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# Case Reports of Inversed Ratio Ventilation

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**Abstract.** The prolongation of inspiratory time from an I : E ratio of 1 : 2 to a ratio of 4 : 1 significantly improved arterial oxygenation in 7 patients with acute respiratory failure due to pulmonary disease or cardiogenic lung edema when positive end-expiratory pressure had failed. The reduction in the respiratory index (y) ($y=-2.02x+9.392$, $r=-0.5732$, $p<0.05$) and the pulmonary shunt ratio (y) ($y=-5.502x+41.329$, $r=-0.5561$, $p<0.05$) were in proportion to the prolongation of inspiratory time (x). A reduction in cardiac output and a concomitant reduction oxygen delivery were observed with the I : E ratio of 4 : 1 but not with an I : E ratio of 3 : 2 or 2.6 : 1 in patients with pulmonary disease. However, two of three patients with cardiac diserse had a reduction in cardiac output and oxygen delivery with an I : E ratio of 1.2 : 1. The results suggest that the improvement of arterial oxygenation proportional to the prolongation of inspiratory time is due to the increase in the end-expiratory lung volume with a short exhalation time. Although this ventilatory pattern may be indicated when the effect of positive end-expiratory pressure on artrerial oxygenation has failed, an unduly long prolongation of inspiratory time is not suitable for patients with cardiac disease.

*Key Words* : Respiration ; IRV, I/E ratio, Mechanical ventilation, Respiratory failure
Oxygenation ; oxygen transport

## Introduction

Positive end-expiratory pressure (PEEP) has been the most frequently used method of mechanical ventilation for improving oxygenation in patients with acute respiratory failure (ARF) [1]. However, it has been observed that a similar or better improvement occurs when the inspiratory : expiratory ratio is increased (inversed ratio ventilation, IRV)[2-4]. It has been suggested that the use of IRV may be preferable to the use of high PEEP because of a more efficient elimination of $CO_2$ and a lower peak pressure, diminishing barotuauma[5]. Therefore, we have used IRV in seven pationts because they could not maintaine an adequate level of arterial oxygen tension ($PaO_2$) on an increased oxygen fraction ($F_IO_2$) of 1.0 with a range of PEEP of 6 to 10 cm $H_2O$. Author compared the effect of the IRV (with I : E ratios of 1.1 : 1, 1.2 : 1, 3 : 2, 2.6 : 1, and 4 : 1) on $Pao_2$ and oxygen delivery ($\dot{D}O_2$) with PEEP at an I : E ratio of 1 : 2 in patients with acute respiratory failure due to pulmonary disease or cardiogenic lung edema.

Table 1. Clinical characteristics of four patients with acute respiratory failure (group 1).

| No | Age (years) | Diagnosis | Mechanical ventilaiton (days) | Outcome |
|---|---|---|---|---|
| 1 | 39 | Systemic lupus erythematosus Interstitial pneumonia | 11 (8th)* | Died |
| 2 | 52 | Post-coronary artery bypass grafting | 8 (2nd) | Died |
| 3 | 54 | Aspiration pneumonia | 71 (3rd) | Survived |
| 4 | 50 | Panperitonitis due to perforation of small intestine Sepsis | 7 (3rd) | Died |

*indicates the day of initiation of IRV.

Table 2. Settings of ventilator and changes in blood gases with different I : E ratio (group 1).

| Patient No | 1 | | 2 | | | 3 | | 4 | |
|---|---|---|---|---|---|---|---|---|---|
| I : E ratio | 1 : 2 | 3 : 2 | 1 : 2 | 3 : 2 | 2.6 : 1 | 1 : 2 | 4 : 1 | 1 : 2 | 4 : 1 |
| Tidal volume (ml) | 467 | 467 | 611 | 833 | 833 | 460 | 460 | 600 | 600 |
| PEEP ($cmH_2O$) | 8 | 10 | 6 | 8 | 8 | 8 | 8 | 10 | 10 |
| PIP ($cmH_2O$) | 26 | 30 | 25 | 42 | 42 | 27 | 27 | 34 | 41 |
| RF (bpm) | 15 | 15 | 18 | 15 | 15 | 25 | 25 | 15 | 15 |
| $F_IO_2$ | 1.0 | 0.75 | 1.0 | 1.0 | 1.0 | 0.9 | 0.9 | 1.0 | 0.6 |
| pH | 7.59 | 7.56 | 7.53 | 7.37 | 7.38 | 7.40 | 7.41 | 7.33 | 7.25 |
| $PaO_2$ (mmHg) | 62 | 88 | 47 | 64 | 93 | 67 | 138 | 81 | 199 |
| $PaCO_2$ (mmHg) | 34 | 35 | 38 | 60 | 57 | 78 | 75 | 44 | 36 |
| $P\bar{v}O_2$ (mmHg) | 34 | 34 | 32 | 33 | 33 | 37 | 37 | 36 | 32 |

RF=respiratory frequency, bpm=breath per minute, other abbreviation refer to text.

Table 3. Changes in pulmonaty mechanics and hemodynamics with different I : E ratio (group 1)

| Patient No | 1 | | 2 | | | 3 | | 4 | |
|---|---|---|---|---|---|---|---|---|---|
| I : E ratio | 1 : 2 | 3 : 2 | 1 : 2 | 3 : 2 | 2.6 : 1 | 1 : 2 | 4 : 1 | 1 : 2 | 4 : 1 |
| $CaO_2$ (ml/dl) | 12.0 | 12.0 | 14.0 | 10.7 | 11.1 | 10.1 | 10.8 | 9.8 | 10.3 |
| $C\bar{v}O_2$ (ml/dl) | 6.1 | 7.5 | 9.8 | 6.6 | 6.6 | 6.7 | 6.8 | 6.4 | 4.5 |
| Hb (g/dl) | 9.9 | 9.6 | 12.5 | 8.6 | 8.3 | 8.2 | 8.1 | 8.8 | 7.9 |
| A-$aDO_2$ (mmHg) | 679 | 410 | 628 | 586 | 563 | 495 | 429 | 588 | 190 |
| RI | 10.0 | 4.9 | 13.4 | 9.2 | 6.1 | 7.4 | 3.2 | 7.3 | 1.0 |
| $\dot{Q}s/\dot{Q}t$ (%) | 34 | 30 | 52 | 39 | 28 | 41 | 28 | 53 | 18 |
| CO (l/min) | 2.5 | 4.2 | 4.4 | 6.7 | 6.7 | 7.0 | 5.7 | 3.9 | 2.7 |
| CI ($l/min/m^2$) | 1.8 | 3.0 | 2.3 | 3.6 | 3.6 | 4.4 | 3.6 | 2.9 | 2.0 |
| HR (BPM) | 105 | 142 | 118 | 118 | 118 | 102 | 103 | 124 | 113 |
| MAP (mmHg) | 86 | 129 | 90 | 60 | 60 | 89 | 84 | 60 | 47 |
| MPAP (mmHg) | 23 | 27 | 22 | 19 | 22 | 27 | 36 | 14 | 16 |
| PCWP (mmHg) | 7 | 8 | 14 | 11 | 11 | 9 | 15 | 7 | 9 |
| PVR ($dynes/sec/cm^5$) | 508 | 366 | 145 | 96 | 132 | 206 | 293 | 144 | 204 |
| $\dot{D}O_2$ (ml/min) | 216 | 366 | 322 | 385 | 400 | 444 | 389 | 284 | 206 |
| $\dot{V}O_2$ (ml/min) | 106 | 138 | 97 | 148 | 162 | 150 | 144 | 99 | 116 |

CI=cardiac index, BPM=beat per min, MPAP=mean pulmonary pressure, HR=heart rate, other abbreviation refer to text.

Table 4. Clinical characteristics of three patients with cardiogenic lung edema (group 2)

| No | Age (years) | Diagnosis | Mechanical ventilation (days) | Outcome |
|---|---|---|---|---|
| 1 | 73 | Aortic stenosis | 1 (1st)* | Died |
| 2 | 80 | Acute myocardial infarction | 25 (1st) | Died |
| 3 | 62 | Hypertensive cardiac failure | 6 (2nd) | Survived |

*indicates the day of initiation of IRV.

Tabel 5. Settings of ventilator and changes in blood gases with different I : E ratio (group 2)

| Patient No | 1 | | 2 | | 3 | |
|---|---|---|---|---|---|---|
| I : E ratio | 1 : 2 | 1.2 : 1 | 1 : 2 | 1.2 : 1 | 1 : 2 | 1.2 : 1 |
| Tidal volume (ml) | 533 | 500 | 500 | 563 | 533 | 500 |
| PEEP ($cmH_2O$) | 6 | 6 | 6 | 6 | 6 | 6 |
| PIP ($cmH_2O$) | 20 | 17 | 26 | 26 | 22 | 22 |
| RF (bpm) | 15 | 18 | 18 | 16 | 15 | 12 |
| $F_IO_2$ | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 0.65 |
| pH | 7.28 | 7.38 | 7.45 | 7.51 | 7.41 | 7.56 |
| $PaO_2$ (mmHg) | 86 | 181 | 48 | 169 | 52 | 145 |
| $PaCO_2$ (mmHg) | 63 | 46 | 41 | 32 | 34 | 34 |
| $P\bar{v}o_2$ (mmHg) | 43 | 36 | 23 | 33 | 35 | 34 |

Abbreviations refer to text and other tables.

Table 6. Changes in pulmonary mechanics and hemodynamics with different I : E ratio (group 2)

| Patient No | 1 | | 2 | | 3 | |
|---|---|---|---|---|---|---|
| I : E ratio | 1 : 2 | 1.2 : 1 | 1 : 2 | 1.2 : 1 | 1 : 2 | 1.2 : 1 |
| $CaO_2$ (ml/dl) | 17.2 | 18.9 | 6.1 | 7.4 | 16.7 | 14.1 |
| $C\bar{v}O_2$ (ml/dl) | 12.8 | 13.8 | 2.9 | 4.9 | 12.1 | 9.3 |
| Hb (g/dl) | 13.7 | 13.8 | 5.2 | 5.2 | 14.2 | 10.4 |
| $A\text{-}aDO_2$ (mmHg) | 564 | 486 | 624 | 295 | 628 | 282 |
| RI | 6.5 | 2.7 | 13.1 | 0.6 | 12.2 | 2.0 |
| $\dot{Q}s/\dot{Q}t$ (%) | 32 | 20 | 47 | 25 | 47 | 15 |
| CO (l/min) | 5.6 | 3.9 | 4.0 | 6.2 | 5.4 | 3.8 |
| CI ($l/min/m^2$) | 3.3 | 2.3 | 2.5 | 3.8 | 3.5 | 2.5 |
| HR (BPM) | 119 | 104 | 73 | 69 | 174 | 110 |
| MAP (mmHg) | 87 | 84 | 50 | 68 | 63 | 77 |
| MPAP (mmHg) | 15 | 10 | 16 | 19 | 20 | 23 |
| PCWP (mmHg) | 9 | 5 | 4 | 11 | 14 | 6 |
| PVR (dynes/sec/$cm^5$) | 86 | 102 | 238 | 103 | 88 | 355 |
| $\dot{D}O_2$ (ml/min) | 584 | 435 | 153 | 281 | 585 | 353 |
| $\dot{V}O_2$ (ml/min) | 162 | 117 | 80 | 95 | 161 | 120 |

Abbreviations refer to text and other tables.

## Materials and Methods

Seven patients suffering from ARF of diverse cause were studied in the Intensive Care Unit. Patients divided into two groups ; group 1 consists of 4 patients with pulmonary disease and group 2 consists of 3 patients with cardiogenic lung edema. Tables 1 and 4 summarize the clinical data of these patients. No patients had any history of lung disease. All patients received mechanical ventilation with PEEP. The cause of pulmonary injury were diverse ; pulmonary bacterial infection was the initial problems in one, aspiration in one, septic lung in one and postcardiac surgery in pulmonary group and cardiogenic lung edema due to congestive heart failure in three cardiac group. All patients had bilateral diffuse chest radiographic findings.

The radial artery was cannulated for the measurement of arterial pressure. A Swan -Ganz triple-lumen balloon tipped flow-directed thermodilution catheter (Edward Laboratories, MA.) was positioned in the pulmonary artery percutaneously through the right internal jugular vein by ultrasonic guidance[6] to measure the right atrial pressure (RA), pulmonary arterial pressure (PA), pulmonary capillary wedge pressure (PCWP) and cardiac output (CO). The catheter position was confirmed by a portable chest radiograph and by visualization of the appropriate were form in the pulmonary artery. Vascular pressure were measured with a Hewlett 1290A transducer at end-expiration. The transducers were positioned at the mid-axillary line with the patients lying supine, and atmospheric pressure was used as a zero reference point. The CO was determined by the thermodilution technique using 10 ml 0f 5% dextrose in water at 0℃. Pulmonary vascular resistance (PVR) was calculated from the Pulmonary vascular resistance (PVR) was calculated from the ratio of the differece between the mean PA and PCWP, and CO. All pressures and CO measurements were on the day of admission to the ICU and then at intervals as needed. Measurements were performed in triplicate and averaged. Arterial and mixed venous samples were drawn anaerobically into heparinized syringes at the same time the pressure were recorded. The $PO_2$, $PCO_2$, and pH in the arterial and mixed venous blood were measured with a blood gas analyzer (178 pH/ Blood Gas Analyzer. Corning Medical and Scientific, MA.). Oxygen saturation ($SaO_2$, $S\bar{v}O_2$) and hemoglobin (Hb) were measured with a Hemoximeter (OSM2, Radiometer, Copenhagen). Oxygen content was calculated from the hemoglobin oxygen-carrying capacity and the amount of dissolved oxygen, as estimated from $PaO_2$ and oxygen solubility. The respiratory index (RI) was calculated as alveolar-arteril oxygen tension difference/$PaO_2$ ($A\text{-}aDO_2$/ $PaO_2$). The intrapulmonary shunt ($\dot{Q}s/\dot{Q}t$) was calculated using the standard shunt equation[7] : $\dot{Q}s/\dot{Q}t = Cc'O_2 - CaO_2/Cc'O_2 - C\bar{v}O_2$, where $Cc'O_2$ is the pulmonary capillary oxygen content, $CaO_2$ is the arterial oxygen content, and $C\bar{v}O_2$ is the mixed venous oxygen content. To calculate $\dot{Q}s/\dot{Q}t$ we assumed that pulmonary capillary oxygen tension was the same as alveolar oxygen tension. The $\dot{D}O_2$ was calculated as the product of $CaO_2$ and CO. Oxygen consumption ($\dot{V}O_2$) was calculated using the Fick principle as follows : $\dot{V}O_2 = CO \times (CaO_2 - C\bar{v}O_2)$. All variables were calculated using a programed calculator.

All patients were sedated with intravenous injection of diazepam or buprenorphine and paralyzed with pancuronium bromide for facilitating the controlled mechanical ventilation. Mechanical ventilation was provided by a Siemens-Elema 900C are shown in Tables 2 and 5. When the $PaO_2$ could not be maintained above 70 mmHg in spite of applying a PEEP of 6 to 10 $cmH_2O$ in pulmonary patients and of 6 $cmH_2O$ in cardiac patients, I : E ratio was increased stepwise from 1 : 2 to 4 : 1 with PEEP of range from 6 to 10 $cmH_2O$ in pulmonary patient and of 6 $cmH_2O$ in cardiac patients measuring the blood gas. The prolongation of inspiratory time was achieved by prolonging the inspiratory time by 67 percent at each step, keeping constant a 20 percent pause time. Following the reduction in $F_IO_2$ to 0.5, when the $PaO_2$ remained above 70 mmHg and patients' chest x-ray films began to show clearing of the alveolar infiltrates, the I : E ratio was gradually decreased to 1 : 2, at which time the paralysis was terminated. Weaning from the ventilator was initiated by synchronized intermittent mandatory ventilation on an $F_IO_2$ of 0.4 with goals of normocarbia, a $PaO_2$ above 70 mmHg and zero end-expiratory pressure. Statistical analysis was not attempted because of too small number of patients.

## Results

Tables 2, 3, 5, and 6 show the ventilator

setting and concomitant blood gas values, systemic and pulmonary hemodynamics, RI, $\dot{V}O_2$, $\dot{D}O_2$ and $\dot{Q}s/\dot{Q}t$. As the I : E ratio increased from 1 : 2 to 4 : 1, all patients showed a significant reduction in $\dot{Q}s/\dot{Q}t$ associated with an increase in $PaO_2$ but no significant change in the $PaCO_2$. Decrease in both $\dot{Q}s/\dot{Q}t$ (x) ($y=-5.502x+41.329$, $r=-0.5561$, $p<0.05$) and RI (x) ($y=-2.02x+9.392$, $r=-0.5732$, $p<0.05$) were in proportion to the prolongation of the inspiratory time (y). The range of increase in $PaO_2$ were from 98% in minimume to 285% in maximum in 7 patients. These improvements become even more significant with continued IRV and the improvement of oxygenation to above 80 mmHg of $PaO_2$ on 100 percent oxygen took just over three hours from the initiation of IRV. Although the CO increased when the I : E ratio was 3 : 2 or 2.6 : 1, it decreased when the I : E ratio was 4 : 1 in pulmonary group. On the other hand, the CO decreaded when the I : E ratio was 1.2 : 1 in 2 of 3 cardiac patients. The reduction in $\dot{Q}s/\dot{Q}t$ with an increase in CO resulted in an increase in $\dot{D}O_2$ during the period when the I : E ratio was 3 : 2 or 2.6 : 1. A decrease in $\dot{D}O_2$ was observed when the I : E ratio was increased to 4 : 1 in pulmonary patients and 1.2 : 1 in 2 cardiac patients. As there was a positive relationship between $\dot{D}O_2$ (y) and $\dot{V}O_2$ ($y=3.719x-109.649$, $r=0.8215$, $p<0.01$), the mixed venous $PO_2$ ($P\bar{v}O_2$) did not changed significanitly with an increase in I : E ratio, from $34\pm6$ mmHg to $34\pm2$ mmHg. Two of 7 patients survived following 6 and 71 days of mechanical ventilation. There were no significant changes in the PA pressure with an increase in I : E ratio, from $20\pm5$ mmHg in control to $22\pm8$ mmHg in IRV. The initial pulmonary hemodynamic data of patients 1, 2, and 3 showed them to meet the criteria for adult respiratory distress syndrome (ARDS) with pulmonary hypertension (mean PA>19 mmHg[8]) and an elevated PVR associated with a normal PCWP.

On the 4th day following IRV (I : E ratio 3 : 2) patient 2 with pulmonary disease developed a pneumothorax ; a chest tube was placed immediately after this insult and was effective. Peak inspiratory airway pressure (PIP) did not increased significantly with an increase in I : E ratio, from $26\pm4$ $cmH_2O$ of I : E ratio of 1 : 2 to $29\pm9$ $cmH_2O$ of 1.2 : 1, 3 : 2, or 4 : 1. The other patients had no complications related to the IRV. A tracheostomy was performed in patient 3 on the 24th day on the ventilator and he was successfully weaned from the ventilator on the 71st day. The other five patients died from multiple organ failure upon ventilator in ICU.

## Discussion

The major findings of this study were that the prolongation of inspiratory time improved the arterial oxygenation in seven mechanically ventilated patients with ARF of diverse causes who were poorly oxygenated on 100% oxygen with a range of PEEP of 4 to 10 $cmH_2O$ ; this improvement became even more significant with continued IRV and was in proportion to the prolongation of inspiratory time. The increase in the I : E ratio from 1 : 2 to 4 : 1 produced a reduction in $\dot{D}O_2$ associated with a decrease in CO, whereas an I : E ratio of 3 : 2 or 2.6 : : 1 provided the increase in $\dot{D}O_2$ with a concomitant increase in CO in pulmonary patients. However, in 2 of 3 cardiac patients, the increase in I : E ratio from 1 : 2 to 1.2 : 1 to produced a reduction in CO and $\dot{D}O_2$. The effect of IRV on arterial oxygenation was not achieved immediately after applying IRV[5] and the improvements became even more significant with continued IRV[9]. In our patients, it took three hour to raise the $PaO_2$ above 80 mmHg on 100% oxygen following initiation of IRV. Ravizza and co-workers [5] also reported that 6 hours of IRV significantly improved oxygenation but 30 minutes IRV had not achieved the same improvement as it had at six hours, and they explained the difference between 6 hours of IRV and 30 minutes IRV by the fact that IRV improves gas exchange by progressive alveolar recruitment. This may explain why some investigators have reported no improvement of $PaO_2$ after IRV, since the results were registered after 15 min[10-11]. Tyler and Cheney [12] suggested in animal

experiments that PEEP is preferable to IRV because the thoracic gas volume was greater with PEEP than with IRV (at an I : E ratio of 4 : 1) for equivalent increase in mean airway pressure. In our study, although PEEP of 10 $cmH_2O$ could not maintain the arterial $PO_2$ above 80 mmHg on 100% of oxygen, adding IRV to PEEP produced a significant improvement in arterial oxygenation. Our results suggest that IRV added to PEEP is the preferable ventilatory pattern for patients with such severe ARF that an adequate $PaO_2$ can not be maintained despite stepwise increases in PEEP. Coles and colleagues[13] suggested that the use of IRV reduces $\dot{Q}s/\dot{Q}t$ in proportion to the change in functional residual residual capacity (FRC) and therefore analogous to the use of PEEP. The PEEP improves gas exchange by preventing terminal airway collapse and increasing the FRC, but it increases peak inspiratory airway pressure (PIP)[1]. Accordingly, high levels of PEEP are reported to be associated with an increased incidence of pulmonary barotrauma (PBT) and a safe upper limit of PEEP has not been established yet[14]. Since, as we observed, PIP does not increase in spite of superimposing IRV on PEEP, IRV has been used successfully to treat patients with ARDS whose airway pressure is already high enough to produce PBT. Baum and colleagues[2] have suggested that IRV may produce a "differential PEEP" which mainly affects the alveolar units with longer time constant. A higher PEEP in poorly ventilated areas is automatically adjusted with IRV, so that it has less effect in areas with a short time constant. Increases in the I : E ratio to produce an increase in the FRC may reduce CO and impair oxygen delivery to the tissue, despite an increase in the $PaO_2$[14]. In this study, we determined the optimal prolonged I : E ratio for producing an improvement in arterial oxygenation without reducing the CO to such an extent that there is a reduction in the overall $\dot{D}O_2$ to the tissue. Our results suggest that the optimal prolonged I : E ratio would be 3 : 2 or 2.6 : 1 to produce better gas exchange without significantly affecting CO in patients with pulmonary disease. On the other hand, in cardiac patients, a slight prolongation of inspiratory time (I : E ratio of 1. 2 : 1) produced a reduction in CO and $\dot{D}O_2$ because of impairment of venous return that depends on the change in mean intrapleural pressure[15]. Despite the reduction in CO and $\dot{D}O_2$, the $P\bar{v}O_2$ did not decreased significantly and was remained within 31 mmHg[16] in all patients with prolonging an I : E ratio. These results indicate that total body tissue oxygenation was probably not impaired. Cole and colleagues[13] advocated IRV-min which increases the I : E ratio to between 1. 1 and 1.7 : 1, and which reduces $\dot{Q}s/\dot{Q}t$ significantly but had no effect on CO so that the $\dot{D}O_2$ is increased. The results of our I : E ratio of IRV in patients with pulmonary disease did not correspond to Cole and colleagues' results. The most likely explanation for the difference between these studies is the difference in the degree of ARF. The stiffer the patient's lung have become, the more pressure will be required to inflate them. Therefore, we should carefully manage respirator adjustments, with stepwise prolongation of the I : E ratio, and closely observe the hemodynamics including measurement of the CO ; this approach will lead to the successful use of IRV. One of seven patients developed pneumothorax during IRV and a chest tube was placed successfully. Petersen and Baier[14] reviewed PBT 171 patients who received treatment for respiratory failure with mechanical ventilation, concluded that the incidence of PBT was 8 percent (fourteen of 171 patients) and that no PBT occurs with a PIP of less than 50 $cmH_2O$. The PIP of our patient with pneumothorax was less than 40 $cmH_2O$, so the higher level of PEEP did not seem to cause it. The PIP decreases and the mean airway pressure (mAP) increases during IRV[12]. Although the explanation for this not clear, fighting the ventilator may cause a transient increase in airway pressure with subsequent alveolar rupture.

In conclusion, our study showed that IRV should be superimposed on PEEP when the effects of PEEP on arterial oxygenation have failed and that the prolongation of inspiratory time should be limited to I : E ratio of 3 :

2 or 2.6 : 1 for preventing a reduction in $\dot{D}O_2$ in patients with pulmonary disease. And, when IRV is applied on patients with cardiogenic lung edema, monitoring for tissue oxygenation is the most important.

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