon dioxide, the latter by both low PaO2 and **high** PaCO2.

Think about such definitions for a moment! By looking at arterial partial pressures of gases, we are side-stepping the main issue, which is *failure of the normal respiratory pump* ! We are basing our definitions (and presumably our decision to ventilate) on a few observed consequences, and we are failing to take the **baseline** state of the individual into account.

I may seem to be arguing semantics, but .. consider a 70 year old patient with chronic obstructive airways disease who lives at an altitude of say 1600 metres above sea level. We might expect a normal seventy year old at this altitude to have a room-air PaO2 of perhaps 57 mmHg. An arterial PO2 of 47 mmHg with a PCO2 of (say) 50 mmHg tells us *absolutely nothing* about our patient! This person in "type II respiratory failure" might be sitting in your consulting rooms talking fairly comfortably to you, or be on the brink of intubation.

Again and again we will find that blind application of "rules" will get us into trouble with ventilatory management of patients. It is most important when assessing the illness of a patient to know something about how they normally function, and then to *look* at the patient. Particular attention should be paid to the respiratory rate, and the overall condition of the patient - is she distressed, sweating, poorly perfused or using accessory muscles of respiration. Such clinical observation is the correct context in which to interpret the "numbers".

We use mechanical ventilation to support patients who cannot sustain ventilation unaided. We normally do this by inserting an endotracheal tube, actively blowing air into the chest, and then allowing the air to passively leave. In so doing, we create a multitude of problems, including:

- Ventilator-associated pneumonia ("VAP");
- Ventilator induced lung injury ("VILI");
- Patient discomfort;
- The potential for cardiovascular compromise;
- The consequences of excessive patient sedation, and, particularly, over-enthusiastic paralysis.

We have invented a vast variety of different ways of mechanically ventilating patients, but in adults there is no clear evidence that any one method is superior to the others. Clearly some are more comfortable to the patient than others, but even here, there is substantial variation. Dogmatic assertions that things must be done in a certain ritualised fashion are more commonly based on arrogance than evidence. Nevertheless, over the past ten or so years, we have perhaps progressed a little in defining approaches to ventilation that cause less harm than we in our ignorance used to cause! This web-page looks at some of the speculation and facts surrounding these 'new' approaches.

Before we being, we might briefly describe a framework that is useful in describing ventilation. This isn't as easy as it sounds, as there is little consensus on how to classify the different modes of ventilation. We'll try not to get too bogged down. Then we will go through various ventilatory strategies that have shown promise in the past few years. These include:

- Changes in pressure profiles , and use of feedback;
- Fast rates ;
- The use of PEEP ;
- Non-invasive ventilation (and use of cuirasses);
- Liquid ventilation ;
- Use of smaller volumes for stiff lungs;
- Modifying our goals (pH, PO2, PCO2);
- Prone positioning;
- TGI (tracheal gas insufflation) and expiratory washout;
- Recruitment Maneuvers ;
- Paralysis, sedation, and approaches to "weaning";

A framework

Ventilator terminology is confusing, especially as some authors have used the same words - "control", "assisted", "spontaneous" and "mandatory" - for different things! We would like to draw your attention to the way Kapadia has simplified ventilator terminology [Postgrad Med J 1998 74 330-5]. He uses three basic terms:

1. The *trigger* - the signal that opens the inspiratory valve, allowing air to flow into the patient;
2. The *limit* - the factor which limits the rate at which gas flow into the lungs;
3. *Cycling* - the signal which stops inspiration AND eventually opens the expiratory valve.

This 'TLC' terminology has the merit of being simple and fairly comprehensive. **Triggering** is straightforward - either the patient initiates a breath, or the machine automatically does so at a given rate. (Different signals from the patient may be used to trigger a breath - older ventilators relied on changes in pressure, but more recently the sneaky approach of detecting changes in flow has been used to decrease the effort that a patient has to employ to trigger a breath

{This may not be better, see [Chest 1997 Jun;111(6):1649-53] also [Crit Care Med 1997 May;25(5):756-60]}

). In addition, some factor must **limit** the rate of gas flow into the lungs - many ventilators deliver gas at a constant flow rate ("flow-limited ventilation"), while others deliver gas up to a preset pressure ("pressure-limited ventilation"). Combinations may be used.

Cycling is complex, as there may be a pause between the cessation of inspiration, and the onset of expiration (as the expiratory valve opens). Without a pause, the signal that ends inspiration is immediately used to trigger expiration; otherwise, the machine inserts a delay between closure of the inspiratory valve, and opening of the expiratory valve. Cycling may be triggered by:

- volume - inspiration stops once the target volume is delivered, and then (often after a *time* delay), expiration starts;
- time - inspiration stops after a preset time interval (often set simply by dialling in, say, respiratory rate and and I:E ratio);
- flow - when flow decreases to a given level, inspiration is shut off.
- pressure (although this is not commonly used on its own);

Kapadia conveniently describes the commonly-used modes of ventilation in his Table 1, which we have abbreviated as follows:

Common modes of ventilation - TLC classification			
Mode	*Trigger*	*Limit*	*Cycling*
Continuous Mechanical Ventilation Assist (CMVa) = Assist Control (A/C) = Volume Control Assist (VCa)	Ventilator or Patient	Flow	Volume (Time controls pause)
Pressure Control Ventilation (PCV)	Ventilator or Patient	Pressure	Time (Time also controls pause)
volume-cycled Synchronised Intermittent Mandatory Ventilation (SIMV)	Ventilator or Patient	Flow (mandatory breath)	Volume (mandatory breath)
pressure-limited SIMV	Ventilator or Patient	Pressure (mandatory breath)	Time (mandatory breath)
Pressure Support (PS)	Patient	Pressure	Flow
CPAP	Patient	Pressure	Flow
CPAP + PS	Patient	Pressure	Flow
SIMV + PS	A combination of synchronised intermittent mandatory ventilation (with the appropriate characteristics of the mandatory breaths) and pressure support (with its characteristics). Note that either type of SIMV mentioned above may be used.		
Note that where CPAP is combined with ventilator triggered modes, confusing terminology			

kicks in again - CPAP is then called "PEEP" (Positive End-Expiratory Pressure).

A/C is a mode that is really only used in theatre, and is an anachronism in the ICU. A fixed number of breaths is delivered, but the patient may also trigger delivery of a full breath. **PCV** used to be seen as a "last resort" in patients with very stiff lungs, running high pressures on SIMV". Many intensivists now view it as a good primary mode of ventilation in patients with poor lung compliance (for example, ARDS), especially when combined with longer inspiratory times (inverse ratio PCV, otherwise known as "PCIRV"). In **SIMV** , breaths are delivered by the machine at a preset rate, and the patient can initiate an (unassisted) breath in between the machine breaths. If the patient starts breathing just before a machine breath was due, then only is a full machine breath delivered. Note that with pure **PS** or pure **CPAP** no machine breaths are delivered - the patient initiates all breaths. On many machines (especially older ones) the work of breathing in CPAP mode is so substantial that this mode should not be used!

In the following sections, we look at new approaches to ventilation (including new modes). We will refer to their TLC characteristics as we examine these modes.

1. Altered pressure profiles and related strategies

Here we will examine two strategies, the first being the use of so-called "BiPAP", and the second the use of feedback to dynamically alter ventilation ("servo-mechanisms").

BiPAP

There are at least five circumstances where the dreaded term "BiPAP" has been used:

1. As a synonym for **CPAP+PS** (Respironics). The CPAP level is termed "E-PAP" and the "I-PAP" is the peak pressure attained. Other frills have been added by some, for example the ability to drop CPAP a fixed number of times every minute! ("Intermittent Mandatory Pressure Release Ventilation" - Cesar).

2. As a synonym for **pressure control** - again, CPAP is called "E-PAP", and "I-PAP" is the pressure control level.

3. With spontaneous breathing at **two different levels of CPAP** (Dräger Evita) - these pressure levels are alternated every five to ten seconds.

4. For a variant of (3) above, where the upper pressure limit is *short* , so the patient only breathes at lower pressure - rather similar to **pressure-limited SIMV** !

5. Another variant of (3), otherwise called **Airway Pressure Release Ventilation** , where the duration of the lower pressure is *short* , so the patient only breathes at the upper pressure!

APRV

Airway Pressure Release Ventilation (APRV) appears to have had a mixed reception. Some experimental studies show little merit (APRV proved inferior to CPAP in maintaining oxygenation and lowering shunt in an oleic acid model of ALI in pigs [Anesth Analg 2001 Apr;92(4):950-8]), while others disagree, showing improved VQ mismatch with unrestricted spontaneous breathing during APRV compared with pressure support [Am J Respir Crit Care

Med 1999 Apr;159(4 Pt 1):1241-8]. Isolated case reports claim benefit in a variety of respiratory disorders.

Feedback

The old-fashioned term "servo" (which just means feedback) is commonly used in ventilators that use feedback to alter settings according to how the patient's lungs respond. In each case, we must define the parameter that is monitored, and the feedback response (alteration) that occurs:

- PRVC - **Pressure-regulated volume control** is like pressure control - the limit is pressure and cycling is time-based, *but* you set a desired volume, and the level of pressure is continually varied to try and achieve this volume, based on results obtained for the last few breaths! A rather cute feature of the Siemens Servo 300.

- **"Auto flow "** is like PRVC, but combines this with the Evita's "BiPAP" (BiPAP type 3, above). Found on the Dräger Evita Dura.

- **"Volume Support "** is a PRVC-like modification of pressure support. The difference between this and PRVC is the difference between PCV and PS - volume support is flow-cycled.

- Similar feedback mechanisms to those employed in volume support are used in **Minimum Minute Ventilation** to ensure a preset minute volume (Hamilton Weolar).

- **Mandatory Rate Ventilation** instead watches the respiratory rate, and increases the level of pressure support as the patient breathes faster.

- ***Mandatory* Minute Ventilation** (not to be confused with Minimum Minute Ventilation) uses feedback to vary ventilator breath rate - if the patient achieves an adequate minute ventilation on his/her own, the ventilator doesn't add any breaths. This therefore differs from SIMV, where the machine always delivers the minimum number of breaths. (Erica Engstrom).

- **Proportion Assist Ventilation** is smart - the ventilator assesses inspiratory effort (based on the current flow and volume signals), and adjusts pressure *during that breath* ! Volume proportional assist ventilation [Crit Care Med 2000 Jun;28(6):1940-6] was found to be more comfortable than PSV in healthy volunteers with artificially decreased respiratory system compliance!

"Automatic Tube Compensation"

During spontaneous breathing, the resistance of an endotracheal tube may contribute substantially to the work of breathing, especially at high flow rates, and with smaller tubes. With 'ATC', inspiratory pressure is increased to compensate for the resistance of the tube, and this compensation is varied with flow, providing full compensation. The work that the patient has to exert in overcoming the resistance of the tube effectively 'disappears'. It has even been claimed that because of this lack of tube resistance, ATC "predicts the patient response after extubation"! (Evita ventilators).

2. Fast Rates

Several modes of high-frequency ventilation have been proposed, including high-frequency oscillatory ventilation (HFOV), and high-frequency jet ventilation (HFJV). HFJV has been mainly used during short procedures such as bronchoscopies. It is not without complications, notably pressure "stacking", hypercarbia, and airway injury owing to difficulty with gas humidification. High-frequency ventilation can be considered ventilator-triggered, flow (HFJV) or pressure (HFOV) limited, and time-cycled. Some commercial machines *allow* rates of up to about forty breaths *per second* (2400/min), although substantially slower rates are usually employed, in the range of 2-15 Hz.

HFOV is interesting. It was first thought of as a ventilatory modality when someone noticed that dogs, when they *pant* , take breaths that are smaller than their dead space. (Dead space in all animals, even giraffes, is generally constant at about 1/3 of resting tidal volume). How, the researcher wondered, do panting dogs maintain oxygenation? To this day, we're still not sure of the answer - at least five different credible explanations have been proposed. Simplistically, the high frequency of panting (and HFOV) increases turbulence and thus mixing and diffusion of oxygen.

HFOV has been extensively used in neonates, and has almost become a 'standard of care' in some centres. There is however controversy about when it should be used, and even whether it is beneficial. Recent meta-analyses question its benefit in neonates, especially now that surfactant can be delivered into the lungs of infants with surfactant deficiency. There is some evidence to suggest that HFOV may be associated with an increased incidence of severe intraventricular haemorrhage, and periventricular leukomalacia.

The largest HFOV trial in neonates (the "HIFI" trial) had negative results, but the methodology employed has been questioned. Clearly, HFOV is tricky. There is experimental evidence to suggest that HFOV at low mean airway pressures may be extremely harmful to the lung, especially if lung compliance is poor, causing extensive barotrauma. "High-volume" strategies are now advocated (i.e. using high mean airway pressures). Inappropriate HFOV strategies probably account for the poor results initially encountered with HFOV in adults. HFOV may well still have a place in ventilation of adults with severe ARDS, but no decent-sized studies have been performed. We wait!

3. PEEP

Many have written about the value of positive end-expiratory pressue (PEEP/CPAP) in critically ill patients, especially in ARDS. There is fairly strong evidence that PEEP is of great value in the management of ARDS, but how to obtain "best" PEEP is very controversial. Everone has his/her own approach! Some have empirically taken +2cm H_2O above the "lower inflection point" (LIP) of a quasi-static pressure-volume curve. This is performed by paralysing the patient and then slowly expanding the lungs using a "super syringe", all the time monitoring airway pressure and volume injected. There are numerous problems with this approach (apart from the time taken and the requirement of total patient paralysis). Among the problems are:

- Inspiratory and expiratory curves differ. Which one should one use?
- In many curves there is no definite "inflection point";
- Some studies have shown poor correlation between recruitment and the LIP.
  A superb article is: [Acta Anaesthesiol Scand 1998 Nov;42(10):1149-56].

Rigorous studies looking at outcome as related to level of PEEP are lacking. Most approaches are anecdotal. Our particular anecdotal approach is to use at least 10cm H_2O PEEP unless there is a good reason to not do so! Patients who become cardiovascularly compromised at such levels are usually grossly behind on fluids. Exceptions to this approach should include asthmatics (where one should generally shy away from PEEP), and patients with chronic obstructive airways disease. Note that in asthmatics with residual auto-PEEP despite slow rates (say 8 breaths per minute), judicious application of a small amount of PEEP to balance the auto-PEEP may in fact be beneficial! Take care with PEEP (although it's probably a *good thing* overall) - recent work suggests that although it works in experimental models of ARDS, its success is at the expense of increased parenchymal stress [J Appl Physiol 2001 May;90(5):1744-53].

4. "Non-invasive Ventilation" and Negative Pressures!

Non-invasive positive-pressure ventilation (NIPPV) has been around for ages, but earlier masks were hellishly uncomfortable, and patient selection was often poor. Kacmarek has reviewed the "level one" evidence that NIPPV works. NIPPV appears to be particularly advantageous (with careful selection) in patients with chronic obstructive pulmonary disease. For a meta-analysis, see [Crit Care Med 1997 25 1685-92]. It has also been used in cystic fibrosis, acute asthma, and patients awaiting lung transplantation. NIPPV may be life-preserving in immunocompromised patients with Pneumocystis carinii or other pneumonias [Chest 1996 109 179-93; Intensive Care Med 1998 24 1283-8; JAMA 2000 283 235-41]. The major advantage of NIPPV is probably the lack of an endotracheal tube - normal defence mechanisms that prevent entry of bacteria into the lung are preserved. [Intensive Care Med 1999 25 567-73; Ann Intern Med 1998 128 721-8; N Engl J Med 1998 339 429-35] There is now compelling evidence that early extubation to NIPPV lowers duration of mechanical ventilation, shortens ICU stay, lowers the incidence of nosocomial pneumonia and improves sixty-day survival [Ann Intern Med 1998 128 721-8].

There are still numerous 'tricks' to establishing successful NIPPV. The patient must be co-operative, and gently informed that you have a mask that will help their breathing. NIPPV must be mechanically feasible. Don't show the patient the head-gear first off - start with a low level of CPAP/PEEP (± PS), and allow them to hold the soft silicone mask gently to their own face. When you have their confidence, then you can adjust settings to an optimal level, and finally strap the mask on. It's often initially quite tricky (or impossible) to get a distressed patient to breathe through a nasal mask - the drive to breathe through the mouth may be overwhelming! NIPPV *may* make large demands on nursing staff, although this has generally not been the case. Successful outcomes tend to correlate with the presence of teeth, younger age, lower APACHE/SAPS scores, and less acidosis. Guidelines (which seem to be a bit of a bland thumb-suck) are:

Consensus Guidelines: NIV for COPD + Acute Respiratory Failure

Step 1: Identify need for ventilatory assistance

A. Acute respiratory distress: symptoms + signs
   a. Dyspnoea - moderate to severe, more than usual;
   b. RR > 24, accessory muscle use, paradoxical breathing
B. Abnormal Gas Exchange

a. $PaCO_2 > 45$ mmHg, pH < 7.35 *or*
b. $PaO_2/FiO_2 < 200$

Step 2: Exclude those at high risk

A. Respiratory arrest
B. "Medically unstable" (shock, uncontrolled cardiac ischaemia, arrhythmias)
C. Cannot protect airway
D. Excessive secretions
E. Agitated/unco-operative
F. Facial anatomic abnormality (trauma, burns, ..)

Modified after [Respir Care 1997 42 364-9]

Negative Pressures

The first ventilators were tank ventilators such as the "iron lung" that relied on extracorporeal application of **negative** pressure, with a rubber seal around the patient's neck. These fell into disfavour for a variety of reasons including upper airway obstruction (obstructive sleep apnoea, even in "normal patients"), claustrophobia, discomfort, lack of triggering in response to patient attempts to breathe, leaks, and sheer bulk (the "iron lung" weighed a ton, even modern cuirasses are bulky). Newer developments in this field have yet to be extensively tested. A recent, thorough review of all aspects of non-invasive ventilation [Am J Respir Crit Care Med 2001 163 540-77] includes a good history of development of tank and cuirass ventilators.

5. Liquid Ventilation

There is a vast amount of experimental literature on liquid ventilation. Most commonly, this is a combination of airway instillation of oxygen-carrying fluorocarbons such as perflubron, with conventional ventilation (partial liquid ventilation, PLV). Unfortunately, clinical studies are few and far between. There is a lot we still need to learn about PLV - for example, distribution of perflubron within the lung is far from homogeneous, tending to gravitate towards dependent areas. Initial improvements in oxygenation need not necessarily be maintained, and falsely high tidal volumes may occur (fixed orifice measurements are up by 7-16%, hot wire 35-41%) [Physiol Meas 2000 Aug;21(3):N23-30] Claimed merits of PLV include improved oxygenation, wash-out of exudates and infectious material, decreased bacterial adhesion [Crit Care Med 1999 27 2741-7], and even (possibly) reduced lung injury [Crit Care Med 1999 27 2500-7].

6. Altered Volumes - "Protective Strategies"

The ARDSNET study is probably the most important paper on ventilation from the last decade. This well-designed study demonstrates unequivocally that lower tidal volumes (6ml/kg ideal weight) are associated with improved outcomes, including 30-day mortality, when compared with more "conventional" volumes (12ml/kg). Even more interesting is the observation that this improvement was in the face of slightly lower arterial partial pressures of oxygen. Also of note is that in this study, pH was corrected with intravenous sodium bicarbonate, to remove pH as an interfering variable. Another interesting question is that of respiratory rate - some have argued by unconvincing analogy that respiratory rates should be

kept low in ARDS, but the ARDSNET group that did better had average rates of 29/min (as opposed to half this rate in controls).

The ARDSNET study is the most important of several studies which have looked at the question of tidal volumes in ARDS, some of the others agreeing with its findings, and other coming up 'negative' for reasons capably discussed in the paper itself [New Engl J Med 2000 May 342 (18) 1301-9].

By the way, note the introduction of the peculiar term "volutrauma". This is a silly and quite unnecessary neologism, as pressure and volume are intimately related. The term seems to arise from a failure to understand that there is an inconstant relationship between transalveolar pressure and transthoracic pressure. Whatever the terminology used, it is now quite clear that alveolar overdistension results in disruption of the alveolar capillary membrane, and florid pulmonary inflammation.

7. Altered Goals - O2, CO2 and pH

Only recently have we really begun to appreciate that "normality" is a far from reasonable goal in the critically ill. Everything in ICU is a trade-off, and previously we were probably far too enthusiastic in our attempts to reach a "normal" saturation of say 93(+)%, a "normal" PCO2 of say 40 mmHg, and a "normal" pH of 7.36 to 7.44.

There is emerging evidence that we may be wrong on all three counts:

1. PaO2 in the ARDSNET study (mentioned above) was allowed to drop to 55mmHg (or an SaO2 of 88%) for protracted periods with quite the opposite of an adverse effect. (Note that in man there is very little evidence that 'oxygen toxicity' is a problem, but we should nevertheless be very cautious about exposing patients to a high FiO2 for a long time);
2. For years now we have accepted a strategy of "permissive hypercapnia" in asthmatic patients (and more recently, in ARDS patients), with apparent benefit;
3. pH is perhaps the most controversial of the lot, but many intensivists would be quite happy with a respiratory acidosis and pH of say, 7.20 or even less. In the ARDSNET study, pH was corrected using bicarbonate infusion, but this was to eliminate pH as an interfering variable. (Some would assert that outcome would have been even better had pH been allowed to become more acidotic).

Note that permissive hypercapnia induces dramatic changes in the cardiovascular system, [Am J Respir Crit Care Med 1997 Nov;156(5):1458-66] and may also tend to worsen PaO2, as shown in a multiple inert gas study on a small number of patients [Am J Respir Crit Care Med 2000 Jul;162(1):209-15]. Correcting the pH does alleviate the haemodynamic effects of permissive hypercapnia [Crit Care Med 1996 May;24(5):827-34], at least in sheep. Some have advocated the use of THAM as an alternative to bicarbonate administration [Am J Respir Crit Care Med 2000 Apr;161(4 Pt 1):1149-53].

8. Prone Positioning

In the absence of large studies on prone positioning, one has to be content with empiric generalisations. Prone positioning does appear to benefit a proportion of patients with severe ARDS. [Intensive Care Med 1997 23 1219-24; Anesth Analg 1995 80 955-60; Anesthesiology

1991 74 15-23; Am Rev Respir Dis 1987 135 628-33] The reason for this is not clear, but may be to do with the heterogeneous nature of the lung injury - "ARDS" seems to preferentially affect the dependent regions of the lungs, so a patient lying on their back may benefit from improved V/Q ratios when flipped over. The benefit of prone positioning may take several hours to become apparent. Nursing is not generally a problem once the nurses have become accustomed to this approach, (but one must take care not to allow undue pressure on the eyes - this should be common sense). It makes sense to try this maneuver in ARDS patients with refractory arterial hypoxia, before indulging in more aggressive heroics. Prone positioning is also an important component of recruitment (see below).

9. Low Dead Space and Tracheal Gas Insufflation

One problem with critically ill patients, especially those with severely diseased lungs, is high VD/VT ratios (large dead space: tidal volume). This may be a substantial problem as we decrease tidal volume in an attempt to limit barotrauma, although most patients tolerate hypercapnia fairly well. Exceptions would be patients with severe associated metabolic acidosis, or those with raised intracranial pressure. Possible solutions are to use specially constructed endotracheal tubes with an internal second lumen to minimise dead space, or even to insufflate gas via a small cannula. Although attractive in concept, there seem to be significant problems, including:

- Pressure measurements;
- "Stacking";
- Potential for damage caused by the inspiratory jet;
- The need (with some systems) to anticipate the problem of VD/VT and put in a special tube early on, or alternative to change the tube on a very unstable patient.

See [Anesthesiology 1997 87 6-17; Am Rev Respir Dis 1993 148 345-51]

10. Recruitment Maneuvers

A variety of approaches have been used to "recruit" collapsed air spaces in the lung. The general consensus seems to be that substantial pressures (40+cmH_2O) are needed to open up such air spaces - simply cranking up the PEEP a few centimetres is not usually adequate, although such "mini-recruitment maneuvers" may cause mild improvements in oxygenation [Intensive Care Med 2000 May;26(5):501-7]. We have elsewhere discussed one approach to recruitment that seems particularly effective. All such approaches should at present be regarded as "experimental", although the results of a successful recruitment maneuver are so dramatic and satisfying that we cannot countenance not trying this maneuver in severely hypoxaemic patients. We can only hope that formal studies of recruitment don't hash things up (and give false negative results) by, for example, failing to turn the patients prone before recruitment is attempted!

11. Paralysis, Sedation, and the Myth of Weaning

As always, one's goal in ventilating a patient should be to minimise intervention and yet support the patient adequately. An awake, comfortable, co-operative patient is infinitely preferable to a heavily sedated patient, which in turn is far, far superior to having to paralyse a heavily sedated

patient. We have only recently begun to realise the carnage that we wreak when we paralyse a patient for a prolonged period (over 24 hours) - so-called "critical illness polyneuropathy" (and/or myopathy) is a dramatic and devastating consequence of aggressive neuromuscular blockade, especially if level of paralysis is not monitored using a nerve stimulator, and/or steroids are administered concomitantly.

The term "weaning from the ventilator" is unfortunate. It conjures up the image of ventilation as something akin to mother's milk, from which we reluctantly 'wean' the patient. This picture may be far from the truth.

It would appear logical to remove the invasive and noxious intervention of ventilation as soon as the patient is ready to have this removed. Here is the catch - our "weaning strategies" are often merely maneuvers that we indulge in to **reassure ourselves** that the patient is ready to have the ventilator, and finally, the endotracheal tube, removed! (Some have called this "liberating the patient from the ventilator", which I find far more honest)!

Clearly however, as a patient improves, modification of our ventilatory strategy may make the patient more comfortable, and this *is* desirable. There are no cast-iron rules. For example, in some intensive care units, pressure control ventilation is still regarded as an approach of last resort, and heavy sedation and paralysis are regarded as necessary concomitants! As the patient improves, he/she is then put "back on SIMV+PS", and the SIMV rate is then progressively "weaned". There is no justification for this approach, (although it often works as well as any other). You will find, with careful experimentation, that some patients on pure pressure support will be *more comfortable* on pressure control ventilation, as with the latter one can set an I:E ratio (cycling is time-dependent and not flow-dependent). Others will be very intolerant of pressure control. Often simple observation of the patient (looking carefully at the pattern of ventilation) will allow one to adjust ventilation appropriately. Newer modes of ventilation such as APRV, volume support and PRVC may be extremely useful in improving patient comfort.

An Approach - Ten Guidelines

What can we conclude from the above? For patients with severe lung disease, there is no one approach - diseases differ, and so must managment. The following seem to us to be reasonable guidelines:

1. Tidal volumes of ~ 6ml/kg ideal body weight should be the standard of care in ventilation for Acute Respiratory Distress Syndrome, and probably also for other patients with reduced pulmonary compliance. Pressure-controlled inverse ratio ventilation should probably be instituted sooner rather than later in patients with anticipated severe lung injury, despite the lack of studies confirming a substantial benefit from this mode of ventilation!

2. Plateau airway pressures should be kept under about 30-35 cmH2O - one should probably aim for plateaus of *not more than* 30 cmH_2O! But note that *volumes* are likely to be a better indicator of potential lung injury than pressures, as pressures are a reflection of total respiratory compliance - most intensivists cannot measure interpleural pressure (or a proxy of this such as intra-oesophageal pressure).

3. PEEP should be tailored to the case, and be used carefully if at all in patients with impaired gas expiration (asthmatics and those with chronic obstructive pulmonary

disease). In ARDS, the suggestion is that relatively high levels of PEEP are advantageous in preventing de-recruitment, but insufficient on their own to substantially "recruit" collapsed air-spaces. There seems to be little value in using values such as the lower inflection point (derived from "super syringe" pressure-volume curves) to determine PEEP.

4. Permissive hypercapnia is an acceptable maneuver, but the role of pH correction is not resolved. Aggressive correction of pH is of unproven value, and should be regarded as experimental.

5. One should **not** aim for a "normal" PaO2 - a haemoglobin saturation of ~ 90% is probably more than adequate, if achieving a higher saturation is likely to compromise the patient in other ways;

6. Respiratory rate is probably of little importance in the pathogenesis of lung injury;

7. Appropriate recruitment maneuvers *may* be life-saving but all the necessary caveats must be adhered to (for example, prone position or upper chest compression during the maneuver). Recruitment maneuvers are more likely to work in patients with so-called "secondary ARDS" (following on, for example, abdominal sepsis).

8. Prone ventilation may be of use in a small subset of patients;

9. High-frequency ventilation (including HFOV, HFPV, HFJV), partial liquid ventilation, and newer variant modes of ventilation such as the different "BiPAPs" should be regarded as experimental at present, although some of these appear attractive and may well be beneficial in select patients. Tracheal gas insufflation and expiratory washout are likewise of dubious value at present.

10. Contrary to the outmoded practice of "flattening" every ventilated patient, one should aim for an awake, co-operative and comfortable patient. Second prize is sedation, and heavy sedation with paralysis comes a dismal third, although all too often in critically ill patients, we still have to resort to such measures. If we *must* paralyse a patient, then we are obliged to check their degree of paralysis using a nerve stimulator (and avoid complete ablation of neuromuscular responses - 2 twitches on a train-of-four is usually quite adequate, and more suppression is usually excessive). Concomitant use of steroids and neuromuscular blocking agents should be considered relatively contraindicated.

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