Effect of Controlled Mechanical Ventilation without Positive End-expiratory Pressure on Right Ventricular Function after Coronary Artery Bypass Graft Surgery

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To evaluate the changes in right ventricular function during controlled mechanical ventilation (CMV) without positive end-expiratory pressure (PEEP) and during spontaneous breathing, we compared right ventricular ejection fraction (RVEF), right ventricular end-diastolic volume index (RVEDVI), and right ventricular end-systolic volume index (RVESVI) using a thermodilution technique after coronary artery bypass graft surgery. Patients were divided into two groups on the basis of changes in RVEDVI from CMV to spontaneous breathing: group U (n = 6) consisted of patients whose RVEDVI increased during spontaneous breathing compared with mechanical ventilation, group D (n = 3) consisted of patients whose RVEDVI decreased during spontaneous breathing compared with mechanical ventilation. PVRI values during CMV in group D were significantly larger than those in group U. Patients in group U showed no increase in RVEDVI, or decrease in RVEF during CMV without PEEP. However, the remaining 3 patients in group D showed an increase in RVEDVI and a decrease in RVEF during CMV. Mean PAP, RAP, RV systolic pressure, RV end-diastolic pressure, PWP, HR, and mean arterial pressure in both groups were comparable, and showed no significant difference at each of the measured points by 24 hrs postoperatively. Then, RVEF, RVEDVI and RVESVI measured by thermodilution technique is useful in evaluating ventricular function at bedside in ICU. (Key words: intermittent positive pressure ventilation, right ventricle, ejection fraction, thermodilution, coronary artery bypass graft)

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Controlled mechanical ventilation with or without positive end-expiratory pressure (PEEP) is associated with an increase in intrathoracic pressure and may decrease cardiac output and arterial pressure[1]. In patients undergoing coronary artery bypass graft surgery, controlled mechanical ventilation with PEEP is believed to depress right ventricular (RV) function[2,3]. In critical clinical conditions, bedside evaluation of RV function is thought to be crucial for assessing RV hemodynamics as well as left ventricular performance, especially in early vulnerable stage after cardiac surgery. The thermodilu-

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tion technique allows repetitive measurement of right ventricular ejection fraction and volumes. Its reproducibility and the good correlation with other methods have already been reported by a number of authors[4–6]. Monitoring RV function continuously at bedside is also reported to be beneficial in patients undergoing cardiac surgery[2,7–9].

This study was designed to investigate the influence of controlled mechanical ventilation without PEEP on RV function, compared with those during spontaneous breathing, by using thermodilution technique and measuring serial changes in RV function from immediately after operation to 24 hrs postoperatively. The other purpose was to evaluate the advantage of monitoring right ventricular ejection fraction (RVEF), right ventricular end-diastolic volume index (RVEDVI), and right ventricular end-systolic volume index (RVESVI) by using thermodilution technique, as bedside monitoring parameters in intensive care unit (ICU).

Subjects and Methods

Nine patients undergoing elective coronary artery bypass graft surgery were studied over 24 hrs postoperatively. Patients with any evidence of tricuspid regurgitation or pulmonary hypertension were excluded from the study. The protocol of this study was approved by the Institutional Ethics Committee on clinical investigation, and an informed consent was obtained from each patient. After induction of anesthesia, a quadruple-lumen balloon-flotation pulmonary artery catheter mounted with a fast response (95-msec) thermistor (Swan-Ganz Thermodilution Ejection Fraction/Volumetric catheter, Model 93A-431H-7.5F, Baxter/Edwards Critical-Care Division, Santa Ana, CA) was inserted via right internal jugular vein, and its proper position verified before each measurement. Cardiac output and ejection fraction were measured by thermodilution technique using a bedside microprocessor (REF-1^{TM} Ejection Fraction/Cardiac Output Computer, Baxter/Edwards Critical-Care Division), and determined at least in triplicate, using its mean values for statistics. Anesthesia was maintained with oxygen and/or air, fentanyl, and enflurane as needed. All patients received controlled mechanical ventilation without PEEP by Servo Ventilator 900C (SIEMENS-ELEMA AB, Solna, Sweden), and were extubated by 24 hrs postoperatively.

To evaluate the influence of controlled mechanical ventilation on RV function, hemodynamic measurements were performed serially at total of 5 points during 24 hrs: 2 points during mechanical ventilation, (1) immediately after admission to ICU and (2) two hours after admission; 3 points during spontaneous breathing, (3) 30 min before extubation, (4) 30 min after extubation, and (5) 24 hrs after admission to ICU. Arterial pressure (radial artery) and heart rate (HR) were monitored continuously. Hemodynamic parameters consisted of right atrial pressure (RAP), right ventricular pressure (RVP), pulmonary arterial pressure (PAP), pulmonary artery wedge pressure (PWP), cardiac output (CO), and RVEF. Cardiac index (CI), stroke volume index (SVI), and pulmonary vascular resistance index (PVRI) were calculated by standard formulas. RVEDVI was calculated by dividing stroke volume index by RVEF, and RVESVI by RVEDVI minus SVI.

For the purpose of the analysis, patients were divided into two groups on the basis of changes in RVEDVI from controlled mechanical ventilation to spontaneous breathing: group U ($n = 6$) consisted of patients whose RVEDVI increased at point (2) during spontaneous breathing compared with point (3) during mechanical ventilation, group D ($n = 3$) consisted of patients whose RVEDVI decreased at point (2) during spontaneous breathing compared with point (3) during mechanical ventilation.

Data are expressed as mean ± SE. The statistical analysis was performed using one-way analysis of variance, followed by Fisher PLSD test for paired data, Mann-Whiteny U test, and χ^2 test. A P value < 0.05 was considered significant.

Table 1. The characteristics of patients studied

	Group U	Group D
Sex (male/female)	3/3	1/2
Age (yrs)	67 ± 8	62 ± 2
Weight (kg)	51 ± 11	50 ± 0.6
Bypass time (min)	157 ± 26	194 ± 71
Aortic cross-clamping time (min)	119 ± 28	157 ± 63
Cathecholamines		
dopamine	0	3*
dotutamine	0	1†
CABG		
LAD + RCA	2	1
LAD + LCx	2	1
LAD + LCx + D1	1	1
LAD + LCx + OM	1	

Data represent mean ± SD. *administered doses 6.2 ± 2.6 $\mu g \cdot kg^{-1} \cdot min^{-1}$, †administered dose 2.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$. LAD: Left anterior descending branch of left coronary artery, RCA: Right coronary artery, LCx: Circumflex branch of left coronary artery, D1: first diagonal branch of left coronary artery, OM: Obtuse marginal branch of left coronary artery.

Results

Table 1 shows the clinical characteristics of patients in both groups. In group D, all patients needed hemodynamic support with dopamine (5.0-10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and/or dobutamine (2 $\mu g \cdot kg^{-1} \cdot min^{-1}$). There was no significant difference between the groups except the administration of cathecholamines (table 1).

RVEDVI in group U increased by 15% at point (2) compared with point (3), RVEDVI in group D decreased by 10% at point (2) compared with point (3). RVEDVI in group D was significantly larger than in group U at point (2) (fig. 1). However, there was no significant difference in RVEDVI between the groups after the ventilator was disconnected from patient. RVEDVI measured at all 3 point during spontaneous breathing tended to be larger in group D than in group U. RVESVI at point (2), as well as during spontaneous breathing, was significantly larger in group D than in group U (fig. 2). RVEF in group U (0.415 ± 0.034) was significantly larger than group D (0.233 ± 0.047) at point (2) during controlled mechanical ventilation (fig. 3). In group D, RVEF at point (2), during controlled mechanical ventilation, decreased by 39% from spontaneous breathing, whereas RVEF in group U was comparable between mechanical ventilation and spontaneous breathing (0.403 to 0.428). RVEF in group D tended to be smaller than in group U at all measured points.

PVRIs at points (1) and (2) during controlled mechanical ventilation in group D were significantly larger than those at (3) and (4) during spontaneous breathing, whereas PVRIs in group U during controlled mechanical ventilation were comparable to

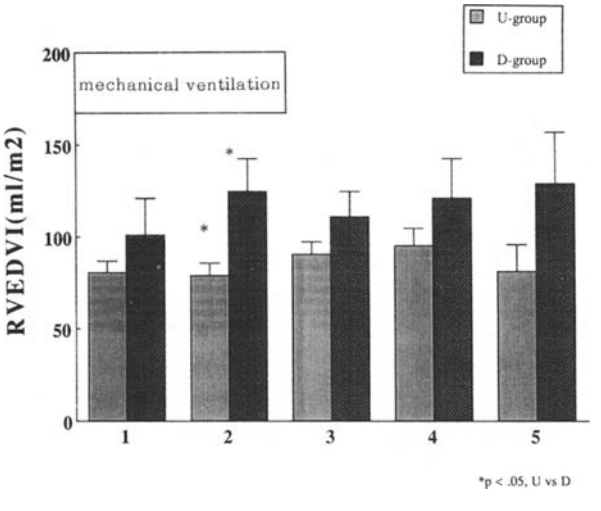

Fig. 1. Serial changes in right ventricular end-diastolic volume index.

(1) immediately after admission to intensive care unit (ICU), (2) two hours after the admission, during mechanical ventilation, (3) 30 min before extubation, (4) 30 min after extubation, and (5) 24 hrs after the admission to ICU under spontaneous breathing. Data represent mean ± SE. *$P < 0.05$, compared between both groups.

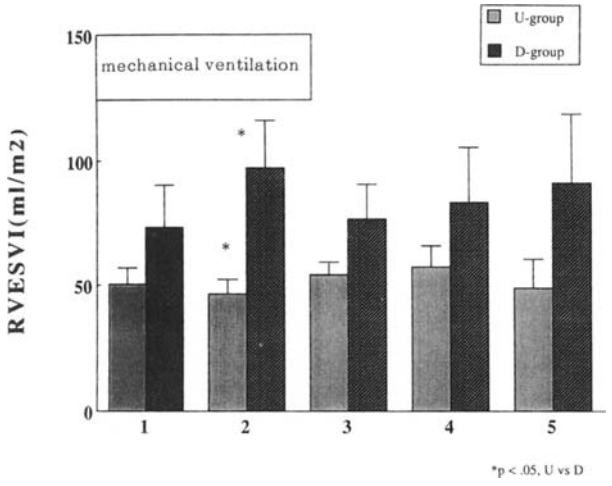

Fig. 2. Serial changes in right ventricular end-systolic volume index.

Data represent mean ± SE. $*P < 0.05$, compared between both groups.

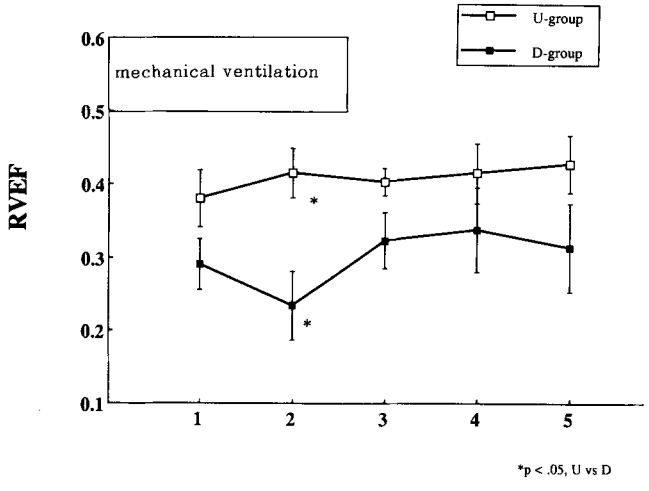

Fig. 3. Serial changes in right ventricular ejection fraction.

Data represent mean ± SE. $*P < 0.05$, compared between both groups.

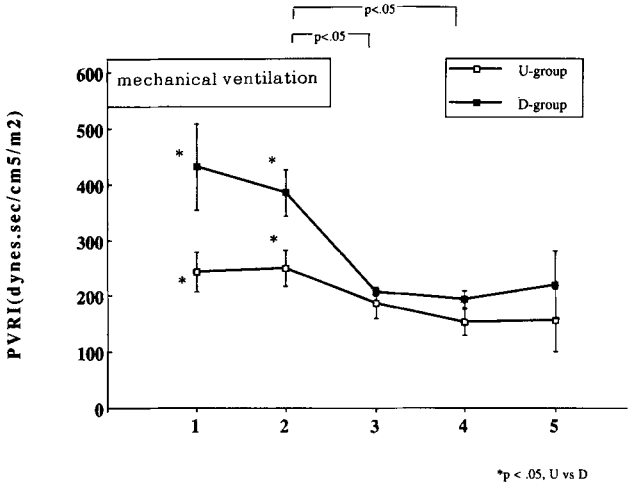

Fig. 4. Serial changes in pulmonary vascular resistance index.

Data represent mean ± SE. $*P < 0.05$, compared between both groups.

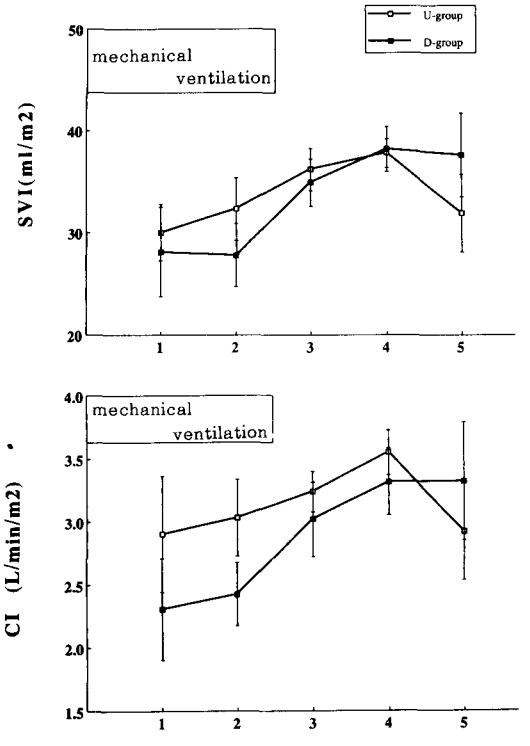

Fig. 5. Serial changes in stroke volume index and cardiac index.

Data represent mean ± SE. $^*P < 0.05$, compared between both groups.

those during spontaneous breathing (fig. 4). In group D, PVRIs during controlled mechanical ventilation were significantly larger than those in group U.

SVI and CI tended to be lower during mechanical ventilation than during spontaneous breathing in both groups. SVI and CI in group D tended to be lower than those in group U. Nevertheless, there was no significant difference between the groups or within each group (fig. 5). Mean PAP, RAP, RV systolic pressure, RV end-diastolic pressure, PWP, HR, and mean arterial pressure in both groups were comparable, and showed no significant difference at each of the measured points by 24 hrs postoperatively (table 2).

Discussion

Controlled mechanical ventilation is an essential therapeutic intervention in patients undergoing cardiac surgery, and during relatively immediate postoperative period. However, controlled mechanical ventilation may affect cardiac performance in critically ill patients. The most common and important hemodynamic effect of artificial ventilation is the decrease in cardiac output by a decreased

Table 2. Hemodynamic data of the patients

		immediately after admission to ICU	2 hours after admission	30 min before extubation	30 min after extubation	24 hours after admission
		mechanical ventilation		spontaneous breathing		
HR	Group U	95 ± 8.6	95 ± 5.2	90 ± 2.7	94 ± 3.3	91 ± 3.0
(min^{-1})	Group D	81 ± 1.7	87 ± 1.3	86 ± 3.5	87 ± 3.4	88 ± 3.3
MAP	Group U	101 ± 4.5	87 ± 4.1	93 ± 5.6	88 ± 3.3	89 ± 3.7
(mmHg)	Group D	108 ± 5.9	94 ± 4.2	99 ± 3.3	96 ± 5.4	90 ± 5.3
MPAP	Group U	15 ± 1.5	17 ± 13	15 ± 1.1	15 ± 1.5	14 ± 2.1
(mmHg)	Group D	18 ± 3.2	20 ± 3.6	17 ± 1.7	16 ± 2.5	18 ± 3.8
PWP	Group U	7 ± 1.2	8 ± 1.5	8 ± 0.9	8 ± 1.4	9 ± 1.5
(mmHg)	Group D	7 ± 3.2	8 ± 3.8	10 ± 1.9	8 ± 2.5	10 ± 2.6
RAP	Group U	6 ± 1.0	7 ± 1.1	6 ± 0.5	7 ± 1.4	6 ± 0.8
(mmHg)	Group D	6 ± 3.7	7 ± 3.3	7 ± 1.7	6 ± 2.4	9 ± 4.2
RVPsys	Group U	29 ± 3.2	29 ± 2.1	28 ± 2.1	30 ± 2.8	28 ± 3.9
(mmHg)	Group D	26 ± 4.4	33 ± 3.0	27 ± 2.6	27 ± 3.9	28 ± 4.3
RVEDP	Group U	4.0 ± 1.3	3.0 ± 0.7	1.5 ± 0.4	1.8 ± 0.9	1.0 ± 0.9
(mmHg)	Group D	3.7 ± 2.3	5.0 ± 3.2	2.3 ± 1.2	2.3 ± 1.5	2.3 ± 1.2

HR: heart rate, MAP: mean arterial pressure, MPAP: mean pulmonary artery pressure, PWP: pulmonary wedge pressure, RAP: right arterial pressure, RVPsys: right ventricular systolic pressure, RVEDP: right ventricular end-diastolic pressure, Data represent mean ± 1 SE.

pressure gradient necessary for right heart venous filling[10]. An increase in transmural PAP is also seen during mechanical ventilation and increases PVR. This increase of PVR leads to an increase in RV afterload, which consequently dilates the right ventricle. PEEP also increases intrathoracic pressure. Jardin et al.[11] reported that with high PEEP, RV loading depresses left ventricular performance through a leftward displacement of the interventricular septum. In patients with depressed RV function or with stenotic right coronary artery, PEEP depresses RV function[12,13]. In patients with already depressed RV contractility, the increased afterload easily deteriorates RV hemodynamic performance.

Anatomical architecture of right and left ventricles, as well as any changes in impedance of pulmonary circulation allow for a remarkable influence of the function of one ventricle upon the other[14,15]. After cardiac surgery, pulmonary circulation is modified by a number of factors due to cardiopulmonary bypass: release of chemical mediators, release of granulocyte protease, tendency to overhydrate, pulmonary vascular endothelial damage, and others. Hence, impedance of the pulmonary circulation may become of major importance of RV function in patients after cardiopulmonary bypass[16]. Monitoring of RV function seems to be essential in detecting early signs of cardiac dysfunction in patients undergoing cardiac surgery, as well as in critically ill patients[4].

In this study, patients were divided into two groups. Most of patients (group U) showed no increase in RVEDVI, or decrease in RVEF during intermittent positive pressure ventilation (IPPV). However, 3 patients (group D) showed an increase in RVEDVI and a decrease in RVEF during IPPV. In group D, PVRIs during IPPV were high compared with those during spontaneous breathing, and also compared with group U. In group D, RV function was believed to have been depressed with IPPV. Schulman et al. reported that PEEP depresses RV function in patients with low RVEF, whereas in patients with normal RVEF, PEEP does not affect RV performance[13]. In our study, patients with increased RVEDVI which was seen during IPPV, had RVEF of 0.290 ± 0.035. This result was in accordance with the observation made by Schulman et al[13].

The reason for the significant increase of PVRI seen during IPPV in 3 out of 9 patients is not clear, although an increase in intrathoracic pressure caused by IPPV without PEEP is thought to have contributed partially. Nevertheless, the different responses of pulmonary circulation exhibited by each of the groups are not explained, because the preoperative patient condition, duration of cardiopulmonary bypass time, and aortic clamping time were comparable between the groups. Doses of dopamine and/or dobutamine used with these 3 patients, less than 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$, was too low to provide pulmonary vasoconstriction[17]. RVEF, RVEDVI and RVESVI measured by thermodilution technique showed apparent changes, whereas routine hemodynamic parameters such as RVP and PAP did not show any changes. Therefore, our results indicate monitoring RVEDVI and RVESVI to be useful in that it may allow earlier intervention of RV hemodynamics, which is in concordance with other clinical studies[2,8,9,12]. We believe that having these information adds an important dimension in deciding which treatment should be applied: volume loading[2] or pharmacological hemodynamic support[18], when treating patients during cardiac surgery. Our results also revealed that monitoring RV hemodynamics is beneficial in assessing postoperative patients during their vulnerable periods in ICU.

In conclusion, the conventional hemodynamic parameters such as right arterial pressure and right ventricular pressure showed no changes during IPPV. In contrast, RVEDVI, RVEF and RVESVI changed, which suggested dilatation of right ventricle. The data in this study showed IPPV without PEEP affected RV function in two different ways: although IPPV did not affect RV function after cardiac surgery in most of the patients, it had a significant effect on other patients. RVEF, RVEDVI and RVESVI measured by

thermodilution technique, therefore, is useful in evaluating ventricular function at bedside in ICU.

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