

**EFFECTS OF PRESSURE CONTROLLED AND
VOLUME CONTROLLED VENTILATION ON
RESPIRATORY MECHANICS AND SYSTEMIC
STRESS RESPONSE DURING PRONE POSITION
SURGERIES**

**DISSERTATION SUBMITTED FOR THE DEGREE OF
DOCTOR OF MEDICINE**

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THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

CERTIFICATE BY GUIDE

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This is submitted to The Tamilnadu DR.M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Doctor of Medicine degree branch X (Anaesthesiology) to be held in MAY 2019.

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INTRODUCTION:

Volume-controlled ventilation (VCV) that utilizes a constant flow to deliver a target tidal volume ensures minute ventilation, but reduced thoracic compliance of the patient may result in high airway pressures and increase the risk of ventilator-induced lung injury. Pressure-controlled ventilation (PCV), with its high and decelerating inspiratory flow pattern, has faster tidal volume delivery and diffused gas distribution. The same tidal volume setting, delivered by pressure controlled ventilation (PCV) versus volume controlled ventilation (VCV), may result in a lower peak airway pressure and reduced risk of barotrauma, but variable tidal volume may be generated.

Prone position during general anesthesia for special surgical operations may be related with increased airway pressure, decreased pulmonary and thoracic compliance that may be explained by restriction of chest expansion and compression of abdomen during prone position . The optimum ventilation mode for anesthetized patients in prone position was not described and studies comparing volume controlled ventilation (VCV) and pressure controlled ventilation (PCV) during prone position are limited.

We hypothesized that pressure controlled ventilation (PCV) instead of volume controlled ventilation (VCV) during prone position could achieve

lower airway pressures and reduce the systemic stress response. In this study, we aimed to compare the effects of pressure controlled ventilation (PCV) and volume controlled ventilation(VCV) modes during prone position on respiratory mechanics, oxygenation, and hemodynamics, as well as blood cortisol, insulin and lactate and glucose levels.

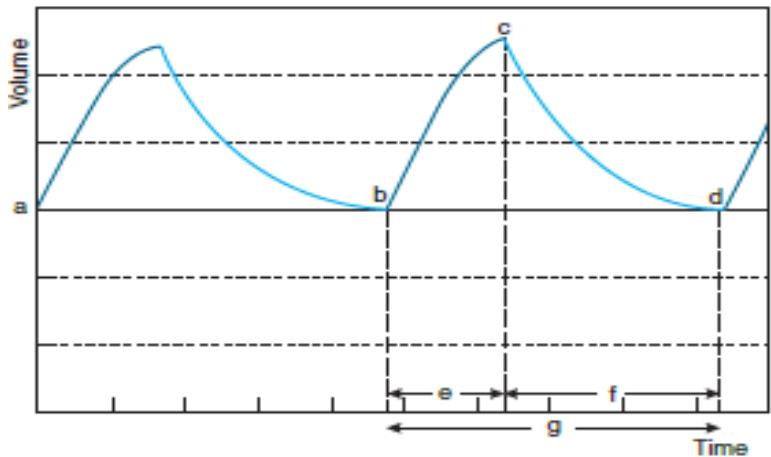
VENTILATION MODES

A mechanical ventilation mode is defined as “a specific combination of breathing pattern, control type, and operational algorithms”. With the advent of microprocessor-controlled ventilators, the variety and complexity of modes has dramatically increased. It is important to understand mechanical ventilation modes in order to match breath delivery to specific clinical application and patient needs.

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Volume-Controlled Ventilation

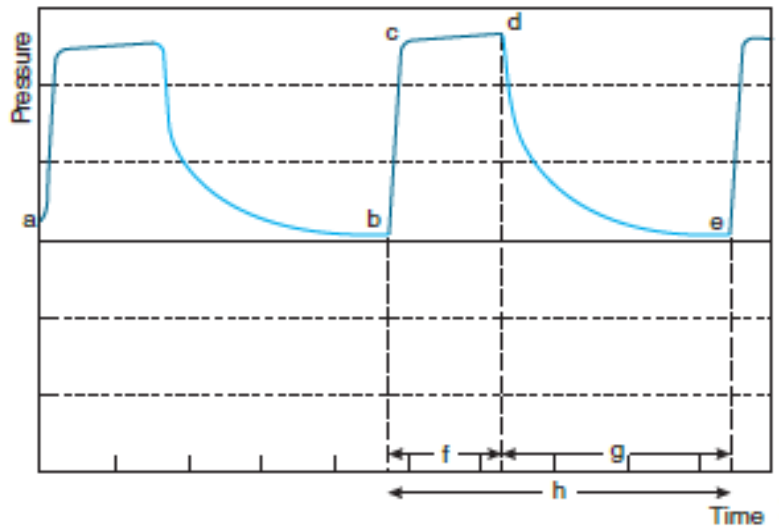
Volume-controlled ventilation allows the clinician to set the volume to delivered with each breath with fixed volume delivery and variable pressure depending upon the patient’s pulmonary compliance and airway resistance, Volume will remain constant in spite of changes in the patient’s condition. The advantage of volume control is the ability to regulate both tidal volume and minute ventilation (a product of tidal volume and frequency).



Volume time scalar in volume control mode (a: beginning inspiration, b: end-expiration/beginning inspiration, c: end-inspiration/beginning expiration, d: end-expiration, e: inspiratory time, f: expiratory time, g: total cycle time.)

Pressure-Controlled Ventilation

The pressure-controlled mode allows the clinician to set a peak inspiratory pressure for each mechanical breath. Since pressure remains constant, volume and minute ventilation will vary with changes in the patient's pulmonary compliance or airway resistance. Should the patient's compliance worsen or airway resistance increase, the peak inspiratory pressure terminates soon and the tidal volume and minute ventilation decreases. The advantage of the pressure-controlled mode is that the lungs can be protected from excessive pressures, preventing ventilator-induced lung injury (VILI).



A pressure-time scalar in pressure-controlled mode. A pressure-time scalar in pressure-controlled mode. a: beginning inspiration, b: end-expiration/beginning inspiration, c to d: pressure plateau, d: end-inspiration/beginning expiration, e: end-expiration, f: inspiratory time, g: expiratory time, h: total cycle time

Airway resistance

Airway resistance is defined as airflow obstruction in the airways. In mechanical ventilation, the degree of airway resistance is primarily affected by the length, size, and patency of the airway, endotracheal tube, and ventilator circuit.

Factors Affecting Airway Resistance

Airway resistance causes obstruction of airflow in the airways. It is increased when the patency or diameter of the airways is reduced.

Obstruction of airflow may be caused by:

- (1) changes inside the airway (e.g., secretions),
- (2) changes in the wall of the airway (e.g., neoplasm of the bronchial muscle structure),
- (3) changes outside the airway (e.g., tumors surrounding and compressing the airway) .

When one of these conditions occurs, the radius of the airway decreases and airway resistance increases. According to the simplified form of Poiseuille's Law, the driving pressure (DP) to maintain the same airflow ($V\dot{\#}$) must increase by a factor of 16-fold when the radius (r) of the airway is reduced by only half of its original size.

- One of the most common causes of increased airway resistance is chronic obstructive pulmonary disease (COPD).
- This type of lung disease includes emphysema, chronic bronchitis, chronic asthma, and bronchiectasis.
- Mechanical conditions that may increase airway resistance include post intubation obstruction and foreign body aspiration.
- Infectious processes include laryngotracheobronchitis (croup), epiglottitis, and bronchiolitis. Table 1-1 lists three categories of clinical conditions that increase airway resistance.
- Normal airway resistance in healthy adults is between 0.5 and 2.5 cm H₂O/L/sec. It is higher in intubated patients due to the smaller diameter of the endotracheal (ET) tube.
- Airway resistance varies directly with the length and inversely with the diameter of the airway or ET tube.
- In the clinical setting, the ET tube may be shortened for ease of airway management, reduction of mechanical deadspace, and reduction of airway resistance.
- However, the major contributor to increased airway resistance is the internal diameter of the ET tube.

- Therefore, during intubation, the largest appropriate size ET tube must be used so that the airway resistance contributed by the ET tube may be minimized.
- Once the ET tube is in place its patency must be maintained, as secretions inside the ET tube greatly increase airway resistance. Besides the ET tube, the ventilator circuit may also impose mechanical resistance to airflow and contribute to total airway resistance. This is particularly important when there is a significant amount of water in the ventilator circuit due to condensation.

TABLE 1

TABLE 1-1 Clinical Conditions That Increase Airway Resistance	
Type	Clinical Conditions
COPD	Emphysema Chronic bronchitis Asthma Bronchiectasis
Mechanical obstruction	Postintubation obstruction Foreign body aspiration Endotracheal tube Condensation in ventilator circuit
Infection	Laryngotracheobronchitis (croup) Epiglottitis Bronchiolitis

Lung compliance

Lung compliance is volume change (lung expansion) per unit pressure change (work of breathing), and it is calculated by

$$C = DV/DP,$$

where C = compliance,

DV = volume change, and DP = pressure change.

Most ventilators can measure and show the static and dynamic compliance values on the data or graphic display

TABLE 2

TABLE 1-2 Method to Measure Static and Dynamic Compliance
(1) Obtain corrected expired tidal volume.
(2) Obtain plateau pressure by applying inspiratory hold or occluding the exhalation port at end-inspiration.
(3) Obtain peak inspiratory pressure .
(4) Obtain positive end-expiratory pressure (PEEP) level, if any.
Static Compliance = $\frac{\text{Corrected Tidal Volume}}{(\text{Plateau Pressure} - \text{PEEP})}$
Dynamic Compliance = $\frac{\text{Corrected Tidal Volume}}{(\text{Peak Inspiratory Pressure} - \text{PEEP})}$

- **Low Compliance.** Low compliance (high elastance) means that the volume change is small per unit pressure change.

- Under this condition, the lungs are stiff or noncompliant. The work of breathing is increased when the compliance is low.
- In many clinical situations (e.g., acute respiratory distress syndrome or ARDS), low lung compliance is associated with **refractory hypoxemia**
- Low compliance measurements are usually related to conditions that reduce the patient's functional residual capacity.
- Patients with noncompliant lungs often have a restrictive lung defect, low lung volumes, and low minute ventilation. This condition may be compensated by an increased frequency.

High Compliance.

- High compliance means that the volume change is large per unit pressure change.
- In extreme high compliance situations, exhalation is often incomplete due to lack of elastic recoil by the lungs.
- Emphysema is an example of high compliance where the gas exchange process is impaired. This condition is due to chronic air trapping, destruction of lung tissues, and enlargement of terminal and respiratory bronchioles.

- High compliance measurements are usually related to conditions that increase the patient’s functional residual capacity and total lung capacity.
- Patients with extremely compliant lungs often develop airflow obstruction, incomplete exhalation, air trapping, and poor gas exchange.

TABLE 3

TABLE 1-3 Clinical Conditions That Decrease the Compliance	
Type of Compliance	Clinical Conditions
Static compliance	ARDS Atelectasis Tension pneumothorax Obesity Retained secretions
Dynamic compliance	Bronchospasm Kinking of ET tube Airway obstruction

Static and Dynamic Compliance

Assessment of compliance can be divided into static compliance and dynamic compliance measurements..

Static Compliance.

Static compliance is calculated by dividing the volume by the pressure (i.e., plateau pressure) measured when the flow is momentarily stopped.

When airflow is absent, airway resistance becomes a non-factor. Static compliance reflects the elastic resistance of the lung and chest wall.

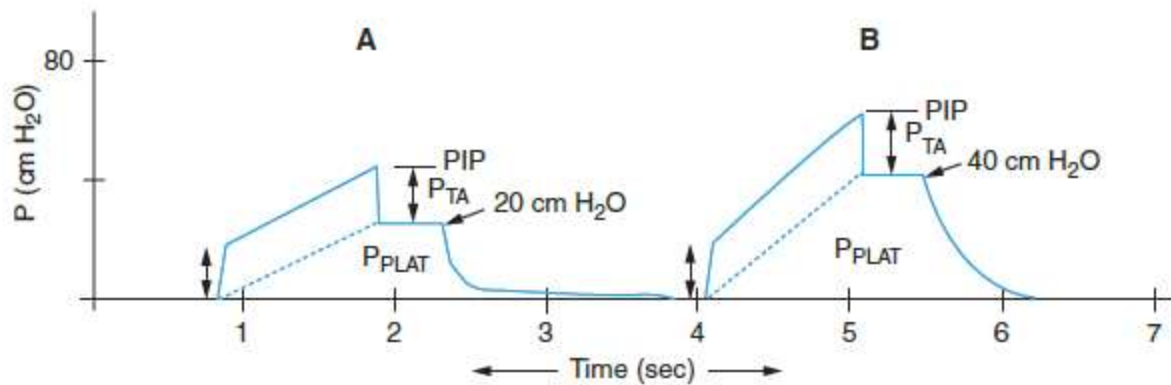
Dynamic Compliance.

Dynamic compliance is calculated by dividing the volume by the pressure (i.e., peak inspiratory pressure) measured when airflow is present. Since airflow is present, airway resistance becomes a factor in the measurement of dynamic compliance. Dynamic compliance therefore reflects the condition of airway resistance (nonelastic resistance) as well as the elastic properties of the lung and chest wall (elastic resistance).

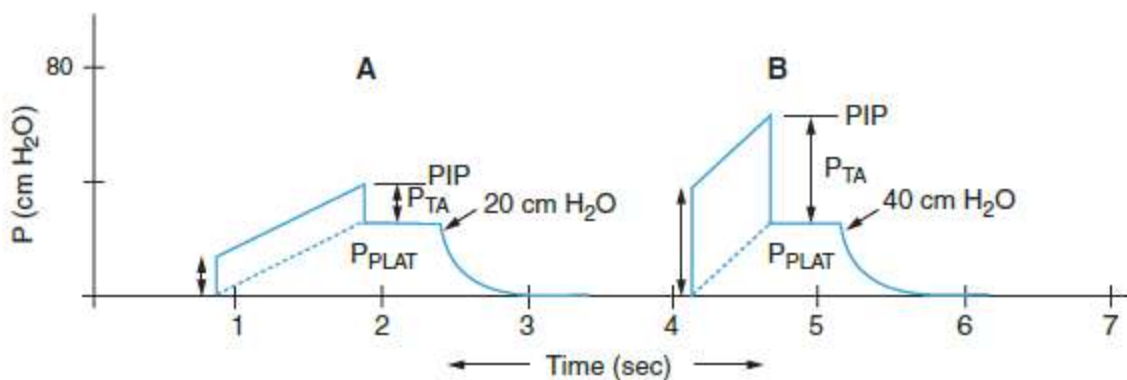
Plateau and Peak Inspiratory Pressure.

In general, conditions causing changes in plateau pressure and static compliance invoke similar changes in peak inspiratory pressure and dynamic compliance.

- For example, atelectasis causes an increase of plateau and peak inspiratory pressures. Since the plateau and peak inspiratory pressures are increased, the calculated static and dynamic compliance measurements are decreased.
- When atelectasis is resolved, the plateau and peak inspiratory pressures return to normal.
- In conditions where the airflow resistance is increased (e.g., bronchospasm), the peak inspiratory pressure is increased while the plateau pressure stays unchanged.
- Since the peak inspiratory pressure is increased, the dynamic compliance is decreased. The static compliance stays the same because there is no change in the plateau pressure.
- When bronchospasm is resolved, the peak inspiratory pressure and dynamic compliance measurements return to their previous states.



In conditions where the lung compliance is decreased (e.g., atelectasis), the plateau pressure (P_{PLAT}) and peak inspiratory pressure (PIP) are both increased (from A to B).



In conditions where the airflow resistance is increased (e.g., bronchospasm), the peak inspiratory pressure (PIP) is increased while the plateau pressure (P_{PLAT}) stays unchanged (from A to B).

VOLUME CONTROLLED VERSUS PRESSURE CONTROLLED

Volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) are not different ventilatory modes, but are different control variables within a mode.

TABLE 4

	PCV	VCV
TIDAL VOLUME	variable	Constant
Peak inspiratory pressure	constant	Variable
P Plateau	Constant	Variable
Flow pattern	variable	Set
Peak flow	variable	Set
Inspiratory time	Set	Set
Minimum rate	Set	Set

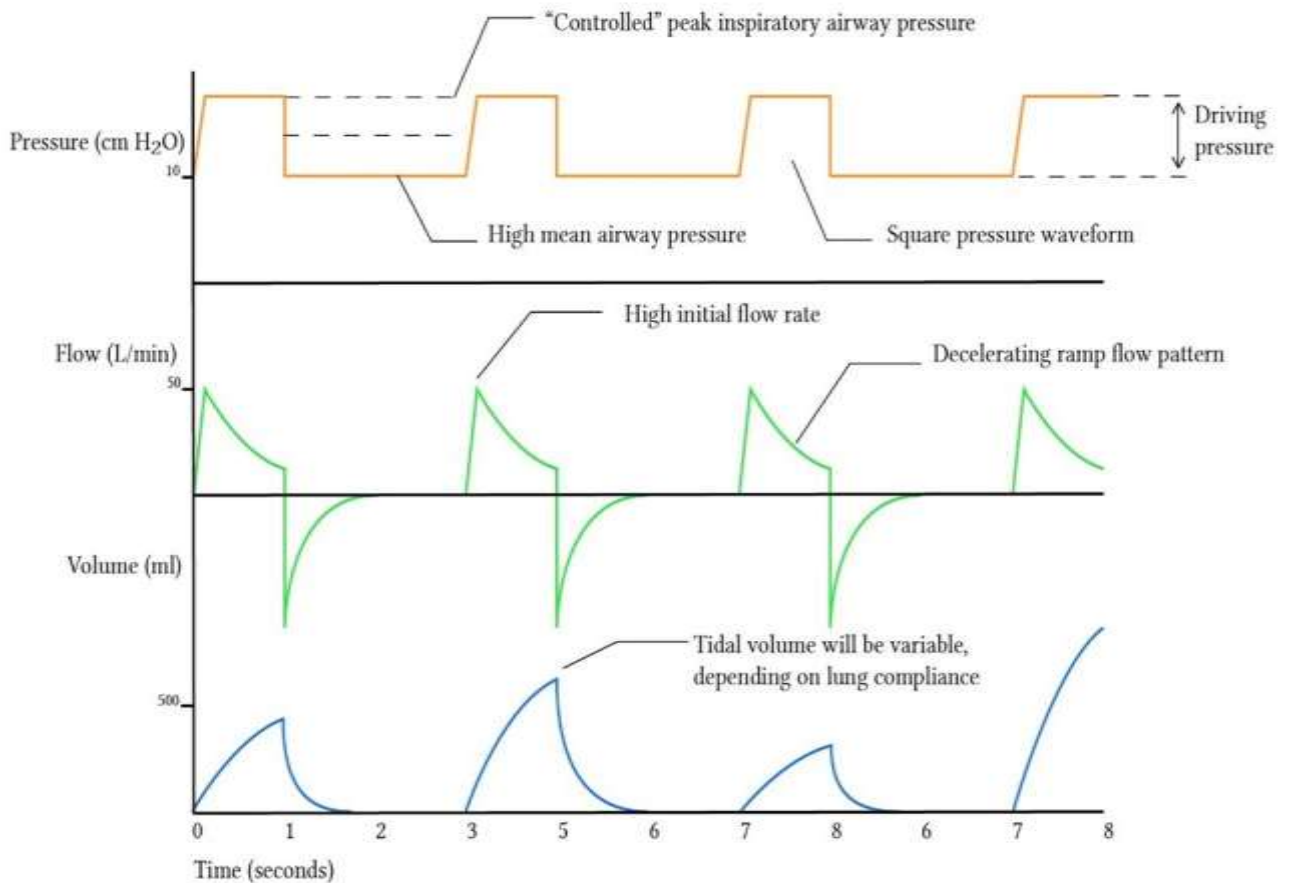
- VCV offers the safety of a pre-set tidal volume and minute ventilation but requires the clinician to appropriately set the inspiratory flow, flow waveform, and inspiratory time.
- During VCV, airway pressure increases in response to reduced compliance, increased resistance, or active exhalation and may increase the risk of ventilator-induced lung injury.
- PCV, by design, limits the maximum airway pressure delivered to the lung, but may result in variable tidal and minute volume. During PCV the clinician should titrate the inspiratory pressure to the measured tidal volume, but the inspiratory flow and flow waveform are determined by the ventilator as it attempts to maintain a square inspiratory pressure profile

Pressure control modes of ventilation

- In a pressure controlled mode of ventilation, the inspiratory pressure is the control variable, and is maintained during the inspiratory phase.
- As a result of this, the pressure waveform is “square”. This increases the mean airway pressure (i.e. the area under the pressure/time graph is greater) During the early inspiratory phase,

the ventilator provides a high inspiratory flow rate to rapidly achieve the pressure limit variable.

- In order to maintain this pressure, the flow rate needs to decrease over the course of inspiration, and it generally takes the shape of a downsloping ramp.
- If the inspiratory time is long enough, the flow will eventually reach zero, as demonstrated in the ventilator graphic below.
- When the flow rate reaches zero, i.e. in the absence of flow, the constant prescribed pressure is in equilibrium with the peak alveolar pressure at the end of the breath and equals the plateau pressure.



Advantages of PCV

PCV has a series of theoretical advantages in ventilating a patient in whom hypoxia and poor lung compliance are major contributors to respiratory failure:

- Increased mean airway pressure, which improves oxygenation (as discussed elsewhere the mean airway pressure is one of the main determinants of the oxygenation improvement associated with positive pressure ventilation). Because the dominant influence on mean airway pressure is PEEP, this is not a massive advantage

until you start using extremely inspiration-heavy I:E ratios, eg. 1:1.5 or 1:1

- Increased duration of alveolar recruitment: with a square pressure waveform, alveoli are opened earlier and remain open for longer, allowing better gas exchange (i.e. the early high airway pressure allows the alveoli to benefit from the high pressure for longer)
- Pressure limited ventilation may protect against barotrauma; the fixed pressure level defends against pressure-induced alveolar injury. In other words, because the pressure level is controlled, there should never be a time where the patient is suffering from extremely high pressures.
- Work of breathing and patient comfort may be improved because the initial high flow rate prevents the "flow starvation" type of patient-ventilator dyssynchrony, where the patient's demand for fresh gas flow goes unmet by the ventilator's inappropriately low flow limit. [Cinella et al \(1996\)](#) found that this was the case at low-moderate tidal volumes, i.e. wherever the pressure control mode competes with a volume control mode which is set to low flow, the patient's work of breathing will be better with a square pressure waveform.

Additionally, the pressure control variable allows for a significant leak. The ventilator will automatically adjust the inspiratory flow rate to accommodate for even a significant leak in an effort to maintain the prescribed pressure level. This has advantages in settings where circuit leak is inevitable, for example during bronchoscopy or while ventilating a patient with a bronchopleural fistula.

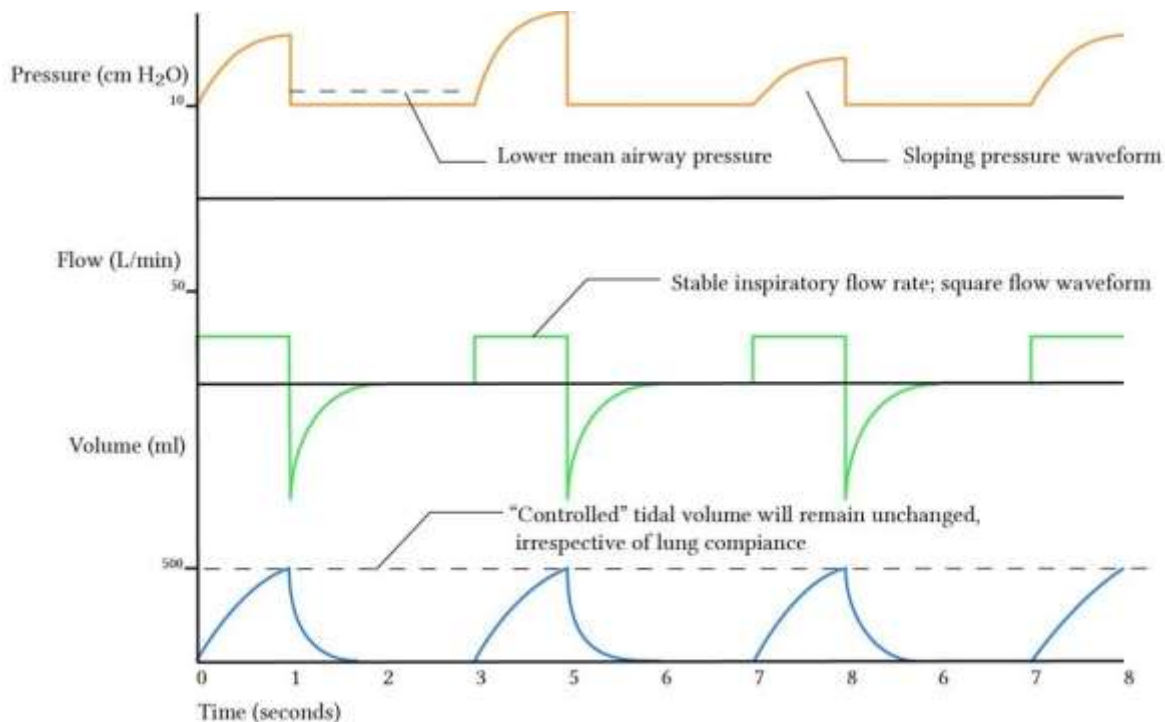
Disadvantages of PCV

- Tidal volume is dependent on respiratory compliance; and it may vary substantially over the course of mechanical ventilation, requiring frequent adjustments. With PCV, without constant attention to the ventilator settings tight control of PaCO₂ may be difficult to achieve.
- Uncontrolled volume may result in volutrauma, i.e. if the lung compliance improves suddenly the ventilator may deliver volumes which distend the most compliant lung units beyond their elastic limit.
- A high early inspiratory flow may breach the pressure limit if airway resistance is high. The high initial flow rate will create a high pressure due to airway resistance, which could be high enough to blow the pressure alarm limit. If airway resistance is a problem, a more gentle inspiratory flow rate might be beneficial.

Volume control modes of ventilation

By definition, in volume control modes the tidal volume is the defined variable which is used by the ventilator to give feedback to the solenoid valve circuits. As volume and flow are inextricably linked, the volume control modes are generally constant flow modes, i.e. the ventilator delivers flow which is constant, and stops this flow when the desired volume is achieved. Because pressure is not controlled or regulated in any way, the pressure waveform takes a parabolic sloping shape as the lungs distend during a breath.

The pressure waveform is highly variable during volume control ventilation, changing shape depending on lung compliance and airway resistance. As a result, it offers a significant amount of information. Interpretation of the pressure waveform is discussed elsewhere.



Advantages of volume control ventilation

- **Guaranteed tidal volumes produce a more stable minute volume.** The reliability of the minute volume makes this mode of ventilation more appropriate in situations where careful control of PaCO₂ is of importance. Volume controlled modes were the standard of care for patients with severe traumatic brain injury where tight PaCO₂ control is necessary.
- **The minute volume remains stable over a range of changing pulmonary characteristics.** If airway resistance fluctuates significantly (eg. in the course of therapy for status asthmaticus) this mode has the advantage of maintaining a reliable minute volume.

- **The initial flow rate is lower than in pressure-controlled modes.** This is an advantage if airway resistance is high; blowing more slowly into the tight bronchi does not produce a high resistance-related early pressure peak, and potentially prevents an early termination of the breath by the pressure alarm limit.

Disadvantages of volume control ventilation

- **The mean airway pressure is lower** with volume control ventilation, due to the slopy shape of the pressure waveform. This can theoretically be a disadvantage in patients who have severe hypoxia; in those people one might want to use a pressure-controlled mode instead. One wa of getting around this is to use an inspiratory pause, which is discussed elsewhere (in summary, it doesn't work and possibly increases the work of breathing).
- **Recruitment may be poorer in lung units with poor compliance.** Units with a long time constant and poor compliance may remain unrecruited until very late in the inspiratory phase when pressure approaches its maximum value. These units will have little time for gas exchange before the ventilator cycles to expiration. From this, one might expect that with a volume-controlled mode the degree of atelectasis will be greater than with a pressure controlled mode, peak aiway pressures being equal.

- **In the presence of a leak, the mean airway pressure may be unstable.** The constant flow used during VCV may not be able to compensate for an intermittent leak. Consider: if the leak flow rate is equal to the inspiratory flow rate, there will be no volume delivered.
- **Insufficient flow may give rise to patient-ventilator dyssynchrony.** In the presence of increased respiratory demand during the course of a breath, the ventilator may not meet the patient's need for increased flow.

REVIEW OF LITERATURE

1.Effects of pressure-controlled and volume-controlled ventilation on respiratory mechanics and systemic stress response during prone position

Oznur Sen,¹ Mefkur Bakan,^{corresponding author}² Tarik Umutoglu,² Nurdan Aydın,¹ Mehmet Toptas,¹ and Ibrahim Akkoc¹

Background

Prone position during general anesthesia for special surgical operations may be related with increased airway pressure, decreased pulmonary and thoracic compliance that may be explained by restriction of chest expansion and compression of abdomen. The optimum ventilation mode for anesthetized patients on prone position was not described and studies comparing volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) during prone position are limited. We hypothesized that PCV instead of VCV during prone position could achieve lower airway pressures and reduce the systemic stress response. In this study, we aimed to compare the effects of PCV and VCV modes during prone position on respiratory mechanics,

oxygenation, and hemodynamics, as well as blood cortisol and insulin levels, which has not been investigated before.

Methods

Fifty-four ASA I-II patients, 18–70 years of age, who underwent percutaneous nephrolithotomy on prone position, were randomly selected to receive either the PCV (Group PC, n = 27) or VCV (Group VC, n = 27) under general anesthesia with sevoflurane and fentanyl. Blood sampling was made for baseline arterial blood gases (ABG), cortisol, insulin, and glucose levels. After anesthesia induction and endotracheal intubation, patients in Group PC were given pressure support to form 8 mL/kg tidal volume and patients in Group VC was maintained at 8 mL/kg tidal volume calculated using predicted body weight. All patients were maintained with 5 cmH₂O PEEP. Respiratory parameters were recorded during supine and prone position. Assessment of ABG and sampling for cortisol, insulin and glucose levels were repeated during surgery and 60 min after extubation.

Results

P-peak and P-plateau levels during supine and prone positions were significantly higher and P-mean and compliance levels during prone position were significantly lower in Group VC when compared with Group PC. Postoperative PaO₂ level was significantly higher in Group

PC compared with Group VC. Cortisol levels were increased with surgery in both groups ($p < 0.05$) and decreased to baseline levels in Group PC while remained high in Group VC in the early postoperative period. Cortisol levels were significantly higher in Group VC during surgery and in the early postoperative period compared with Group PC.

Conclusion

When compared with VCV mode, PCV mode is associated with lower P-peak and P-plateau levels during both supine and prone positions, better oxygenation postoperatively, lower blood cortisol levels during surgery in prone position and in the early postoperative period. We concluded that PCV mode might be more appropriate in prone position during anesthesia.

2.Comparison of Pressure- and Volume-Controlled Ventilation in Laparoscopic Surgery: A Meta-analysis of Randomized Controlled Trial.

[Wang JP¹](#), [Wang HB](#), [Liu YJ](#), [Lou XP](#), [Wang XD](#), [Kong Y](#).

PURPOSE:

Volume-controlled ventilation (VCV) has been the traditional mechanical ventilation mode in laparoscopic surgery. Pressure-controlled ventilation (PCV) has been used more frequently in recent years, especially for patients with complicated conditions; however, evidence on whether

PCV is superior to VCV is still lacking. A meta-analysis was used to compare the effects of PCV and VCV on respiratory and hemodynamic parameters during laparoscopic surgery.

METHODS:

PubMed and Embase were each searched from their inception to December 2014 for randomized controlled trials comparing the effects of PCV and VCV on respiratory and hemodynamic parameters during laparoscopic surgery. Standard mean difference (SMD) with 95% confidence interval (CI) was calculated using a random effect model. Outcomes were assessed at three times: preoperative (T1), intraoperative (T2) and postoperative (T3). Respiratory mechanics (including peak airway pressure, plateau pressure, mean airway pressure, compliance, airway resistance, minute volume, end-tidal CO₂ tension and tidal volume) and hemodynamic parameters (including heart rate and mean arterial pressure) were calculated.

RESULTS:

Eight randomized controlled trials with a total of 428 participants, 214 cases using PCV and 214 cases using VCV, were included in the meta-analysis. No significant differences were detected between the groups in terms of hemodynamic parameters. In contrast, with respiratory mechanics, PCV was slightly but significantly associated with lower peak

airway pressure, higher compliance, lower airway resistance at T1, lower peak airway pressure, higher compliance, higher mean airway pressure at T2, lower peak airway pressure, lower mean airway pressure and higher end-tidal CO₂ tension at T3. For the rest of respiratory parameters, there were no statistical differences between the groups. Subgroup analysis by morbidly obese, type of operations and quality of studies, showed similar results.

CONCLUSIONS:

Our meta-analysis suggests that hemodynamic parameters are similar in patients who underwent laparoscopic surgery with PCV and VCV, but patients who had PCV exhibited mildly better respiratory data.

3.Effects of pressure-controlled and volume-controlled ventilation on respiratory mechanics and systemic stress response during laparoscopic cholecystectomy.

[Sen O¹](#), [Umutoglu T²](#), [Aydın N¹](#), [Toptas M¹](#), [Tutuncu AC³](#), [Bakan M²](#).

Abstract

Pressure-controlled ventilation (PCV) is less frequently employed in general anesthesia. With its high and decelerating inspiratory flow, PCV has faster tidal volume delivery and different gas distribution. The same tidal volume setting, delivered by PCV versus volume-controlled

ventilation (VCV), will result in a lower peak airway pressure and reduced risk of barotrauma. We hypothesized that PCV instead of VCV during laparoscopic surgery could achieve lower airway pressures and reduce the systemic stress response. Forty ASA I-II patients were randomly selected to receive either the PCV (Group PC, n = 20) or VCV (Group VC, n = 20) during laparoscopic cholecystectomy. Blood sampling was made for baseline arterial blood gases (ABG), cortisol, insulin, and glucose levels. General anesthesia with sevoflurane and fentanyl was employed to all patients. After anesthesia induction and endotracheal intubation, patients in Group PC were given pressure support to form 8 mL/kg tidal volume and patients in Group VC was maintained at 8 mL/kg tidal volume calculated using predicted body weight. All patients were maintained with 5 cmH₂O positive-end expiratory pressure (PEEP). Respiratory parameters were recorded before and 30 min after pneumoperitonium. Assessment of ABG and sampling for cortisol, insulin and glucose levels were repeated 30 min after pneumoperitonium and 60 min after extubation. The P-peak levels observed before (18.9 ± 3.8 versus 15 ± 2.2 cmH₂O) and during (23.3 ± 3.8 versus 20.1 ± 2.9 cmH₂O) pneumoperitoneum in Group VC were significantly higher. Postoperative partial arterial oxygen pressure (PaO₂) values are higher (98 ± 12 versus 86 ± 11 mmHg) in Group PC. Arterial carbon dioxide pressure (PaCO₂) values (41.8 ± 5.4 versus

36.7 ± 3.5 mmHg) during pneumoperitonium and post-operative mean cortisol and insulin levels were higher in Group VC. When compared to VCV mode, PCV mode may improve compliance during pneumoperitoneum, improve oxygenation and reduce stress response postoperatively and may be more appropriate in patients having laparoscopic surgery.

4. The effect of the prone position on pulmonary mechanics is frame-dependent.

[Palmon SC](#)¹, [Kirsch JR](#), [Depper JA](#), [Toung TJ](#).

Abstract

By compressing the abdomen and restricting chest wall movement, the prone position compromises pulmonary compliance. For spine surgery, placing the anesthetized patient into the prone position increases the risk of improper ventilation. In this study, we tested the hypothesis that the compromise in pulmonary compliance is related to the patient's body habitus and the surgical frame used to support the patient while in the prone position. Seventy-seven adult patients were divided into three groups according to body mass index: normal (n = 36) < or = 27 kg/m², heavy (n = 21) 28-31 kg/m², and obese (n = 20) > or = 32 kg/m². Patients were placed in the prone position supported by chest rolls, a Wilson frame, or the Jackson spinal surgery table (Jackson table)

according to the surgeon's preferences. Peak airway pressure (at the proximal endotracheal tube), pleural pressure (esophageal balloon), and mean arterial pressure were recorded in the supine position and prone position within 15 min of the turn. Dynamic mean (\pm SD) pulmonary compliance (mL/cm H₂O) decreased when turning from the supine to the prone position in all three body mass groups when using chest rolls (normal 37 \pm 5 to 29 \pm 6; heavy 43 \pm 2 to 34 \pm 4; obese 42 \pm 8 to 32 \pm 6) or the Wilson frame (normal 39 \pm 6 to 32 \pm 7; heavy 43 \pm 16 to 34 \pm 10; obese 36 \pm 11 to 28 \pm 9). The dynamic pulmonary compliance was not altered in patients positioned on the Jackson table. Regardless of body habitus, using the Jackson table for prone positioning was not associated with a significant alteration in pulmonary or hemodynamic variables. We conclude that moving patients from the supine to the prone position during anesthesia results in a decrease in pulmonary compliance that is frame-dependent but that is not affected by body habitus.

IMPLICATIONS:

We hypothesized that compromise in pulmonary compliance in the prone position is related to the patient's body mass index and the surgical frame used. In this study, we demonstrated that prone positioning during anesthesia results in a decrease in pulmonary

compliance that is frame-dependent but that is not affected by body mass index.

5. Pressure controlled vs. volume controlled ventilation during prone position in high-level spinal cord injury patients: a preliminary study

[Mirum Kim](#),¹ [Jieun Kim](#),¹ [Song Hwa Kwon](#),² and [Gunn-Hee Kim](#)¹

Prone positioning during operation is associated with significant challenges to anesthesiologists. Wilson Frame was introduced to reduce abdominal pressures. However, when it is applied inappropriately, it can increase peak airway pressure (PIP) and decrease dynamic compliance (C_{dyn}) by compromising the diaphragm movement [1].

Spinal cord injury (SCI) patients have a probability to get pressure sore operations in prone position under general anesthesia. Patients with quadriplegia have decreased chest wall and lung compliance, increased abdominal wall compliance. Their rib cage stiffness and paradoxical chest wall movements also result in an increase in the work of breathing. Expiratory muscle function is more compromised than inspiratory muscle function among subjects with quadriplegia and high paraplegia, which can result in ineffective coughing, accumulation of mucus and atelectasis. Vagal activity is high and baseline airway caliber is reduced in patients with quadriplegia. Pulmonary function in SCI is compromised by most lesions of the spinal cord, even in those with paraplegia .

Therefore, it is questionable which ventilator mode is more effective on systemic oxygenation and safer during prone position in these patients, but these issues have not been well established. We are to investigate which ventilation method is effective to decrease peak inspiratory pressure (PIP) and to maintain systemic oxygenation.

This study was performed from January 2013 to March 2014. After obtaining the Institutional Review Board's approval (IRB No. H-1302/027-001), 20 adult patients scheduled for debridement under general anesthesia were enrolled in this study. Patients who were neurologically stable SCI patients (quadriplegia and high level [above T4] paraplegia) for more than 2 years were included. Patients who were older than 75 years, or had severe pulmonary disease were excluded. Patients were randomized to either volume-controlled ventilation (VCV, n = 9) or pressurecontrolled ventilation (PCV, n = 9) group.

Patients were not premedicated. Patients were applied noninvasive blood pressure cuff, electrocardiogram, pulse oximetry in the operating room. General anesthesia was induced with 40 mg lidocaine, 1.5 mg/kg propofol, and 0.5 mg/kg rocuronium. After tracheal intubation with plain endotracheal tube, anesthesia was maintained with 0.8 to 1.2 vol% sevoflurane in N₂O and oxygen (FIO₂ = 0.5). Patient's lung was ventilated as follows; VCV group ventilated 10 ml/IBW (ideal body

weight) and PCV group ventilated at PIP adjusted to same tidal volume with VCV group. Thirty minutes after induction, arterial PaO₂, mean blood pressure (MBP), heart rate (HR), peak inspiratory pressure (PIP), mean inspiratory pressure (P_{mean}), respiratory rate (RR) and expired tidal volume (V_T) were recorded. Then, patient position was changed to prone and the same procedure was repeated as above.

Statistical analyses were performed with SPSS 16.0 (SPSS Inc., Chicago, IL). Data were presented as means ± SD or number of patients. Data between VCV and PCV group were compared using a paired t test after pairing patients. A P < 0.05 was considered statistically significant.

There were no statistical differences in age, body mass index, male-to-female ratio, quadriplegia -to- high level paraplegia ratio, anesthesia time, and operation time between the two groups.

The PIP increased after prone positioning in both groups. PIP in PCV group was lower than in VCV group. The RR and C_{dyn} decreased after prone positioning in both groups. The PaO₂ also decreased after prone positioning but SpO₂ was maintained above 98% in all patients during the study. There were no statistical differences in MBP and HR in both groups ([Table 1](#)).

Table 1

Mean Values of Intraoperative Variables

	VCV (n = 9)		PCV (n = 9)	
	Supine	Prone	Supine	Prone
V _T (ml)	667.4 ± 77.6	674.2 ± 82.1	652.4 ± 63.1	652.4 ± 63.1
PIP (cmH ₂ O)	15.1 ± 3.4	19.4 ± 4.4* (P = 0.001)	13.3 ± 3.4 [†] (P = 0.014)	17.1 ± 5.5* [†] (P = 0.001)(P = 0.043)
Pmean (cmH ₂ O)	4.2 ± 0.9	4.6 ± 0.8	4.8 ± 1.5	5.3 ± 1.0
ETCO ₂ (mmHg)	34.6 ± 1.3	33.8 ± 1.4	33.7 ± 1.5	32.9 ± 0.6
PaO ₂ (mm Hg)	349.5 ± 98.7	279.9 ± 106.5* (P = 0.001)	347 ± 90	217.5 ± 60* (P = 0.002)
RR (breaths/min)	9.9 ± 0.9	8.3 ± 1.3* (P = 0.005)	9.7 ± 1.3	7.9 ± 1.2* (P = 0.009)
MV (L/min)	6.4 ± 1.0	5.6 ± 0.6* (P = 0.017)	6.2 ± 1.6	5.2 ± 0.9 (P = 0.05)
Cdyn (ml/cmH ₂ O)	46.6 ± 13	36.4 ± 10.1* (P < 0.001)	51.5 ± 12.2 [†] (P = 0.039)	39.8 ± 10.5* (P < 0.001)
HR (beats/min)	86 ± 16.7	89.8 ± 9.5	95 ± 5.6	90 ± 14.2
MBP (mmHg)	79 ± 13.4	87.3 ± 14.0	85 ± 10.5	78 ± 11.3

Values are means ± SD. Cdyn: dynamic compliance of the respiratory system, ETCO₂: end-tidal carbon dioxide tension, MV: minute volume, PCV: patients with pressure-controlled ventilation, Pmean: mean airway pressure, Ppeak: peak airway pressure, Prone: at 30 minutes after the prone positioning, RR: respiratory rate, Supine: after induction of anesthesia in the supine position, VCV: patients with volume-controlled ventilation, V_T: tidal volume. *P < 0.05, compared with supine position within the group. †P < 0.05, compared with VCV group.

In the present study, ventilation in PCV mode decreased PIP more in the prone position, but this does not mean to improve systemic oxygenation. Although SpO₂ was maintained over 98% during operation time, it is difficult to conclude that due to a decreased PIP, PCV mode is more beneficial in quadriplegia or high-level paraplegia patients.

We presumed that our SCI patients have decreased pulmonary function. There are several studies supporting this presumption [3,4,5]. Almenoff et al. [3] presented SCI/pulmonary function investigation, studying 165 male military veteran outpatients. They found statistically significant correlations of SCI level with forced vital capacity (FVC), forced expired volume in 1 second (FEV₁), and peak expiratory flow rate (PEFR). Linn et al. [4] reported that the higher the SCI level, the more decreased in percent predicted forced vital capacity (FVC %). They investigated the relationship between SCI level and FVC % with 455 patients. Especially in SCI patients above the T4 level, the FVC % presented below 80% of normal findings [5]. Since our study included quadriplegia or above T4 level paraplegia patients, it may be postulated that at least FVC %, FEV₁, and PEFR were decreased in our patients. Another limitation of this study is small sample size. For resolving this problem, we used pairing methods in which two patients in each group were paired according to age, height, weight, and spinal cord injury level. Since the data showed normal distribution, we could use a paired t-test. Further larger-scale studies to explore these issues will be required.

In conclusion, it is suggested that when high-level SCI patients are ventilated under general anesthesia in prone position, PCV may decrease PIP more than VCV but oxygenation effect seems to be similar in both ventilation

Aim and objective:

To compare the perioperative hemodynamic values HR, SAP, MAP, and respiratory parameters during mechanical ventilation ETCO₂, P peak, P plateau, P mean and perioperative arterial blood gas PH, PaCO₂, PaO₂ and systemic stress response by cortisol, insulin, glucose, and lactate in adults undergoing spine surgical procedures

Statistical analysis

A pilot study was conducted to determine the sample size.

- In the power analysis, according to the results of this pilot study, a sample size of 30 patients in each group was calculated to be sufficient to achieve 80% power and an α value of 0.05 by taking a delta value of 3.5 and a standard deviation of 4.8 into consideration.
- SPSS software was used for statistical analysis
- Descriptive statistics were given in terms of numbers and percentages for categorical variables, and in terms of the mean, standard deviation and the median for the numerical variables.
- Comparison of two independent groups of variables was carried out using the Student T test when meeting the normal distribution

criteria, or by the Mann–Whitney U test when these criteria were not met.

- Relationship between numerical variables was assessed by means of the Spearman Correlation Analysis.
- The differences between categorical variables were evaluated by the Chi square analysis. Statistical α (alpha) significance level was accepted with the 'p' value below 0.05.

Material and methodology:

60 patients, undergoing elective spine surgical procedures under general anaesthesia are included in study.

Patients were randomly selected, by opening sealed envelopes, to receive either PCV (Group PC: n = 30) or VCV (Group VC: n = 30) mode of ventilation during anesthesia.

- On arrival at the operating room, standard monitoring was applied consisting of ECG, pulse oximetry and temperature.
- Intravenous midazolam 0.03 mg/kg was administered.
- After Allen test and local anesthetic infiltration, a cannula was placed to the radial artery and arterial pressure was monitorized.

- Blood sampling was made for baseline arterial blood gases (ABG) analysis and for cortisol, insulin and glucose levels to assess the systemic stress response.
- Anesthesia was induced with propofol 2 mg/kg and fentanyl 2 µg/kg. Suxamethonium 2mg/kg was given to facilitate tracheal intubation (with a reinforced endotracheal tube).
- Anesthesia was maintained with 0.5–1.0 MAC of sevoflurane in a mixture of oxygen and N₂O. Fentanyl 0.5–1 µg/kg was added to maintain systolic arterial pressure within $\pm 20\%$ of the baseline value.
- Atracurium 0.5mg/kg of loading dose and 0.1mg/kg of maintenance dose was administered to maintain train-of-four (TOF) at 0 and 1.
- The ventilatory parameters were set as respiratory rate: 12 breaths/min, inspiratory time /expiratory time: 1/2, positive end-expiratory pressure (PEEP): 5 cmH₂O and FiO₂: 50 %, which were constant during anesthesia in both groups.
- Patients in Group PC were given pressure support to form 8 mL/kg tidal volume (pressure support level was adjusted to maintain the same tidal volume during anesthesia); while Group

VC was maintained at 8 mL/kg tidal volume. Tidal volume was calculated using predicted body weight in both groups.

- Heart rate, arterial pressures, end-tidal carbon dioxide pressure (EtCO₂), peak, plateau and mean airway pressures (P-peak, P-plateau, P-mean respectively) were recorded after intubation during supine position.
- All patients were transferred to classic prone position on a flat table. Head was in neutral position, the arms were raised beside the head, parallel chest rolls were placed from shoulder to hip on both sides, the legs were bent at the knees and all pressure points (forehead, elbow, knees, etc.) were padded. After surgery was started and at the 30th minute of prone position, respiratory and hemodynamic parameters were recorded again and sampling for ABG, cortisol, insulin and glucose levels were repeated.
- For postoperative analgesia, paracetamol 1 g and tramadol 100 mg were administered and skin incisions were infiltrated with 10–15 ml of bupivacaine 0.5 % before closure.
- Anesthesia was maintained until the end of surgery. Neuromuscular blockade was antagonized with neostigmine 0.04 mg/kg and glycopyrolate 0.01 mg/kg and tracheal extubation was carried out when the patient was fully awake.

- Hemodynamic parameters were recorded and blood sampling for ABG, cortisol, insulin and glucose levels were repeated for the last time 60 min after extubation (without supplemental oxygen).

INCLUSION CRITERIA: 1.ASA 1 & 2

2.Men and women

3. Aged 18 TO 70 years ,

4.PROCCEDURE - spine surgical

procedures under general anaesthesia

EXCLUSION CITERIA:

1.ASA 3 & 4

2. history of cardiac, pulmonary, hepato-renal, endocrine, cerebrovascular and neuromuscular diseases

3.h/o thoracic surgery

4.emergency cases

5.bleeding/coagulation disorder

6. age<18,>70

7. obese,bmi>30kg/m²

8. patient refusal

STUDY DESIGN:

Randomised controlled prospective study

STUDY PLACE:

Govt Rajaji hospital, Madurai medical college.

COLLABORATING DEPARTMENT:

Department of orthopaedics and spine surgeries

PERIOD OF STUDY: 1YEAR

STATISTICAL ANALYSE PLAN

SPSS SOFTWARE

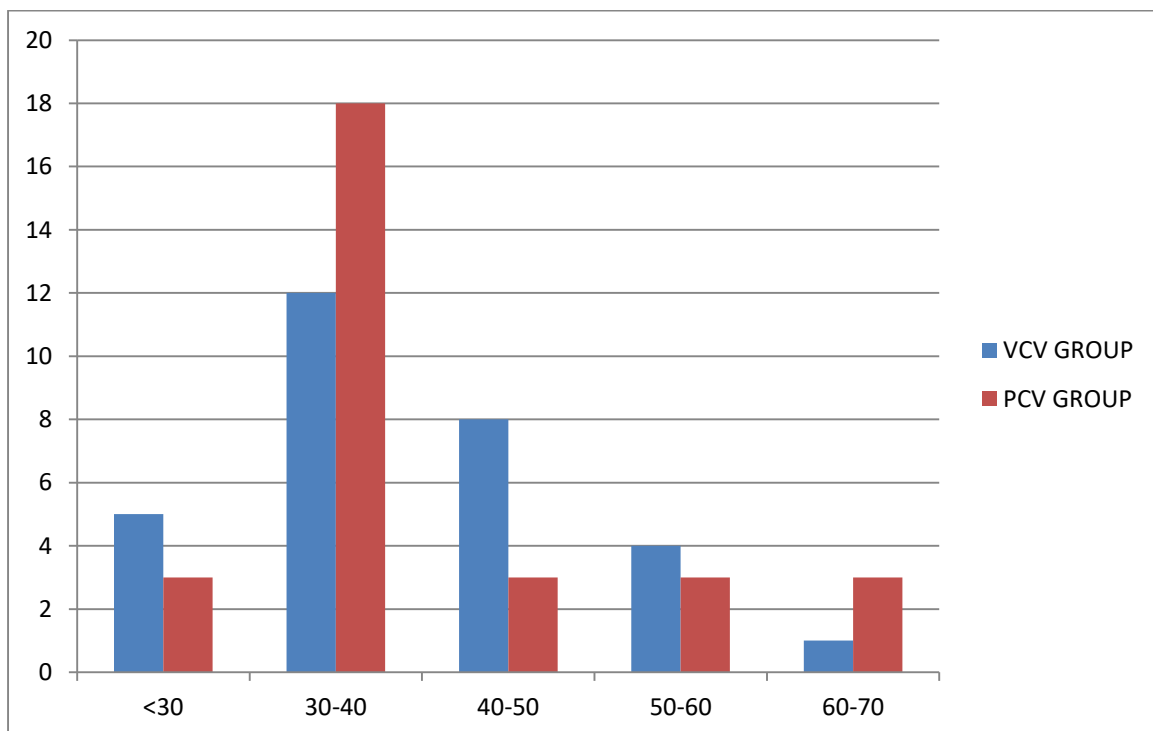
CONFLICT OF INTEREST :NIL

FINANCIAL SUPPORT :NIL

OBSERVATIONS

TABLE 5- AGE DISTRIBUTION

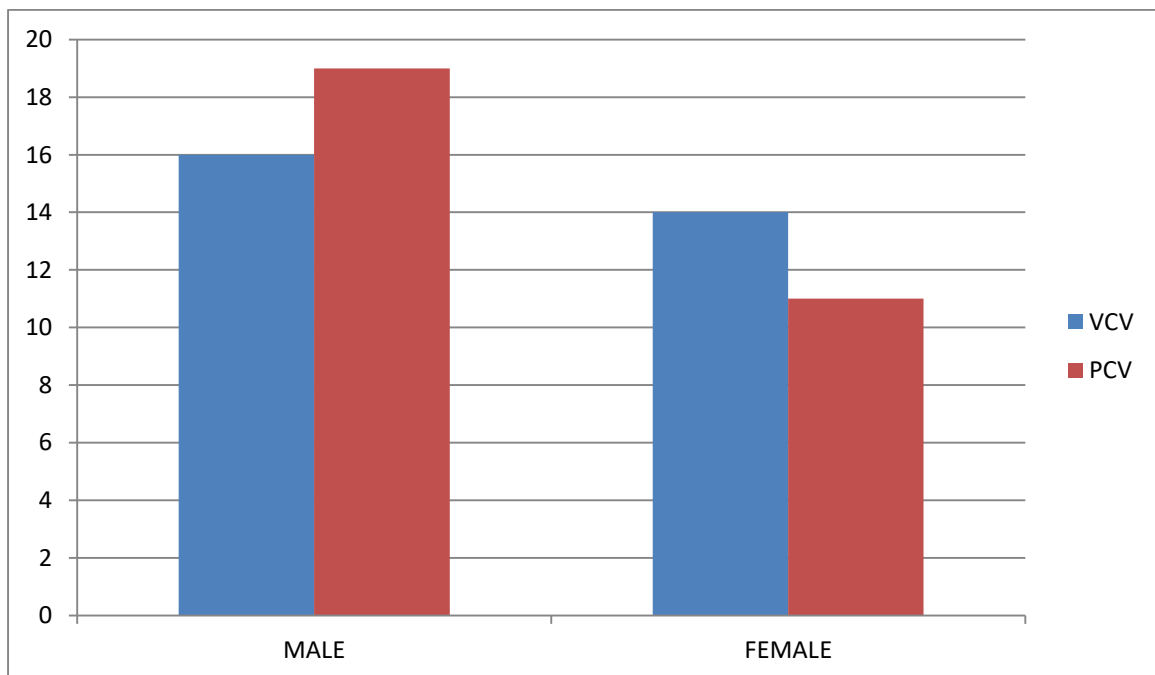
AGE	VCV GROUP	PCV GROUP
<30	5	3
30-40	12	18
40-50	8	3
50-60	4	3
60-70	1	3
MEAN±SD	39.27±9.45	36.63±7.31
P VALUE	0.232317	
	NOT SIGNIFICANT	



INFERENCE: both groups are comparable with age distribution

TABLE 6 - SEX DISTRIBUTION

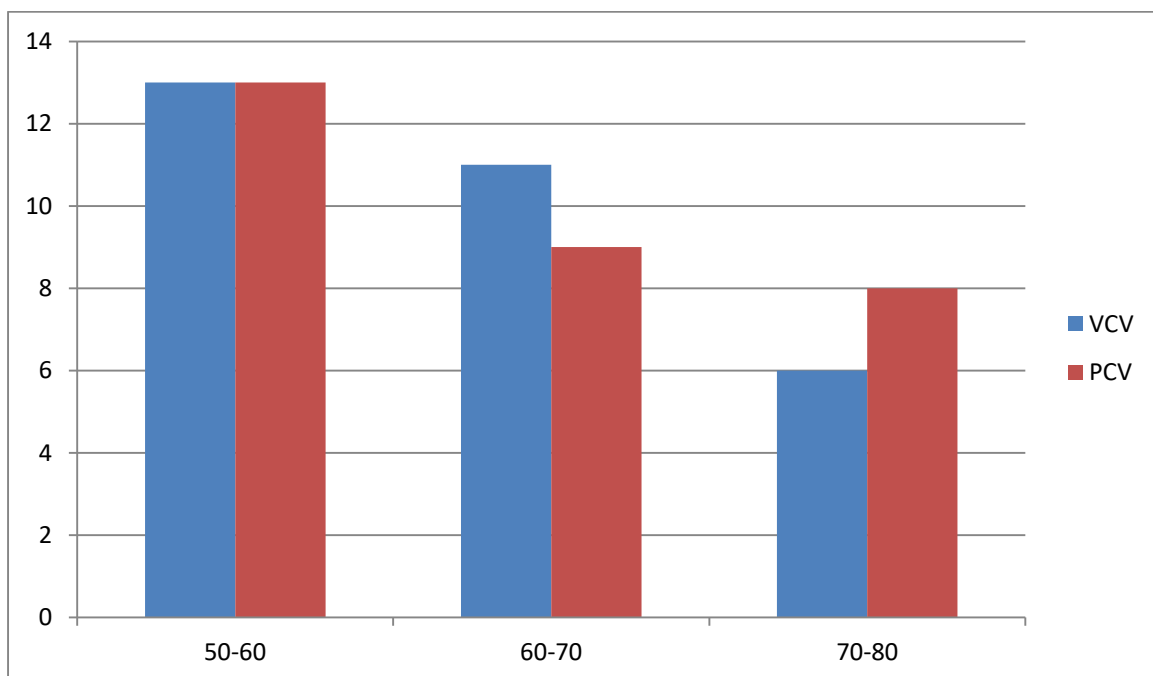
	VCV	PCV
MALE	16	19
FEMALE	14	11
TOTAL	30	30



INFERENCE: sex distribution is comparable in both groups

TABLE 7 - WEIGHT DISTRIBUTION

WEIGHT	VCV	PCV
50-60	13	13
60-70	11	9
70-80	6	8
MEAN + SD	60.27±5.3	60.8±5.5
P VALUE	0.141878	
	NOT SIGNIFICANT	



INFERENCE: weight distributions are comparable for both groups

TABLE 8**DURATION OF SURGERY**

DURATION (MINS)	VCV	PCV
<90	3	9
90-100	10	10
>100	17	11
TOTAL	30	30
MEAN	102.9	98.33
SD	9.95	11.38
P	0.1031 NOT SIGNIFICANT	

INFERENCE: Both the groups were comparable in duration of surgery

- The mean time for duration of surgery was comparable in both the Groups, for VCV mean duration were 102.9 ± 9.95 minutes and for Group PCV mean duration were 98.33 ± 11.38 minutes. P value of 0.1031 which is insignificant.

DURATION OF SURGERY

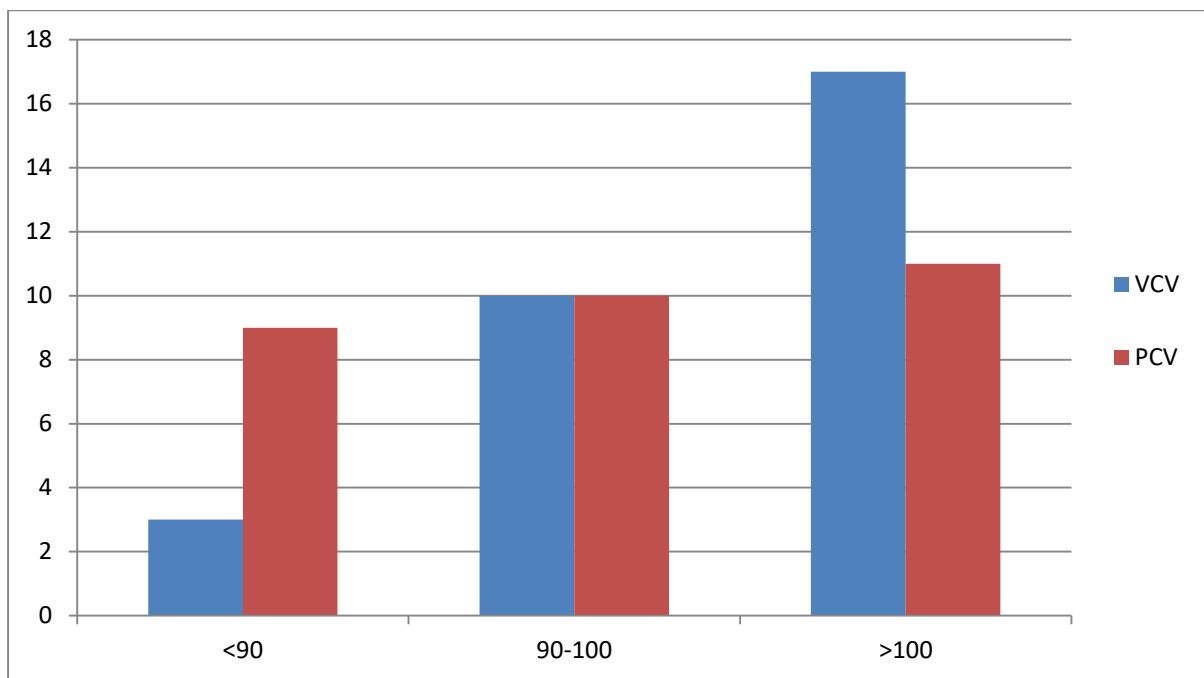


TABLE 9

	HEART RATE		
	PREOP	PRONE	POSTOP
VCV	79.07±7.91	86.4±6.83	88.9±5.94
PCV	79.83±7.47	84.47±6.94	88.63±5.56
P VALUE	0.35	0.14	0.43
	NS	NS	NS

TABLE 10

	SYSTOLIC ARTERIAL PRESSURE		
	PREOP	PRONE	POSTOP
VCV	122.33±11.01	118.07±6.92	132.1±6.62
PCV	123.57±8.7	119.47±6.98	130.87±5.3
P VALUE	0.32	0.22	0.2145056
	NS	NS	NS

TABLE 11

	MEAN ARTERIAL PRESSURE		
	PREOP	PRONE	POSTOP
VCV	88.63±3.93	92.07±4.73	86.93±4.6
PCV	88.63±3.93	92.07±4.73	86.77±4.66
P VALUE	0.5 NS	0.5 NS	0.44 NS

TABLE 12

	PaO2			PaCO2		
	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
VCV	90.57±3.31	168.37±18.25	90.83±2.73	39.9±2.92	37.13±4.66	42.57±3.87
PCV	90.43±3.15	166.7±16.17	90.6±2.63	40.13±1.91	37.73±3.84	42.9±3.51
P VALUE	0.44	0.35	0.37	0.36	0.29	0.36
	NS	NS	NS	NS	NS	NS

TABLE 13

	PH			ETCO2	
	PREOP	PRONE	POSTOP	SUPINE	PRONE
VCV	7.37±0.03	7.4±0.04	7.39±0.03	33.87±1.66	35±2.21
PCV	7.37±0.03	7.39±0.05	7.39±0.04	33.77±1.76	34.73±2.16
P VALUE	0.48	0.2	0.38	0.41	0.32
		NS	NS	NS	NS

TABLE 14

	P PEAK		P PLATEAU		P MEAN	
	SUPINE	PRONE	SUPINE	PRONE	SUPINE	PRONE
VCV	27.13±4.05	25.8±1.35	22.53±1.36	23.23±1.1	11.77±0.68	12±0.69
PCV	23.87±1.33	26.83±1.49	21.8±0.89	22.67±0.8	12±0.74	12.4±0.67
P VALUE	<0.05	0.003297	0.01	0.01	0.11	0.01
	significant	significant	significant	significant	NS	significant

TABLE 15

	CORTISOL(mg/dl)			INSULIN micu/ml		
	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
VCV	12.04±1.03	24.9±2.16	27.48±2.0 9	7.43±1.12	8.53±0.9 3	13.25±1.9 2
PCV	12.04±1.03	23.74±2.42	25.63±3.3 5	7.43±1.12	8.16±1.2 9	12.98±1.5
P VALUE	0.5	0.03	0.01	0.5	0.1	0.27
	NS	significant	significant	NS	NS	NS

	GLUCOSE (mg/dl)			LACTATE (mmol/l)		
	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
VCV	91.77±6.01	114.9±8.06	128.2±6.51	2.58±0.39	2.86±0.33	2.84±0.41
PCV	91.77±6.01	109.8±9.2	125.23±8.22	2.58±0.39	2.82±0.32	2.81±0.34
P VALUE	0.5	0.01	0.06	0.5	0.32	0.37
	NS	SIGNIFICANT	NS	NS	NS	NS

RESULTS

Respiratory parameters during mechanical ventilation are shown in Table [12,13](#). Perioperative arterial blood gas analysis, cortisol, insulin, glucose and lactate levels are shown in Table [13,14,15](#). No statistically difference was found in HR, systolic arterial pressure, mean arterial pressure, EtCO₂, Pao₂, PaCO₂, pH, and lactate levels between two groups. P-peak and P-plateau levels during supine and prone position were significantly higher in Group VCV when compared with Group PCV. P-mean levels during prone position were higher in Group PCV when compared with Group VCV.

Cortisol levels are significantly higher in prone position and postop periods in VCV group when compared to PCV group. Glucose levels are significantly higher in postop periods in VCV group compared to PCV group

DISCUSSION

Major findings of the present study are PCV mode was associated with lower P-peak and P-plateau levels during both supine and prone positions, and higher P-mean levels during prone positions when compared with VCV mode.

Mean airway pressure during inspiratory phase of respiration determines the recruitment of the collapsed alveoli and distribution of perfusion also it is a critical factor at gas exchange.

PCV maintains higher P-mean levels, which may improve oxygenation. Postoperative PO₂ levels, which were significantly higher in Group PC compared with Group VC, may indicate less alveolar de-recruitment.

Cortisol levels were increased with surgery in both groups, while this increase was significantly higher in Group VC. The lower cortisol levels with PCV mode usage may be due to the deceleration flow form, lower P-peak, higher oxygenation and higher compliance levels which may result in lower incidence of atelectasis and pulmonary strain.

It has been showed that; elevated respiratory pressures could lead to acute lung injury (ALI) (Licker et al. [2003](#); Oeckler and Hubmayr [2007](#)), higher P-peak levels could lead to lung edema after lobectomy (Van der Werff et al. [1997](#)), and also minimal increases in P-peak can result the

postoperative ALI risk (Fernandez-Perez et al. [2009](#)). Studies reported that P-plateau lower than 35 cmH₂O was associated with lower incidence of death and barotrauma in patients (Amato et al. [1998](#); Boussarsar et al. [2002](#)).

Regarding insulin levels we could not demonstrate a significant difference between groups. Literature regarding systemic stress response during general anesthesia with different ventilation modes is limited.

The meta-analysis that compared PCV and VCV modes during laparoscopic surgery by Wang et al. ([2015](#)) concluded that patients had PCV mode had lower P-peak and resistance accompanying higher compliance and P-mean levels. Although with subgroup analysis revealed the same results including morbid obese patients who underwent different kind of operations.

Another meta-analysis by Jiang et al. ([2016](#)) included 27 trials with 1643 cases that compared PCV and VCV modes on different positions (supine, prone and lateral) and conditions (laparoscopic surgery, one lung ventilation, etc.); concluded that PCV mode was associated with increased oxygen index and decreased alveolo-arterial oxygen difference (A-aDO₂). Subgroup analysis defining the effect of PCV mode on oxygenation concluded that patients having one-lung ventilation or

laparoscopic surgery, and obese patients significantly benefit from the use of PCV, but patients on special positions did not. However, there is insufficient data comparing PCV and VCV modes during prone position.

Jo et al. ([2012](#)) found lower P-peak levels during PCV in both supine and prone positions when compared with VCV. Kim et al. ([2014](#)) compared the PCV and VCV modes during prone position in high-level spinal cord injury patients and found that P-peak increased after prone positioning in both groups, but this increase was significantly higher in VCV group compared with PCV group. In our study, compliance levels were lower in both groups in prone position when compared with supine. Compliance levels in prone position were found to be statistically higher in PCV group. Jo et al. ([2012](#)) found similar results like our study additionally they found higher compliance levels in both supine and prone positions during PCV ventilation.

Conclusion

According to our findings, when compared to VCV mode, PCV mode is associated with lower P-peak and P-plateau levels during both supine and prone positions, better oxygenation postoperatively, lower blood cortisol levels during surgery in prone position and in the early postoperative period. We concluded that PCV mode might be more appropriate in prone position during anesthesia.

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PROFOMA

PATIENT AGE

WEIGHT (KG)

GENDER M/ F

GROUP PC / VC

	PREOP(before induction)	PRONE(30 min after prone position)	POSTOP(60 min after extubation without supplemental O2)
HEART RATE			
SAP			
MAP			
PaO2			
PaCO2			
PH			
ETCO2			
P Peak			
P Plateau			
P mean			
CORTISOL			
INSULIN			
GLUCOSE			
LACTATE			

MASTER CHART

VCV GROUP

	NAME	AGE	SEX	WEIGHT	ASA	HEART RATE		
						PREOP	PRONE	POSTOP
1	RAJAVEL	38	M	58	2	78	76	82
2	MALARVIZHI	42	F	62	2	85	76	78
3	RAMALAKSHMI	38	F	65	1	76	89	87
4	UMADEVI	33	F	59	1	79	76	87
5	ARUNKUMAR	35	M	49	1	66	83	82
6	MUTHUMARI	48	M	52	2	78	76	76
7	SUBRAMANI	30	M	53	2	78	82	84
8	RAMANI	46	F	58	2	85	87	88
9	VIGNESH	27	M	56	1	76	99	98
10	TAMILSELVI	40	F	58	2	79	93	90
11	KANNAN	40	M	58	2	66	82	88
12	KARTHIK	33	M	60	1	78	82	88
13	RAMYA	39	F	62	1	86	90	92
14	DHIVYA	24	F	64	1	92	88	99
15	MANIKANDAN	29	M	59	1	67	88	90
16	ANNAMALAI	32	M	59	2	71	83	89
17	MANICKAM	52	M	56	2	64	87	88
18	SIVAGAMI	32	F	64	2	78	89	90
19	LAKSHMI	53	F	65	2	94	86	98
20	SANTHOSH KUMAR	60	M	63	1	76	87	88
21	VEERAIYA	29	M	62	2	76	78	78
22	RAGU	49	M	60	1	78	89	87
23	MUTHUSAMY	42	M	70	2	89	83	98
24	KANAMMAL	30	F	59	2	83	95	98
25	REVATHI	52	F	72	1	82	98	94
26	SHANKAR	39	M	68	1	88	98	90
27	PUNITHA	45	F	63	2	76	78	90
28	MARIYAMMAL	28	F	65	2	75	93	90
29	MURUGAN	37	M	59	1	78	87	88
30	LEELAVATHY	56	F	50	2	95	94	92

SYSTOLIC ARTERIAL PRESSURE			MEAN ARTERIAL PRESSURE			PaO2		
PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
132	112	120	88	90	86	90	172	92
138	124	138	89	92	90	94	156	95
144	128	140	92	88	98	88	166	96
136	124	138	94	86	94	89	154	92
122	118	130	86	86	86	90	165	89
138	114	130	86	90	82	92	172	88
124	114	130	96	84	82	92	172	92
110	106	130	88	94	80	90	178	91
120	124	130	90	84	82	91	180	92
110	112	122	86	92	84	95	168	94
116	124	130	92	84	86	96	156	95
118	112	136	86	98	88	99	156	91
144	128	130	90	86	90	90	168	93
136	124	146	94	92	92	91	135	90
122	118	130	92	92	94	88	156	89
138	114	132	96	98	86	87	148	88
124	114	130	86	96	82	89	149	87
110	108	123	84	97	82	90	167	90
120	126	130	80	94	84	91	168	93
110	114	126	90	90	86	97	170	91
116	126	146	88	92	90	92	146	86
114	124	134	84	92	84	89	145	88
124	114	120	88	99	86	87	158	86
110	108	130	86	98	88	90	160	89
120	126	140	90	92	90	88	190	90
110	114	136	84	90	92	82	190	88
116	126	128	82	92	94	88	198	92
114	124	140	90	97	86	90	200	95
124	114	132	90	98	82	90	210	93
110	108	136	92	99	82	92	198	90

PaCO2			PH			ETCO2	P PEAK		
PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP	32	33	24	23
41	42	45	7.37	7.32	7.35	33	34	24	24
42	41	46	7.38	7.34	7.39	34	35	24	25
35	36	47	7.33	7.35	7.38	35	36	30	26
36	37	43	7.35	7.36	7.39	32	37	22	24
38	38	45	7.33	7.37	7.38	32	38	22	25
39	42	48	7.34	7.38	7.4	33	33	23	26
36	45	43	7.35	7.33	7.41	34	34	32	26
35	42	44	7.34	7.38	7.42	35	35	33	26
41	32	45	7.36	7.39	7.38	36	36	34	29
43	31	47	7.37	7.4	7.43	37	37	27	25
44	30	47	7.38	7.45	7.42	33	38	28	25
46	31	48	7.39	7.48	7.4	34	32	29	25
45	38	49	7.4	7.39	7.39	35	32	30	28
41	39	45	7.42	7.4	7.38	36	32	24	29
38	43	44	7.41	7.48	7.4	37	33	24	25
39	31	43	7.39	7.44	7.49	31	34	32	25
40	36	42	7.38	7.42	7.4	32	35	33	25
38	45	42	7.35	7.41	7.49	33	36	34	25
37	38	42	7.36	7.39	7.3	34	37	25	25
38	39	40	7.38	7.39	7.38	35	38	26	26
40	40	41	7.39	7.4	7.41	36	32	27	27
41	41	41	7.32	7.46	7.39	32	32	23	28
37	42	40	7.33	7.4	7.38	33	32	24	26
38	34	38	7.36	7.48	7.39	34	33	25	25
39	36	39	7.39	7.39	7.38	35	34	35	25
40	38	37	7.35	7.37	7.39	31	35	27	25
41	31	36	7.36	7.39	7.37	32	36	25	28
42	30	38	7.38	7.39	7.39	33	37	23	25
43	32	35	7.39	7.38	7.37	34	38	24	25
44	34	37	7.4	7.36	7.39	35	39	25	26

P PLATEAU				CORTISOL(mg/dl)			INSULIN micu/ml		
P MEAN				PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
22	23	13	11	11.98	21.66	28.65	6.8	9.8	10.4
23	25	12.2	11	11.2	24.67	28.9	6.9	8.6	9.8
24	24	11	11	12.23	26.65	28.56	7.2	11	13.4
25	22	13	12	12.88	27.23	27.56	7.4	7.6	14.4
21	22	12	13	10.98	23	25.56	8.2	9.8	15.6
22	22	13	12	11.98	24.3	26.68	9.2	8.4	11.6
22	23	12	11	12.23	25.3	27.56	6.4	7.6	12.4
23	24	12	12	12.28	26.3	27.67	5.8	7.3	12.7
24	22	12	12	13.4	22.3	29.98	6.4	7.8	12.8
25	22	12	12	14.23	22.65	29.98	7.8	6.4	9.8
23	22	11	12	12.43	23.65	29.98	7.9	7.4	13.4
24	23	12	12	12.34	24.65	24.45	7.2	7.8	14.3
21	23	12	12	11.43	17.34	26.45	8.4	8.6	13.4
21	23	13	13	10.45	23.45	24.45	8.5	8.6	15.2
21	24	12	12	10.46	23.24	28.46	7.6	8.4	9.8
21	25	11	11	10.45	24.24	28.38	7.4	8.4	12.24
21	22	11	12	10.43	26.46	21.23	8.9	8.6	12.6
22	23	12	12	11.45	27.46	24.39	7.9	8.8	12.3
23	24	12	13	11.47	26.45	28.38	8.2	8.9	12.4
24	25	11	12	11.57	26.45	29.4	9.2	9.6	13.5
21	21	11	12	11.68	25.46	28.49	8.7	9.8	13.5
21	22	11	13	12.56	26.45	29.4	8.5	7.8	13.6
21	23	11	12	12.56	26.45	28.49	8.9	8.8	16.4
22	23	11	12	12.68	26.45	29.47	7.6	9.4	12.4
23	23	12	14	12.78	26.45	27.49	6.3	7.8	13.4
24	24	13	12	12.56	26.45	28.48	5.9	9.2	13.5
25	25	12	11	13.56	25.45	27.49	5.2	8.6	16.7
22	24	11	12	14.23	26.56	28.49	5.6	8.4	17.4
22	24	12	12	11.34	26.34	24.46	6.5	8.6	15.4
23	24	12	12	11.34	23.34	25.47	6.4	8.2	13.2

GLUCOSE (mg/dl)			LACTATE (mmol/l)		
PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
94	110	113	2.83	2.85	2.97
98	112	118	2.02	3.02	3.14
99	118	120	2.08	2.08	2.2
100	119	122	2.22	2.32	2.2
102	119	130	2.02	2.22	2.34
88	121	139	2.13	2.23	2.35
98	122	129	2.13	2.45	2.57
90	123	130	2.82	2.92	3.04
93	129	122	2.99	3.02	3.14
94	130	128	2.98	3.03	3.15
95	122	128	2.97	3.04	2.24
96	110	129	2.98	3.12	3.24
98	118	127	2.87	3.22	2.21
100	100	130	2.78	3.22	3.34
96	108	132	2.98	3.21	3.33
98	104	132	2.99	3.21	2.22
93	102	134	2.66	2.98	3.1
88	103	137	2.78	2.99	3.11
89	104	139	2.99	3.02	3.14
90	105	136	2.78	2.97	2.28
80	109	132	2.76	2.92	3.04
82	110	120	2.77	2.9	3.02
83	112	122	2.56	2.78	2.9
84	120	128	2.87	2.98	3.1
85	122	130	2.98	3.02	3.14
86	120	132	2.02	3.12	3.24
87	118	136	2.21	2.98	3.1
88	119	122	2.01	2.88	3
89	120	120	2.08	2.78	2.9
90	118	129	2.04	2.24	2.36

PCV GROUP

S.NO	NAME	AGE	SEX	WEIGHT	ASA	HEART RATE		
						PREOP	PRONE	POSTOP
1	VAIRAMANI	48	M	58	2	78	78	83
2	MUTHUMURUGAN	30	M	62	2	85	76	78
3	SUBBU	32	M	65	1	76	90	87
4	SURYA	26	M	59	1	79	76	88
5	SAROJA	42	F	49	1	66	83	89
6	VIKRAM	26	M	52	2	78	76	90
7	NAGAMMAL	38	M	53	2	78	83	86
8	MUTHUMARI	37	F	58	2	79	87	88
9	SADHASIVAM	37	M	56	1	80	99	98
10	AJITHA	30	F	58	2	81	94	90
11	MUTHAMMAL	39	F	58	2	82	82	88
12	NAGAMANI	40	F	60	1	79	83	78
13	MUTHUMARI	38	F	62	1	86	84	92
14	GIRIDHARAN	32	M	64	1	92	88	89
15	ARUMUGAM	50	M	59	1	67	88	90
16	MANI	50	M	59	2	68	85	89
17	BALRAJ	30	M	56	2	69	87	78
18	KARUPPAIYA	37	M	64	2	70	89	90
19	PANDIYAMMAL	55	F	65	2	94	76	98
20	KARTHICK	32	M	63	1	76	77	87
21	INDHRA	38	F	72	2	76	78	88
22	RAJAMMAL	39	F	60	1	78	79	89
23	RASATHI	40	F	70	2	89	80	78
24	ARUMUGAM	38	M	55	2	83	95	98
25	MUTHUMARI	39	F	72	1	82	95	98
26	AJITHKUMAR	28	M	68	1	88	95	90
27	GIRIDHRAN	29	M	63	2	78	78	90
28	PANDY	39	M	65	2	85	79	92
29	VIKRAM	30	M	59	1	78	80	88
30	DHANUSHKODI	30	M	60	2	95	94	92

SYSTOLIC ARTERIAL PRESSURE			MEAN ARTERIAL PRESSURE			PaO2		
PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
132	112	120	88	90	86	88	172	92
138	124	138	89	92	90	94	156	90
144	128	140	92	88	98	88	165	96
136	124	138	94	86	94	90	154	90
122	128	130	86	86	86	90	165	89
135	114	131	86	90	82	92	172	87
124	114	132	96	84	82	92	167	92
123	122	133	88	94	80	90	178	91
120	124	134	90	84	81	90	180	92
110	112	135	86	92	82	95	168	94
116	124	136	92	84	86	96	156	95
122	112	136	86	98	88	97	156	91
122	130	130	90	86	90	90	168	93
134	124	130	94	92	92	91	135	88
125	123	130	92	92	94	88	156	89
138	114	130	96	98	86	87	148	90
124	114	130	86	96	82	89	149	87
123	108	131	84	97	83	90	167	90
120	128	132	80	94	84	90	155	93
110	114	133	90	90	86	97	170	91
116	126	134	88	92	90	92	146	86
123	124	134	84	92	84	89	145	88
124	115	120	88	99	84	87	158	86
118	108	121	86	98	86	90	166	88
120	128	122	90	92	90	89	188	90
124	114	123	84	90	92	82	190	90
116	126	128	82	92	94	88	198	92
114	124	129	90	97	86	90	197	95
124	118	130	90	98	82	90	188	93
110	108	136	92	99	83	92	188	90

PaCO2			PH			ETCO2	
PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP	32	33
42	42	42	7.37	7.33	7.35	32	34
42	41	43	7.37	7.33	7.38	32	34
41	41	41	7.33	7.33	7.38	32	34
40	41	44	7.35	7.32	7.39	33	37
39	38	45	7.33	7.32	7.38	32	38
38	38	45	7.34	7.32	7.42	33	33
37	38	45	7.35	7.33	7.41	34	34
39	42	44	7.34	7.33	7.42	34	35
40	42	45	7.36	7.39	7.33	36	36
39	31	47	7.37	7.4	7.43	37	37
38	32	48	7.37	7.42	7.42	37	38
37	33	49	7.39	7.42	7.35	34	32
42	38	49	7.4	7.42	7.39	35	32
41	38	45	7.42	7.4	7.38	36	32
41	43	46	7.41	7.48	7.4	36	33
41	31	47	7.41	7.44	7.49	31	33
42	32	42	7.38	7.44	7.4	32	35
42	33	43	7.35	7.41	7.49	32	36
42	38	44	7.36	7.39	7.3	32	37
38	39	40	7.36	7.39	7.38	35	38
39	39	41	7.39	7.4	7.41	36	32
40	41	42	7.32	7.43	7.39	32	32
37	42	40	7.33	7.4	7.38	32	32
38	43	41	7.36	7.48	7.39	34	33
39	36	42	7.39	7.39	7.38	35	33
40	37	37	7.39	7.37	7.39	35	35
41	38	36	7.36	7.39	7.37	32	36
42	39	37	7.38	7.39	7.39	33	36
43	32	38	7.38	7.38	7.37	34	36
44	34	39	7.38	7.36	7.39	35	39

P PEAK		P PLATEAU		P MEAN	CORTISOL		
					11	PREOP	PRONE
25	26	22	23	13	11	11.98	21.66
22	27	22	23	11	11	11.2	24.67
23	28	22	24	13	12	12.23	26.65
24	29	21	24	12	12	12.88	27.23
25	24	21	22	13	12	10.98	23
25	25	21	22	13	13	11.98	24.3
25	26	22	22	13	14	12.23	21.02
25	27	22	22	13	13	12.28	20.43
25	28	22	22	12	11	13.4	22.3
25	29	21	22	13	13	14.23	22.65
23	25	22	22	11	12	12.43	20.45
23	26	23	22	13	12	12.34	19.45
23	27	24	23	12	12	11.43	17.34
23	28	21	23	12	12	10.45	23.45
25	29	21	23	12	13	10.46	23.24
26	25	21	22	12	13	10.45	25.46
22	26	21	22	11	13	10.43	23.24
22	27	21	22	12	12	11.45	25.24
22	28	22	23	12	12	11.47	26.45
22	25	23	23	12	12	11.57	26.45
26	26	24	23	11	13	11.68	25.46
22	27	21	21	12	13	12.56	26.45
23	28	21	22	11	13	12.56	23.46
24	29	21	23	11	12	12.68	22.45
25	25	21	23	11	12	12.78	23.46
26	26	22	23	12	12	12.56	24.45
24	27	22	23	13	13	13.56	25.45
25	28	22	22	12	13	14.23	26.56
23	29	22	24	11	12	11.34	26.34
24	25	22	24	12	13	11.34	23.34
24	26	23	24	12	12		

POSTOP	INSULIN micu/ml			GLUCOSE (mg/dl)			LACTATE (mmol/l)		
	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP	PREOP	PRONE	POSTOP
28.65	6.8	9.8	10.2	94	100	110	2.83	2.85	2.97
28.9	6.9	9.8	11.2	98	100	110	2.02	2.85	2.97
21.3	7.2	11	11.2	99	100	110	2.08	2.08	2.2
27.56	7.4	10.2	11.2	100	119	110	2.22	2.32	2.44
25.56	8.2	9.8	15.6	102	120	130	2.02	2.22	2.34
26.68	9.2	8.4	11.6	88	121	139	2.13	2.23	2.35
27.56	6.4	7.6	11.6	98	122	129	2.13	2.45	2.57
27.67	5.8	7.3	11.6	90	123	120	2.82	2.92	2.98
29.98	6.4	7.8	12.8	93	100	120	2.99	2.92	3.04
20.22	7.8	6.4	12.67	94	100	120	2.98	2.92	2.37
20.22	7.9	6.4	13.4	95	100	120	2.97	3.04	3.16
20.22	7.2	6.4	14.3	96	100	129	2.98	3.12	3.24
26.45	8.4	8.6	13.4	98	100	127	2.87	3.22	2.23
24.45	8.5	9.6	11.4	100	100	130	2.78	3.22	3.34
28.46	7.6	8.4	15.2	96	108	124	2.98	3.21	2.22
21.3	7.4	8.4	12.24	98	102	124	2.99	3.21	3.33
21.23	8.9	8.6	12.6	93	102	134	2.66	2.98	2.8
21.23	7.9	6.4	12.3	88	103	137	2.78	2.98	2.8
21.23	8.2	6.4	12.4	89	104	139	2.99	2.98	2.8
29.4	9.2	6.4	13.5	90	102	136	2.78	2.97	3.09
28.49	8.7	6.4	13.5	80	109	124	2.76	2.92	3.04
22.32	8.5	7.8	13.6	82	110	124	2.77	2.9	3.02
28.49	8.9	8.8	12.6	83	112	124	2.56	2.78	2.9
29.47	7.6	9.4	12.4	84	120	128	2.87	2.78	2.9
27.49	6.3	7.8	13.4	85	122	130	2.98	2.78	2.9
28.48	5.9	7.8	13.5	86	120	132	2.02	2.78	2.9
27.49	5.2	7.8	16.7	87	118	126	2.21	2.98	3.1
28.49	5.6	8.4	14.6	88	119	122	2.01	2.88	2.9
24.46	6.5	8.6	15.4	89	120	120	2.08	2.78	2.9
25.47	6.4	8.2	13.2	90	118	129	2.04	2.24	2.36



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**ETHICS COMMITTEE
 CERTIFICATE**

Name of the Candidate : Dr.Dineshkumar S.N
 Designation : PG in MD., Anaesthesia
 Course of Study : 2017- 2020
 College : MADURAI MEDICAL COLLEGE
 Research Topic : Effects of Pressure controlled
 and volume controlled ventilation
 on respiratory mechanics and
 systemic stress response during
 prone position
 Ethical Committee as on : 02.07.2019

The Ethics Committee, Madurai Medical College has decided
 to inform that your Research proposal is accepted.

[Handwritten Signature] Member Secretary
[Handwritten Signature] Chairman
[Handwritten Signature] Dean/Convenor
Prof Dr V Nagaraajan
 MNAMS, D.M., Dsc.(Neuro), Dsc (Hons)
 CHAIRMAN
 Madurai Medical College
 Madurai
DEAN
 Madurai-20



Urkund Analysis Result

Analysed Document: DINESHKUMAR DESSERTATION .docx (D57915544)
Submitted: 10/30/2019 10:57:00 AM
Submitted By: dheenadr@gmail.com
Significance: 21 %

Sources included in the report:

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