PRESSURE CONTROL VENTILATION: A REVIEW FOR THE INFORMED CLINICIAN

by David Wheeler RRT, NPS

The critical reader might ask, "Why is Wheeler discussing PCV?" The inherent implication being that this is such a mundane topic due to the fact that…"I already know everything I need to know about Pressure Control." Indeed, the informed clinician may be very well versed in all aspects of Pressure Control Ventilation, (PCV). I find however, that many knowledgeable practitioners, when asked probing questions are decidedly wanting in their current theoretical and clinical appreciation of this complex form of mechanical ventilation.

The clinical realization of PCV demands a comprehensive knowledge and understanding of the relationship between flow, time, pressure, compliance, resistance and the pulmonary time constant. In clinical application, PCV varies in a number of complex ways. The most widely employed variant of PCV is; patient or machine triggered, pressure limited and may be flow, volume or time cycled. PCV tidal volumes will vary with collective mechanical forces of the total respiratory system.

The delivered tidal volume will be inversely related to the compliance of the lung. A rough estimation of tidal volume is obtained by mutinying inflation pressure times lung compliance. The informed clinician will note that this number denotes the potential for volume as pressure transfer is dependent upon endogenous time constants. I must comment that to understand fully PCV one must have a working knowledge of the pulmonary time constant.

The Pulmonary Time Constant (TC, RC, t, Kt), is the product of the airways resistance times the static lung compliance in seconds. One time constant is the period that will allow for 63% of pressure to equilibrate. The time constant is the natural consequence of the lungs inherent tendency for expansion under pressure and resistance to airflow. The time constant will fluctuate in response to changes in resistance and compliance. In non-homogenous disease states, Time Constants may vary regionally. Indeed, gas moves into and out of the lung at a rate and in a fashion that is conditioned by the collective impedance of individual lung units and the pressure gradient between the ventilation system and the patient. (Yes, Anita this is patientcentric).

Lung units with high compliance will have a longer time constant; as will lung units with high airways resistance. Conversely, lung units with low compliance and low airways resistance will have a shorter time constant. The time constant of any individual lung unit may alter with the phase of ventilation and will vary with changes in the patient-ventilator system. (Think locally but treat globally.) Three to five time constants are required for adequate pressure equilibration within the patient-ventilator system.

During the initiation of the inspiratory phase of PCV, gas flows in a decaying fashion into the patient-ventilator circuit up to a clinician pre-selected pressure target. Once the target pressure limit has been reached, flow decay is flattened and terminal flow maintains a linear pressure graphic profile for the remaining inspiratory time.

Inspiratory flow is determined by the pressure gradient between the ventilation unit and the lung. Should the gradient be large the initial flow has a greater velocity than that which occurs in states of decreased pressure gradients. As the gradient between machine pressure and target pressure narrows inspiratory flow decays. Generally speaking, the inspiratory flow is estimated by dividing the pressure gradient by the inspiratory resistance. The decay in the inspiratory flow pattern is a predictable result of this diminishing pressure gradient. (In states of airflow obstruction clinician pre-set pressure targets may be achieved with lower terminal flow velocities.) Inspiratory flow, in a rapidly decaying graphic profile, continues until conditions relating to pressure and time are met.

The mindful clinician should remain aware of the fact that the inspiratory time (Ti) of the positive pressure profile is pre-set by the clinician. Decreasing the inspiratory portion of the duty cycle may lead to cessation of flow prior to satisfaction of three to five inspiratory time constants and a commensurate decrease in potential tidal volume. Lengthening Ti beyond the satisfaction of time constants, (past the point of pressure equilibration), will increases mean airway pressure however, this may not result in greater inspiratory volumes

The initial pressure gradient is also the greatest, which results in both high initial flow rates and a large fraction of the tidal volume being delivered early in the inspiratory cycle. This unique yet disproportionate delivery of tidal volume early in the inspiratory phase raises the mean airway and alveolar pressures. Both the MAP and PALV may be elevated in PCV as compared to volume targeted ventilation. Indeed both MAP and PALV may influence, to a great degree, the potential for oxygenation.

Tidal volume potential is determined by an applied positive pressure and inspiratory time but is primarily influenced by pulmonary system resistance and compliance. In systems presenting with high resistance flow decay is protracted commensurate with

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resistive elements. One must note that in such states initial flow profiles present with a shallower slope whilst terminal flow profiles decay with a pronounced linear shape. An examination of the graphic profile will reveal the appropriateness of Ti. Should the flow-time graphic depict gas delivery throughout the entire inspiratory cycle, increasing Ti should produce the largest tidal volume as the added Ti is acting to fulfill unsatisfied time constants

Tidal volume depends primarily on the pressure applied over the duty cycle and the compliance of the respiratory system. In states with decreased compliance tidal volume decreases and as compliance improves tidal volume will increase. This concept is extremely important. The re-perfusion of the pulmonary capillary bed, for example, can create dramatic and instantaneous changes in compliance and the patient-centered clinician must be mindful that any condition that alters lung compliance will have a dramatic effect on tidal volume. Volume kills.

Machine cycling frequency may influence tidal volume. The higher the respiratory rate the less efficient the mechanical potential for rectilinear pressure profiles and subsequent potential tidal volume may be diminished. One must be acutely aware that at extreme rates the potential for pressure delivery may fall due to a decline in net driving pressures in the face of iatrogenically induced auto-PEEP, diminished pressure gradients and critically reduced Ti. High respiratory rates may adversely alter potential gas exchange by altering dead space to tidal volume ratios. The obvious exception being in significant restrictive disease states where tidal volumes are potentially small and increased frequencies would result in a net increase in minute ventilation. The discrete clinician will note that the diameter of the airways will influence rate of flow. Realize that if the resistance is high, the flow will be lower and if the resistance is low flow potential will increase.

Prolonged Ti is to be avoided as it has bee associated with changes in lung architecture due to protracted alveolar shear stress. The role of shear and stress forces, and the potential contribution of these forces to ventilator-associated lung injury may be contraindications for the use of PCV in heterogeneous lung injury. Clearly, lower peak airway pressures produced by decaying flow velocity profiles does not guarantee protection in stress or shear force related ventilator associated lung injury, (VALI). The mechanism of inherently prolonged exposure to alveolar wall stress, vis a vie the unique flow velocity profile, may be responsible for VALI in PCV.

Having said the above I must add that, PCV with the inherent decaying flow velocity profile and decreased pressure limits may create the environment for a more uniform distribution of forces within the lung. This character may act to reduce the risk for ventilator induced lung injury. The decaying waveform may generate other advantages for the patient; increased MAP at reasonable volumes may increase oxygenation in diffuse lung states; improve global compliance and lessen the work of breathing. Improvements in lung compliance that result from the decaying flow velocity profile are in proportion to the percentage of potentially recruitable lung and the critical opening pressure of atelectatic lung units.

Decaying flow velocity profiles may have some bearing on the non-linear viscoelastic lung mechanics. Viscoelastance is the mechanical effect seen in the pressure drop present from the moment of cessation of flow to equilibrium of volume. This global pressure gradient is due to regional time constant disparity and tissue viscoelastic properties. This phenomenon has been associated with the severity of lung injury. PCV has demonstrated a predisposition for smaller viscoelastic pressure drops than most forms of Volume Control.

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PCV may lower ventilator imposed work of breathing in patients clinically manifesting elevated respiratory drive and increased respiratory rates. With flow rates that vary and are determined by pressure gradients, a change in patient inspiratory flow demand is satisfied by changes in delivered flow. This character of PCV flow eliminates the clinical event of flow starvation one may find in volume ventilation.

There are several aspects of PCV that seduce clinicians towards utilization of this fundamentally physiologically based mode. The variable and rapidly decaying flow velocity profile thought to reduce patient work of breathing, flow hunger, enhance the distribution of ventilation and limit peak airway pressures are very attractive character traits. PCV is known to create an increased mean airway pressure and potential for improved oxygenation status; with more volume delivered in the early portion of the inspiratory time PCV maintains the lung at a higher volume allowing for the potential recruitment of alveolar units for gas exchange.

I must comment that PCV has not demonstrated a clear superiority to other modes of mechanical ventilation. However, PCV is linked with lower peak airway pressures, a greater relationship with the fundament mechanical concepts of the lung, the potential for improved gas exchange and potential for the satisfaction of aberrant flow demands. PCV has not demonstrated greater lung saving potential.

This column is really a clinical brief intended to make the reader a bit more familiar with the notion and characteristics of PCV in the abstract sense. You my dear reader are encouraged to seek greater depths of knowledge and understanding at you leisure.

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Board of Registered Polysomnographic Technologists eliminates six-month waiting period and announces 2008 examination dates and deadlines.

(McLean, VA, September 20, 2007) — The Board of Registered Polysomnographic Technologists (BRPT) announced today the immediate elimination of a waiting period between exam attempts. "The Board is very pleased to be able to lift the exam waiting period requirement between exam attempts," explained Bonnie Robertson, CRT, RPSGT. "We believe that this will make it easier for applicants to schedule their exams." In addition, the board announced 2008 exam date windows. The dates of the exams will be:

- **March 17-29, 2008**
- **June 16-28, 2008**
- **September 15-27, 2008**
- **December 1-13, 2008.**

Those eligible to take the examination include professionals with 18 months of paid clinical experience in polysomnography, credentialed professionals with 6 months of paid clinical experience in polysomnography from a BRPT-accepted health-related field such as nursing, respiratory care and electroneurodiagnostics, or graduates of programs with special recognition in polysomnography, as accredited by the Commission on Accreditation of Allied Health Education Programs (CAAHEP).