Editorial II

Pressure support ventilation and the critically ill patient with muscle weakness

The case reports by Kannan and colleagues in this issue of British Journal of Anaesthesia serve to highlight that we should remain alert for inspiratory triggering failure during pressure support ventilation (PSV) in patients with severe muscle weakness. Despite improvements in the ability of ventilators to respond to the patient’s own breathing pattern, inspiratory triggering failure may also be encountered in patients with chronic obstructive airways disease (COAD) or acute lung injury, who have high intrinsic positive end-expiratory pressure (PEEPi) in addition to possible muscle fatigue. There is also the potential for expiratory triggering failure and mismatches in tidal exchange in PSV.

Used for respiratory support and weaning, PSV has an inspiratory phase initiated by muscle effort ($P_{\text{mus}}$). The trigger—depending upon the machine and its options—is either the transient airway pressure drop below a set threshold, or the ventilator-sensed difference between the instantaneous inspiratory and expiratory flows, superimposed upon a base or bias flow commonly set to 10 litre min$^{-1}$. The flow ‘sensitivity’ setting of 2 litre min$^{-1}$ may be considered to be equivalent to a pressure ‘sensitivity’ setting of 1 cm H$_2$O. Whether at a flow sensitivity setting of 1 litre min$^{-1}$ or at a pressure sensitivity of 1 cm H$_2$O, cardiogenic oscillations can result in spurious autotriggering in nearly one in five patients, and even in the brain dead. In contrast, when the trigger setting is less sensitive, triggering may fail in the presence of muscle weakness or impaired central drive. This could also occur when tracheobronchial secretions episodically increase the inspiratory resistance. Inspiratory triggering may fail in patients with a rapid respiratory rate and short expiratory time, and in patients with significant intrinsic PEEP and a corresponding delay in the fall in airway pressure on expiration. Deviation from the one-to-one response which is a ventilatory response following each breathing effort, increases at higher ventilatory frequencies and higher levels of pressure support and lower levels of patient effort.

A less sensitive trigger setting will also tend to increase the work of breathing, although it has been pointed out that an inadequate flow rate delivery at the onset of inspiration, such as in the Tyco PB7200, was as significant in terms of increased respiratory work. Some newer ventilators incorporate a rise time control to be used in conjunction with graphics to ensure rapid attainment of the pressure plateau, but if too rapid, over-pressure causes premature cut-out of the inspiratory phase.

The tidal volume exchange in PSV is independent of inspiratory effort at the lowest levels of $P_{\text{mus}}$ and neural expiration commences before the mechanical phase cycles to expiration. Should respiratory demand increase, there is not invariably an increase in minute ventilation. If the patient is able to respond with a muscular effort equivalent to 5 cmH$_2$O ($P_{\text{mus}}$), there is no increase in tidal exchange but rather an increase in inspiratory flow rate with shortening of the mechanical inspiratory phase and an associated increase in the work of breathing. At greater levels of $P_{\text{mus}}$, when the timing of termination of neural and mechanical inspiration coincide, tidal exchange can increase by 50%. It can be imagined that satisfactory blood gases obtained during a period of high respiratory work may not be sustained either during sleep or if fatigue supervenes. Another study on patients and with computer simulation by the same team, showed that there is a phase of great instability of tidal volume across changes in inspiratory $P_{\text{mus}}$ between 3 and 12 cm H$_2$O, becoming more evident the greater the pressure support levels were above 10 cm H$_2$O. This is largely related to failed triggered breaths as the fall in ventilator breaths matched a paradoxical rise in tidal volume. Whilst the actual blood gases were thus similar in both the PSV and proportional assist ventilation (PAV) modes, the tidal volume and ventilator rate responses were only consistent in the PAV. However, this latter mode would probably provide totally inadequate tidal volumes at low levels of $P_{\text{mus}}$.

Determinants of the termination of the inspiratory phase vary according to the ventilators but typically they are a decline in the inspiratory flow rate to a fixed percentage of the peak flow rate, ranging between 5 and 25% (Siemens Servo900, Bird 8400ST), or to an absolute flow of 5 litre min$^{-1}$ (Tyco PB7200). Most ventilators have a back-up means of terminating the inspiratory phase such as an over-pressure of 1.5 cm H$_2$O, or if the total inspiratory time exceeds 3 s or 80% of one breath cycle.

Factors which may lead to a lag in inspiratory to expiratory cycling include both patient and circuit characteristics, such as a leak past the cuff of the tracheal tube. Ventilator-related factors are illustrated by the failure of the Tyco PB7200 to cycle at the target flow threshold in a workbench set-up; cycling was only initiated by over-pressure at end-inspiration. Such an occurrence in patients will increase the work of breathing and contribute to an unsettled pattern of breathing. A terminal inspiratory pressure overshoot is diagnostic of the onset of neural expiration, except in the tetraplegic patient lacking expiratory muscle activity, in whom a weak inspiratory effort will also combine to extend the mechanical inspiratory time.

Being simple to set up, PSV has become widely used for the patient with an adequate ventilatory drive who is not so ill as to require muscular paralysis and controlled venti-
lating, especially as it probably prevents or helps restore deconditioned respiratory muscles, \(^{10}\) and the patient does not appear to ‘fight’ the ventilator in the same way as with synchronized intermittent mandatory ventilation (SIMV). It has also been advocated for a faster weaning in comparison with SIMV, although a meta-analysis\(^ {11}\) has found spontaneous breathing trials equally effective, suggesting that adherence to procedures are as important as the actual weaning technique.

The multi-centre study of Estaban and colleagues,\(^ {12}\) enrolled 546 patients of whom a quarter were classed as difficult to wean on the first trial of spontaneous breathing. After randomizing the latter patients into four groups, the median time to wean differed by only a day or two; the two trials of spontaneous breathing groups weaned at 3 days each, the PSV group at 4 days and the synchronized intermittent mandatory ventilation group at 5 days. At 14 days, there were still between 10 and 20% who had failed to wean and no outcome was given for this group of slower patients.

Neurological disorders associated with profound respiratory muscle weakness include the gamut of acute weakness syndromes,\(^ {13,14}\) undiagnosed motor neuron disease,\(^ {15}\) and spinal cord injury. In one series, 62% of difficult-to-wean patients had neuromuscular abnormalities.\(^ {16}\) There were only two or three patients described as ‘neurological’ in each of the Estaban sub-groups and their individual progress was not documented. It leaves open the question of how best to mechanically ventilate and wean a tetraplegic patient, who may require weeks or months of respiratory support whilst avoiding diaphragm atrophy.

Diaphragm preservation in tetraplegia varies between the patient with only one functioning hemidiaphragm and the C5 transection patient with innervation of both diaphragms. In one study looking at the period of phrenic nerve function in 107 artificially ventilated tetraplegic patients, the ‘early’ group who never lost diaphragm activity after injury were fully weaned from ventilation by graduated ventilator-free breathing time after an interval of 37 (25) days mean (SD).\(^ {17}\) Peterson and colleagues\(^ {18}\) retrospectively studied 42 tetraplegics whose respiratory management alternated between the assist/control mode on the ventilator and increasing periods of ventilator-free breathing. They found that in these patients, without acute lung injury, final assist/control tidal volumes greater than 20 ml kg\(^ {-1}\) were associated with a faster mean wean time of 37 as against 58 days, and attributed this to better prevention and clearing of atelectasis. It might, therefore, be prudent if PSV were used to include some deeper mandatory volume controlled breaths and, if necessary, incorporate a dead space to prevent hypocapnia.

Another reason for a mandatory back-up rate is that peripheral muscle fatigue in acute tetraplegia is associated with adaptive central fatigue,\(^ {19}\) which may manifest as periodic breathing or sleep apnoea. The tetraplegic patient who has lost all intercostal breathing has also lost the ability to share the work of breathing between the diaphragm and intercostal muscles, a pattern known as respiratory alternans, which predisposes to fatigue. A raised carbon dioxide blood gas tension in neurological patients breathing with CPAP or PSV is always indicative of muscle weakness or fatigue. Much can be gained by adopting an empirical bedside approach in which a value of the ratio of independent tidal volume to voluntary vital capacity of over 0.4 be predictive of fatigue,\(^ {20}\) assuming a degree of linearity between the volume and transdiaphragmatic pressure responses.

Sub-clinical and clinical muscle fatigue may be identified by the spectral shift in the EMG, but this technique is not widely available and the techniques of phrenic nerve stimulation, transdiaphragmatic and mouth occlusion pressures\(^ {13}\) are all relatively difficult. There is, however, a strong case to undertake more systematic electrophysiological studies of peripheral nerve and muscle in intensive care patients, in view of the finding that three-quarters of those with proven axonal polynuropathy had diaphragmatic impairment, with reduction of the compound diaphragm action potential or increased latency of phrenic nerve conduction.\(^ {21}\)

In the clinical situation, it may be difficult to separate out the effect of central drive, peripheral fatigue, and simple muscle weakness whether in COAD or in neurological disease. Fluoroscopy of the diaphragm may be helpful, and the sniff test is reliable in pure tetraplegia as there is no paradoxical relaxation of the abdominal musculature on inspiration. Ultrasound examination may be more convenient but should be tempered by its limitations in visualizing the left hemidiaphragm and the possibility of false readings due to relative movements between the probe and abdominal wall.\(^ {22}\)

However, bedside neurological examination supplemented by regular spirometry of both tidal volume and vital capacity remains central to clinical appraisal of progress in weak patients. These measurements are integral to the technique of weaning by graduated ventilator-free breathing, with the prescribed rate of increase in ventilator-free time being adapted to the patient accordingly. In a slow wean, the patient benefits psychologically from his perceived active participation in the wean programme. Cuff deflation is also beneficial for clearance of mucus and for speech, all of which contribute to the success of this technique, even compared with the more widely known PSV.

J. W. H. Watt
Spinal Injuries Unit
Southport and Ormskirk Hospital NHS Trust
Town Lane
Southport PR8 6PN
UK

374
References

8. Giannouli E, Webster K, Roberts D, Younes M. Response of ventilator-dependent patients to different levels of pressure support and proportional assist. Am J Respir Crit Care Med 1999; 159: 1716–25