## Editorial

## Inspiratory pressure support: does it make a clinical difference?

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When new techniques, pharmacologic agents or approaches to mangement are introduced their clinical effciency is always questioned. It has always been most difficult to answer this question with reference to mechanical ventilatory techniques. Each new entry on the list of available techniques has created controversy regarding its efficiency. Inspiratory Pressure Support (IPS) introduced in 1981 on the Seimens Servo 900C and the Engstrom Erica (referred to as Inspiratory Assist) ventilators [1] is no exception. In spite of the availability of IPS on almost every new adult mechanical ventilator marketed [2] and its widespread application, little scientific information is available to guide our use of IPS. Questions regarding indications, methods of application and possible physiologic effects have not been conclusively answered.

Inspiratory Pressure Support is a form of pressurelimited mechanical ventilation, somewhat similar to, but more sophisticated than conventional IPPB. With IPS, the patient's inspiratory efforts activate a pressure-limited breath. Once activated, a flow of gas sufficient to meet the patient's inspiratory demands enters the circuit as the exhalation valve closes, allowing the system pressure to rapidly approach the set IPS level. Normally, a pressure plateau is established and maintained until the patient's inspiratory flow decreases to a ventilator-specific minimal level, at which time exhalation occurs. As a result, patients exert significant control over the process of ventilation. They determine the frequency, inspiratory flow, and the inspiratory time. Patient effort, IPS level, and the total patient/system impedances combine to determine the tidal volume.

Since IPS is a form of partial ventilatory support, one would suspect that increasing levels of IPS would produce effects similar to conventional mechanical ventilation. This was first demonstrated by McIntyre, [3] who was able to maintain the same level of ventilation using IPS (up to 42 cmH<sub>2</sub>O) as could be achieved with SIMV. Viale et al. [4] have demonstrated that IPS in postoperative patients without chronic pulmonary disease can reduce oxygen consumption to levels seen with controlled ventilation. This has also been described by Brochard et al. [5] in patients with chronic pulmonary disease who fail to wean. Others have described a decrease in respiratory rate, [3, 6] an increase in tidal volume, [6, 7] a decrease in use of accessory muscles [5, 8, 9] and a general improvement in patient tolerance of mechanical ventilation as IPS is applied [2, 10].

The most enlightening data regarding alteration of the work of breathing, reduction in the activity of ventilatory muscles, and the efficiency of gas exchange with IPS, has come from Brochard et al. [5, 8, 9, 10] They have demonstrated marked reductions in lung plus imposed work of breathing in patients who are failing to wean. In a group of six patients with chronic obstructive pulmonary disease, IPS was titrated from zero to 20 cmH<sub>2</sub>O, with lung plus imposed work decreasing from  $20.6 \pm 19.4$  J/min to  $5.8 \pm 2.7$  J/min [9]. In addition, in a group of eight patients recovering from ventilatory failure who were non-weanable, they demonstrated marked improvements in gas exchange with IPS [7]. During demand flow spontaneous ventilation, PO<sub>2</sub> fell by  $20\pm30$  mmHg and PCO<sub>2</sub> increased by  $11 \pm 14$  mmHg from controlled ventilation settings. However, when 10 cmH<sub>2</sub>O of IPS was applied, PO<sub>2</sub> fell by only  $1 \pm 15$  mmHg and the PCO<sub>2</sub> increased by  $5\pm7$  mmHg. Brochard et al. also demonstrated an alteration in high/low frequency distribution of EMG activity of the diaphragm (using an esophageal electrode) and the sternomastoid muscle (using a skin electrode) as IPS is increased [9]. We have also noted similar changes in the magnitude of the integrated EMG signals from electrodes directly implanted in the costal and crural diaphragms of awake sheep 24 to

48 h post-thoracotomy, as IPS was increased [12]. This preliminary data supports the widely held opinion that IPS unloads ventilatory muscles during pressure supported ventilation.

In this issue, Tokioka et al. [13] address the use of IPS as a mode of ventilation in acute respiratory failure. They compared the efficiency of IPS to assist/control ventilation. With IPS they were able to maintain the same level of gas exchange, but with a lower peak airway pressure  $(38 \pm 4 \text{ cm H}_2\text{O} \text{ vs.})$  $42\pm7$  cmH<sub>2</sub>O), a larger tidal volume (908±179 ml vs  $633 \pm 96$  ml), and a lower respiratory rate ( $15 \pm 4/min$ vs.  $24 \pm 5$ /min) than with assist/control. This article highlights one of the primary indications for the use of IPS, provision of mechanical ventilation, and adds to the increasing amount of literature advocating pressure-limited approaches to ventilation. The data provided by Tokioka et al. is consistent with the findings of McIntyre [3]. We have also used IPS on selected patients who were difficult to manage on volume-limited approaches to ventilation. In our patients, volumelimited ventilation without excessive peak airway pressures could not be achieved without sedation and paralysis. The use of IPS resulted in markedly decreased peak airway pressures and improved gas exchange. IPS should be used with caution during the acute phase of ventilatory failure. Since IPS is a true assist mode, no back-up rate is provided. It is true that a few ventilators have apneic ventilation modes (Puritan-Bennett 7200, Bear 5) and most have apneic alarms; but apneic ventilation may not be activated for 20-60 and alarms may not be noticed for even longer periods. Unless a patient has an intact ventilatory drive and requires only minor sedation, IPS for ventilatory maintenance must be employed cautiously.

With the introduction of modes permitting partial ventilatory support (IMV/SIMV) came the recognition that ventilator systems and artificial airways may impose a considerable workload on the spontaneously breathing patient. Reduction of this imposed work of breathing is another indication for IPS [2]. As demonstrated by Brochard et al. [9], titration of IPS can bring about unloading of the ventilatory muscles to non-fatiguing level. From Brochard's analysis of high/ low frequency EMG activity, it was noted that non-fatiguing activity of the diaphragm and sternomastoid muscles corresponded to a lung plus imposed workload of < 10 J/l. The utility of maintaining ventilatory loads at < 10 J/l does require further investigation. Numerous authors [14, 15, 16] have attempted to correlate ventilatory laods in this range (with the ability to wean from ventilatory support. The problem is that measuring work at the bedside is extremely complex and time-consuming. Fiastro et al. [17], utilizing lung models and volunteers, have provided algorithms for determining IPS levels based on ventilatory drive (peak spontaneous inspiratory flow) and endotracheal tube size. Similar data (determined using lung models) have been recently reported by McMahon et al. [18] and Robinson et al. [19]. All of these descriptions provide reasonable starting points for the titration of IPS but none take into account alterations in ventilatory drive once IPS has been instituted.

Using our present knowledge of pressure support to reduce the imposed work of breathing, we prefer to employ the data provided by Brochard et al. [9, 11] in determining the optimal IPS level. Our goals with IPS in this regard are two-fold: 1) to eliminate the clinical signs of impending or actual ventilatory muscle fatique; that is, titrating IPS until paradoxical breathing and use of accessory muscles (particularly the sternomastoid muscle) is eliminated; and 2) to establish the ventilatory pattern expected once the acute process is reveresed and extubation feasible. In most patients, this represents a spontaneous tidal volume of 300 to 600 ml with a respiratory rate of 15 to 25/min. Normally, from 5 to 20 cmH<sub>2</sub>O IPS is needed, with most patients requiring 10-15 cmH<sub>2</sub>O.

The effect that IPS has on weaning is difficult to quantify objectively and scientifically. Since IPS can reduce ventilatory load to a nonfatiguing level during CPAP trials, it should assist in muscle retraining without permitting fatigue. This should theoretically decrease the time course for weaning. It can be assumed that any approach that allows retraining without fatigue will have the same result.

A number of different approaches may be used when IPS is employed to facilitate weaning. McIntyre [3] recommends initially ventilating patients at IPS<sub>max</sub> (defined as the IPS level required to produce a tidal volume of 10-12 ml/kg) then gradually decreasing IPS over time to a minimal level as clinical status improves, with subsequent discontinuation. We commonly use SIMV plus IPS (to overcome imposed work) during periods where complete support (rest) is required; then, periodically provide "CPAP trials" with IPS to recondition ventilatory muscles, gradually increasing the trial periods until no SIMV is required, then extubating when the minimal level of IPS needed to overcome imposed work has been achieved. Similar results are obtained by alternating IPS levels; that is, using IPS<sub>max</sub> during rest periods and a lower level of IPS during "CPAP trials". Unfortunately, no specific data is available to guide the application of IPS during weaning. We believe the use of the modified "CPAP trials", as described above, to be most advantageous.

Does IPS make a clinical difference? We believe it does, primarily in that group of patients with ventilatory muscle dysfunction (acute or chronic), those with inappropriately small endotracheal tubes who require lengthy ventilator management and those patients acutely ventilated for respiratory failure where high peak airway pressures may be a concern, or who consistently oppose volume-limited approaches to ventilation. As with any new therapeutic modality, discussion seems to generate more questions than answers. The introduction of IPS raises many issued concerning approaches to weaning and methods of optimizing ventilatory muscle function, as well as providing ventilatory support in general. The data provided by Tokioka et al. will clarify some of the questions about its physiologic effects and application. Its use should be encouraged but should be based on available scientific data.

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