https://doi.org/10.48047/AFJBS.6.Si3.2024.2179-2195

African Journal of Biological Sciences

Hemodynamic Effects of Pressure-Regulated Volume-Controlled Versus Volume-Controlled Ventilation Mode in Patients with Diastolic Dysfunction Undergoing Radical Cystectomy- A Cross Over Randomized Study

Tarek S. Ibrahim, Tamer A. Abdelsalam, Mahmoud M. Othman And Moustafa M. Gad

[a]Anesthesia, Surgical Intensive Care and Pain management Department, Faculty of Medicine, Mansoura university, Egypt

Corresponding author: Tarek S. Ibrahim

Email: tarekabdelkarem927@gmail.com Phone: +201553538602

Article History

Volume 6, Issue Si3 2024

Received: 12/03/2024

Accepted: 18/05/2024

Published: 20/06/2024

Doi: 10.48047/AFJBS.6.Si3.2024.2179-2195

Abstract

Background: Management of hemodynamics remains one of the core tasks in perioperative and critical care settings. The basis of hemodynamic management in patients undergoing major surgery is formed by a rational titration of fluids, vasopressors and inotropes.

Objective: The objective of this study was to investigate whether, the pressure regulated volume-controlled mode (PRVC) in comparison with the volume-controlled mode in patients with diastolic dysfunction, was associated with better hemodynamic alterations and different vasopressors support during anesthesia for radical cystectomy.

Methods: This study was a randomized, cross-over single blinded study and included 86 adult patients of both sexes with American Society of Anesthesiologists (ASA) physical status I - II with diastolic dysfunction, the patients were randomly assigned to group 0 (VCV-PRVC) and group 1 (PRVC-VCV) in a cross-over manner according to a computer-generated randomization sequence. Hemodynamic variables included stroke volume, stroke volume variation, stroke index, cardiac output, cardiac index, systemic vascular resistance, systemic vascular resistance index (SVRI), thoracic fluid content, and corrected flow time, were measured by a Portable noninvasive cardiometry.

Results: There were no statistically significant differences in demographic data such as age, sex, ASA physical status, body weight, height, body surface area (BSA), duration of surgery, anesthesia, cardiac output, and cardiac index between the two modes of ventilation. Stroke volume (SV) was significantly higher in the VCV group than in the PRVC group at the 3-hour mark from the start of the resection. Regarding fluid status parameters (SVV, FTc, and TFC), there was no statistically significant difference in stroke volume variation (SVV) or (FTc) between both modes. As regards to crystalloids, colloids, blood and plasma transfusions, blood loss, and total fluids, there were no statistically significant differences between both modes.

Conclusion: no significant differences in CO, CI, SV, SVI, and SVV. Both VCV and PRVC ventilation modes could be used in patients with diastolic dysfunction undergoing major abdominal surgery, showing

Keywords: Pressure-Regulated Volume-Controlled, volume-controlled, hemodynamics, Stroke Volume, Stroke Index, Cardiac Index, Systemic Vascular Resistance, Systemic Vascular Resistance Index.

1. Introduction

Diastolic function is a combination of ventricular chamber compliance, active myofilament relaxation, and elastic recoil of systolic potential energy. Diastole is classically divided into four stages, isovolumetric relaxation, early rapid filling, late slow filling, and atrial contraction. Isovolumetric relaxation refers to the rapid decrease in LV pressure with little or no change in volume acting as LV suction and ends with the opening of the mitral valve and early LV filling, while late slow filling is more dependent on ventricular compliance**[1]**.

Diastolic dysfunction represents an abnormality in left ventricular relaxation and/or compliance that changes the onset, rate and extent of LV pressure decline and filling during diastole. These changes cause an abnormal relation between LV pressure and volume so that higher filling pressures are required to maintain normal LV end-diastolic volume and cardiac output **[2].** Ventricular compliance is affected by numerous factors, including the accumulation of cytoskeletal collagen, which increases with age (over 60 years), longstanding wall stress beyond its physiological reserve, for example, during episodes of uncontrolled systemic hypertension, long-term DM, obesity, cardiac ischemia or atrial fibrillation (AF), and various neuro-humoral factors. Less common causes are infiltrative diseases, pericardial constriction, and effusion **[3].**

Diastolic dysfunction is thought to be a risk factor for postoperative consequences after noncardiac surgery, including greater mortality and a higher incidence of serious cardiovascular events including myocardial infarction, pulmonary edema, ventricular fibrillation, or primary cardiac arrest and complete heart block. However, the underlying mechanism by which diastolic dysfunction raises the risk of postoperative complications is unknown. Anesthetic drugs may cause altered hemodynamic function and subsequent impairment of diastolic function in patients with diastolic dysfunction, which may be linked to a higher incidence of postoperative problems **[4].**

Positive pressure ventilation is required for anesthesia, and significant volume shifts occur intraoperatively and postoperatively, especially in major surgery. The interactions of positive pressure ventilation with the cardiovascular system are complex and affect both ventricles **[5].**

Mechanical ventilation impacts heart and hemodynamics by altering ITP and lung volume The effects of different ventilatory modes on patient´s hemodynamics requires further investigations; the increase of the intrathoracic pressure can decrease venous return and then preload resulting in decreased cardiac output **[6].**

Whereas Volume-controlled ventilation ensures the delivery of a defined tidal volume and uses a square waveform flow delivery method that produces high peak airway pressures in low-compliance states**.** On the other hand, PRVC used numerical response mechanisms to control tidal volume, aiming for a fixed volume with decelerating flow. It calculated lung compliance and adjusts inspiratory pressure based on previous breaths, achieving the target volume with the lowest inspiratory pressure **[7,8].**

Management of hemodynamics remains one of the core tasks in perioperative and critical care settings. The basis of hemodynamic management in patients undergoing major surgery is formed by a rational titration of fluids, vasopressors and inotropes **[9].** The use of

dynamic parameters to guide intravenous and inotropic therapy is frequently applied with the intention to optimize peri-operative hemodynamic profiles and maximize O_2 delivery in patients undergoing major abdominal surgery **[10].**

Though the hemodynamic effect of mechanical ventilation on diastolic dysfunction is still unclear. The optimal ventilation mode for anesthesia of patient with diastolic dysfunction remains a subject of debate. To the best of our knowledge, no previous studies have been done to evaluate the hemodynamic profiles non-invasively in diastolic dysfunction patients under either VCV or PRVC mode.

1.1 Hypothesis

We hypothesized that PRVC mode might have less unexpected effects on hemodynamic parameters with lesser doses of vasopressors in patients with diastolic dysfunction undergoing major abdominal surgery.

1.2 Objective

The aim of this study was to investigate whether PRVC, compared with VCV was associated with better hemodynamic optimisation profile and less vasopressor requirement in patients with diastolic dysfunction underwent major surgery.

2. Patients and Methods

2.1 Design

This study was a randomized, cross-over single blinded study.

2.2 Setting

This study was conducted at Anesthesia, Surgical Intensive Care and Pain management Department, Faculty of Medicine, Mansoura university, after obtaining approval from the Institutional Review Board (IRB) of faculty of medicine at Mansoura University (code no:MD2107497R1) and was registered at clinical trial .gov with ID number**:** (NCT05048199).

2.3 Patients

This study included 86 adult patients of both sexes with American Society of Anesthesiologists (ASA) physical status I - II diagnosed with diastolic dysfunction. The patients at risk of diastolic dysfunction like (long term hypertension, diabetes mellitus, aged above 60 years and atrial fibrillation) were included and scheduled for radical cystectomy and urinary diversion for muscle invasive urinary bladder carcinoma at Mansoura Urology and Nephrology center.

Patients with a body mass index (BMI) < 25 and > 35, any contra-indications to epidural anesthesia (patient's refusal, local skin infection, previous spine surgery and coagulopathy), those with known allergy to local anesthetics, patients with major cardiovascular problems (mitral or aortic valve disease, any implanted mechanical cardiac device, with ejection fraction < 40 % and arrythmias other than atrial fibrillation), renal impairment with serum creatinine >1.8 mg/dl and patients with hepatic dysfunction were excluded from the study.

All patients were assessed on the day before surgery by detailed history taking, thorough clinical examination, and basal laboratory investigations [Complete blood count (CBC), Coagulation profile (prothrombin time and INR), liver and renal function tests (liver

enzymes, bilirubin, albumin and s. creatinine], electrocardiograph (ECG) and echocardiography.

2.4 Randomization

After taking informed consent, patients were randomly assigned to two groups; group 0 (VCV-PRVC) and group 1 (PRVC-VCV) in a cross-over manner, according to a computer-generated randomization sequence.

After anaesthesia induction and endotracheal intubation in group 0 (VCV-PRVC), the patient was ventilated by VC mode, which continued till the end of radical cystectomy. Then, the ventilation mode changed to PRVC mode during urinary diversion till the end of surgery. The PRVC mode was initiated in group 1 (PRVC-VCV) and then switched to VCV in the same manner as group 0.

Pre-oxygenation for 5 minutes was done in all patients and they received 0.02 mg/Kg midazolam, 1-2 µg/Kg fentanyl, 2-2.5 mg/kg Propofol slowly until loss of verbal contact. rocuronium 0.6 mg/kg to facilitate proper placement of endotracheal tube. The tidal volume in both groups was set to deliver 6-8 mL/kg of ideal body weight. The respiratory rate (RR) was adjusted to maintain an end tidal CO2 (ETCO2) level of 30–35 mmHg, the inspiratory to expiratory time (I: E) ratio was 1:2 and PEEP 5-8 cmH2O via FLOW-i anesthesia machine (Maquet Critical Care AB-Röntgenvägen 2 SE-171 54-Solna Sweden).

Anesthesia was maintained in all patients using (1-1.5) minimum alveolar concentration (MAC) of sevoflurane in O_2/air mixture with FIO_2 about 40-45%. Also, analgesia was maintained by incremental epidural boluses according to requirements. The formulas to calculate the IBW were: 50 + 0.91 [height (cm) -152.4] for men and 45.5 + 0.91 [height (cm) - 152.4)] for women.

After induction of anesthesia all patients received, Colloid aliquots of 500 mls 6% hydroxyethyl starch 130/ 0.4 (Voluven, Fresenius kabi, Deutschland GmbH, Bad Homburg, Germany) within 10 minutes; ringer acetate was then infused during operation at a basal rate of 4- 6 ml/Kg/h to obtain an appropriate urinary output of 1 cc/kg/h. hemodynamic optimization with fluids and vasopressor relies on SV optimisation, CI and MAP**.** A decrease in SV > 10% over 10 min prompted giving 250 mls fluid boluses of colloid with simultaneous assessment of mean arterial Pressure (MAP), when the MAP reached less than 65, patients were given 2-4 mL of intravenous ephedrine, which has a concentration of 2.5 mg/ml.

After 5 minutes, their Mean Arterial Pressure (MAP) was checked. If the MAP was not at least 65 mmHg, the same dose of ephedrine was administered again, up to a maximum of three times, with each dose given at five-minute intervals. if the MAP had not reached 65 mmHg after three doses of ephedrine at five-minute intervals norepinephrine infusion (.03-.4 µg/kg/min) was started intraoperatively supposed that CI > 2.5 L/min/m2, if CI < 2.5 L/min/m2 dobutamine infusion initially (5 µg/kg/min) was started. In both groups, the total rate of 6% hydroxy ethyl starch (Voluven) administration for 24 hours must not exceed 25

ml/kg. If there is a need to give fluid boluses, Ringer acetate could replace voluven administration.

2.5 Monitoring

Hemodynamic variables included stroke volume (SV), stroke volume variation (SVV), stroke index (SI), cardiac output (COP), cardiac index (CI), systemic vascular resistance (SVR), systemic vascular resistance index (SVRI), thoracic fluid content (TFC), corrected flow time (FTc), and index of contractility (ICON) were measured by a Portable noninvasive cardiometry (ICON®, OSYPKA medical).

These hemodynamic variables were obtained before induction of anesthesia (Basal; T1), post-intubation (T2), just before skin incision (T3), every hour till the end of resection of the urinary bladder (1h; T4A, 2hs; T4B, 3hs; T4C and end of resection; T4D), 10 minutes after switch between both modes (T5), every hour till the end of the urinary diversion (1h; T6A, 2hs; T6B, 3hs; T6C and end of diversion; T6D), after extubation (T7) and postoperative at 2hs and 12hs PACU (P1, P2). At the end of surgery, residual neuromuscular blockade was reversed using neostigmine 50 µg/kg and atropine 20 µg/ kg IV. After fulfilling the criteria of extubation (sustained hand grip, sustained eye opening for 5 seconds and sustained head or leg lift) patients were extubated, transferred to PACU for 24 hours and discharged to the ward when Alderte scored more than 9. Total volumes of fluid administered either crystalloids, voluven, blood or blood products all-over surgery were recorded.

2.6 Sample size calculation

Sample size calculation was based on power analysis using G*power 3.1.9.7 statistical software. power analysis was based on data extraction from a previous study by (Kahl et al. 2021) **[11]**. They assumed that a difference of 0.4/µg/kg/30 min of vasopressor consumption between groups was clinically meaningful. Sample size will be based on that clinically significant difference of vasopressor in patients with diastolic dysfunction with standard deviation equal to 1µg/kg/30min. This difference has created an effect size equal to 0.4 Assuming to have a study of power 74 % and a two-sided test with an α error of 0.05, 39 patients in group 0 (VCV-PRVC) and 47 patients in group 1 (PRVC-VCV) (total 86 patients) were allocated.

2.6 Statistical Analysis

Statistical analysis was performed using SPSS version 27 statistical software. For continuous data, normality was first assessed with the Kolmogorov-Smirnov test. quantitative parametric data, presented in the form of mean (SD), and then analyzed with the student t-test. Data that didn't have a normal distribution, as well as ordinal data, were analyzed with the Mann-Whitney U test. The qualitative variables were expressed in terms of percentage (%) and frequency and were analysed using the chi-square test. All P values presented was 2-sided and values of less than 0.05 were considered significant.

3. Results

This prospective randomized cross-over study included 86 patients of both genders, with ASA physical status I and II, who were undergoing open radical cystectomy. There were no statistically significant differences in demographic data such as age, sex, ASA physical

status, body weight, height, body surface area (BSA), duration of surgery, and anesthesia between the two modes of ventilation being studied **(Table 1).**

In terms of blood flow hemodynamic parameters (CO, CI, SV, and SI), there was no statistically significant difference between the ventilation modes in cardiac output (CO), except at the 3-hour from the start of the resection, where the VCV group had significantly higher CO than the PRVC group (P value= 0.02) **(Fig. 1).** The cardiac index (CI) showed no statistically significant difference between the two modes **(Fig. 2).** The stroke volume (SV) was significantly higher in the VCV group than in the PRVC group at the 3-hour mark from the start of the resection (P value= .03), but there was no significant difference at other time intervals **(Fig. 3).** The stroke volume index (SVI) showed no statistically significant difference between the modes. Regarding fluid status parameters (SVV and FTc), there was no statistically significant difference in the corrected flow time (FTc) between both modes at all time points **(Fig. 4).** Similarly, there was no statistically significant difference in stroke volume variation (SVV%) between both modes at all time points **(Fig. 5).** In terms of vascular resistance, as determined by systemic vascular resistance (SVR) and systemic vascular resistance index (SVRI), there was no statistically significant difference at any time interval between both modes**.**

There were no statistically significant differences observed between the modes in terms of intraoperative inotropic support (dobutamine) or vasopressor support (norepinephrine or total ephedrine dose) **(Table 2).** In terms of crystalloids, colloids, blood and plasma transfusions, blood loss, and total fluids, there were no statistically significant differences between the two modes**.** Regarding the incidence of complications such as major cardiac problem, cardiac acute kidney injury (3 patients represented with clinical presentation of acute renal failure and none needed dialysis), burst abdomen, pneumonia, intestinal obstruction, pulmonary edema, and postoperative ventilation, no statistically significant difference was found between the groups**.**

Table 1. Demographic data of the study's groups:

Parameter	**Group 0 (n=39)**	**Group 1 (n=47)**	***P value***
	M (S. D)	**M (S. D)**	
Age (years)	63.2 (6.79)	64.4 (7.02)	**0.43**
Height (cm)	168.1 (9.36)	168.4 (8.34)	**0.88**
Weight (kg)	79.7 (12.82)	79.9 (16.39)	**0.96**
BMI (kg/m^2)	28.23 (4.25)	28.12 (5.21)	**0.91**
Sex (male %)	35 (89.7%)	42 (89.4%)	**0.95**
BSA (m^2)	1.9 (0.18)	1.9 (0.20)	**0.95**
Surgery's duration (hours)	8.4 (0.53)	8.5 (0.52)	**0.29**
Anaesthesia's duration (hours)	8.9 (0.53)	9.0 (0.52)	**0.29**
ASA			
ASA 1	22 (53.8%)	26 (55.3%)	**0.56**
ASA 2	17 (43.6%)	21 (44.7%)	
Diastolic dysfunction			
Grade 1	42 (97.7%)	39 (90.7%)	***0.36***
Grade 2	1 (2.3%)	4 (9.3%)	
Diversion			

W-ileal neobladder	19 (48.7%)	21 (44.7%)	**0.09**
Ileal loop	15 (38.5%)	25 (53.2%)	
Ureterostomies	5 (12.8%)	1 (2.1%)	

Group 0 (VCV-PRVC), Group 1(PRVC-VCV), BSA: body surface area, BMI: Body mass index, Grade 1 DD: impaired relaxation (E/A <0.8), Grade 2 DD: normal or pseudonormal relaxation (E/A 0.8-1.5), Grade 3 DD: restrictive (E/A >2). Data are presented in mean (SD) or number (%). *Significant inter group difference P < 0.05.

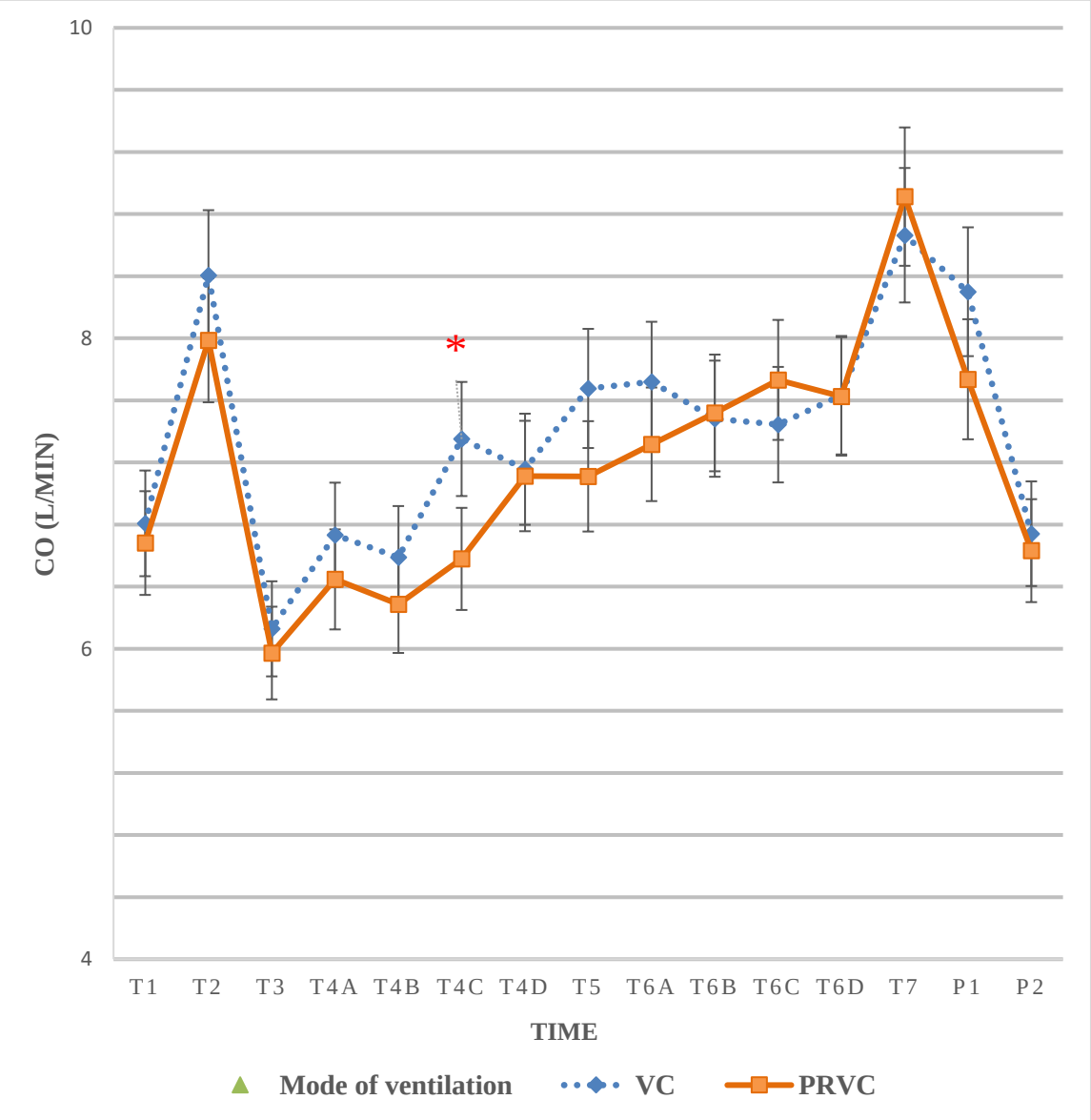

Figure 1. Perioperative changes in cardiac output (CO; l/min) with both modes. (T1) Basal; (T2) post-intubation; (T3) at skin incision; (T4A) 1h from start of resection; (T4B) 2hs from start of resection; (T4C) 3hs from start of resection; (T4D) end of resection; (T5) 10 mins after switch; (T6A) 1h from start of diversion; (T6B) 2hs from start of diversion; (T6C) 3hs from start of diversion; (T6D) end of diversion; (T7) after extubation; (P1) PACU 2hs; (P2)

PACU 12hs. VCV: Volume-controlled, PRVC: Pressure-regulated volume controlled. Data are presented in mean (SD). * Significant inter group difference P < 0.05.

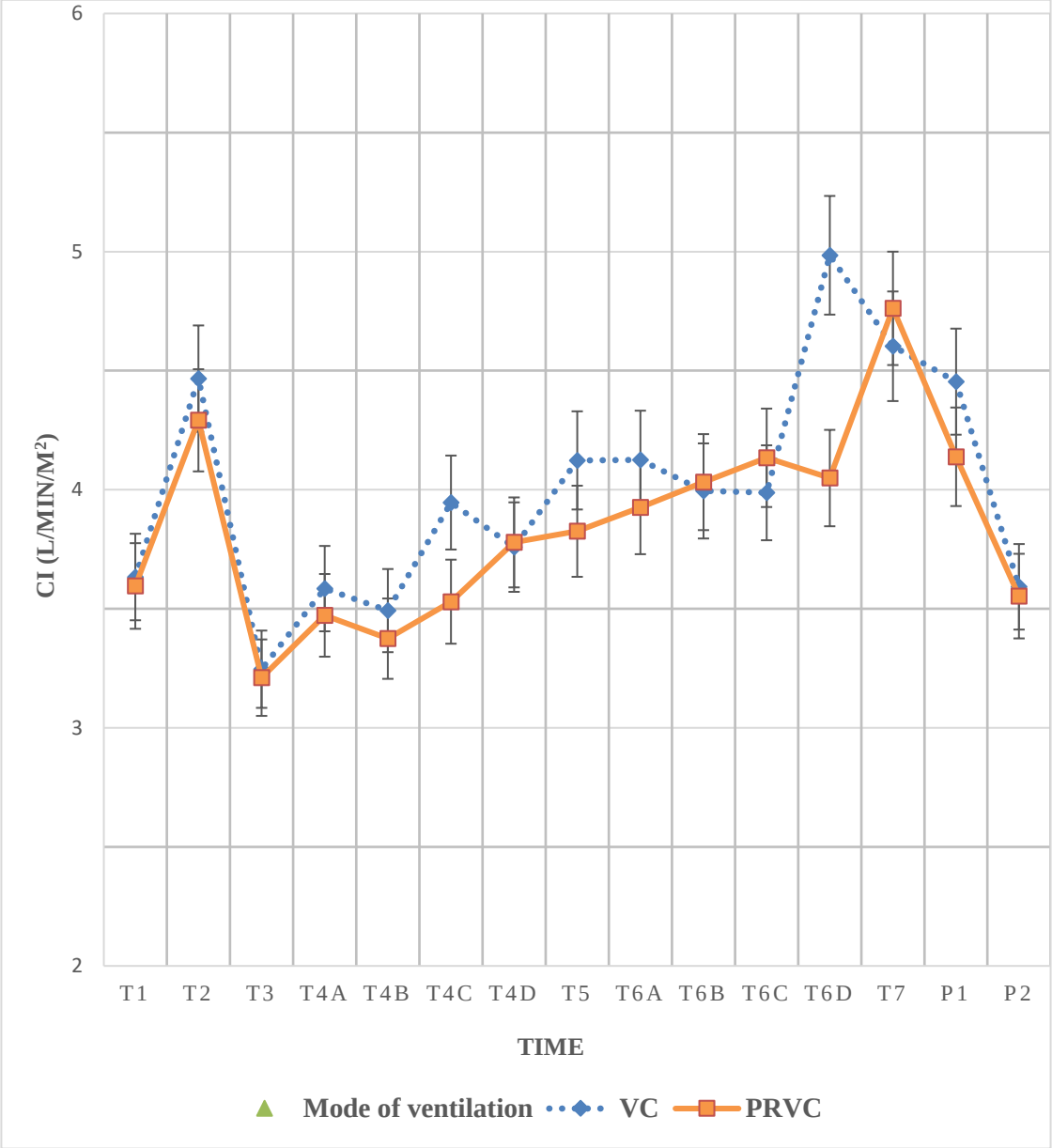

Figure 2. Perioperative changes in cardiac index (CI; L/min/m2) with both modes. (T1) Basal; (T2) post-intubation; (T3) at skin incision; (T4A) 1h from start of resection; (T4B) 2hs from start of resection; (T4C) 3hs from start of resection; (T4D) end of resection; (T5) 10 mins after switch; (T6A) 1h from start of diversion; (T6B) 2hs from start of diversion; (T6C) 3hs from start of diversion; (T6D) end of diversion; (T7) after extubation; (P1) PACU 2hs; (P2) PACU 12hs. VCV: Volume-controlled, PRVC: Pressure-regulated volume controlled. Data are presented in mean (SD). * Significant inter group difference P < 0.05.

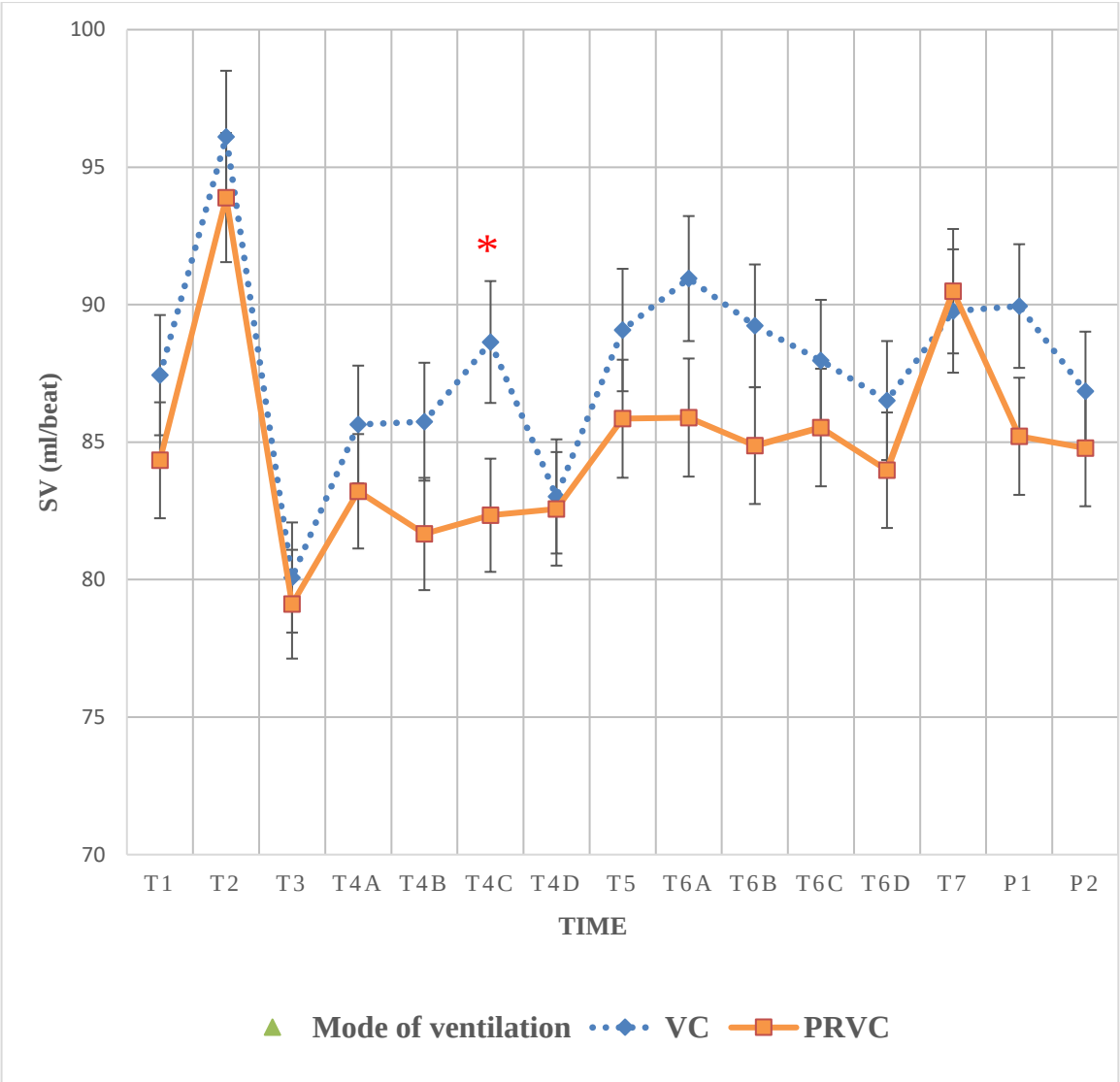

Figure 3. Perioperative changes in stroke volume (SV; ml/beat) with both modes. (T1) Basal; (T2) post-intubation; (T3) at skin incision; (T4A) 1h from start of resection; (T4B) 2hs from start of resection; (T4C) 3hs from start of resection; (T4D) end of resection; (T5) 10 mins after switch; (T6A) 1h from start of diversion; (T6B) 2hs from start of diversion; (T6C) 3hs from start of diversion; (T6D) end of diversion; (T7) after extubation; (P1) PACU 2hs; (P2) PACU 12hs. VCV: Volume-controlled, PRVC: Pressure-regulated volume controlled. Data are presented in mean (SD). * Significant inter group difference $P < 0.05$.

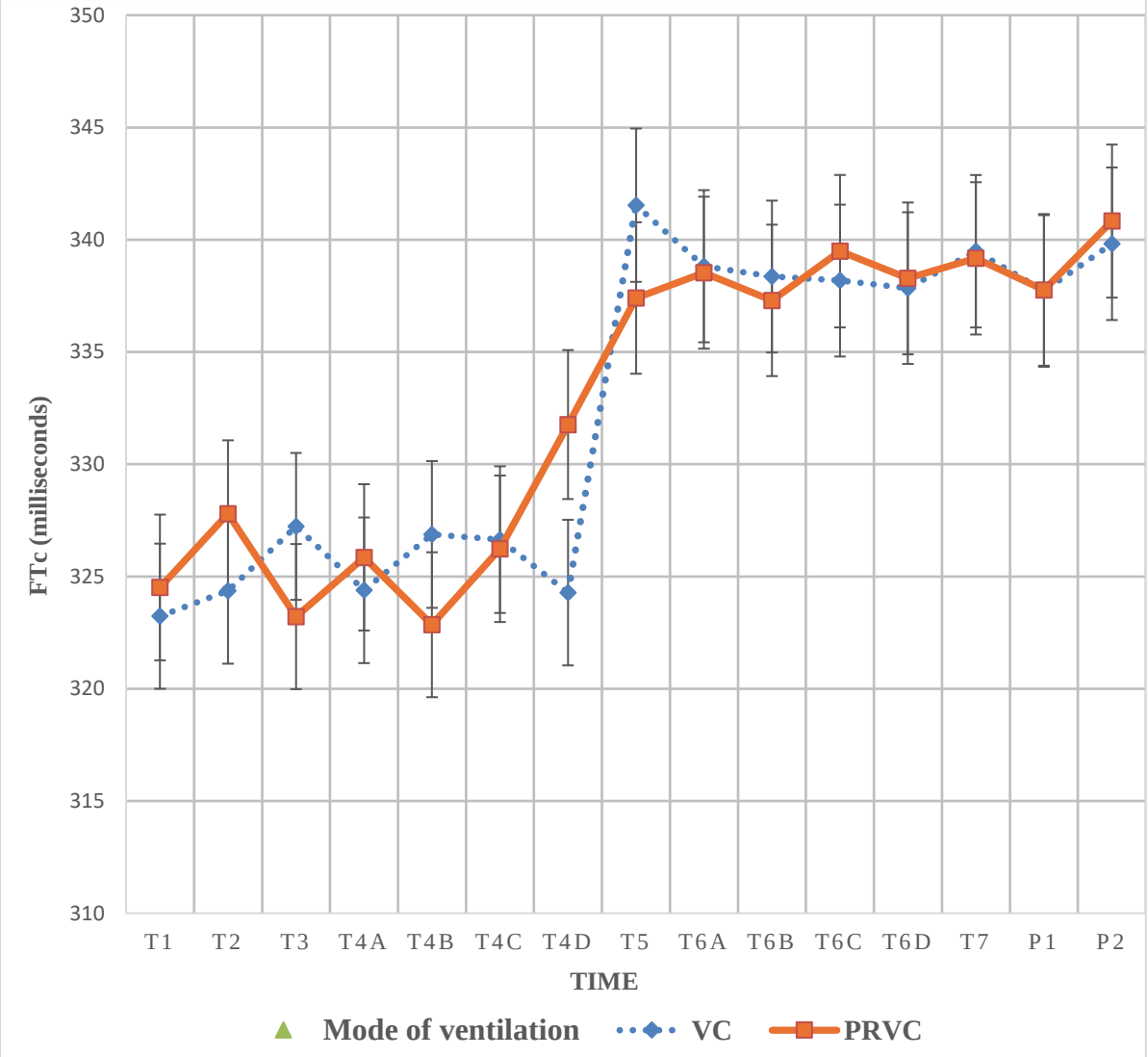

Figure 4. Perioperative changes in corrected flow time (FTc; milliseconds) with both modes. (T1) Basal; (T2) post-intubation; (T3) at skin incision; (T4A) 1h from start of resection; (T4B) 2hs from start of resection; (T4C) 3hs from start of resection; (T4D) end of resection; (T5) 10 mins after switch; (T6A) 1h from start of diversion; (T6B) 2hs from start of diversion; (T6C) 3hs from start of diversion; (T6D) end of diversion; (T7) after extubation; (P1) PACU 2hs; (P2) PACU 12hs. VCV: Volume-controlled, PRVC: Pressure-regulated volume controlled. Data are presented in mean (SD). * Significant inter group difference $P < 0.05$.

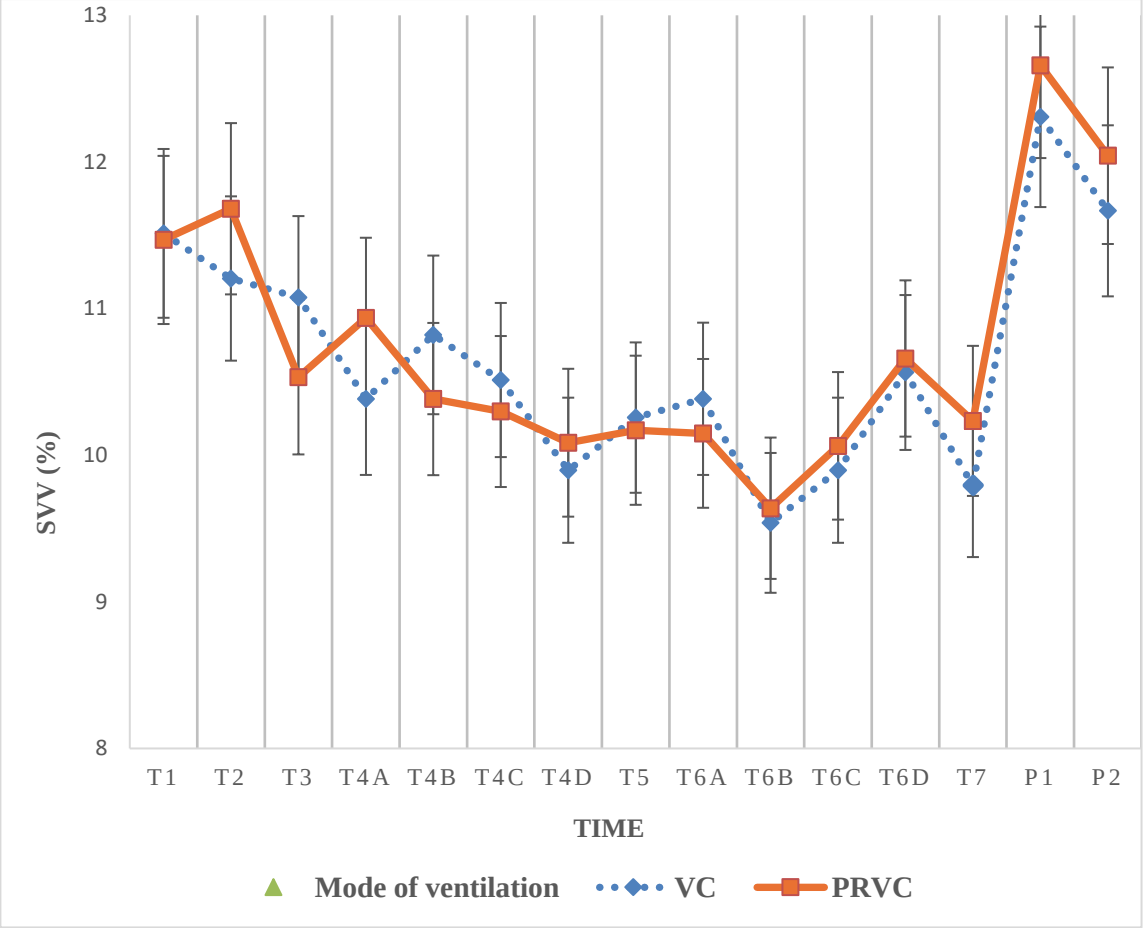

Figure 5. Perioperative changes in stroke volume variation (SVV; %) with both modes. (T1) Basal; (T2) post-intubation; (T3) at skin incision; (T4A) 1h from start of resection; (T4B) 2hs from start of resection; (T4C) 3hs from start of resection; (T4D) end of resection; (T5) 10 mins after switch; (T6A) 1h from start of diversion; (T6B) 2hs from start of diversion; (T6C) 3hs from start of diversion; (T6D) end of diversion; (T7) after extubation; (P1) PACU 2hs; (P2) PACU 12hs. VCV: Volume-controlled, PRVC: Pressure-regulated volume controlled. Data are presented in mean (SD). * Significant inter group difference $P < 0.05$.

Table 2. Intraoperative Circulatory support measures in both studied groups:

	Group 0 (n=39)	Group 1 (n=47)	*P value*
Norepinephrine (.03- .4µg/kg/min)	**0 (0%)**	**2 (4.3%)**	**0.16**
Dobutamine (5 µg/kg/min)	**0 (0%)**	**3 (6.4%)**	**0.08**
Ephedrine (5-10mg bolus)	**27 (69.2%)**	**32 (68.1%)**	**0.91**
Total ephedrine (mg)	**9.87 (7.299)**	**10.53 (10.121)**	**0.73**

Group 0 (VCV-PRVC), Group 1 (PRVC-VCV), h: hour, PACU: post-anesthesia care unit. Data are presented in mean (SD) or number (%). *Significant inter group difference P < 0.05.

4. Discussion

In the present study, several hemodynamic parameters were measured using a noninvasive electrical bioimpedance. This cross over study demonstrated that both VCV and PRVC modes could be utilized in patients with diastolic dysfunction safely with similar perioperative hemodynamics.

Contrary to expectations of the detrimental effects of diastolic dysfunction and mechanical ventilation on the hemodynamic parameters, the current study revealed that CO, CI, SV and SVI did not show significant difference between both modes. This was consistent with previous study results which distinguished non-significant hemodynamic changes in CO, CI, SVI and SV over time between VCV and PCV-VG despite the expected hemodynamic changes in spine surgery that occur during the prone position **[12],** another study showed that CO, CI and SVI had no significant difference between VCV and PCV, although Ppeak was higher in VCV compared to PCV **[13]. Lee et al**. in another study found no significant difference in CO, CI, SVI and SV between VCV mode and pressure-mediated ventilation (PCV or PCV-VG) despite of significant effects of laparoscopic surgery in the Trendelenburg position on venous return and hemodynamic **[14]. Balick-weber et al**. used TEE to assess systolic and diastolic functions during laparoscopic procedures and realized non-significant difference between VCV and PCV on LV end-systolic wall stress or LV preload mainly after switching from VCV to PCV**[15]**. In agreement with our findings **Othman et al**. found no significant changes in CO and SV between PCV when was compared to VCV mode in head trauma patients admitted in icu when the same ventilation strategy and PEEP were applied **[16].**

Although, in diastolic dysfunction, decreased LV compliance rises LV end-diastolic pressure which may be aggravated by increased preload, afterload, and tachycardia during surgery. also, stress significantly increases LV filling pressure with further increase in left atrial pressure to maintain adequate stroke volume. Unfortunately, the net consequence is a decrease in SV, CO, and CI, and inotropic and vasopressor support may be required to maintain these hemodynamics**[4,17]**. This may increase the postoperative cardiac complications, especially in elderly patients with weakened stroke volume, impaired coronary blood flow leading to subendocardial ischemia and hemodynamic **[18].** In addition, positive pressure ventilation and intrathoracic pressures changes may deteriorate myocardial perfusion and diastolic function in patients with pre-operative impaired LV relaxation **[19].** also, PEEP may lead to impaired LV relaxation and may be associated with poor right ventricular (RV) function and possible RV dilatation **[20].** This insignificant difference may be due to similar mean airway pressures of the two modes with the same PEEP value (6-8 cmH_2O), and that adequate fluid management strategy in all patients with similar amounts of iv fluids was also present. The similar fluid and $PRBC_S$ administered between the two groups could potentially stabilize hemodynamics without the need for vasopressors or inotropes.

Regarding the fluid status and responsiveness, there was no significant difference in SVV, this study found that SVV changes that could predict fluid responsiveness over time in both VCV and PRVC were almost equal. **Lee et al.** found that SVV could predict fluid responsiveness during both VCV and PCV modes, he concluded that SVV can be used to guide fluid management during both VCV and PCV modes, but the optimal threshold values of SVV were different: 11% for VCV and 14% for PCV **[21].** Another study found that the change in SVV values over time were not significantly different between pressure-mediated ventilation (PCV or PCV-VG) and VCV during laparoscopic gynecological surgery **[14]. Zitzmann et al**. and his colleagues showed the same results between VCV and PCV at any fluid status, except for baseline where VCV had lower SVV than PCV, however it was an experimental animal study **[22].**

Current study had showed that There was no statistically significant difference in systemic vascular resistance (SVR) and systemic vascular resistance index (SVRI) between the two ventilation modes, indicating equivalent levels of vascular resistance. Several studies compared pressure-mediated ventilation modes with VCV and conducted results that complied with current findings, **Othman et al**. found no significant changes in SVR between VCV and PCV modes in severe head trauma patients with stable hemodynamics **[16]**. **Kocis et al**. compared PRVC and VCV, and their effects on SVR in infants post cardiac surgery using pulmonary artery catheter and found no significant changes in SVR, while PRVC mode resulted in a 19% decrease in peak inspiratory pressure, it did not adversely affect SVR **[23].** SVRI values were lower in the (PCV-VG) group at the onset of peritoneal insufflation, SVRI remained higher in the volume-controlled ventilation (VCV) group throughout pneumoperitoneum during laparoscopic surgeries in exaggerated Trendelenburg position, although the difference was not statistically significant **[24]**. **Peng et al.** found that both VCV and PCV modes did not show a significant impact on SVRI, indicating similar effects on systemic vascular resistance during the surgery during lumbar spine surgery**[25] .**

As regards to intraoperative inotropic support and vasopressor support, where no statistically significant differences were found between the two modes, only 3 patients required intra-operative norepinephrine infusion started at (0.03- 0.4µg/kg/min), two of whom with grade 2 diastolic dysfunction started infusion shortly after induction, continued throughout resection and discontinued after cystectomy, the other patient started infusion during start of lateral wall resection and discontinued at the end of surgery. Two patients had wall motion abnormalities in ECHO with EF 45-50%, one of them had grade 2 diastolic dysfunction required dobutamine infusion throughout cystectomy and discontinued before extubation. We thought that the need for the vasopressors or dobutamine was related to either excessive response to epidural boluses during surgery especially with hypovolemia that might present or surgery-related factors with extensive blood loss and fluid shifting imbalances, the impact of diastolic dysfunction or mechanical ventilation related issues were not of that importance. In conformity with current findings. **Kahl et al**. investigated whether patients with diastolic dysfunction require higher doses of norepinephrine during general anaesthesia for noncardiac surgeries, the results showed that diastolic dysfunction didn't lead to impaired hemodynamic function requiring increased vasopressor support during anaesthesia. Furthermore, diastolic function, as measured by the E/e' ratio, didn't worsen under anesthesia **[11]**. Other studies showed non-significant difference between dual-controlled ventilation and VCV during laparoscopic surgeries in

Trendelenburg position regarding either vasoactive drug used with both of them **[26]**, or total ephedrine dose in mgs consumed intra-operative **[27]**.

This study found no significant differences in the total amount of fluids including crystalloids, colloids, blood, or plasma transfusions administered intraoperatively between both modes, suggesting fluid optimization alone wasnot enough for hemodynamic optimization. Factors like contractility and vascular tone also played a role. The similarity in surgery duration, anesthetic technique, surgeon experience, blood loss, and goal-directed maintenance fluid administration may also explain that no group needed more fluids than the other.

Regarding the incidence of complications such as acute kidney injury (AKI), burst abdomen, pneumonia, intestinal obstruction, pulmonary edema, and postoperative ventilation, there were no statistically significant difference was found between both VCV and PRVC. In concomitant with what the current study found, **Sahutoglu et al**. recorded a non-significance between PCV-VG and VCV as regard respiratory, cardiac, renal and git events post-operatively after one-lung ventilation procedures **[28].** Also, no significance between PCV-VG and VCV regarding pneumonia and acute respiratory distress post-thoracic surgeries was recorded by **Mahmoud et al [29].**

Limitations and Recommendations

The study had some limitations that should be acknowledged. First, it was a single-centred study. Second, relatively small sample size and may have limited the statistical power to detect significant differences between the ventilation modes. Third, the study did not measure the real impact of diastolic dysfunction on hemodynamic parameters as almost all of the patients selected for the study had grade 1 diastolic dysfunction which is common among elderly aged. Fourth, the study did not measure ventilation modes on postoperative pulmonary function, gas exchange, or inflammatory response, which are important outcomes in patients undergoing major abdominal surgery. Finally, the study did not assess the long-term outcomes or quality of life of the patients after the surgery. Therefore, future studies should include larger and more diverse samples, compare different ventilation modes with a control group, measure more comprehensive and clinically relevant outcomes, and follow up the patients for longer periods of time.

5. Conclusion

The findings of this study suggest that PRVC and VCV could be used safely in patients with diastolic dysfunction undergoing major surgery, there were no major differences in CO, CI, SV, SVI and SVV. Also, there was no significant difference as regards fluid status, contractility and cardiac function, vascular resistance, tissue oxygenation, and clinical outcomes between both modes .

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Disclosure statement

The authors stated no conflict of interest.

References:

[1] George EP Godfrey, Marcus JE Peck. Diastolic dysfunction in anaesthesia and critical care, BJA Education. 2016;16(9): 287–291.doi.org/10.1093/bjaed/mkw007.

[2] Fayad A, Ansari MT, Yang H, Ruddy T, Wells GA. Perioperative Diastolic Dysfunction in Patients Undergoing Noncardiac Surgery Is an Independent Risk Factor for Cardiovascular Events: A Systematic Review and Meta-analysis. Anesthesiology. 2016;125(1):72-91. doi:10.1097/ALN.0000000000001132.

[3] Sharkey A, Mahmood F, Matyal R. Diastolic dysfunction - What an anesthesiologist needs to know?. Best Pract Res Clin Anaesthesiol.2019;33(2):221-228. doi:10.1016/j.bpa.2019.07.014.

[4] Cios TJ, Klick JC, Roberts SM. Managing Diastolic Dysfunction Perioperatively. Semin Cardiothorac Vasc Anesth. 2023;27(1):42-50. doi:10.1177/10892532221142441.

[5] Juhl-Olsen P, Hermansen JF, Frederiksen CA, et al. Positive end-expiratory pressure influences echocardiographic measures of diastolic function: a randomized, crossover study in cardiac surgery patients. Anesthesiology. 2013;119(5):1078-1086. doi:10.1097/ALN.0b013e3182a10b40.

[6] Mahmood SS, Pinsky MR. Heart-lung interactions during mechanical ventilation: the basics. Ann Transl Med. 2018;6(18):349. doi:10.21037/atm.2018.04.29

[7] Lee JY, Park HY, Jung WS, et al. Comparative study of pressure- and volume-controlled ventilation on stroke volume variation as a predictor of fluid responsiveness in patients undergoing major abdominal surgery. J Crit Care. 2012;27(5):531.e9-531.e5.31E14. doi:10.1016/j.jcrc.2011.11.012.

[8] Singh G, Chien C, Patel S. Pressure Regulated Volume Control (PRVC): Set it and forget it?. Respir Med Case Rep. 2019;29:100822. doi:10.1016/j.rmcr.2019.03.001.

[9] Vos JJ, Kalmar AF, Hendriks HGD, et al. The effect of fluid resuscitation on the effective circulating volume in patients undergoing liver surgery: a post-hoc analysis of a randomized controlled trial. J Clin Monit Comput. 2018;32(1):73-80. doi: 10.1007/s10877-017-9990-5.

[10] Lopes MR, Oliveira MA, Pereira VO, et al. Goal-directed fluid management based on pulse pressure variation monitoring during high-risk surgery: a pilot randomized controlled trial. Crit Care. 2007;11(5):R100. doi: 10.1186/cc6117.

[11] Kahl U, Vens M, Pollok F, et al. Do Elderly Patients With Diastolic Dysfunction Require Higher Doses of Norepinephrine During General Anesthesia for Noncardiac Surgeries? A Prospective Observational Study. Anesth Analg. 2021;132(2):420-429. doi:10.1213/ANE.0000000000005304 .

[12] Lee JM, Lee SK, Kim KM, et al. Comparison of volume-controlled ventilation mode and pressure-controlled ventilation with volume-guaranteed mode in the prone position during lumbar spine surgery. BMC Anesthesiol. 2019;19(1):133. doi: 10.1186/s12871-019-0806-7.

[13] Fantoni DT, Ida KK, Lopes TF, et al. A comparison of the cardiopulmonary effects of pressure controlled ventilation and volume controlled ventilation in healthy anesthetized dogs. J Vet Emerg Crit Care (San Antonio). 2016 Jul;26(4):524-30. doi: 10.1111/vec.12485.

[14] Lee JM, Lee SK, Rhim CC, et al. Comparison of volume-controlled, pressure-controlled, and pressure-controlled volume-guaranteed ventilation during robot-assisted laparoscopic gynecologic surgery in the Trendelenburg position. Int J Med Sci. 2020;17(17):2728-2734. doi: 10.7150/ijms.49253.

[15] Balick-Weber CC, Nicolas P, Hedreville-Montout M, et al. Respiratory and haemodynamic effects of volume-controlled vs pressure-controlled ventilation during laparoscopy: a cross-over study with echocardiographic assessment. Br J Anaesth. 2007;99(3):429-35. doi: 10.1093/bja/aem166.

[16] Othman MM, Farid AM, Mousa SA, et al. Hemodynamic Effects of Volume-Controlled Ventilation Versus Pressure-Controlled Ventilation in Head Trauma Patients:A Prospective Crossover Pilot Study. ICUDirector.2013;4(5):223-231. doi: 10.1177/1944451613491491.

[17] Roche-Campo F, Bedet A, Vivier E, et al. Cardiac function during weaning failure: the role of diastolic dysfunction. Ann Intensive Care. 2018 Jan 9;8(1):2. doi: 10.1186/s13613-017-0348-4.

[18] Jun IJ, Kim J, Kim HG, et al. Risk factors of postoperative major adverse cardiac events after radical cystectomy: implication of diastolic dysfunction. Sci Rep. 2019;1;9(1):14096. doi: 10.1038/s41598-019-50582-6.

[19] Chin JH, Lee EH, Kim WJ, et al. Positive end-expiratory pressure aggravates left ventricular diastolic relaxation further in patients with pre-existing relaxation abnormality. Br J Anaesth. 2013;111(3):368-73. doi: 10.1093/bja/aet061.

[20] Sanfilippo F, Scolletta S, Morelli A, et al. Practical approach to diastolic dysfunction in light of the new guidelines and clinical applications in the operating room and in the intensive care. Ann Intensive Care. 2018 ;8(1):100. doi: 10.1186/s13613-018-0447-x.

[21] Lee JY, Park HY, Jung WS, Jo YY, Kwak HJ. Comparative study of pressure- and volume-controlled ventilation on stroke volume variation as a predictor of fluid responsiveness in patients undergoing major abdominal surgery. J Crit Care. 2012;27(5):531.e9-531.e5.31E14. doi:10.1016/j.jcrc.2011.11.012.

[22] Zitzmann A, Bandorf T, Merz J, et al. Pressure- vs. volume-controlled ventilation and their respective impact on dynamic parameters of fluid responsiveness: a cross-over animal study. BMC Anesthesiol. 2023;23(1):320. doi: 10.1186/s12871-023-02273-z.

[23] Kocis KC, Dekeon MK, Rosen HK, et al. Pressure-regulated volume control vs volume control ventilation in infants after surgery for congenital heart disease. Pediatr Cardiol. 2001;22(3):233-237. doi:10.1007/s002460010210 .

[24] Yılmaz H, Kazbek BK, Köksoy Ü C, et al. Hemodynamic outcome of different ventilation modes in laparoscopic surgery with exaggerated trendelenburg: a randomised controlled trial. Braz J Anesthesiol. 2022;72(1):88-94. doi: 10.1016/j.bjane.2021.04.028. Epub 2021 May 12.

[25] Peng Z, Xia J, Yin N, et al. The effects of volume-controlled ventilation versus pressure-controlled ventilation on hemodynamic and respiratory parameters in patients undergoing lumbar spine fusion surgery: a randomized controlled trial. Ann Palliat Med. 2021;10(9):9553-9563. doi: 10.21037/apm-21-1932.

[26] Li J, Ma S, Chang X, et al. Effect of pressure-controlled ventilation-volume guaranteed mode combined with individualized positive end-expiratory pressure on respiratory mechanics, oxygenation and lung injury in patients undergoing laparoscopic surgery in Trendelenburg position. J Clin Monit Comput. 2022 Aug;36(4):1155-1164. doi: 10.1007/s10877-021-00750-9. Epub 2021 Aug 26.

[27] Park JH, Park IK, Choi SH, et al. Volume-Controlled Versus Dual-Controlled Ventilation during Robot-Assisted Laparoscopic Prostatectomy with Steep Trendelenburg Position: A Randomized-Controlled Trial. J Clin Med. 2019 Nov 21;8(12):2032. doi: 10.3390/jcm8122032.

[28] Sahutoglu C, Kocabas S, Askar FZ. Effects of pressure-controlled ventilation-volume guaranteed on airway pressure, oxygenation, and postoperative complications in one-lung ventilation: a prospective randomized-controlled study. Int J Clin Exp Med. 2018;11(12):13681-13688.

[29] Mahmoud K, Ammar A, Kasemy Z. Comparison Between Pressure-Regulated Volume-Controlled and Volume-Controlled Ventilation on Oxygenation Parameters, Airway Pressures, and Immune Modulation During Thoracic Surgery. J Cardiothorac Vasc Anesth. 2017 ;31(5):1760-1766. doi: 10.1053/j.jvca.2017.03.026.