Hemodynamic Effects of High-frequency Jet Ventilation in Dogs with Acute Right Coronary Arterial Ligation and Pulmonary Arterial Banding

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The hemodynamic effects of high-frequency jet ventilation (HFJV), synchronized with diastole, and intermittent positive-pressure ventilation (IPPV) were studied in 10 dogs with acute right-sided myocardial ischemia and elevated right ventricular pressure. Myocardial ischemia was produced by ligation of the proximal right coronary artery (RCA), then the right ventricular pressure was elevated to facilitate the ischemia by banding the main pulmonary artery. Before and 1, 2, 3, and 5 hr after the RCA ligation, cardiorespiratory variables for each ventilatory mode and creatine phosphokinase MB isoenzyme (CPK-MB) were measured. During HFJV compared with IPPV: there were significant increases in stroke index and left ventricular stroke work index at all ischemic periods, and decreases in peak and mean airway pressures and pulmonary vascular resistance at all ischemic periods, and in the product of systolic right ventricular pressure and heart rate at 2 hr, 3 hr, and 5 hr. The difference in mean airway pressure between IPPV and HFJV correlated significantly with those in cardiac index and stroke index ($r = 0.575$ and 0.779, respectively). CPK-MB was significantly greater at 3 hr and 5 hr than that before RCA ligation. These findings suggest that HFJV synchronized with diastole offers hemodynamic advantages over IPPV to ischemic right ventricle with constricted pulmonary artery, mainly due to lowering the mean airway pressure. (Key words: HFJV synchronized with diastole, IPPV, hemodynamic effects, right myocardial ischemia, pulmonary arterial banding)

(Ushijima K, Oka Y, Weinberg P et al.: Hemodynamic effects of high-frequency jet ventilation in dogs with acute right coronary arterial ligation and pulmonary arterial banding. J Anesth 4: 232–241, 1990)

It is well known that increased intrathoracic pressure associated with intermittent positive-pressure ventilation (IPPV) may cause a lowering of right ventricular filling pressures and a decrease in central blood volume[1–3], a decrease in biventricular end-diastolic volumes[4], an increase in pulmonary vascular resistance[5], an alteration in cardiac geometry[6], a shift of the interventricular septum[7], and a decrease in myocardial contractility[8], leading to a reduction in cardiac output. Therefore, high-frequency jet ventilation (HFJV) may be expected to cause less hemodynamic impairment than IPPV, because of its possibility of lowering airway pressure[9,10]. There are several reports on the hemodynamic advantages of HFJV over IPPV, suggesting that HFJV may assist right heart function mechanically or by changing pre- and/or afterload[11–15].

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Furthermore, we have demonstrated that the function of the dilated right ventricle was maintained better with HFJV, particularly when the peak airway pressure was synchronized with the diastolic phase of the cardiac cycle, than with IPPV in dogs with a chronically banded pulmonary artery[16]. However, there is no report on the influence of HFJV on the function of ischemic right heart. In this study, the cardiorespiratory effects of HFJV synchronized with diastole were compared with those of IPPV in dogs with acute right-sided myocardial ischemia and elevated right ventricular pressure.

Materials and Methods

Ten female mongrel dogs weighing 20 to 29 (23.9 ± 2.4) kg were used. Following the induction of anesthesia with 10 $mg \cdot kg^{-1}$ of intravenous (iv) sodium thiopental, the animals were intubated with a 9.0-mm ID endotracheal tube (Hi-Lo Jet, National Catheter Corporation, Argyle, NY). Standard limb electrocardiograph (ECG) leads were attached; ECG was continuously monitored throughout the experiment. The femoral artery was cannulated for pressure monitoring and blood gas sampling. Two 7-Fr flow-directed thermodilution catheters (Swan-Ganz, American Edwards Laboratories, Santa Ana, CA) were positioned in the pulmonary artery via a jugular vein to measure pulmonary arterial pressure, pulmonary artery wedge pressure (PAWP), central venous pressure (CVP), and cardiac output (CO), and in the right ventricle through a femoral vein to measure right ventricular pressure. Anesthesia was maintained with continuous iv drip of fentanyl citrate (15 $\mu g \cdot kg^{-1} \cdot hr^{-1}$). A lactated Ringer's solution was administered through a peripheral vein at a constant rate of 5 $ml \cdot kg^{-1} \cdot hr^{-1}$. Muscular paralysis was obtained with continuous iv drip of vecuronium bromide (0.1 $mg \cdot kg^{-1} \cdot hr^{-1}$).

Following a median sternotomy and pericardiotomy, 2 $mg \cdot kg^{-1}$ of bolus lidocaine was intravenously given for prevention of ventricular arrhythmia, and the proximal right coronary artery (RCA) was ligated. Then lidocaine was continuously infused at a rate of 0.1 $mg \cdot kg^{-1} \cdot min^{-1}$. About 10 min after the RCA ligation, in order to facilitate the myocardial ischemia, the main pulmonary artery was banded to approximately double the systolic right ventricular pressure by degrees in 10 to 20 min. The heart was observed for 10 to 20 min and the sternotomy was closed. The pericardium and the accidentally cut mediastinal pleura were left open.

Before sternotomy and at 1, 2, 3, and 5 hr after RCA ligation, IPPV and HFJV were alternately performed in random order, and the cardiorespiratory variables mentioned below were measured for each mode of ventilation. A 15-min period for each ventilatory mode was used to allow stabilization of hemodynamics and blood gases. IPPV was provided with a Bird Mark 7 (Bird Corporation, Palm Springs, CA) using a high-pressure oxygen gas for driving source at an inspired oxygen fraction ($F_{I_{O_2}}$) of 1.0. A Pa_{CO_2} of 40 ± 5 torr was maintained at a respiratory rate of 8 to 10 $breaths \cdot min^{-1}$ and an inspiratory/expiratory (I:E) ratio of 1:2. The animals were always ventilated with IPPV except during the periods of HFJV. HFJV was accomplished with a ventilator (MSKCC, New York, NY) in which an electronically controlled solenoid valve periodically interrupted the flow from a high-pressure oxygen source. Before sternotomy, the respiratory rate was fixed at 100 $breaths \cdot min^{-1}$ without synchronization, because the heart rate (HR) was so low that the respiratory rate would be deficient if HFJV was synchronized with the cardiac cycle. For the ischemic periods, the solenoid valve was triggered by the R wave of ECG after a variable delay adjusted so that the peak airway pressure was synchronized with the diastolic phase of the cardiac cycle. Jet flow was delivered via the insufflation lumen in the side wall of the endotracheal tube. Driving pressure was adjusted to obtain a Pa_{CO_2} of 40 ± 5 torr at an I:E ratio of 1:2. An $F_{I_{O_2}}$ of 1.0 was assured by connecting the endotracheal tube to the circuit of an anesthesia machine with a flow of oxygen (5

Table 1. Value of creatine phosphokinase MB isoenzyme in each period

	Before sternotomy	1 hr	2 hr	3 hr	5 hr
Mean ± SD	1.1 ± 1.7	1.8 ± 2.3	2.2 ± 2.7	5.5 ± 3.4*	13.7 ± 8.3**
Range	0 – 5	0 – 7	0 – 8	0 – 12	4 – 35

*$P < 0.01$ different from the period before sternotomy, and $P < 0.05$ different from 1 hr and 2 hr.

** $P < 0.01$ different from all other periods.

Table 2. Respiratory variables during IPPV and HFJV in each period

		Before sternotomy (BS)	1 hr	2 hr	3 hr	5 hr
PIP (mmHg)	IPPV	11.0 ± 1.3[a]	11.4 ± 1.0	12.3 ± 1.8	12.3 ± 1.1	12.7 ± 1.3
	HFJV	5.7 ± 0.9**	5.4 ± 2.3**	5.9 ± 1.9**	5.7 ± 1.9**	5.4 ± 1.7**
EEP (mmHg)	IPPV	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
	HFJV	0.6 ± 0.6*	0.2 ± 0.3	0.3 ± 0.5	0.2 ± 0.2*	0.2 ± 0.2*
$\overline{Paw}$ (mmHg)	IPPV	2.5 ± 0.8[b]	2.8 ± 0.8	3.1 ± 1.0	3.2 ± 0.4	3.5 ± 0.7
	HFJV	2.4 ± 0.6	1.8 ± 0.7*	2.0 ± 0.4*	2.0 ± 0.5*	2.0 ± 0.4**
Pa_{O_2} (torr)	IPPV	464 ± 67[c]	311 ± 99	314 ± 85	333 ± 77	284 ± 126
	HFJV	432 ± 67[d]	294 ± 95	319 ± 113	321 ± 101	267 ± 128
$\dot{Q}sp/\dot{Q}t$ (%)	IPPV	10.4 ± 4.6[c]	16.9 ± 4.4	18.1 ± 5.0	17.0 ± 3.3	18.0 ± 7.9
	HFJV	13.5 ± 5.2[e]	18.0 ± 5.0	16.5 ± 6.0	16.2 ± 5.3	18.1 ± 5.1
DO_2I	IPPV	466 ± 133	497 ± 92	453 ± 119	462 ± 140	508 ± 161
($ml \cdot min^{-1} \cdot m^{-2}$)	HFJV	507 ± 160	530 ± 88	488 ± 114	480 ± 148	566 ± 242

(mean ± SD)

IPPV vs. HFJV in each period; *$P < 0.05$ and **$P < 0.01$.

Comparisons between periods for each ventilatory mode;

[a] $P < 0.05$ BS vs. 5 hr.

[b] $P < 0.01$ BS vs. 5 hr, and $P < 0.05$ BS vs. 3 hr.

[c] $P < 0.01$ BS vs. all other periods.

[d] $P < 0.01$ BS vs. 1 hr and 5 hr, and $P < 0.05$ BS vs. 2 hr and 3 hr.

[e] $P < 0.05$ BS vs. 1 hr and 5 hr.

$l \cdot min^{-1}$). Arterial blood gases were analyzed at any time, and when metabolic acidosis existed, 8.4% sodium bicarbonate (body weight ×0.2× (base excess) ×1/2 ml) was given. As a result, arterial pH and base excess could be kept within the normal limits at every period in obtaining the experimental data. No catecholamine was administered and core temperature measured in the pulmonary artery was kept at 37.0 ± 0.5°C by the use of a heating pad throughout the study.

The following cardiorespiratory variables were measured for each ventilatory mode at each period: peak inspiratory (PIP), end-expiratory (EEP), and mean ($\overline{Paw}$) airway pressures; arterial and mixed-venous blood gas tensions and hemoglobin concentrations; systolic, diastolic, and mean arterial and pulmonary arterial pressures; systolic (RVSP), end-diastolic (RVEDP), and systolic mean (mRVP) right ventricular pressures; HR, PAWP, CVP, and CO. Airway pressure was measured through an orifice at the distal tip of the endotracheal tube, i.e., at 7 cm below the level of the jet injection port. CO was measured by thermodilution with a computer (9520A, American Edwards Laboratories, Santa Ana, CA) using triplicate

Table 3. Cardiovascular variables during IPPV and HFJV in each period

		Before sternotomy (BS)	1 hr	2 hr	3 hr	5 hr
RVSP (mmHg)	IPPV	23.3±4.6[a]	49.1±9.2	48.0±12.5	45.5±8.2	45.4±7.9
	HFJV	23.4±7.0[a]	48.9±8.8	45.6±9.6	43.9±7.3	44.6±9.0
RVEDP (mmHg)	IPPV	3.7±2.1[b]	5.9±3.1	5.8±1.5	6.3±1.4	6.1±1.1
	HFJV	3.9±2.1[c]	6.3±2.9	6.1±3.1	6.7±1.1	6.8±1.7
mRVP (mmHg)	IPPV	13.5±2.6[a]	28.4±5.0	27.0±5.4	25.9±4.0	26.1±3.8
	HFJV	13.6±4.5[a]	27.1±4.1	26.6±4.9	25.8±3.6	26.3±4.4
CVP (mmHg)	IPPV	2.5±2.1[d]	4.7±2.1	5.2±1.7	5.3±1.6	5.5±2.3
	HFJV	2.9±2.8[e]	4.7±1.8	5.0±2.2	5.5±1.8	5.9±2.1
HR (beats·min^{-1})	IPPV	63±12[a]	110±17	108±14	109±15	117±18
	HFJV	64±9[a]	107±14	103±14	104±17	110±19
RVSP×HR	IPPV	1474±407[a]	5402±1466	5119±1600	4959±1259	5314±1363
(mmHg·beats·min^{-1})	HFJV	1516±549[a]	5244±1350	4607±1097*	4611±1257**	4851±1320*
CI (l·min^{-1}·min^{-2})	IPPV	2.30±0.49	2.29±0.29	2.13±0.39	2.17±0.57	2.31±0.70
	HFJV	2.49±0.61	2.46±0.37	2.34±0.57	2.27±0.66	2.57±1.07
SI (ml·m^{-2})	IPPV	36.5±4.8[a]	21.3±3.5	19.6±1.9	20.2±5.0	20.0±6.1
	HFJV	38.0±6.2[a]	23.2±3.6*	22.7±3.2*	22.0±5.8**	23.7±9.1*
LVSWI (g·m·m^{-2})	IPPV	46.3±9.7[a]	26.1±5.6	25.0±7.1	26.5±8.3	27.2±11.1
	HFJV	51.4±9.8[f]	30.2±6.3*	29.3±6.0*	29.5±8.5*	34.4±18.1*
PVRI	IPPV	201±69[g]	271±74[h]	285±75	329±68	351±78
(dyne·sec·m^2·cm^{-5})	HFJV	189±61[b]	234±60**,[h]	246±76**	286±48*	311±68**

(mean±SD)

IPPV vs. HFJV in each period; *$P < 0.05$ and **$P < 0.01$.
Comparisons between periods for each ventilatory mode;
[a] $P < 0.01$ BS vs. all other periods.
[b] $P < 0.01$ BS vs. 3 hr and 5 hr, and $P < 0.05$ BS vs. 2 hr.
[c] $P < 0.01$ BS vs. 3 hr and 5 hr.
[d] $P < 0.01$ BS vs. 2 hr and 3 hr, and $P < 0.05$ BS vs. 1 hr and 5 hr.
[e] $P < 0.05$ BS vs. 3 hr and 5 hr.
[f] $P < 0.01$ BS vs. 1 hr, 2 hr and 3 hr, and $P < 0.05$ BS vs. 5 hr.
[g] $P < 0.01$ BS vs. 3 hr and 5 hr, and $P < 0.05$ BS vs. 1 hr and 2 hr.
[h] $P < 0.05$ 1 hr vs. 5 hr.

injections of 5 ml of room-temperature 5% dextrose, and recorded as the mean of 3 measurements. During IPPV, all intravascular pressure measurements were taken at end-expiration, while CO measurement was not timed to the respiratory cycle. All pressures were measured in the supine position with pressure transducers (Model 800, Bentley Trantec Incorporation, Irvine, CA) which had been zero-referenced at the mid-thoracic line, and simultaneously displayed with ECG on a multichannel recorder. Mean pressure values were obtained by planimetric integration of the traces. Intrapulmonary physiologic shunt ratio ($\dot{Q}sp/\dot{Q}t$), oxygen delivery index ($D_{O_2}I$), arteriovenous oxygen content difference, oxygen consumption index, cardiac index (CI), stroke index (SI), left ventricular stroke work index (LVSWI), and systemic and pulmonary (PVRI) vascular resistance indexes were calculated with standard formulae. The product of RVSP and HR (RVSP × HR) was also calculated. Right ventricular stroke work index (RVSWI) was calculated using the following equation:

$$RVSWI = 1.36\ (mRVP - RVEDP) \times SI/100$$

Canine oxygen hemoglobin saturation was

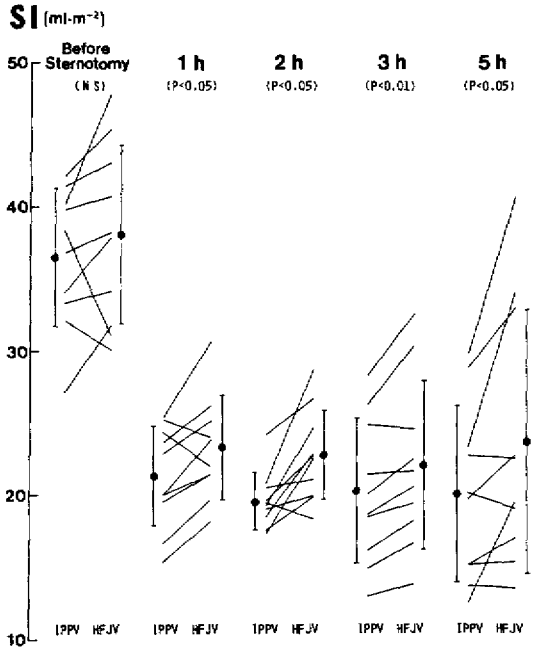

Fig. 1. Comparative values of stroke index (SI) during intermittent positive-pressure ventilation (IPPV) and high-frequency jet ventilation (HFJV) in each dog at each period. Mean values with standard deviations in both ventilatory modes at each period are also shown.

derived from the nomogram prepared by Rossing et al.[17], and body surface area was calculated with the equation presented by Stahl[18]. Creatine phosphokinase MB isoenzyme (CPK-MB) was also measured just before the sternotomy and at 1, 2, 3, and 5 hr after the RCA ligation. Gross autopsy was performed at the end of the experiment.

Data are reported as mean values with standard deviations. Results were analyzed using the Wilcoxon signed-rank test for paired data between IPPV and HFJV in each period. Comparisons between the periods for CPK-MB and for each cardiorespiratory variable in each ventilatory mode were performed by the Mann-Whitney U test. Furthermore, relationships between $\Delta P\overline{aw}$ and ΔCI, ΔSI, or $\Delta PVRI$, as well as between ΔPIP and ΔCI, ΔSI, or $\Delta PVRI$, were analyzed with linear regressions by the least-square methods; where Δ means the algebraical difference in each variable between IPPV and HFJV ($n = 50$). A value of $P < 0.05$ was regarded as statistically significant.

Results

Status of the dogs

Immediately after the RCA ligation, about 30 to 40% of the right ventricular surface area appeared cyanotic, and following the pulmonary artery banding, the enlargement of the cyanotic area and the dyskinesia of the right ventricular wall were observed in all cases. Although we did not quantitatively assess the areas of myocardial injury/infarction, CPK-MB increased significantly at 3 hr and 5 hr as compared with that before sternotomy ($P < 0.01$, table 1). Even at the end of the experiment, all animals had a cyanotic right ventricle although some showed the reductions in size and intensity of the myocardial cyanosis. Arrhythmia that might possibly affect the results of measurement was not detected. At autopsy, the left heart and the cardiac valves were normal, and neither visceral congestion nor ascites was observed, while some hemothorax, pneumothorax, and atelectasis were observed in all animals.

Comparison between IPPV and HFJV (tables 2 and 3)

During HFJV as compared with IPPV, there were significant increases in EEP before sternotomy and at 3 hr and 5 hr, SI and LVSWI at all ischemic periods, and decreases in PIP at all experimental periods, $P\overline{aw}$ at all ischemic periods, RVSP × HR at 2 hr, 3 hr, and 5 hr, and PVRI at all ischemic periods. The other variables showed no significant differences. The comparative values of SI during IPPV and HFJV in each animal at each period are shown in figure 1.

Comparison between periods (tables 2 and 3)

There were significant increases in RVSP, mRVP, HR, and RVSP × HR, and decreases in Pa_{O_2}, SI, and LVSWI at all ischemic periods as compared with those before sternotomy in both ventilatory modes. PIP at 5 hr, and $P\overline{aw}$ at 3 hr and 5 hr significantly increased during IPPV as compared with

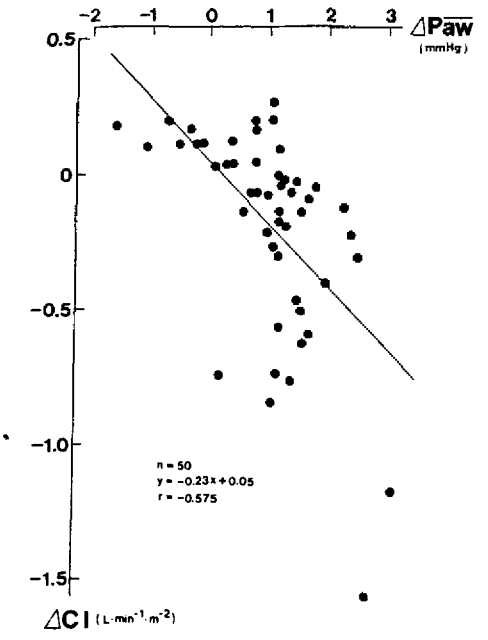

Fig. 2. Relationship between $\Delta P\bar{a}w$ and ΔCI. The line of regression fits the equation: $y = -0.23x + 0.05$, where $y = \Delta CI$ and $x = \Delta P\bar{a}w$. $P < 0.01$. Abbreviations: $P\bar{a}w$; mean airway pressure, CI; cardiac index.

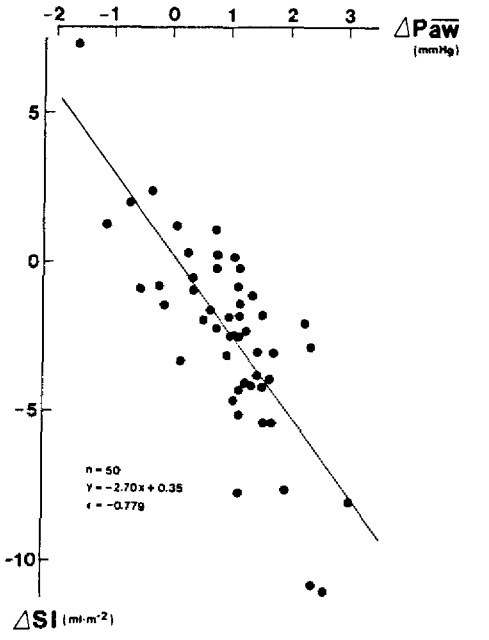

Fig. 3. Relationship between $\Delta P\bar{a}w$ and ΔSI. The line of regression fits the equation: $y = -2.70x + 0.35$, where $y = \Delta SI$ and $x = \Delta P\bar{a}w$. $P < 0.01$. Abbreviations: $P\bar{a}w$; mean airway pressure, SI; stroke index.

those before sternotomy, while there were no significant differences in those during HFJV. $\dot{Q}sp/\dot{Q}t$ increased significantly at all ischemic periods during IPPV and at 1 hr and 5 hr during HFJV as compared with that before sternotomy. During IPPV, RVEDP at 2 hr, 3 hr, and 5 hr, and CVP at all ischemic periods increased significantly in comparison with those before sternotomy, while the increases in those during HFJV were observed only at 3 hr and 5 hr. PVRI at all ischemic periods during IPPV and at 2 hr, 3 hr, and 5 hr during HFJV was significantly greater than that before sternotomy. There was no significant fall in CI even during the ischemic periods.

Relationship between the differences in airway pressure and hemodynamic parameters

There were significant correlations between $\Delta P\bar{a}w$ and ΔCI ($\Delta CI = -0.23$ ($\Delta P\bar{a}w$) + 0.05, r = 0.575, $P < 0.01$, figure 2), and between $\Delta P\bar{a}w$ and ΔSI ($\Delta SI = -2.70$ ($\Delta P\bar{a}w$) + 0.35, r = 0.779, $P < 0.01$, figure 3). However, there was no significant correlation between $\Delta P\bar{a}w$ and $\Delta PVRI$, and ΔPIP did not significantly correlate with any of ΔCI, ΔSI, and $\Delta PVRI$.

Discussion

A change in airway pressure can cause a corresponding change in cardiac performance, and it is generally believed that mean rather than peak airway pressure affects cardiac function[19]. Although there are many controversial reports on the hemodynamic effects of HFJV compared with those of IPPV under both normal and pathological conditions[10–13,15,16,19–23], it seems possible to explain the differences in their results mainly by the difference in $P\bar{a}w$. Some showed that the hemodynamic advantages of HFJV over IPPV were directly related to the extent that $P\bar{a}w$ was lowered[11,13,15,21], and others revealed that the hemodynamic effects were similar when comparable lev-

els of $\overline{Paw}$ were applied between HFJV and IPPV[19,20], meanwhile cardiac function was depressed during HFJV when $\overline{Paw}$ was higher than during IPPV[22]. In our study, $\Delta\overline{Paw}$ significantly correlated with ΔCI and ΔSI, but ΔPIP did not, suggesting that the decrease in $\overline{Paw}$ might contribute partly to the increase in SI during HFJV as compared with IPPV though the increase in CI during HFJV was statistically insignificant.

Despite the close relation between $\Delta\overline{Paw}$ and ΔSI, $\Delta\overline{Paw}$ was only 1.0 ± 0.9 (−1.6 to 3.1) mmHg. This suggests that some factors other than $\Delta\overline{Paw}$ might also influence ΔSI. Such factor can be the cardiac depressant, negative inotropic, reflex that originates in the lung as demonstrated by Shepherd[24]. The magnitude of this reflex is dependent on the volume of gas insufflated in the lung[24,25], which may be smaller during HFJV than during IPPV[26]. Thus this cardiac depressant reflex might be less evident in HFJV than in IPPV. Furthermore, Whittenberger et al. showed that the influence of the state of lung inflation on the pulmonary vascular resistance and the consequent pulmonary blood flow is stronger at low levels of pulmonary blood flow than at high levels[5]. In our study, pulmonary blood flow, SI, markedly decreased at all ischemic periods as compared with that before sternotomy, suggesting that the decrease in level of pulmonary blood flow caused by the myocardial ischemia and the pulmonary artery banding might be another possible factor that made the difference in SI between IPPV and HFJV at all ischemic periods.

Several studies suggested that HFJV may support right heart function mechanically or by increasing preload and/or decreasing afterload, because of its possibility of maintaining oxygenation and alveolar ventilation without such an increase in intrathoracic pressure as associated with IPPV[11−15]. Our results demonstrate a significant decrease in PVRI during HFJV from that during IPPV and the corresponding increase in SI at all ischemic periods. These findings suggest that the decrease in right ventricular afterload might partly contribute to the increase in SI during HFJV, although there was no significant linear correlation between $\Delta\overline{Paw}$ and ΔPVRI. On the other hand, transesophageal echocardiographic dimensional analyses reveal that the decrease in CO associated with the increased intrathoracic pressure during positive end-expiratory pressure (PEEP, 10 to 20 cmH_2O) ventilation may be primarily caused by the mechanical reduction in right ventricular and right atrial distensibility, or end-diastolic dimensions, i.e., reduced right ventricular preload without impairment of the right ventricular systolic function[27,28]. In our study, RVEDP and CVP at the ischemic periods were elevated earlier during IPPV than during HFJV, suggesting that the right ventricular function might be depressed sooner during IPPV. However, the differences in RVEDP and CVP between IPPV and HFJV in each period were not statistically significant. Therefore, the participation of right ventricular preload in the hemodynamic improvement during HFJV was uncertain.

Studies on the hemodynamic effects of HFJV synchronized with specific phases of the cardiac cycle are also controversial[11,16,29]. We have demonstrated that HFJV had hemodynamic advantages over IPPV particularly when the peak airway pressure was synchronized with diastole of the cardiac cycle in dogs with a dilated right ventricle caused by chronical banding of the main pulmonary artery[16]. Because of this reason we chose the mode of HFJV synchronized with diastole at the ischemic periods.

The product of systolic arterial pressure and HR, so-called double index, has been widely used as a simple index of myocardial oxygen consumption, although the relationship between double index and myocardial oxygen consumption is so dependent upon loading conditions that this index has been largely discarded. We employed expediently the product of RVSP and HR to evaluate the myocardial oxygen consumption of the right ventricle. The significantly lower value of RVSP × HR during HFJV than that during IPPV suggests that HFJV may reduce

the oxygen consumption of ischemic right ventricle.

The canine heart is rich in collateral circulation so that it is rather difficult to make the myocardium ischemic. Therefore, we loaded the right ventricle with excessive pressure by banding the main pulmonary artery in addition to the RCA ligation to make sure that the right-sided myocardial ischemia was created. In consequence, myocardial cyanosis continued at least for 5 hr and the increase in CPK-MB was observed in all animals at 5 hr at the latest. However, some animals showed reductions in size and intensity of myocardial cyanosis, and the standard deviations in CI and SI showed a tendency to increase with time, suggesting that there may be a considerable difference in collateral circulation among the animals.

HFJV has been suggested to be hemodynamically superior to IPPV under various pathological conditions of right ventricular loading, i.e., right ventriculotomy[14], pulmonary embolism[15], and right ventricular dilatation with constricted pulmonary artery[16]. In our study during the ischemic periods, the right ventricle had a remarkably decreased SI, but HR reversely increased so that CI did not significantly fall despite the sustained ischemia and pressure overload. Moreover, RVEDP and CVP remained within the normal limits. These findings as well as the findings at autopsy indicate that the animals may not have had severe right heart failure even at the ischemic periods.

Long-term HFJV can cause microatelectasis resulting in the increase in $\dot{Q}sp/\dot{Q}t$ and the decrease in Pa_{O_2}[13,16,19,20], nevertheless HFJV often produces PEEP[30] as shown also in our study. There were no significant differences in Pa_{O_2} and $\dot{Q}sp/\dot{Q}t$ between IPPV and HFJV in our study, probably because of the relatively short duration of HFJV.

The decrease in Pa_{O_2} and the increases in PIP, $P\bar{a}\bar{w}$, $\dot{Q}sp/\dot{Q}t$, and PVRI with time are probably caused by the hemothorax, pneumothorax, and atelectasis observed at autopsies. However, the increases in PIP and $P\bar{a}\bar{w}$ with time were observed only during IPPV, suggesting that HFJV can be performed without increasing airway pressure even while IPPV needs more airway pressure to maintain the comparable levels of oxygenation and alveolar ventilation with HFJV.

There are several limitations in this study. We demonstrated a close correlation between $P\bar{a}\bar{w}$ and SI. However, we did not measure the pleural pressure, which might in reality affect the cardiac performance. In subjects with large airway resistance, large increase in $P\bar{a}\bar{w}$ may occur; however, this increase will not be transmitted to the pleural space and therefore will not affect cardiac performance as much as in subjects with normal airways. There is a possibility that the following factors might affect the results: 1) the pericardium and also the mediastinal pleura were left open to observe the status of myocardium as needed; this open chest-open pericardium model could affect the results of IPPV versus HFJV; 2) the quantitative assessment of RCA ligation and the anatomic measurement of the areas of myocardial injury/infarction were not evaluated; and 3) the level of anesthesia induced with thiopental and maintained with constant infusion of fentanyl was not determined during the study. In our model, the right ventricular impedance would be due to both pulmonary vascular impedance and the pulmonary artery banding. The measurement of pulmonary arterial pressure proximal to the band, which allows assessment of the relative resistance imposed by the band to the resistance by the pulmonary vasculature, was not performed. The gradient between RVSP and systolic pulmonary arterial pressure (SPAP) was approximately double that between SPAP and PAWP. If the pulmonary artery banding represented the majority of right ventricular impedance, it might be clear that attributing hemodynamic changes between IPPV and HFJV to 1 mmHg changes in $P\bar{a}\bar{w}$ or to 20–50 $dyne \cdot sec \cdot m^2 \cdot cm^{-5}$ changes in PVRI is tenuous at best. Finally, we used an I:E ratio of 1:2 during IPPV, which is used most frequently in mechanical ventilation. If we had employed an I:E ratio of 1:3 and the same ventilatory rate during IPPV, we might

probably have achieved the identical Pa_{CO_2} and $P\bar{a}w$, and also hemodynamics to those during HFJV.

Our study seems to show the potential importance, and future studies in this area should be performed, with respect to the cardio-respiratory care of patients with right heart problems, e.g., congenital heart diseases, right heart or biventricular dysfunction immediately following myocardial revascularization, and right ventricular dysfunction with respiratory failure in intensive care unit setting.

In conclusion, HFJV synchronized with diastole offers hemodynamic advantages (increases in stroke volume and left ventricular stroke work associated with decreases in afterload and myocardial oxygen consumption) over IPPV to the ischemic right ventricle with constricted pulmonary artery.

(Received Feb. 2, 1990, accepted for publication Mar. 9, 1990)

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