

# Positive End Expiratory Pressure Prevents Loss Of Respiratory Compliance During Low Tidal Volume Ventilation In Acute Lung Injury Patients

V Gupta, A Gupta, A Mehta, R Wakloo, R Angral, V Sarswat, P Darswal, R Lahori, B Kapoor, S Gupta, D Kumar

## Citation

V Gupta, A Gupta, A Mehta, R Wakloo, R Angral, V Sarswat, P Darswal, R Lahori, B Kapoor, S Gupta, D Kumar. *Positive End Expiratory Pressure Prevents Loss Of Respiratory Compliance During Low Tidal Volume Ventilation In Acute Lung Injury Patients*. The Internet Journal of Anesthesiology. 2008 Volume 20 Number 2.

## Abstract

A total of 120 patients were enrolled in the present study over a period of 3 years from (2005 – 2008) . Pressure controlled ventilation (PCV) and volume controlled ventilation (VCV), with an average tidal Volume (VT) of  $8.2 \pm 0.4$  ml/kg was applied at three levels of PEEP (5, 10, 15 cm H<sub>2</sub>O). Before each PCV & VCV period, lung volume history was standardized by manual hyperinflation maneuvers. The compliance was measured from the beginning at time 0 (start), 10, 20 & 30 (end) min of each PCV & VCV period. Three gas exchange and hemodynamic data were collected in the end. After statistical analysis results showed progressive decay in compliance with both PCV & VCV modes at 5 & 10 cm H<sub>2</sub>O. At PEEP 5 cm H<sub>2</sub>O compliance decrease was higher during PCV mode than VCV mode and at PEEP 15 cm H<sub>2</sub>O, compliance did not decay significantly. The values of PaO<sub>2</sub> were significant but statistically insignificant in PCV mode at 15 cm H<sub>2</sub>O than VCV mode. Hemodynamic data did not differ between PCV & VCV mode.

## INTRODUCTION

In recent years, the use of low (<10 ml/kg) tidal Volume (VT) is getting more and more frequently recommended in the management of acute lung injury patients [1,2,3,4].

Experimental evidence, indeed indicate that high peak air way pressure level [5] and / or lung overdistension [6,7] may not lead only to overt barotrauma but also severe alveolar and microvascular damage.

In the past, low tidal volume ventilation has been associated with unstable lung mechanics and altered gas exchange [8]. Infact, a number of studies have reported progressive compliance decay during anaesthesia with low tidal volume ventilation [9,10,11]. These finding have been mainly ascribed to air way collapse. Low Vt has been shown to also cause a compliance loss in respiratory failure patients [12,13,14,15,16].

A loss of respiratory compliance could then be a major drawback of those ventilatory strategies focused on preventing barotrauma by use of low Vt. This compliance decay will become obvious with an increase in airway pressure during volume control ventilation. There are several advantages of pressure controlled ventilation than volume

controlled ventilation, as limiting the peak inflating pressure delivered by the ventilator will limit the trans alveolar pressure produced, there by reducing the ventilator induced lung injury.[6]. The decelerating flow used to produce pressure controlled ventilation is thought to improve distribution of gas flow [13]. When compared with volume controlled ventilation, there is rapid improvement in lung compliance and oxygenation [14]

We wondered whether an adequate PEEP level could prevent the progressive loss of lung compliance during low VT ventilation. Experimental evidence suggests that PEEP may indeed optimize respiratory compliance during low VT ventilation [15]. In respiratory failure, Suter et al [15] reported evidence suggesting that the smaller the VT -the higher the PEEP level needed to optimize lung mechanics. Mauliz Cereda et al studied effect of three PEEP level (5, 10, 15 cm) on compliance decay related to the relatively low VT (7 to 9 ml/kg) in 8 ALI cases. As number of cases was too small, we designed the present study to be conducted in Govt. Medical College, Jammu in Intensive Care Unit under Department of Anaesthesiology and ICU over a time span of 3 years to investigate the effects of three PEEP levels (5, 10, 15 cm

## **Positive End Expiratory Pressure Prevents Loss Of Respiratory Compliance During Low Tidal Volume Ventilation In Acute Lung Injury Patients**

---

H<sub>2</sub>O) on compliance decay in low VT (7 to 9 ml/kg) ventilation in both pressure control & volume control Ventilation.

### **MATERIAL AND METHODS**

After approval from the hospital ethical committee and informed consent, 120 patients with ALI were enrolled in this study according to following criteria : Lung injury score of 2.5 and more, No H/O chronic obstructive lung disease, No active air leak, Pulmonary artery occlusion pressure less than 18 mm Kg., Stable hemodynamic during previous 6hrs. All patients were on supine position and undergoing VCV by a ventilator (Puritan Bennett 840, 7200 Series, and Evita Dura-2). All patients were under I/V sedation by continuous infusion of midazolam or propofol or combination of both drugs & paralyzed with I/V bolus of pancuronium bromide. All patients were orally intubated with PCV cuffed tubes no. 7.5 to 8.5 ID

Respiratory rate, expiratory minute volume, VT, PEEP and FiO<sub>2</sub> had been selected by critical care specialist in ICU in order to optimize blood gas values while minimizing barotrauma risk. In particular, relatively low tidal volume (8ml/kg) was adopted, following current practice in one unit. Table 1 shows relevant clinical data at entry into study. All patients had an arterial cannula and a central line in place for hemodynamic monitoring and blood sampling. Mean airway pressures, PEEP total, PEEP intrinsic were recorded from the ventilator.

The patients were randomized in to 6 groups

- Group I : PCV 5 cm H<sub>2</sub>O
- Group II: PCV 10cm H<sub>2</sub>O
- Group III: PCV 15 cm H<sub>2</sub>O
- Group IV: VCV 5 cm H<sub>2</sub>O
- Group V: VCV10 cm H<sub>2</sub>O
- Group VI: VCV15 cm H<sub>2</sub>O

Prior to the study, all patients were undergoing low Vt VCV, the standard controlled ventilation mode in our centre. Before the beginning of study inspiratory time was set at 33% of total respiratory pause (plateau) thus leaving Vt and RR at the level previously set by clinical criteria. All ventilatory parameters, unless specified were kept constant through out the study. The Pt. underwent PCV and VCV at 5, 10 & 15 Cm H<sub>2</sub>O PEEP. All combinations of two ventilatory modes and of the three PEEP levels were applied in random order in all the patients, for period lasting 30 min.

each. In order to standardize lung volume history, airway secretions were aspirated and ten manual inflations of approximately 1 litre were performed before each PCV, VCV period. A silicon resuscitator equipped with a mechanical PEEP valve, set a minimal 10 cm H<sub>2</sub>O was used. Manual inflations have been proven effective to maximize system compliance and oxygenation during general anaesthesia [1017]. The tidal volume clinically in use before study was the target of both PCV & VCV. Each PCV & VCV period started (start) immediately after having adjusted the ventilator to deliver the target Vt, was kept constant during each PCV period, but not between PEEP level.

We performed 3 end-inspiratory and 3 end-expiratory occlusions, at start, after 10 min, 20 min and at end. Each occlusion lasted 3 sec and obtained by priming appropriate button on ventilator, airway pressure and flow signals were recorded. At end arterial samples were collected for ABG. Hemodynamic data was recorded. All data in the table are expressed as mean ± SD. All statistics comparison was performed using student "t test

### **STATISTICS**

Data was analyzed with the help of a computer software SPSS12.0 for windows. All data was expressed as MEAN + SD to assess the effect of time. Start data were compared with end data for each PEEP and ventilatory mode (VCV & PCV). To assess the effect of ventilatory mode, PCV and VCV data were compared for each PEEP level. Student t test was used to evaluate statistically significant difference between variance a p value of less than .05 was considered statistically significant. All analysis was done in accordance to intention to treat principle and all p-values reported are to tailed.

### **RESULTS**

120 patients were enrolled in this study aged b/w 20-40 yrs of any sex, 120 pts were divided randomly in 6 groups with 20 pts in each group. After recruitment maneuver, compliance were recorded at start, 10 min, 20 min & 30 min with both ventilator modes at PEEP 5, 10 & 15 H<sub>2</sub>O thrice and mean of 3 values were taken. Intergroup comparison in pressure control ventilation with PEEP 5, 10 & 15 H<sub>2</sub>O showed statistically significant compliance decay in 5 and 10 cm H<sub>2</sub>O PEEP from start to end but decay of compliance was statistically insignificant in 15 cm of H<sub>2</sub>O PEEP.

**Positive End Expiratory Pressure Prevents Loss Of Respiratory Compliance During Low Tidal Volume Ventilation In Acute Lung Injury Patients**

**Figure 1**

Table 1: Demographic data expressed as mean ± SD

GROUPS	AGE	SEX(M)	(F)	WEIGHT	INTUBATED
PCV 5	38.00 ±7.7	19	1	50.1±5.5	2
PCV 10	39.50 ±11	18	2	50.5±5.15	1
PCV 15	38.75 ±7.85	20	0	52.2±6.30	3
VCV 5	38.5 ±8	20	0	50.4±7.0	2
VCV10	39.0 ±7.6	19	1	50.8±6.0	1
VCV15	38.0 ±8.1	19	1	51.75±7.2	3

**Figure 2**

Table 1B (Data represented as average mean)

GROUPS	Diagnosis				FiO2	VE	Vt	RR
	trauma	sepsis	FESS	BN				
PCV 5	18	1	1	1	1.0	7	8.4	12
PCV 10	17	2	0	1	0.9	8.5	8.2	15
PCV 15	17	3	0	0	0.7	8.1	8.6	16
VCV 5	19	1	0	0	0.9	7.9	8.8	14
VCV10	16	1	1	2	0.85	14.1	8.6	22
VCV15	16	3	0	1	0.6	6.8	8.0	12

T = trauma, S= sepsis, FESS=fat embolism shock syndrome, BN=bacterial pneumonia  
 FiO2=fraction of oxygen in inspiratory Ve=minute volume  
 Vt=tidal volume,  
 RR=respiratory rate

**Figure 3**

Table 2: Intergroup comparison b/w PCV and VCV at PEEP 5 cm of H2O

Group	Start			End		
	PCV	VCV	t, P	PCV	VCV	t, P
Tidal vol	507.5 ± 17	511.5±21.3	.80 ,.42	502 ± 25.6	513±19.2	.23, .81
Compliance	42.7 ± 2.0	33.55±1.82	15.12 1.07	39.35±1.66	31.9±1.77	13.70,1.12
PEEP	0.51 ± 0.23	0.51 ± 0.14	1.8, 1.11	0.34 ± 0.18	0.43 ± 0.18	1.44, 0.156
PELI	22.85±1.46	27.85±1.05	11.68, 1.72	22.7 ± 1.58	27.3 ± 1.26	10.13, 1.35
PIP	23.5±1.39	21.85±0.87	4.47, 6.98	23.5 ± 1.39	21.7 ± 1.08	4.5, 5.19
MAP	11.1±1.07	10.7±0.92	1.26, 0.21	10.7 ± 1.29	10.4 ± 0.68	1.07, 0.2
PAO2	82.0 ± 4.0	82.3±3.9	0.24,0.81	86.5 ± 4.93	83.8 ± 4.32	1.84, 0.07
PACO2	60 ± 3.8	59±4.1	0.80,0.42	55 ± 4.32	56.4 ± 4.78	0.97, 0.33

**Figure 4**

Table 3: Intergroup comparison b/w PCV and VCV at PEEP 10 cm of H2O

Group	Start			End		
	PCV	VCV	t, P	PCV	VCV	t, P
Tidal vol	511±27.7	509.5±19.0	0.26,0.791	510±16.4	485±17.3	4.67,3.61
Compliance	33.1±1.58	33.3±1.97	0.35, 0.726	31.5±1.55	30.4±1.66	2.65, 0.011
PEEP	0.30 ± 1.13	0.38±0.22	1.24, 0.22	0.3±0.12	0.25±0.18	0.97, 0.33
PELI	27.5±1.05	27.75±1.01	0.76, 0.44	28.3±2.92	27.7±1.21	0.84, 0.401
PIP	23.8±4.95	27.45±1.23	3.19, 0.004	23.7±4.50	27.55±1.1	3.69, 0.001
MAP	15.95±1.79	17.05±0.94	2.42, 0.01	15.85±1.75	16.75±0.8	2.06,0.04
PAO2	129 ± 19.3	124 ± 20.1	.802, 0.42	138.5±23.3	125.65±10	2.24, p0.03
PACO2	56.1 ± 3.2	55.4 ± 2.92	.72, 0.43	54.9±4.64	53.2±2.14	1.48, p0.145

**Figure 5**

Table 4: Intergroup comparison b/w PCV and VCV at PEEP 15 cm of H2O

Group	Start			End		
	PCV	VCV	t, P	PCV	VCV	t, P
Tidal vol	511.5±15.6	518.5±24	1.88, 0.28	513.2±16.3	515.7±21.5	0.421, 0.62
Compliance	28.8±1.91	29±1.21	0.52, 0.60	28.5±1.19	29±1.38	1.58,0.62
PEEP	0.32±0.15	0.35±0.11	0.80,0.42	0.29±0.17	0.34±0.11	0.94, 0.35
PELI	40.9±0.91	34.55±4.07	6.8, 1.02	41.1±1.02	35.35±1.75	12.6, 3.13
PIP	35±1.55	35.9±1.07	2.13, 0.03	35.45±0.82	36.05±1.0	1.95, 0.05
MAP	20.95±0.82	22.45±1.76	3.44,0.001	20.75±0.85	22.2±1.28	4.21, 0.001
PAO2	192 ± 3.2	204 ± 10.5	1.64,0.10	145 ±11.2	138 ± 9.6	6.1, 3.3,07
PACO2	58.2 ± 4.20	54.6 ± 2.12	1.50, 0.14	53.8±3.20	50.85±1.08	3.89,0.07

**Figure 6**

Table 5: compliance in Pressure control ventilation represented as mean ± SD with in the same group(start + end)

	PEEP 5 cm H2O	PEEP 10 cm H2O	PEEP 15 cm H2O
Start	42.7 ± 2.0	33.1 ± 1.58	28.8 ± 1.19
End	39.35 ± 1.66	31.75 ± 1.55	28.5 ± 1.19
t-value t(19)	7.27	9	1.83
p value	0.0001	HS	NS

**Figure 7**

Table 5: compliance in Volume control ventilation represented as mean  $\pm$  SD with in the same group (start + end)

	PEEP 5 cm H2O	PEEP 10 cm H2O	PEEP 15 cm H2O
Start	33.5 $\pm$ 1.82	33.3 $\pm$ 1.97	29.5 $\pm$ 1.26
End	31.9 $\pm$ 1.77	30.4 $\pm$ 1.66	29.15 $\pm$ 1.38
t-value (t19)	12.56	14.22	0.6
p value	HS	HS	NS

In VCV group compliance decay was statistically significant in PEEP in 5 and 10 cm H2O insignificant in 15 cm of H2O PEEP. Decay of compliance observed was progressive in time but a plateau stage reached at 30 min.

Intergroup comparison in PCV & VCV with PEEP 5, 10 & 15 cm of H2O showed compliance decay in group with 5 cm H2O. PEEP was highly significant during PCV than during VCV, even if end compliance values were comparable.

At 10 cm of H2O PEEP no difference in compliance was found at both start and end. As expected the compliance decay was associated with increase in  $P_{el}$ ,  $i$  & PIP during VCV and with decrease in VT during PCV. Raising PEEP also increases PIP,  $P_{el}$ ,  $i$  & MAP. No statistically significant difference observed in intrinsic PEEP.

At PEEP 15 cm H2O, PaCO2 was lower with PCV as compared with VCV, despite a comparable VT but not statistically significant. No differences could be shown in hemodynamics, oxygenation between modes.

## DISCUSSION

The results of this study showed that at PEEP 5 and 10 cm H2O, compliance decayed progressively from start to end during both VCV and PCV modes however a PEEP 15 cm H2O this decay in compliance was prevented in both PCV & VCV modes. Further a PEEP 15 cm H2O, PaCO2 was lower in PCV than in VCV. The observed decay of compliance confirms the classic studies reporting a progressive compliance decrease during anesthesia [9]. Low VT ventilation fosters compliance decay while hyperinflation maneuvers may restore compliance [10]. Recently, studies have reported progressive compliance decay and lung volume loss during high-frequency oscillation and low Vt ventilation in rabbits [14]. The use of

relatively low VT was probably the cause of the compliance decay in our study also. Indeed, in ALI patients, compliance has been reported to be lower with low VT than with high VT [12]. The compliance decay during constant low VT anesthesia has been mainly related to alveolar collapse [810]. Since in ALI patients the lung is typically prone to atelectasis, it is likely that the compliance decay observed was also due to airspace collapse [18]. Surfactant alteration at low lung volumes may indeed favor alveolar collapse [19]. Moreover, airway closure occurs at higher transpulmonary pressure in edematous lungs than in normal lungs [20]. Thus, progressive compliance loss in ALI patients may occur despite low (5 cm H2O) or moderate (10 cm H2O) levels of PEEP. Moreover, cyclic collapse and reopening of unstable alveoli is likely when PEEP is inadequate to avoid end-expiratory alveolar closure [21]. Such instability causes alveolar collapse and compliance decay.

A high PEEP may stabilize alveolar recruitment and provide constant compliance and prevent compliance decay. At PEEP level of 15 cm of H2O, airway pressures were adequate to preserve the lung volume recruited during manual inflations this was probably high enough to keep most of recruitable alveoli open [22]. Moreover, at PEEP 15 cm H2O,  $P_{el}$ ,  $i$  reached a level approximately 35 cm H2O reportedly sufficient to warrant an almost complete alveolar recruitment [23].

We observed higher compliance decay with PCV compared with VCV at 5 cm H2O PEEP. However, the compliance values at end were not significantly different from the ones observed during VCV. Furthermore, we found insignificant difference in PaCO2 and PaO2 between PCV and VCV which indicates decreased alveolar recruitment with VCV than PCV mode. The compliance improvement by PEEP 5 cm H2O during PCV was likely due to the decelerated flow waveform typical of PCV, [2124]. Since VT values were comparable, this finding accounts for a dead-space reduction. Mechanical ventilation with inspiratory flow deceleration reduces dead-space fraction compared with constant flow inflation [2425] thanks to an improved distribution of inspired gas [26]. In addition, rapid alveolar inflation produces longer residence time of inspired gas. This enhances intrapulmonary gas mixing [27] and improves CO2 exchange [28].

At PEEP lower than 15 cm H2O, however, the VT decay offsets the advantage of a lower dead-space fraction,

resulting in comparable alveolar ventilations.

## CONCLUSIONS

In ALI patients ventilated with relatively low VT, we observed compliance decay in time that was prevented by a 15 cm H<sub>2</sub>O PEEP level. Progressive compliance loss during mechanical ventilation is the result of ventilatory strategies that allow the development of atelectasis. Experimental evidence suggests, however, that alveolar instability worsens lung injury. Therefore, ventilatory patterns focused on lung volume recruitment are recommended as a safer choice. In our patients, a relatively high PEEP level (15 cm H<sub>2</sub>O) was required to prevent the compliance decay. The problem is left open of whether the advantages achieved by optimizing PEEP in such way could balance the potential risks of higher airway pressures. We have no direct indications about the clinical relevance of the relatively minor changes observed in the present study.

## References

1. Hickling KG. Ventilatory management of ARDS: can it affect the outcome? *Intensive Care Med* 1990; 16:219-26
2. Mancebo J. PEEP, ARDS, and alveolar recruitment. *Intensive Care Med* 1992; 18:383-85
3. Marini JJ. Re-targeting ventilatory objectives in adult respiratory distress syndrome: new treatment prospects-persistent questions. *Am Rev Respir Dis* 1992; 146:2-3
4. Tuxen DV. Permissive hypercapnic ventilation. *Am J Respir Crit Care Med* 1992; 150:870-74
5. Kolobow T, Moretti MP, Fumagalli R, et al. Severe impairment in lung function induced by high peak airway pressure during mechanical ventilation: an experimental study. *Am Rev Respir Dis* 1987; 135:312-15
6. Dreyfuss D, Soler P, Basset G, et al. High inflation pressure pulmonary edema: respective effects of high airway pressure, high tidal volume, and positive end-expiratory pressure. *Am Rev Respir Dis* 1988; 137:1159-64
7. Hernandez LA, Peevy KJ, Moise AA, et al. Chest wall restriction limits high airway pressure-induced lung injury in young rabbits. *J Appl Physiol* 1989; 66:2364-68
8. Pontoppidan H, Geffin B, Lowenstein E. Acute respiratory failure in the adult (third of three parts). *N Engl J Med* 1972; 287:799-806
9. Mead J, Collier C. Relation of volume history of lungs to respiratory mechanics in anesthetized dogs. *J Appl Physiol* 1959; 14:669-78
10. Bendixen HH, Hedley-White J, Chir B, et al. Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. *N Engl J Med* 1963; 269:991-96
11. Egbert LD, Laver MB, Bendixen HH. Intermittent deep breaths and compliance during anesthesia in man. *Anesthesiology* 1963;24:57-60
12. Blanch L, Fernandez R, Valles J, et al. Effect of two tidal volumes on oxygenation and respiratory system mechanics during the early stage of adult respiratory distress syndrome. *J Crit Care* 1994; 9:151-58
13. Rappaport SH, Shipnerr, yoshilara G, et al randomized prospective trial of pressure limited v/s volume control ventilation in severe respiratory failure- *crit care med* 1994;22:22-32.
14. Bond DM, McAloon J, Froese AB. Sustained inflations improve respiratory compliance during high-frequency oscillatory ventilation but not during large tidal volume positive-pressure ventilation in rabbits. *Crit Care Med* 1994; 22:1269-77
15. Murray JF, Matthay MA, Luce JM, et al. An expanded definition of the adult respiratory distress syndrome. *Am Rev Respir Dis* 1988; 138:720-23
16. Suter PM, Fairley HB, Isenberg MD. Effect of tidal volume and positive end expiratory pressure on compliance during mechanical ventilation. *Chest* 1978; 73:158-62
17. Tweed WA, Chong KY, Lim E, et al. Large tidal volume ventilation improves pulmonary gas exchange during lower abdominal surgery in Trendelenburg position. *Can J Anaesth* 1991; 38:989-95
18. Gattinoni L, Mascheroni D, Torresin A, et al. Morphological response to positive end expiratory pressure in acute respiratory failure: computerized tomography study. *Intensive Care Med* 1986; 12:137-42
19. Brown ES, Johnson RP, Clements JA. Pulmonary surface tension. *J Appl Physiol* 1959; 14:717-20
20. Frazer DG, Stengel PW, Weber KC. The effect of pulmonary edema on gas trapping in excised rat lungs. *Respir Physiol* 1979; 38:325-33
21. Marini JJ. Ventilation of the acute respiratory distress syndrome: looking for Mr. Good mode. *Anesthesiology* 1994; 80:972-75
22. Gattinoni L, D'Andrea L, Pelosi P, et al. Regional effects and mechanism of positive end-expiratory pressure in early adult respiratory distress syndrome. *JAMA* 1993; 269:2122-27
23. Ranieri VM, Eissa NT, Corbeil C, et al. Effects of positive end expiratory pressure on alveolar recruitment and gas exchange in patients with the adult respiratory distress syndrome. *Am Rev Respir Dis* 1991; 144:544-51
24. Al-Saady N, Bennet ED. Decelerating inspiratory flow waveform improves lung mechanics and gas exchange in patients on intermittent positive-pressure ventilation. *Intensive Care Med* 1985;11:68-75
25. Baker AB, Restall R, Clark BW. Effects of varying inspiratory flow waveform and time in intermittent positive pressure ventilation: emphysema. *Br J Anaesth* 1982; 54:547-54
26. Jansson L, Johnson B. A theoretical study on flow patterns of ventilators. *Scand J Respir Dis* 1972; 53:237-46
27. Modell HI, Cheney FW. Effects of inspiratory flow pattern on gas exchange in normal and abnormal lungs. *J Appl Physiol* 1979; 46:1103-07
28. Knelson JH, Howatt WF, DeMuth GR. Effect of respiratory pattern on alveolar gas exchange. *J Appl Physiol* 1970; 29:328-31

**Author Information**

**Vishal Gupta**

Senior resident, Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Ajay Gupta**

Senior resident, Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Anjali Mehta**

Senior resident, Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Renu Wakloo**

Senior resident, Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Rajesh Angral**

Senior resident, Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Vikas Sarswat**

P.G. Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Poonam Darswal**

P.G. Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Rohit Lahori**

P.G. Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**B.B. Kapoor**

H.O.D. Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Satyadev Gupta**

Ex-H.O.D. Anaesthesia, Deptt of Anaesthesiology and Critical Care, GMC Jammu

**Dinesh Kumar**

Asst Proff, Deptt of Community Medicine, GMC Jammu