High-Frequency Jet Ventilation Improves Cardiac Function After the Fontan Procedure

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Pulmonary vascular resistance is an important determinant of cardiac output after the Fontan procedure and is adversely affected by elevated mean airway pressure. High-frequency jet ventilation (HFJV) is an alternate form of mechanical ventilation which supports gas exchange at lower mean airway pressure. This study was performed to determine if HFJV could lower mean airway pressure and pulmonary vascular resistance and result in an increase in cardiac output after the Fontan procedure. We prospectively evaluated 13 patients ranging in age from 0.9 to 8.5 years (mean, 3.9 years) and in weight from 6.2 to 20.1 kg (mean, 13.9 kg). Right atrial, left atrial and pulmonary artery catheters were used to measure hemodynamic parameters. Cardiac index was measured by dye dilution technique, and pulmonary vascular resistance was calculated. The patients were stabilized on mechanical ventilation to achieve a $Paco_2$=30±5 mm Hg, and baseline hemodynamic and respiratory measurements were made. HFJV was begun at settings adjusted to achieve similar gas exchange. Respiratory and hemodynamic measurements were repeated after 30–60 minutes of HFJV. Mechanical ventilation was then resumed at baseline settings, and measurements were repeated 0.5–1 hour later. There was no significant change in gas exchange. HFJV resulted in a 50% reduction in mean airway pressure (9.2±0.2 cm H_2O to 4.6±0.1 cm H_2O, $p<0.001$), a 59% reduction in pulmonary vascular resistance (3.82±0.36 to 1.52±0.16 Woods units, $p<0.001$), and a 25% increase in cardiac index (2.32±0.12 l/min/m^2 to 2.91±0.12 l/min/m^2, $p<0.001$). These data demonstrate that HFJV favorably affects ventilatory and hemodynamic parameters and may be a preferable means of ventilation in patients after the Fontan procedure. (*Circulation* 1991;84[suppl III]:III-364–III-368)

The surgical treatment of patients with a univentricular heart has undergone a significant evolution. In 1949 Robdand and Wagner[1] published their report on the first experimental right ventricular bypass. It was not until 9 years later that Glenn[2] described the first superior vena cava to the distal right pulmonary artery shunt. With this operation, Glenn showed that systemic venous blood could be diverted directly into the pulmonary arteries. In 1971 Fontan and Baudet[3] described a procedure in which the total systemic venous return was diverted to the pulmonary arteries in a patient with tricuspid atresia. Since that description there have been multiple modifications in the original technique. These modifications along with advances in anesthesia and postoperative care have allowed successful application of the Fontan procedure to a number of complex lesions.[4]

Intrathoracic pressure can directly alter pulmonary blood flow by altering pulmonary vascular resistance.[5] Furthermore, cardiac output after the Fontan procedure may be affected by even minor changes in pulmonary vascular resistance due to the absence of a pulmonary ventricle. High-frequency jet ventilation (HFJV) can support gas exchange at a low mean airway pressure.[6,7] We hypothesized that by decreasing intrathoracic pressure, HFJV might enhance pulmonary blood flow and thereby improve cardiovascular hemodynamics after the Fontan procedure. This study was undertaken to compare the effects of HFJV and conventional mechanical ventilation on the hemodynamics in the immediate postoperative period.

Methods

Patient Selection

Informed consent was obtained in all patients, and the study protocol was approved by the institutional

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review board at the University of Michigan Medical Center. Eligible patients consisted of those undergoing the Fontan procedure who weighed less than 25 kg. This weight limit was established after preliminary animal trials in which we demonstrated that adequate ventilation was possible using the Bunnell life-pulse ventilator in animals who weighed up to 25 kg. Residual left to right shunting and anastomotic gradients were excluded by intraoperative pressure measurements and mixed venous and pulmonary artery saturations. Each patient served as his/her own control.

Fifteen patients were entered into the study. Two patients were removed because of frequent manipulations in inotropes in one and unreliable cardiac output measurements in the other. The remaining 13 patients constitute the study group. These patients ranged in age from 0.9 to 8.5 years (mean, 3.9 years) and weight from 6.2 to 20.1 kg (mean, 13.9 kg). They had the following preoperative diagnoses: double inlet left ventricle in four, tricuspid atresia in three, unbalanced atrioventricular septal defect with left ventricular hypoplasia in three, hypoplastic left heart syndrome in two, and pulmonary atresia with intact ventricular septum in one.

Procedure

Radial, femoroarterial, and internal jugular catheters were placed in all patients. All patients had an intra-atrial baffle which directed inferior vena caval blood to the superior vena cava. Superior vena cava was then transected and both ends sutured to the pulmonary artery to complete the total cavopulmonary connection. The technical details of the Fontan procedure were altered slightly in some patients to suit the individual anatomy.

At the conclusion of the operation right atrial, left atrial, and pulmonary arterial catheters were placed. A triple lumen Mallinkrodt endotracheal tube custom made for HFJV was inserted. The patients were then transferred to the intensive care unit and standard ventilation was performed with the Servo 900 C ventilator adjusted to provide normalization of arterial blood gases.

Measurements

After admission to the intensive care unit all patients were allowed to reach a steady state during which no manipulations in inotropic or chronotropic therapy were performed. Ventilation was adjusted to achieve moderate alkalosis ($Paco_2$=30±5 mm Hg) and normoxia (Pao_2=90±10 mm Hg). All patients were atrioventricular sequentially paced to maintain a constant rhythm. Following stabilization baseline hemodynamic and respiratory parameters were measured. Hemodynamic measurements included heart rate and central venous, aortic, left atrial, and pulmonary artery pressures. When no gradient existed between the pulmonary artery and right atria, right atrial pressures were substituted for pulmonary artery pressures. Respiratory parameters included arterial oxygen saturation, arterial blood gases, mean airway pressure (measured via the triple lumen tube in the trachea), peak airway pressure, tidal volume, end-expiratory pressure, respiratory rate, inspiratory and expiratory time, and flow rate. All patients were paralyzed and ventilated with 100% inspired oxygen during the study.

Cardiac output was measured by the dye dilution technique in triplicate using methods previously described.[8] Pulmonary vascular resistance was calculated by subtracting right atrial pressure from left atrial pressure and dividing by the cardiac index.

The patients were then placed on a Bunnell life-pulse high-frequency jet ventilator. The rates of HFJV varied between 250 and 400 beats/min. Ventilation was adjusted to achieve a $Paco_2$ of ±5 mm Hg of baseline values. Inspiratory time was held constant at 0.20 msec and peak inspiratory pressure was adjusted to achieve equivalent alveolar ventilation as observed on conventional volume ventilation. Hemodynamic and respiratory parameters were repeated after 30 minutes of HFJV. The patients were then returned to conventional ventilation, and measurements were repeated 30 minutes later.

Analysis

Data are presented as mean±SEM. Hemodynamic and respiratory variables were compared using analysis of variance.

Results

The mean time to reach steady state was 3.3 hours (2.5–5 hours). The hemodynamic and respiratory data are summarized in Table 1. There was no statistically significant difference between the $Paco_2$ prior to HFJV ($Paco_2$=27±1 mm Hg) compared with that obtained during HFJV ($Paco_2$=26±1 mm Hg) or after return to conventional ventilation ($Paco_2$=27±1 mm Hg). However, at equivalent alveolar ventilation, HFJV resulted in a 50% reduction in mean airway pressure. The mean airway pressure during conventional mechanical ventilation was 9.2±0.2 cm H_2O and fell to 4.6±0.1 cm H_2O during HFJV. Following return to conventional mechanical ventilation at comparable $Paco_2$, the mean airway pressure returned to baseline values (9.2±0.2 cm H_2O). (Figure 1).

Before HFJV, right atrial pressure was 14±1 mm Hg and ranged from 11 mm Hg to 19 mm Hg. This was reduced by 21% during HFJV to a mean of 11±1 mm Hg, $p<0.001$. After return to conventional ventilation, right atrial pressure again returned toward baseline values (13±1 mm Hg). Left atrial pressure increased from 5±1 mm Hg on mechanical ventilation to 7±1 mm Hg during HFJV, $p<0.001$. Again, after return to conventional ventilation there was a return of the left atrial pressure to pre-HFJV levels. Consequently, there was a reduction of the calculated pulmonary arterial vascular resistance to 1.56±0.16 Woods units from 3.82±0.36 Woods units during conventional

TABLE 1.

	$PaCO_2$ (mm Hg)			Mean airway pressure (cm H_2O)			Right atrial pressure (mm Hg)			Left atrial pressure (mm Hg)			Cardiac index (l/min/m²)			PVR (Woods units)		
Patient	Pre	HFJV	Post	Pre	HFJV	Post	Pre	HFJV	Post	Pre	HFJV	Post	Pre	HFJV	Post	Pre	HFJV	Post
1	26	22	24	9.2	4.2	8.9	14	12	15	5	6	5	2.45	3.00	2.40	3.67	2.00	3.75
2	28	23	25	8.7	4.3	9.1	12	12	11	6	8	6	2.17	2.92	2.30	2.77	1.37	2.17
3	28	27	24	9.3	4.7	9.5	19	16	18	9	11	9	2.13	2.80	2.16	4.70	1.79	4.17
4	27	25	25	9.1	4.3	9.3	11	9	12	4	5	3	2.90	3.60	3.80	2.41	1.11	2.37
5	24	26	26	8.6	4.1	8.3	13	9	13	4	6	4	2.20	2.97	2.28	4.09	1.01	3.95
6	26	24	26	8.9	4.4	8.7	12	10	12	6	6	5	2.71	3.25	2.73	2.21	1.37	2.56
7	26	26	28	10.3	5.1	10.3	13	9	11	5	6	4	2.06	2.68	2.10	3.88	1.12	3.33
8	30	26	28	10.7	5.3	10.0	15	12	13	6	8	6	2.13	2.87	2.09	4.23	1.39	3.35
9	26	27	26	10.0	4.9	9.7	12	9	13	5	6	6	2.27	2.91	2.10	3.08	1.03	4.29
10	25	27	27	9.1	4.8	9.0	16	12	13	7	8	5	1.85	2.35	1.50	4.87	1.70	5.33
11	27	32	33	8.1	4.3	9.6	13	12	14	5	5	4	2.21	2.23	2.15	3.62	3.14	4.65
12	28	27	29	8.9	4.3	9.0	16	11	16	3	6	4	1.83	2.53	1.90	7.10	1.98	5.26
13	26	28	30	9.0	4.6	8.7	14	12	14	4	7	4	3.30	3.67	3.20	3.03	1.26	3.13
Mean	27	26	27	9.2	4.6	9.2	14	11	13	5	7	5	2.32	2.91	2.36	3.82	1.56	3.72
±SEM	1	1	1	0.2	0.1	0.2	1	1	1	1	1	1	0.12	0.12	0.16	0.36	0.16	0.28
p				-														
vs. Pre		<0.001	0.71		<0.001	0.73		<0.001	0.316		<0.001	0.29		<0.001	0.64		<0.001	0.65
vs. HFJV			<0.001			<0.001			<0.001			<0.001			<0.001			<0.001

PVR, during high-frequency jet ventilation; Pre, during conventional mechanical ventilation before high-frequency jet ventilation; HFJV, during conventional mechanical ventilation after high-frequency jet ventilation; Post, pulmonary vascular resistance.

mechanical ventilation ($p<0.001$, Figure 2). After return to conventional mechanical ventilation, there was an increase in pulmonary vascular resistance to 3.72±0.28 Woods units. Cardiac index rose from 2.32±0.12 l/min/m² during conventional mechanical ventilation to 2.91±0.12 l/min/m² during HFJV ($p<0.001$, Figure 3) and returned to baseline levels after conventional mechanical ventilation was resumed (2.36±0.16 l/min/m²). There was no significant change in oxygen saturation during the study, with saturation during both periods of mechanical ventilation measuring 100±1% versus 99±1% during HFJV. There was no relation between increase in cardiac index and age or diagnosis.

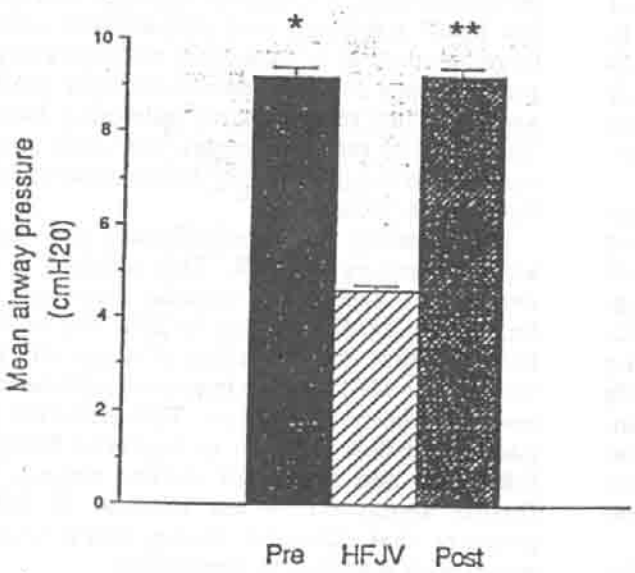

FIGURE 1. *Mean airway pressure prior to high-frequency jet ventilation (Pre), during high-frequency jet ventilation (HFJV) and after high-frequency jet ventilation (Post). Values presented are mean±SEM.* *p<0.001 *vs. HFJV;* **p<0.001 *vs. HFJV.*

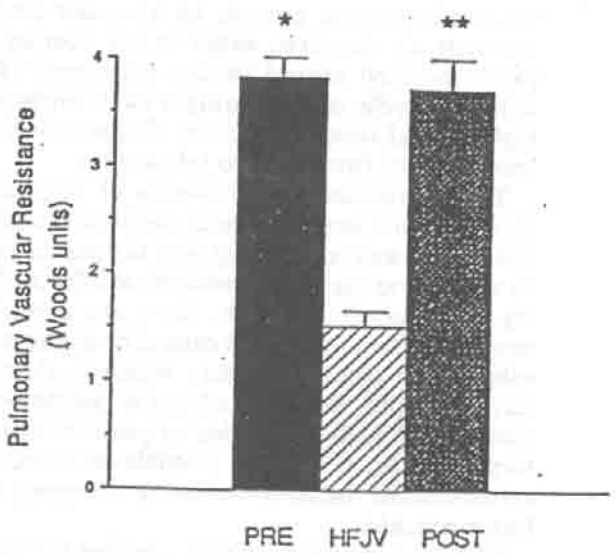

FIGURE 2. *Pulmonary vascular resistance before high-frequency jet ventilation (PRE), during high-frequency jet ventilation (HFJV) and after high-frequency jet ventilation (POST). Values presented are mean±SEM.* *p<0.001 *vs. HFJV;* **p<0.001 *vs. HFJV.*

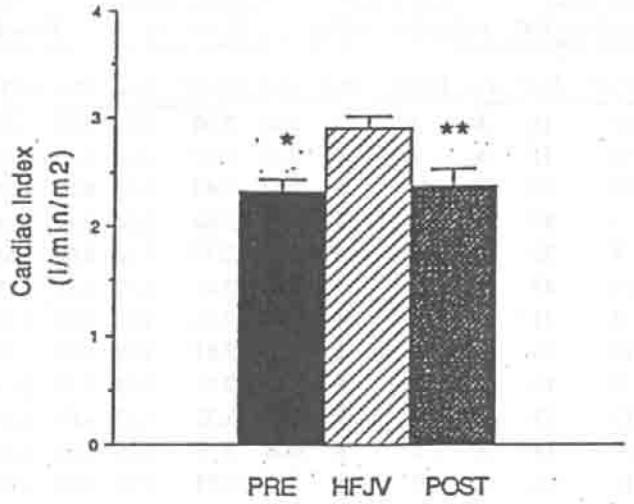

FIGURE 3. *Cardiac index before high-frequency jet ventilation (PRE), during high-frequency jet ventilation (HFJV) and after high-frequency jet ventilation (POST). Values presented are mean±SEM.* *$p<0.001$ *vs. HFJV;* **$p<0.001$ *vs. HFJV.*

Discussion

Despite recent advances in the surgical treatment of patients with a univentricular heart, there remains a significant morbidity and mortality associated with the Fontan procedure.[4] Cardiac output is critically dependent on the pulmonary vascular resistance following this procedure, and even minor elevations in resistance may lead to the onset of significant low cardiac output. Furthermore, pulmonary vascular resistance is usually the most labile immediately following the operation secondary to fluid overload, atelectasis, and myocardial dysfunction. Augmenting the cardiac output requires significant amounts of volume loading to provide an adequate preload to the systemic ventricle, often at the cost of significantly elevated central venous pressures. This may initiate a cycle of increasing airway pressures and higher blood volumes in order to maintain the pressure gradient from right to left atrium.

The interactions and influence of respiration on cardiovascular hemodynamics has been the subject of much study and controversy over the past century.[9–11] In addition to the direct mechanical effects of changing intrathoracic pressure, there are several autonomic and humoral effects caused by changes in lung volume and pleural pressure which further modify cardiac output. With the advent of mechanical ventilation and such modalities as positive end-expiratory pressure, it became possible to directly alter cardiovascular hemodynamics by changing respiratory mechanics.

HFJV is a new technology available for managing patients with respiratory insufficiency.[12,13] The effects of HFJV on cardiovascular hemodynamics are controversial and are the subject of current research.[14] HFJV is a proven, safe mode of ventilation for newborn infants with barotrauma. A major advantage of HFJV when compared with conventional mechanical ventilation is its ability to provide equivalent gas exchange at reduced mean airway pressures.[6] Since there is a close relationship between respiratory function and cardiac performance in infants and children, attempts have been made to use HFJV to assist postoperative pediatric cardiac surgical patients.[15,16] Previous studies performed in children following repair of ventricular septal defect, transposition of the great arteries, and tetralogy of Fallot have produced conflicting results. Weiner and colleagues,[17] using HFJV in four patients, demonstrated that adequate ventilation could be maintained at lower peak and mean airway pressures and that cardiac index improved. In contrast, Vincent and associates[15] studied 20 patients after surgery and observed no change in cardiac index. Modest increases in pulmonary vascular resistance and pulmonary artery pressure were seen in their study.

Previous reports have investigated the effects of HFJV in patients with biventricular hearts. Our study is unique in examining HFJV in patients without a ventricular dependent pulmonary circulation, in which the effects of ventilation and pulmonary vascular resistance would be even more pronounced. We hypothesized that in patients following the Fontan procedure, lowering the mean airway pressure would result in improved cardiac output due to enhanced pulmonary blood flow. This theory was previously suggested by studies of experimental right ventricular dysfunction in dogs.[18]

Elevations in ventilatory volumes and pulmonary vascular resistance lead to reduced pulmonary blood flow. Left ventricular preload is dependent on the volume of blood that is provided to it by the right side of the heart.[19] Therefore, left ventricular output will be limited when the ability of the right heart to fill the left heart is reduced with conventional ventilation; large fluctuations in inspiratory and expiratory airway pressure may result in decreased right heart filling and emptying, thus reducing pulmonary blood flow. These effects may be exaggerated when there is no ventricle to compensate, as seen in patients following the Fontan procedure.

In our study, HFJV significantly reduced mean airway pressure by 50%. This resulted in a 59% decrease in pulmonary vascular resistance and an increase in cardiac output by 25%. We believe this indicates that the reduction of mean airway pressure during HFJV led to improved right heart filling and pulmonary blood flow. The increased pulmonary blood flow resulted in improved filling of the left heart and increased cardiac output. This is further supported by the increase in left atrial pressure that occurred during HFJV when compared with mechanical ventilation.

Future Investigation

The role of HFJV in the management of patients following cardiac surgery needs to be further investigated. Our study demonstrated that cardiac function can be transiently improved using HFJV after

the Fontan procedure. Further studies examining the extended use of HFJV in patients following the Fontan procedure need to be performed to determine if morbidity and mortality are improved. In addition, evaluation of HFJV in patients with isolated right ventricular failure, isolated left ventricular failure, or biventricular failure is currently underway. This information may be helpful to define the specific type of ventilation for specific cardiovascular pathophysiology.

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