Ultrasound Monitoring of Right Ventricular Haemodynamics in Children with Complications of Respiratory Syncytial Virus Infection

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Article ID: WMC002785
Article Type: Research articles
Submitted on: 26-Dec-2011, 06:20:17 AM GMT  Published on: 26-Dec-2011, 09:54:50 AM GMT
Article URL: http://www.webmedcentral.com/article_view/2785
Subject Categories: CARDIOLOGY
Keywords: Cardiac Load, Haemodynamic Monitoring, Children, Respiratory Syncytial Virus


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Source(s) of Funding:
The study was supported by Research Program MSM 0021620819 and Specific University Research SVV 262802 of the Ministry of Education and Charles University in Prague, Czech Republic.

Competing Interests:
The authors declare that they have no competing interests.
Ultrasound Monitoring of Right Ventricular Haemodynamics in Children with Complications of Respiratory Syncytial Virus Infection

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Abstract

Background: The aim of this study was to verify the benefits of a separate evaluation of pulmonary and systemic haemodynamics for the management of treatment. For the purposes of this study, we selected the data of the time period from 2006 to 2010.

Methods: A total of 53 children, average age 1.82 years (SD 1.06) were included in the study and divided according to Lung Injury Score (LIS) and Predicted Risk Index Scoring of Mortality (PRISM). Group A (n= 25) included patients with LIS?1.5 points; PRISM?20 points and group B (n= 28) patients with LIS 1.0-1.4 points; PRISM 10-19 points. A transthoracic echocardiography (TTE) combined with ultrasound cardiac output monitoring (USCOM) was used. The myocardial performance indices (MPI RV/LV), pulmonary and systemic vascular resistance (PVR; SVR), cardiac index (CI RV/LV) and other parameters were collated one hour after initiation of therapy (time-1) and after 48 hours of treatment (time-2) for statistical evaluation. All the data were compared within groups and between groups using the distribution-free Wilcoxon's and two-way ANOVA tests.

Results: A total of 232 TTE and USCOM examinations were performed. At time-1 higher median values of MPI RV (0.32, SD 0.01 vs. 0.21, SD 0.01; p
Conclusion: The RSV infection was complicated by the adverse change of pulmonary haemodynamics. Right ventricular afterload increased, depending on the duration of hypventilation. Haemodynamic monitoring provides valuable real-time information to improve efficiency of therapy.

Introduction

Acute bronchiolitis (BOA) is the most common manifestation of human RSV infection in children [1]. The disease manifests as progressive tachypnoea, expiratory-inspiratory dyspnoea without response to bronchodilators or steroids, grunting, chest retraction, increased work of breathing, hypoventilation and cyanosis. High dynamic resistance of airways leads to alveolar hyperinflation, or conversely to alveolar consolidates and extrapulmonary complications, such as pneumonia, acute respiratory distress syndrome, pulmonary arterial hypertension, fluidothorax, or right-sided heart failure [2]. Effective protection against this serious infection is not reliable. Active immunisation is not possible for multiple RSV genome polymorphisms.

A permanent prophylaxis with monoclonal antibodies or antiviral therapy has not produced the expected results [3]. A reliable, effective treatment can only be addressed by the consequences of RSV infection. Our research was motivated by the clinical experience. In children with respiratory insufficiency due to RSV infection, ventilatory support is extremely difficult and prolonged. The adverse haemodynamic changes induced severe complications that complicate the course of disease and extend the overall duration of treatment.

Aim of the study
The aim of this prospective clinical study was to verify the benefits of a separate evaluation of pulmonary and systemic hemodynamics for the management of effective treatment in children with a complicated course of RSV infection.

Methods

In accordance with the rules of the Helsinki Declaration (revised in 2004) and with the approval of the Ethics Committee of the Faculty of Medicine in Pilsen, we conducted a prospective and crossover comparative clinical study at the Department of Paediatrics-PICU, Faculty of Medicine of Charles University in Pilsen in 2006-2010.

Inclusion criteria were identification of RSV, predicted paediatric risk index scoring of mortality (PRISM) of more than 10 points and a value of lung injury score (LIS) above 1.0 point [4-5]. Exclusion criteria included an end of mechanical ventilation within 48 hours of initiation.

To rapidly identify the RSV infection the Rapid-VIDI test (www.vidia.cz) was used.

Protective conventional mechanical lung ventilation was conducted based on the principle of positive
pressure ventilation (Evita-4, Dräger Medical; Germany or Avea, Bird Viasys Healthcare, Care Fusion; USA). Non-conventional forms of ventilation utilised high-frequency oscillatory ventilation (HFOV; SensorMedics 3100A, Viasys Healthcare; The Netherlands), exogenous surfactant replacement (ESR; Curosurf, Chiesi Farmaceutici, S.p.A., Parma, Italy), inhalation of nitric oxide for selective vasodilatation (INO; Pulmnox-Mini NO, Messer Griesheim; Austria) or tracheal gas insufflation (TGI) and prone position ventilation (PPV) to eliminate carbon dioxide [6-7]. Mechanical ventilation was accompanied by volume infusion therapy, nutrition and continuous analgaesic-sedation without neuromuscular block; a combination of sufentanil (0.8–1.3 µg/kg/hr) with midazolam (0.2–0.3 mg/kg/hr) intravenously. To facilitate the mucociliary clearence, mucolytic drugs (acetylcysteinum 10.0-20.0 mg/kg/day, ambroxoli hydrochloride 1.0-1.5 mg/kg/day) were administered intravenously and inhalation of a solution of 3% sodium chloride was used. Antibiotics were used causally according to the results of microbiological analysis of tracheal aspirate. Noradrenalin (0.01-0.25 µg/kg/min.), dobutamine (3.0-8.0 µg/kg/min.), milrinone (0.35–0.75 µg/kg/min.) intravenously or orally sildenafil (1.0–2.0 mg/kg/day) were used based upon the actual hemodynamic changes. Significant pleural and pericardial fluid collections were drained. The drains (Vygon, SA; France and Arrow ECS; Czech Republic) were introduced under the ultrasonography control. Permanent suction was used for the complete evacuation of fluid in a closed system (Atrium Ocean, Mediform; Czech Republic).

The haemodynamics were monitored simultaneously for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects, and flow with dual colour Doppler imaging was used to explore for regurgitation, valves or septal defects.
Doppler flow with high resolution was used to measure the velocity of the atrioventricular valve inflow and the time interval between valvular closure and opening. The data obtained were used to calculate MPIs [17-19]. The USCOM device (Spacelabs Healthcare; Australia) uses state-of-the-art electronics, ultrasonics and signal processing to accurately measure cardiac flow with a 2.2 MHz transducer. Using the probe device, we measured the pulmonary artery annulus flow from the left parasternal approach. Flow rate of the aortic annulus was measured from the jugular approach. The continuous wave Doppler-based technique permits real-time, beat-to-beat, serial measurement of right and left-sided haemodynamic parameters.

Statistical analysis
All measured parameters were presented as a median with standard deviation (median ± SD). For qualitative analysis and a comparison of the distribution of studied parameters of groups, a Student's t-test and distribution-free Wilcoxon's test were applied. The overall development of studied parameters over time was compared between both groups using the two-way ANOVA test. A level of statistical significance of P< 0.05 was accepted. All the data were analyzed using statistical software Statistica® software (StatSoft, Tulsa, U.S.A.).

Results
In the period 2006-2010 a total of 247 children with dyspnoea and identified RSV infection were admitted to our clinic. 53 children (21.45%) had a complicated course of RSV infection. Clinical characteristics on admission to our PICU are listed in Table 1.

Table 1 Characteristics of the RSV study population on admission (n= 53)
Legend: N, number; NS, not significant
Risk factors in group A consisted of congenital heart defect with significant left-right shunt in five children, bronchopulmonary dysplasia in the seven preterm infants, and in two cases, encephalopathy. In group B there were two children with bronchopulmonary dysplasia and one congenital heart defect combined with encephalopathy. Complications at admission were the five severe cases of the acute respiratory distress syndrome combined with the multiple organ dysfunction syndromes, unilateral fluidothorax in six children, one case combined with cardiac tamponade in group A. Group B had only one case of unilateral fluidothorax.
A total of 232 TTE and USCOM examinations were performed.
At time-1, higher average values of MPIRV (0.32, SD 0.01 vs. 0.21, SD 0.01; p
In group A were higher AaDO2 (16.74, SD 5.11 vs. 12.21, SD 4.70; PPPD/VT (52.30, SD 11.08 vs. 36.42, SD 6.49; Pawe (1.89, SD 0.81 vs. 1.18, SD 0.47; PRV (0.34, SD 0.07 vs. 0.28, SD 0.03; PRV (67.43, SD 6.32 vs. 46.77, SD 4.03; PPP2/FiO2 (216.55, SD 78.90 vs. 295.04, SD 67.69; PRV (0.86, SD 0.19 vs. 1.12, SD 0.26; PRV (11.81, SD 1.59 vs. 15.68, SD 2.60; PRV (1.07, SD 0.04 vs. 1.31, SD 0.11; PRV (3.11, SD 0.17 vs. 4.59, SD 0.63; P
At time-2 fewer differences in the data between the two groups were found. During the 48 hours, quality of ventilation and haemodynamics in both groups improved. However, some differences between the groups persisted. Group A maintained a higher VI (26.64, SD 13.06 vs. 20.75, SD 8.47; PD/VT (29.14, SD 5.55 vs. 23.81, SD 5.80; Pawe (1.22, SD 0.81 vs. 0.77, SD 0.16, PRV (0.27, SD 0.03 vs. 0.23, SD 0.02; PRV (1.07, SD 0.04 vs. 1.31, SD 0.11; PRV (3.89, SD 0.12 vs. 4.47, SD 0.24; P
Table 2 Differences in right ventricle load during the study period in group A (n = 25)
Legend:
SW, stroke work (mJ); Vpk, peak velocity of flow (ml/s); Vti, velocity time integral (cm); PVR, pulmonary vascular resistance (dyn.sec/cm5); PVRI, pulmonary vascular resistance index (dyn.sec/cm5/m2); CO, cardiac output (l/min.); CI, cardiac index (l/min/m2); CPO, cardiac power output (W); CVP, central venous pressure (mmHg). All data are presented as mean ± standard deviation.
Table 3 Differences in right ventricle load during the study period in group B (n = 28)
Unmanageable multiple organ failure syndrome in combination with severe brain damage was the cause of one infant death after five days of treatment in group A. Another patient of the same group survived cardiac tamponade and recovered completely. The echocardiographic findings of this patient show in Figures 1A-B. The real monitoring of the right ventricle cardiac output is shown in Figure 2.
Figure 1A-B The development of echocardiographic views of the four-month-olds infant
Legend:
In Figure1A, echocardiography view in the left parasternal short axis on admission. The pericardial fluid collection compressed all cardiac chambers. Re: Significant cardiac tamponade. Figure 1B, the echocardiography view in the left parasternal short axis. Re: After 24 hours of treatment there has been a significant regression of pericardial fluid collection and cardiac compression.
Figure 2 Ultrasound cardiac output monitoring (USCOM) of the right ventricular loud
that the complications of the RSV infection come many of these works, we cannot fully identify. We believe therapy of RSV infection [24-25]. With the conclusions RSV immunoglobulin and palivizumab for the urgent compared [21-23]. Many works recommended ribavirin, who did and did not respond to bronchodilators were review, studies that dichotomised patients into those factor. In a recent Cochrane collaboration systematic airway obstruction due to RSV infection, i.e. time insufficiency or failure is dependent on the duration of resistance. The development of respiratory the lower airways increases the dynamic airway diffuse obliteration of the smallest segments of the significant obstruction of the body immune system. Local inflammatory response leads to activation of macrophages, release of the proinflammatory mediators and the formation of specific IgE antibodies. The result of these pathophysiological processes is diffuse obliteration of the smallest segments of the lower respiratory track. [20]. Significant obstruction of the lower airways increases the dynamic airway resistance. The development of respiratory insufficiency or failure is dependent on the duration of airway obstruction due to RSV infection, i.e. time factor. In a recent Cochrane collaboration systematic review, studies that dichotomised patients into those who did and did not respond to bronchodilators were compared [21-23]. Many works recommended ribavirin, RSV immunoglobulin and palivizumab for the urgent therapy of RSV infection [24-25]. With the conclusions of these works, we cannot fully identify. We believe that the complications of the RSV infection come many days after virus incubation, which is not actual prophylactic and antiviral management. The efficacy of these products by treatment of the complicated course of the RSV infection lacking in support of used as evidence based medicine. For these reasons, in this study antiviral therapy and prophylaxis of the immunological were not included in the treatment of acute conditions. Nevertheless, our treatment strategy was successful. Ventilatory support and consistent sputum evacuation have contributed to the decline in the dynamic airway resistance and adjustment of ventilation parameters during the 48 hours. Many high-quality studies addressed in detail the need for urgent treatment of the RSV infection, but do no emphasise the issue of blood circulation [26-27]. On the contrary, our previous work documented the benefits of echocardiographic monitoring of haemodynamic changes in children [17]. For the contribution of our new study, we consider the finding of effective treatment of complications of RSV infection requiring parallel modifications of ventilation and blood circulation. Severe respiratory failure is a good time to change strategy and convert from conventional to unconventional mechanical ventilation. This unconventional strategy will not increase the load of blood circulation and will support the respiratory lung function.

Discussion

The human respiratory syncytial virus (RSV) is classified in the family Paramyxoviridae, subfamily Pneumovirinae. It occurs in two subtypes with multiple polymorphisms. The genome of a single coil of viral RNA is composed of eleven structural proteins and two non-structural proteins. Structural proteins penetrate into syncytia and paralyse intercellular communication. Nonstructural proteins inhibit interferon IFN-1 synthesis. High invasiveness and immunosuppressive activity are typical features of the RSV. Droplet transmission of RSV infection usually has a seasonal occurrence. During the 2-7 day asymptomatic incubation period, the RSV replicates and floods the entire respiratory tract mucosa. Infected mucosa with cytolsis, necrosis and loss of cilia are a potent inducer of the body immune system. Local inflammatory response leads to activation of macrophages, release of the proinflammatory mediators and the formation of specific IgE antibodies. The importance of the MPIs, CO and other haemodynamic parameters as a noninvasive, easily obtainable, and reproducible tool for the assessment of cardiac load in the setting of clinical intensive care. It should be emphasised that quality testing is for all methods of mechanical ventilation has limitations related to methodology, parameter selection and investigator experience. Choosing the optimal axis of the Doppler probe may be a limitation of the investigator, depending on his experience. This limitation applies to both the methodologies of the study, i.e. TEE and USCOM. On the contrary, measurement of the time interval between the opening and closing of the atrioventricular valves is not investigator dependent. We feel that any potential inaccuracies of this method are counterbalanced when the same investigator performs all the measurements in each subject [17, 29]. All the above limitations do not detract from the importance of the MPIs, CO and other haemodynamic parameters as a noninvasive, easily obtainable, and reproducible tool for the assessment of cardiac load in the setting of clinical intensive care. It should be emphasised that quality testing is for all methods of monitoring haemodynamics dependant on the hands-on investigator experience. Based on our experience and the experience of others, the measurement of haemodynamic changes in children [17]. For the contribution of our new study, we consider the finding of effective treatment of complications of RSV infection requiring parallel modifications of ventilation and blood circulation. Severe respiratory failure is a good time to change strategy and convert from conventional to unconventional mechanical ventilation. This unconventional strategy will not increase the load of blood circulation and will support the respiratory lung function.

Limitations

The authors are aware that echocardiography during mechanical ventilation has limitations related to methodology, parameter selection and investigator experience. Choosing the optimal axis of the Doppler probe may be a limitation of the investigator, depending on his experience. This limitation applies to both the methodologies of the study, i.e. TEE and USCOM. On the contrary, measurement of the time interval between the opening and closing of the atrioventricular valves is not investigator dependent. We feel that any potential inaccuracies of this method are counterbalanced when the same investigator performs all the measurements in each subject [17, 29]. All the above limitations do not detract from the importance of the MPIs, CO and other haemodynamic parameters as a noninvasive, easily obtainable, and reproducible tool for the assessment of cardiac load in the setting of clinical intensive care. It should be emphasised that quality testing is for all methods of monitoring haemodynamics dependant on the hands-on investigator experience. Based on our experience and the experience of others, the measurement of haemodynamic changes in children [17]. For the contribution of our new study, we consider the finding of effective treatment of complications of RSV infection requiring parallel modifications of ventilation and blood circulation. Severe respiratory failure is a good time to change strategy and convert from conventional to unconventional mechanical ventilation. This unconventional strategy will not increase the load of blood circulation and will support the respiratory lung function.

Clinical Implications
The pulmonary vascular bed of premature, chronic lung diseases or congenital heart defects with left-right shunt and hypoxicemic children rapidly reacts by increasing pulmonary vascular resistance. The higher pulmonary vascular resistance is causally related to the development of pulmonary and extrapulmonary complications, including adverse effects on blood circulation. The blood circulation of children with RSV infection is not preload dependent, as in most critically ill patients. It depends exclusively on the right ventricle afterload. The study demonstrated that the data from the separate monitoring of pulmonary and systemic hemodynamics were easily interpretable. A high pulmonary vascular resistance increased the right ventricle afterload (PVR). The increased end-diastolic volume of the right ventricle activates the neuro-humoral regulation. Higher concentrations of the endogenous catecholamines in the myocardium of the right ventricle supported the inotropic activity of the myocardium (Vpk, ET %) and contributed to an increase in stroke work (SW), and stabilised the declining right ventricular cardiac output (CO, CI). By adequate saturation of endogenous catecholamines, the myocardium didn’t require further increase of inotropes. By reducing the right ventricular afterload a selective pulmonary vasodilation stabilised the cardiac output.

The aim of the study was met. Easily available information and their correct interpretation have contributed to the successful treatment of a complicated course of RSV infection in the study population.

List of abbreviations

ALI/ARDS: acute lung injury and acute respiratory distress syndrome; BOA: bronchiolitis acuta; CO: cardiac output (according to the Teichholtz modified formula); CI: cardiac index; CPO: cardiac power output; CTI: cardiothoracic index; ET%: ejection time percent; FT: flow time; FTC: flow time corrected; INO: inhaled nitric oxide; LV MPI: left ventricle myocardial performance index; MD: minute distance; PICU: Paediatric Intensive Care Unit; Pmn: mean pressure gradient; PPV: prone position ventilation; PRISM: Predicted Risk Index Score of Mortality; PVR: pulmonary vascular resistance; RSV: Respiratory Syncytial Virus; RV MPI: right ventricle myocardial performance index; SV: stroke volume; SVI: stroke volume index; SVR: systemic vascular resistance; SVV: stroke volume variation; SW: stroke work; TGI: tracheal gas insufflation; TTE: transthoracic echocardiography; UO: urine output; Vpk: peak velocity of flow; USCOM: Ultrasound Cardiac Output Monitoring; Vti: velocity time integral.

Conclusions

The course of RSV infection was complicated by the adverse change of pulmonary haemodynamics. Right ventricular afterload increased, depending on the duration of hypoxia from the hypoventilation. For safe handling of blood circulation up to date and easily evaluable haemodynamic information are required. Reliable control of pulmonary haemodynamics and management of therapy complications provide the two non-invasive haemodynamic monitoring methods.

References

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Illustrations

Illustration 1

Table 1 Characteristics of the RSV study population on admission (n= 53)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group A N= 25</th>
<th>Group B N= 28</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years; median±SD</td>
<td>0.80 ± 0.42</td>
<td>1.30 ± 0.58</td>
<td>0.05</td>
</tr>
<tr>
<td>Gender, male/female N</td>
<td>14 / 11</td>
<td>11 / 17</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight, Kg; median±SD</td>
<td>8.83 ± 6.54</td>
<td>9.05 ± 6.77</td>
<td>NS</td>
</tr>
<tr>
<td>Risk factors, N (%), median±SD</td>
<td>15 (60.0)</td>
<td>3 (10.7)</td>
<td>0.001</td>
</tr>
<tr>
<td>Treatment before admission, days;</td>
<td>6.17 ± 1.06</td>
<td>3.54 ± 2.04</td>
<td>0.05</td>
</tr>
<tr>
<td>Complications, N (%), points; median±SD</td>
<td>11 (44.0)</td>
<td>1 (3.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>PRISM score, points; median±SD</td>
<td>20.73 ± 2.25</td>
<td>14.75 ± 1.56</td>
<td>0.01</td>
</tr>
<tr>
<td>LIS score, points; median±SD</td>
<td>2.18 ± 0.33</td>
<td>1.31 ± 0.17</td>
<td>0.01</td>
</tr>
</tbody>
</table>
Illustration 2

Table 2 Differences in right ventricle load during the study period in group A (n = 25)

Table 2

<table>
<thead>
<tr>
<th>Parameter</th>
<th>TIME-1</th>
<th>TIME-2</th>
<th>Value</th>
<th>P&lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>MPI RV</td>
<td>0.34 ± 0.07</td>
<td>0.29 ± 0.03</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>SW RV</td>
<td>67.43 ± 6.32</td>
<td>55.33 ± 2.20</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Vpk RV</td>
<td>0.86 ± 0.19</td>
<td>1.21 ± 0.20</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Vti RV</td>
<td>11.81 ± 1.59</td>
<td>14.51 ± 1.05</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>CO RV</td>
<td>1.07 ± 0.04</td>
<td>1.23 ± 0.06</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Cl RV</td>
<td>3.11 ± 0.17</td>
<td>3.89 ± 0.12</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>PVR</td>
<td>4152.23 ± 915.99</td>
<td>3420.23 ± 515.34</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>PVRI</td>
<td>1517.52 ± 436.57</td>
<td>1301.62 ± 236.57</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>12.66 ± 1.58</td>
<td>9.72 ± 1.94</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>
Illustration 3

Table 3 Differences in right ventricle load during the study period in group B (n = 28)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>TIME-1</th>
<th>TIME-2</th>
<th>Value</th>
<th>P&lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>MPI RV</td>
<td>0.28 ± 0.03</td>
<td>0.23 ± 0.02</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>SW RV</td>
<td>46.77 ± 4.03</td>
<td>57.04 ± 2.01</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Vpk RV</td>
<td>1.12 ± 0.26</td>
<td>1.18 ± 0.31</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Vti RV</td>
<td>15.68 ± 2.60</td>
<td>13.72 ± 1.32</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>CO RV</td>
<td>1.31 ± 0.11</td>
<td>1.30 ± 0.09</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Ci RV</td>
<td>4.59 ± 0.63</td>
<td>4.47 ± 0.24</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>PVR</td>
<td>3452.07 ± 506.15</td>
<td>3122.05 ± 431.19</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>PVRI</td>
<td>4775.05 ± 357.44</td>
<td>4441.25 ± 408.50</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>11.87 ± 1.06</td>
<td>10.47 ± 1.11</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>
Illustration 4

Table 4 Characteristics of the study population during hospitalisation (n= 53)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group A</th>
<th>Group B</th>
<th>Value s</th>
<th>P&lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconventional ventilation</td>
<td>N (%)</td>
<td>N (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TGI</td>
<td>12</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HFOV</td>
<td>5</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>INO</td>
<td>7</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PPV</td>
<td>6</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESR</td>
<td>2</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Associated bacterial infection</td>
<td>N (%)</td>
<td>N (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>8 (32.0)</td>
<td>6 (21.4)</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Associated viral infection</td>
<td>N (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 (8.0)</td>
<td>3 (10.7)</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Ventilation time</td>
<td>Days: median±SD</td>
<td>9.05 ± 5.33</td>
<td>6.35 ± 1.75</td>
<td>0.05</td>
</tr>
<tr>
<td>Total duration of hospitalization</td>
<td>Days: median±SD</td>
<td>10.0 ± 1.82</td>
<td>8.5 ± 1.84</td>
<td>0.05</td>
</tr>
</tbody>
</table>
Illustration 5

Figure 1A: The development of echocardiographic views of the four-month-olds infants
Illustration 6

Figure 1B: The development of echocardiographic views of the four-month-olds infants
Illustration 7

Figure 2: Ultrasound cardiac output monitoring (USCOM) of the right ventricular loud
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