Managing hemodynamics during high frequency oscillatory ventilation

**Introduction**

There is an inextricable link between hemodynamic characteristics and response to ventilation settings. Therefore, a careful evaluation of cardiovascular function in critically ill patients with pulmonary disease is an important aspect of their ventilatory management. It is also then important to understand and utilize those tools and measurements that will enhance our understanding of these inter-relationships.

**Matching ventilation/perfusion (V/Q)**

The goal of breathing is to match alveolar ventilation with pulmonary perfusion to obtain an ideal V/Q balance as it is the major determinant of PaO$_2$ and PaCO$_2$. While our ventilation management determines the “V” component, there are three major determinants of pulmonary “Q”. They are myocardial function, the pulmonary blood volume, and pulmonary vascular resistance (PVR).

There are many factors which play a role in PVR. The lung is not a passive participant in PVR but plays an active role affecting PVR by lung volume (Figure 1). The lung parenchyma not only tethers the airways, but tethers the extra alveolar vessels. At low lung volume, there is a reduction in radial traction reducing the cross-sectional area of the extra alveolar vascular bed, increasing PVR in these vessels. As lung volume increases, PVR falls to its minimal level at an optimum lung volume. However, if mean airway pressure (Paw) continues to increase lung volume resulting in over distention of the alveoli, this results in compression of the intra-alveolar vessels which lack perivascular support resulting in a rise of PVR. Because of this direct action by the ventilator on a major component of hemodynamic performance, it is therefore important to track hemodynamic responses while changing Paw.

**Clinical cardiovascular monitoring heart rate**

Monitoring the heart rate is an essential element for evaluating the cardiovascular status. The pediatric heart rate is influenced by age and disease process.

![Figure 1.](image)

Neonates have the added influence of gestational age and weight. Additional influences on heart rate include central nervous system dysfunction, autonomic dysfunction, stress, drugs, sepsis, anemia, hyperthermia, and endocrine dysfunction (e.g., thyroid). Since cardiac
output equals stroke volume times heart rate, monitoring changes in heart rate can reflect an acute change in the patient’s condition or trend impending problems.

Patients in respiratory failure frequently have an increase in heart rate to improve cardiac output and to compensate for decreased oxygenation. Although, increasing heart rate is an effective mechanism for increasing the cardiac output, there is a point when increasing the heart rate will cause the cardiac output to decrease as the decreased time for ventricular filling and coronary blood flow effect cardiac efficiency.

Bradycardia can be associated with episodes of apnea and hypoxia. Other physiologic dysfunctions associated with a decrease in heart rate include cardiac ischemia, mechanical defects, hypovolemia, and increased vascular resistance. On rare occasions, it has been seen secondary to a reflex response to high PEEP or Paw.

**Blood pressure**

Arterial blood pressure is the dynamic measurement of the force of blood against the wall of the artery reflected during systole (tension during LV contraction), and diastole (relaxation pressure during ventricular diastole). Pulse pressure, the difference between the systolic and diastolic, reflects the ventricular ejection amplitude. Normal values vary in the pediatric patient by age, sex, and method of monitoring. Neonates have the additional effects of gestational age, birth weight, and postnatal age to consider.

Hypotension may result from true low blood volume, low effective blood volume secondary to peripheral vasodilatation, or left ventricular dysfunction. Hypotension induced reduction in oxygenation delivery may result in tissue hypoxia and elevation of lactic acid levels. Hypertension, or an elevated blood pressure, can be the result of increased peripheral vascular resistance or fluid overload. Pulse pressure can be increased with the left-to-right shunt from a patent ductus arteriosus (PDA) or decreased by shock, hypovolemia, or heart failure.

In the pediatric population, considerable information can be obtained from the arterial pressure tracing. The shape of the tracing, pulse pressure, and location of the dicrotic notch, all give pertinent hemodynamic information about the patient.

**Central venous pressure**

Central venous pressure (CVP) is the pressure obtained from a catheter inserted into the superior vena cava or the right atrium. It is crucial when using this pressure to be certain of the catheter’s placement (Figure 2). In the neonate, slight displacement of the catheter from the right atrium can result in fictitious measurements. These errors can result from placing the catheter across the foramen ovale into the left atrium, or by placing it too far into the right atrium with partial obstruction from the intra-atrial septum. If the catheter is not inserted far enough, the pressure may reflect hepatic pressures. Both an AP and lateral chest x-ray for placement is very helpful. In our nursery, we document UVC placement with ultrasonography.

About 70 percent of the time, the correct placement is approximately T9 on AP chest x-ray. With normal cardiac status, CVP reflects right atrium pressure, which correlates with right ventricular pressure. CVP is used to evaluate pre-load to the right ventricle. It allows us to evaluate hemodynamic states such as intravascular volume, intra-thoracic pressure, and response to volume replacement.

The normal range for CVP in the neonate and pediatric patient varies, ranging from 0 to 8 mmHg. However, in our experience, we cannot usually use high frequency oscillatory ventilation (HFOV) effectively if the CVPs in the 0 to 3 mmHg range, but may require CVPs of 5 to 8 mmHg to allow increasing Paw above the critical opening pressure of the lung.
CVPs decrease in hypovolemia or with any mechanism causing delayed venous return to the right heart. Increasing levels of Paw without blood volume expansion can also cause decreases in venous return. Increases in CVPs are seen in right heart failure, hypervolemia, or myocardial dysfunction.

While the validity of CVP in the critically ill or mechanically ventilated patient has been questioned, in our experience, if the CVP is placed in the appropriate position (entrance of right atrium), it is very valuable in evaluating right ventricular preload.

**Pulmonary artery occlusion or “wedge” pressure**

Pulmonary artery occlusion or “wedge” pressure is used for measuring the preload for the left ventricle. The Swan-Ganz catheter introduction by right heart catheterization into the pulmonary artery (PA) is the standard method in pediatrics and adults for evaluating pulmonary vascular hemodynamics. The indications for a PA catheter include discriminating cardiac and noncardiac causes of pulmonary infiltrates, titration of fluid therapy, evaluation of pharmacologic interventions in pulmonary hypertension, and determining the etiology of hypotension.

To obtain a pulmonary capillary wedge pressure (PCWP), the distal lumen of the catheter is used. Balloon inflation isolates the distal tip of the catheter from upstream pulmonary arterial pressure creating a static, nonflowing column of blood distal to the balloon. Without flow there is no pressure drop along the column of fluid. The pressure at the catheter tip measures pressure in the pulmonary vein. Because there is normally minimal resistance in the pulmonary veins, the pressure measured approximates the pressure in the left atrium.

**Oxygen delivery**

Oxygen tension (PaO₂) is the partial pressure of oxygen in the arterial blood. PaO₂ is affected by FiO₂, alveolar ventilation, diffusion defects, V/Q mismatch, and shunt (intrapulmonary or intracardiac). Oxygen is carried by the blood in two ways. Although we frequently focus on PaO₂, this dissolved oxygen (0.0031 mL/torr) is only a small fraction (< 5 percent) of O₂ carried in the blood. The remaining and most significant portion is carried bound with the hemoglobin (Hb) as determined by the O₂ saturation. Each gram of Hb can carry 1.34 mL of O₂ and therefore, the arterial oxygen content (CaO₂) or the total amount of oxygen carried is dramatically affected by changes in hemoglobin.

Normal CaO₂ is approximately 20 mL O₂/dl blood (vol%). To emphasize the difference between PaO₂ and CaO₂ (CaO₂ = O₂ carried by Hb + dissolved O₂), consider the aspects of varying Hb concentration on the following patients:

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>The CaO₂ for a child with a Hb of 15 gm, PaO₂ of 100 torr and O₂ Sat of 97%:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CaO₂ = O₂ carried by Hb + dissolved O₂</td>
</tr>
<tr>
<td></td>
<td>= (15 x 1.34 x 0.97) + (0.0031 x 100)</td>
</tr>
<tr>
<td></td>
<td>= 19.50 ml O₂/dl + 0.31 ml O₂/dl</td>
</tr>
<tr>
<td></td>
<td>= 19.81 ml O₂/dl</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patient 2</th>
<th>The CaO₂ for a child with a Hb of 8 gm, PaO₂ of 100 torr and O₂ Sat of 97%:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CaO₂ = O₂ carried by Hb + dissolved O₂</td>
</tr>
<tr>
<td></td>
<td>= (8 x 1.34 x 0.97) + (0.0031 x 100)</td>
</tr>
<tr>
<td></td>
<td>= 10.40 ml O₂/dl + 0.31 ml O₂/dl</td>
</tr>
<tr>
<td></td>
<td>= 10.71 ml O₂/dl</td>
</tr>
</tbody>
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These two examples demonstrate the dramatic fall in blood oxygen content that occurs with the fall in Hb. Although both patients have the same PaO₂ and O₂ Sat, the second patient must almost double cardiac output to maintain the same oxygen delivery as the first patient.

**Oxygen extraction**

The Arterial-Venous difference (A-VDO₂) is the amount of O₂ extracted by the tissues and is commonly used in the ICU as an indirect measurement of cardiac output. The A-VDO₂ is derived from oxygen content calculations from arterial and mixed venous blood drawn simultaneously. The normal CaO₂ is approximately 20 vol% while the CvO₂ is 15 vol%. Therefore only 5 mL O₂ in each 100 mL is extracted for utilization by the tissues. An increase in the A-VDO₂ indicates that either tissue extraction has increased or cardiac output has decreased. Conversely, a decrease in the A-VDO₂ usually indicates an increase in cardiac output. Ideally, a true venous sample should be drawn from the pulmonary artery. If drawn from the right atrium, there is an increased probability for poor venous mixing and loss of data reliability.
Cardiac output in pediatric patients

Methods used to directly measure cardiac output in pediatric patients include the Fick equation, dye indicator-dilution and most commonly by the use of thermal dilution. The thermal dilution technique utilizes injection of chilled 0.9 percent sodium chloride into the right atrium while the temperature of the blood is constantly measured in the pulmonary artery with a thermistor-tipped catheter. The warming of the saline reflects the blood flow heat transfer. This is an accurate method of determining cardiac output that allows repeated measurements, however, consideration must be given when administering volume to infants and small children relative to the potential for fluid overload. The newest thermal dilution technology replaces cold boluses as an indicator with pulses of energy from a thermal filament in the catheter. This eliminates the potential of fluid overload and can provide continuous measurements.

Cardiac performance can also be assessed clinically by observation of indirect signs, frequently presented as the signs of poor organ perfusion. Signs and symptoms of low cardiac output include cool and pale extremities (skin), oliguria (renal), and decreased level of consciousness (CNS). Variables that may be used indirectly to measure the adequacy of cardiac output include: systemic blood pressure, right atrial pressure (CVP), left atrial pressure (LAP), urine output, blood pH and lactate levels, and arterial venous oxygen difference (A-VDO2).

Because pediatric patients have greater variation in size, it is important to normalize cardiac output by the cardiac index (CI = cardiac output per square meter of body surface area). The CO in children is higher per kilogram of body weight than the adult, resulting in a higher CI. When comparing the child’s cardiac function to the adult, it is important to note that the range of increase in stroke volume is much smaller and therefore a child’s cardiac output is more directly proportional to heart rate. Tachycardia in the pediatric patient is the most efficient way to increase cardiac output.

Echocardiographic evaluation of cardiovascular function

The heart is a parallel pump that delivers blood into two circuits, the right heart to the lungs and the left heart to the systemic circulation. It is critical that these parallel pumps work in sync to meet the metabolic demands of the body for oxygen and nutrients and the removal of the byproducts of metabolism. In the neonate where signs and symptoms of respiratory distress, sepsis, and metabolic derangements may mimic those of congenital heart disease, it may be more difficult to arrive at a correct hemodynamic interpretation at the bedside without more advanced technology. In complex heart-lung interactions, the only true methods to evaluate myocardial function are with an echocardiogram and pulmonary artery catheter.

Left ventricular performance

The stroke volume component of CO is affected by cardiac preload, myocardial contractility and afterload. Stroke volume will rise when preload or myocardial contractility improves and fall when afterload increases. The measurement of left ventricular end-diastolic volume (LVED) reflects preload. This volume affects the stretch of the myocardial fiber length. Preload decreases with blood loss, inadequate fluid maintenance and during positive pressure ventilation. Fluid volume replacement will usually correct a decreased preload in a healthy myocardium. Fluid overload increases preload and can be treated with diuretics. Preload can be evaluated using CVP and circumferential fiber shortening (VFCc).

Afterload is the resistance to blood flow from the ventricle and is effected by aortic pressure, radius of the ventricle, peripheral vascular resistance and the viscosity of the blood. It increases with hypertension and an enlarged ventricle. Decreases in afterload are seen with anemia, vasodilation and central shunting of blood volume. Pharmacologic agents such as nitroprusside that alter vascular tone will also change right and left ventricular afterload. Left ventricular afterload can be evaluated measuring left ventricular posterior wall stress. Laplace’s Law states that wall stress of a chamber is equal to the pressure times the cavity diameter/wall thickness. Combining blood pressure with left ventricular wall thickness and cavity dimensions allows calculation of peak systolic wall stress (PSS).

The force of contraction of the heart, reflected in the efficiency of myocardial fiber shortening, is called contractility. Left Ventricular Shortening Fraction (LVSF) and Ventricular Ejection Fraction (VEF) are best estimated when the left ventricle is circular. In the premature infant, there is intraventricular septal flattening causing
distortion of the shape of the left ventricle and a decrease in circularity. This results in a shortening fraction in the neonate which can underestimate the differences in regional ventricular function. Decrease in contractility occurs with hypoxia, acidosis, with certain drugs and glucose abnormalities. Inotropic drugs will usually improve contractility and cardiac output once preload deficits have been corrected. Common inotropic agents utilized to improve contractility include Dopamine, Dobutamine, Epinephrine, Isoproterenol, Norepinephrine and Aminon. The ratio of PSS and VCFc allows better evaluation of myocardial function by reducing the effect of preload and postload. Other echocardiographic measures that may assist in evaluation of left ventricular function include Left Ventricular Cardiac Output calculated from the velocity time integral aortic valve area and heart rate, Left Ventricular Volume, and Left Ventricular Systolic Time Interval (LVSTI) as an index of LVS function.

Right ventricular performance

In the neonate, the right heart is more difficult to evaluate echocardiographically. The right ventricle lies directly beneath the sternum, has an irregular crescent shape, and trabeculated walls. Useful right ventricular performance measurements include right ventricular afterload (estimated by measuring right ventricular end diastolic diameter-RVEDD), Right Systolic Time Interval (RSTI), and Right Ventricular Output (calculated from the velocity time integral x pulmonary valve area x heart rate).

Pulmonary artery pressure

Echocardiography is also an ideal noninvasive method for estimating pulmonary artery pressure and evaluating the degree of pulmonary artery hypertension. Right ventricular systolic time intervals can reflect elevated pulmonary vascular resistance. Echocardiography can be used to measure the velocity and direction of flow across the PDA. Use of the tricuspid regurgant jet across the tricuspid valve and CVP in a simplified Bernoulli’s equation gives an estimate of the Systolic Pulmonary Artery Pressure (SPAP). This method has been shown to be more reliable than the other echocardiographic methods.

Ductus venous

The ductus venous can be assessed ultrasonographically for flow pattern. The flow pattern reflects the portocaval pressure gradient and the pressure on the right side of the heart and in the pulmonary arteries.

Shunts

Patterns of flow across a Patent Foramen Ovale (PFO) or ductus arteriosus can be recorded by color flow and pulsed-wave Doppler studies. The size of the defect, direction of shunt, and velocity can be estimated. Contrast echocardiography to evaluate PFO flow using an agitated saline flush can be performed if color flow is unsatisfactory.

Hemodynamics in high frequency oscillatory ventilation

Abnormal respiratory function treated with assisted mechanical ventilation can alter lung volume, pulmonary vascular resistance, or intrathoracic pressure. All of these can have significant effects on normal and abnormal circulatory function. During conventional mechanical ventilation (CMV) increasing the tidal volume can decrease venous return. Increasing PEEP is associated with decreased cardiac output, although cardiac function has generally been observed to be normal with PEEP.

The decreased cardiac output with PEEP appears to be due to decreased venous return. Administration of fluids can usually overcome the decreased venous return and restore cardiac output.

Figure 3. Effects of PEEP on the determinants of venous return

The decreased venous return with increasing PEEP can be explained in Figure 3. As lung volume increases with PEEP, intrathoracic pressure increases by an amount determined by chest wall compliance and the lung volume. This effect is transmitted to the right atrium, where it decreases venous return. As PEEP increases from zero to 20 (PEEP}
20 = cardiac function curve with PEEP 20), there is a progressive rightward shift in the cardiac function curves (ZEEP = cardiac function curve with zero end expiratory pressure; PEEP 10 and PEEP 20 = cardiac function curve with 10 and 20 cmH2O PEEP). The point of intersection between the cardiac function and venous return curves shifts from Point A to B to C. Point D is a theoretical point of intersection between the original venous return curve and the cardiac function curve on PEEP, which would have been the situation had mean circulatory pressure not increased with PEEP. Thus, increased mean circulatory pressure buffered the effects of increased PEEP on cardiac output and venous return. There is also a decrease in the downward slope of the venous return curve with PEEP which suggests an increase in the resistance to venous return as well. In addition, Fessler demonstrated a vascular waterfall in the Inferior Vena Cava (IVC) associated with mechanical compression of the lower lobes of the right lung on the IVC, causing a flow limitation. Vedeng found PEEP applied to the right lung alone limits cardiac output more than PEEP to the left lung alone. This effect may be explained on the basis of either direct mechanical compression by the inflating lung on the right ventricle and right atrium with greater increases in right atrial pressure and hence greater decreases in venous return or exceeding a critical closing pressure of the inferior vena cava.

There have been relatively few studies in the literature concerning cardiovascular effects of HFOV, especially in immature subjects.

The ventilatory strategy used in all pathophysiologies except gross air leak is an optimum lung volume strategy. Because the tidal volumes are very small in HFOV, the lung volumes remain near constant. Mean airway pressure is increased on the inflation limb of the pressure volume above the critical opening pressure of the lung. Lung volume is maintained above the critical closing pressure of the lung on the deflation limb of the hysteresis curve. This results in a higher PEEP than seen in CMV. However, because in CMV there is an expiration time with a lower pressure (usually a low PEEP), it is more forgiving in its effect on venous return for the same blood volume than the higher PEEP seen in HFOV. Although when HFOV Paw is similar to that of CMV, several authors found no significant differences in cardiac output, organ blood flow, or metabolic status.

Traverse, et. al., found impairment of hemodynamics on HFOV in the cat model. They found that cardiac output decreased and pulmonary vascular resistance increased with increasing Paw. Although the response was diminished in animals with decreased respiratory system compliance, it was not eliminated.

Kinsella, et. al., investigated the hemodynamic consequences of ventilator strategies for HFOV and CMV in the baboon model of HMD using the radio labeled microsphere technique. Measuring cardiac output and organ blood flow at three time points (3, 8, and 23 hours) showed no significant differences in left ventricular output, effective systemic flow, organ blood flow, or central venous pressure. The HFOV strategy resulted in significant improvement in oxygenation during the 24 hours of treatment without adverse effect on left ventricular output, cerebral blood flow, or central venous pressure.

Nicol, et. al., studied changes in aortic blood flow and blood pressure in normal and surfactant deficient rabbits under varying ventilator strategies of Conventional Tidal Volume Ventilation (CMV), Inverse Ratio Ventilation (IRV), High Frequency Positive Pressure Ventilation (HFV) and High Frequency Oscillatory Ventilation (HFOV). Each strategy was also studied at PEEP (Paw) levels of 0, 5 and 10 cmH2O. In the injured lungs, when 10 cmH2O of distending pressure was applied, PaO2 increased with all modes, but the aortic blood flow was significantly reduced (p < 0.05) with a 40 percent reduction in calculated oxygen delivery during CMV and IRV as compared to HFV or HFOV. They concluded that hemodynamic stability is better maintained during HFV or HFOV when high PEEP levels are required for oxygenation.

Minton, et. al., compared the hemodynamics in 100 newborn infants with birth weights less than 2,500 grams with RDS randomized to either CMV and surfactant or HFOV and surfactant. The strategy used on HFOV was an optimum lung volume strategy. They found no difference in cardiac output or right and left ventricular hemodynamics if close attention was paid to blood volume expansion.

We have subsequently reported on 299 patients on HFOV who underwent 1,420 echocardiograms in the first 21 days of life. In these patients (GA 22.5 to 42 weeks and BW 310 to 5,200 grams) we found a slight
increase in cardiac output with advancing gestation age and birth weight increasing from 280 cc at 23 weeks to 300 cc at 41 weeks. Mean cardiac output was 314 cc/kg/mm with a standard deviation of 28 cc/kg/mm. There did not appear to be any effects on left or right hemodynamics, cardiac output, or incidence of patent ductus arteriosus or foramen ovale shunting in these patients. The cardiovascular effects of a patent ductus arteriosus (in a premature baboon model of HMD treated with CV or HFOV) were studied by Yoder, et. al. There were no significant differences between the two forms of ventilation when MAP, HR, CO, or modes of ventilation were compared.

Gutierrez, et. al., evaluated eight of their pediatric patients who had been managed with HFOV for severe respiratory failure and had pulmonary artery catheters in place. With significant 50 percent increases in mean airway pressures from CMV to HFOV (20.9 cmH₂O to 30 cmH₂O), the only observed effect was a mean reduction in heart rate of 20 beats/min. Oxygen delivery, cardiac index, and mean systemic arterial blood pressure did not change significantly following the change to HFOV with the exception of one patient.

**Blood volume expansion and inotropic support**

Venous return is influenced by the blood volume and a reduction will cause a decrease in cardiac output and hypotension. It is important to correct hypovolemia by fluid replacement. Decreases in cardiac output may also occur in patients with normal volume status and inotropic drugs may be used to improve their myocardial function.

Measurement of the left atrial pressure (LAP) provides the most accurate guide to fluid replacement and is the earliest indicator of hypovolemia. CVP reflects the right ventricular end diastolic pressure. Although CVP is often a poor correlate of left-sided filling pressure, its measurement is important in the assessment of right-sided pressure of the patients with right-sided obstructive lesions.

Preload is best assessed by the measurement of LAP. If filling pressures are low, volume should be administered using normal saline, lactated Ringer’s, Plasmatate, plasma, 5 percent albumin, or blood transfusion. In the neonate, usual fluid replacement is administered with 5 to 10 percent glucose at a rate of 80 to 100 mL/kg/day. When cardiac output is compromised, a fluid challenge may be required until stabilization occurs.

Once the preload is optimized, cardiac output and systemic blood pressure should be measured again. If the output is still low, but the BP is now elevated, a peripheral vasodilator should be used to decrease ventricular afterload (nitroprusside + nitroglycerin). Preload should be carefully monitored during the infusion of vasodilators as sudden peripheral vasodilatation may increase the need for volume administration. If the cardiac output and blood pressure remain low after preload optimization, then contractility should be increased by the use of inotropic agents. Several drugs that are used have both inotropic and vasoactive properties.

Right arterial pressure (RAP) of less than 6 mmHg may be challenged with fluid bolus of 10 mL/kg. Pressures between 6 to 10 mmHg may require a fluid bolus of 5 mL/kg and pressures of greater than 10 mmHg may respond to a bolus of 3 mL/kg. All volume is infused over 30 to 60 minutes. If the RAP continues to be within 2 mmHg of the original measurement repeat the fluid bolus. The bolus can be repeated times three until cardiac output improves which will be reflected by an increase in RAP greater than 2 mmHg than the original pressure. If there is no improvement, an echocardiogram should be performed for spatial anatomy, myocardial function, and chamber dimensions.

**Inotropic support**

**Dopamine**

1. Low Dose: 2 to 5 μg/kg/min IV. Low dose Dopamine increases renal perfusion and has a minimal effect on heart rate and cardiac output.

2. Intermediate Dose: 5 to 15 μg/kg/min IV. Effects include increases in renal perfusion, heart rate, cardiac contractility and cardiac output.

3. High Dose: > 20 μg/kg/min IV. High doses increase systemic vascular resistance and support arterial BP. High doses of Dopamine decrease renal perfusion.

4. Maximum Dose: 20 to 50 μg/kg/min IV.

**Dobutamine**

1. Continuous IV infusion: 2.5 to 15 μg/kg/min. Dobutamine is an effective beta adrenergic and increases arterial BP and myocardial contractility. Tachycardia and hypertension may occur.

2. Maximum Dose: 40 μg/kg/min.
Intractable Shock

Patients on HFOV that experience intractable shock in the face of maximum inotropic support and adequate fluid administration should be considered for alternative treatment. These alternatives include transitioning the patient back to conventional mechanical ventilation or transfer to an ECMO facility.

In our experience, careful selection of mean airway pressures to optimize lung inflation, adequate fluid administration with appropriate inotropic agent and use of all the monitoring techniques described in this article, has limited cases of intractable shock to a handful in more than 600 infants we have treated with the SensorMedics 3100A.

References


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