

# Right Ventricular Dysfunction in Septic Shock

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**Background:** Right ventricular dysfunction (RVD) is common in critically ill patients and the presence of this condition affects patients' outcomes. Improving the knowledge background and establishing the incidence of RVD in septic shock patients would render the management more efficacious. This study was performed to evaluate the incidence and outcomes of RVD in septic shock patients.

**Material and Method:** A single center, retrospective observational study was performed in the Medical ICU, Siriraj Hospital, Mahidol University between January 2007 and October 2009. Patients with septic shock in whom pulmonary artery catheter (PAC) was inserted were included in the study.

**Results:** The PAC was placed in 118 patients during the study period. The patients' mean age was  $58.0 \pm 18.5$  years and 71 of them (59.3%) were male. The mean body mass index was  $25.0 \pm 6.6$  Kg/m<sup>2</sup> and the mean APACHE II score was  $26.1 \pm 7.7$ . The admission diagnoses were severe sepsis or septic shock (70%), severe pneumonia (38%), acute respiratory distress syndrome (21%). Twenty one patients (17.8%) meet the diagnosis criteria of RVD. The hospital mortality in RVD patients tended to be higher than the non-RVD patients (81.0% vs. 60.8%,  $p$  0.06). Although similar proportions of both group received ventilatory support, the RVD patients had lower tidal volume and had higher peak airway pressure. Also the RVD group had lower PaO<sub>2</sub>/FiO<sub>2</sub> ratio. In addition, the RVD group had lower cardiac output and more frequently underwent renal replacement therapy.

**Conclusion:** In patients with septic shock, the incidence of RVD is substantial. The significant factors associated with RVD include low tidal volume and high peak airway pressure. Measures to prevent the alteration in lung compliance in septic shock patients may prevent RVD and improve patients' outcomes.

**Keywords:** Septic shock, Right ventricular dysfunction, Right ventricle, Pulmonary artery catheter

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Acute right ventricular dysfunction (RVD) is one of the important causes of hemodynamic instability among the patients admitted in intensive care units<sup>(1,2)</sup>. The decompensation of right ventricle (RV) function may occur in the acute-on-chronic settings<sup>(3)</sup>; for example, in COPD patients with acute exacerbation or in patients with cor-pulmonale who develop bacterial pneumonia. Acute right ventricular dysfunction is also presented in specific critical care conditions, such as acute respiratory distress syndrome (ARDS) especially in the patients requiring high level of positive end expiratory pressure (PEEP)<sup>(4-9)</sup>, acute right ventricular infarction<sup>(10,11)</sup> and in patients with severe sepsis/septic shock<sup>(12-15)</sup>. The pathophysiologic basis of RVD

arises from its anatomic characteristics. Since RV is a crescentic chamber covered with thin layer of myocardial tissue which wraps around the interventricular septum, its' ability to compensate with the elevation of RV afterload is poor. The conditions which increase pulmonary vascular resistance (PVR) will lead to RV failure and rising of the RV end diastolic pressure (RVEDP). Rigorous volume resuscitation in these patients will aggravate this condition, causing RV dilatation and shifting of the interventricular septum toward the LV chamber. This will impair LV diastolic function and decrease systemic blood flow<sup>(15,16)</sup> which result in poor organ perfusion, persistent shock and multiorgan failure. We report here the characteristics and outcomes of patients with right ventricular dysfunction determined by hemodynamic data obtained from pulmonary artery catheters (PAC) among the septic shock patients in the medical intensive care unit of a 2,300-bed Tertiary, University hospital in Thailand.

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## **Material and Method**

### **Patients**

The medical record forms of the patients with the diagnosis of septic shock, according to the American Collage of Chest Physicians/Society of Critical Care Medicine (1992) criteria<sup>(17)</sup>, admitted to a 12 bed medical, non-coronary, intensive care unit of Siriraj hospital, Bangkok, Thailand, between January 2007 and October 2009 were reviewed. The hemodynamically unstable patients, with PAC insertion, were included in this study. The patients' baseline characteristics (including age, sex, underlying conditions, APACHE II score in first 24 hours after ICU admission and diagnosis of acute event), hemodynamic parameters within the 1<sup>st</sup> hour after PAC insertion and treatment variables (including ventilator setting, vasoactive agents and renal replacement therapy) were recorded. The indication for PAC insertion was persistent inadequate tissue perfusion, specified by at least one of these following criteria: alteration in conscious level, urine output less than 0.5 ml/kg/hour, SCVO<sub>2</sub> less than 70% or elevated serum lactate more than 4 mmol/l, despite receiving at least 2,000 ml of isotonic crystalloid solution infusion and moderate dose of dopamine or norepinephrine.

### **Hemodynamic parameters measurement**

The PAC used in this study was 7Fr Thermodilution Swan-Ganz catheter, Edwards Lifesciences LLC, Irvine, USA. The PAC was inserted via an 8Fr Fast Cath introducer sheath. The patients were in supine position and the pressure transducers were leveled at the mid chest of the patients before zeroing. The pressure transducer was connected to the monitoring system (Phillips Medizin system, IntelliVue MP70, Boellingen, Germany). The hemodynamic parameters were measured within the 1<sup>st</sup> hour after PAC insertion. The pulmonary artery pressure (PAP), pulmonary artery occlusive pressure (PAOP), right ventricular pressure (RVP) and right atrial pressure (RAP) or central venous pressure (CVP) were measured by waveform analysis of the pressure tracing, recorded on the monitoring system at the end of expiratory phase. Cardiac output (CO) was measured by thermodilution technique, using 3 of 5 injections of 10 ml of cold temperature (0-4°C) of normal saline solution. The value beyond 15 percent of average, or the highest and lowest values, was eliminated before calculation of the average values. Systemic blood pressure was measured via arterial line when presented; otherwise the noninvasive sphygmomanometer was used. The type and dosage

of vasopressors were recorded at the same time as hemodynamic parameters measurement.

### **Diagnosis of right ventricular dysfunction and outcomes**

Right ventricular dysfunction was considered if the patients' hemodynamic parameters meet all of the following criteria: 1) Right atrial pressure > 12 mmHg, 2) Mean pulmonary artery pressure more than 30 mmHg, 3) Pulmonary vascular resistance more than 250 dyne/sec/cm<sup>5</sup>, 4) Pulmonary occlusive pressure less than 18 mmHg<sup>(18,19)</sup>.

The patients' outcomes were recorded as the mortality in the intensive care unit, the 28 days mortality and the hospital mortality.

### **Ethical consideration**

This study was reviewed and approved by the Siriraj hospital's ethical committee, using the Declaration of Helsinki.

### **Statistical analysis**

The patients' baseline characteristics, ventilator parameters and hemodynamic parameters were reported as mean  $\pm$  standard deviation (SD) and percentage. The comparison between the patients with RVD (RVD group) and those without (non RVD group) was performed by using the Pearson's Chi square test or the Fisher's exact test for categorical data and by independent sample t-test for continuous variables. To identify the predicting factors for RVD, the difference of parameters between RVD and non-RVD group with  $p < 0.1$  were included to perform the multivariate analysis by the Binary logistic regression model. The  $p$ -value of less than 0.05 was accepted as statistically significant. The SPSS version 17 was used for statistical analysis.

### **Results**

Among 441 patients admitted in a non-cardiac medical intensive care unit during 1<sup>st</sup> January 2007 to 31<sup>st</sup> October 2009, 118 of them were included. The patients' mean age was  $58.0 \pm 18.5$  years and 59.3 percent were male. Most of the patients had at least one co-morbid condition. History of hypertension was the leading condition, followed by diabetes mellitus, chronic renal failure, atherosclerotic heart disease, congestive heart failure, chronic lung disease and malignancy, respectively. The leading diagnosis at present to ICU admission was severe sepsis or septic shock (70%), followed by severe pneumonia (38%)

and acute respiratory distress syndrome (21%). Twenty one patients (17.8%) meet all of the 4 criteria for diagnosis of right ventricular dysfunction. No significant proportional difference in patients' baseline characteristic was noted between the patients with right ventricular dysfunction (RVD group) and those without (non RVD group), except that patients with RVD had higher body mass index. However, there was a trend of increased incidence of malignancy among the patients with RVD. The details of patients' baseline characteristics, are shown in Table 1. Table 2 demonstrates the information regarding treatment modalities. Most of the patients (87%) were intubated and ventilated. The patients with RVD had lower tidal volume (TV), higher peak airway pressure and lower PaO<sub>2</sub>/FiO<sub>2</sub> ratio. The magnitude of positive end expiratory pressure (PEEP) and FiO<sub>2</sub> were similar in both groups. Every patient needed at least one of vasopressors, namely, dopamine, dobutamine, norepinephrine or adrenaline, in varying dose. A higher proportion of RVD patients (almost two-third) needed renal replacement therapy while only one-third of non-RVD group received this support. The patients' hemodynamic parameters were demonstrated in Table 3. The RVD group had significantly higher pulmonary artery pressure, higher pulmonary vascular resistance (PVR) and also, high systemic vascular resistance

(SVR). In addition, they had lower pulmonary occlusive pressure, lower stroke volume and lower cardiac output/cardiac index. The right ventricular pressure, including right ventricular systolic and diastolic pressure, was measured from only 44 patients. The reasons why right ventricular pressure was not routinely measured was to avoid the risk of ventricular tachycardia and ventricular fibrillation provoked by the tip of a pulmonary catheter.

#### **Predisposing factors and prognosis of RVD**

Comparing with the patients without right ventricular dysfunction, the patients with RVD had significant higher body mass index, lower PaO<sub>2</sub>/FiO<sub>2</sub> ratio, received higher peak inspire pressure and lower tidal volume and received renal replacement therapy in higher proportion. The multivariate analysis was performed by forward stepwise logistic regression model, including all of the significant difference parameters (p-value < 0.05) as mention above plus the other factors which had p-value < 0.1 (age and underlying of malignancy). The peak inspire pressure, PaO<sub>2</sub>/FiO<sub>2</sub> and tidal volume were reclassified into the interval of 5 cmH<sub>2</sub>O, 50 score and 100 milliliter, sequentially before analyses. Only peak inspire pressure (Odds ratio = 2.42, 95% CI 1.19-4.96, P = 0.015) and tidal volume (Odds ratio = 0.27, 95% CI 0.09-0.8, P

**Table 1.** Patients' baseline characteristics in patients with right ventricular dysfunction (RVD) and without (non RVD).

Clinical variables	Non RVD (n = 97)	RVD (n = 21)	p
Age (year)	56.6 ± 18.6	64.7 ± 16.8	0.06
Sex (% of male)	60.8	52.4	0.48
Height (cm)	161.1 ± 8.4	159.3 ± 9.8	0.44
Body mass index (kg/m <sup>2</sup> )	25.6 ± 7.6	28.3 ± 7.4	0.04
APACHE II score	25.5 ± 7.6	28.3 ± 7.4	0.13
Underlying conditions (%)			
Hypertension	44.3	47.6	0.81
Diabetes mellitus	37.1	33.3	0.81
Chronic renal insufficiency	18.6	33.3	0.15
Coronary artery disease	16.5	19.0	0.75
Congestive heart failure	10.3	14.3	0.7
COPD	9.3	9.5	1.0
Other chronic lung disease	5.2	9.5	0.61
Malignancy	6.2	19.0	0.08
Others	19.5	14.3	0.7
Diagnosis			
Severe sepsis/septic shock	70.1	71.4	1.0
Pneumonia	37.1	47.6	0.46
ARDS	21.6	19.0	1.0
Others	27.8	42.9	0.2

**Table 2.** Treatment variables of patients with and without right ventricular dysfunction (RVD).

Treatment variables	Non RVD (n = 97)	RVD (n = 21)	p
Ventilator support (%)	88.7	81.0	0.5
Peak inspire pressure* (cm H <sub>2</sub> O)	18.5 ± 6.1	24.0 ± 5.8	0.002
PEEP* (cm H <sub>2</sub> O)	6.8 ± 3.3	7.5 ± 3.4	0.4
Tidal volume* (ml)	483.9 ± 106.6	390.0 ± 91.3	0.003
Minute ventilation* (l/min)	11.1 ± 2.8	10.0 ± 2.2	0.2
FiO <sub>2</sub> *	56.0 ± 19.9	65.9 ± 24.9	0.1
PaO <sub>2</sub> /FiO <sub>2</sub> *	263.0 ± 174.9	172.8 ± 95.1	0.01
Hemodynamic support (%)	100.0	100.0	1.0
Dopamine (%)	34	38.1	0.6
Low (< 5 mcg/kg/min)	9	12.5	
Moderate (5-10 mcg/kg/min)	51.5	62.5	
High (> 10 mcg/kg/min)	39.4	25	
Dobutamine (%)	16.5	19.0	0.5
Low (< 5mcg/kg/min)	18.8	25	
Moderate (5-10mcg/kg/min)	68.7	75	
High (> 10mcg/kg/min)	12.5	0	
Norepinephrine (%)	63.9	85.7	0.14
Low (< 0.1mcg/kg/min)	38.7	27.8	
High (≥ 0.1mcg/kg/min)	61.3	72.2	
Adrenaline (%)	10.3	0	0.16
Renal replacement therapy (%)	34.0	61.9	0.03

\* The information recorded from patients ventilated with mechanical ventilators.

**Table 3.** Patients hemodynamic parameters, recorded with in first hour after pulmonary artery catheter insertion.

Parameters	Non RVD (n = 97)	RVD (n = 21)	p
Heart rate (/min)	102.3 ± 33.4	106.0 ± 32.7	0.4
Systolic arterial pressure (mmHg)	117.4 ± 23.4	112.4 ± 24.0	0.39
Diastolic arterial pressure (mmHg)	63.9 ± 16.0	59.2 ± 14.0	0.19
Mean arterial pressure (mmHg)	79.8 ± 16.0	74.8 ± 14.6	0.18
RA pressure (mmHg)	12.9 ± 5.1	14.6 ± 5.3	0.19
RV systolic pressure* (mmHg)	35.6 ± 11.4	38.1 ± 9.4	0.47
RV diastolic pressure* (mmHg)	11.4 ± 5.4	14.9 ± 6.4	0.11
PA systolic pressure (mmHg)	41.5 ± 11.3	51.2 ± 13.8	0.006
PA diastolic pressure (mmHg)	22.9 ± 5.9	27.2 ± 6.4	0.008
Mean PA pressure (mmHg)	29.6 ± 7.2	36.1 ± 8.7	0.003
Pulmonary occlusive pressure (mmHg)	16.4 ± 6.0	14.2 ± 2.8	0.012
Stroke volume (ml)	57.4 ± 23.8	35.8 ± 15.0	< 0.001
Systemic vascular resistance (dyne/sec/cm <sup>5</sup> )	1,085.6 ± 500.1	1,583.9 ± 899.0	0.02
Pulmonary vascular resistance (dyne/sec/cm <sup>5</sup> )	218.0 ± 111.5	520.6 ± 250.7	< 0.001
Cardiac output (l/min)	5.9 ± 2.6	3.8 ± 1.8	< 0.001
Cardiac index (l/min/m <sup>2</sup> )	3.5 ± 1.5	2.3 ± 0.9	< 0.001

\* The information recorded from only 44 patients.

= 0.018) were significant factors to predict RVD among hemodynamically unstable patients admitted in medical ICU. (Table 4)

The present of right ventricular dysfunction in patients admitted in the medical intensive care unit tends to associate with higher mortality rate. Table 5

**Table 4.** Predisposing factors for right ventricular dysfunction.

Variables	No RVD (n=97)	RVD(n=21)	Univariate p- value	Multivariate analysis Odds ratio (95% CI), p
Age (year)	56.6±18.6	64.7±16.8	0.06	1.04 (0.99-1.09), p = 0.1
BMI (kg/m <sup>2</sup> )	25.6±7.6	28.3±7.4	0.04	0.98 (0.81-1.18), p = 0.82
Malignancy(%)	6.2	19.0	0.08	0.29 (0.03-2.5), p = 0.26
PIP (cm H <sub>2</sub> O)	18.5±6.1	24.0±5.8	0.002	2.42 (1.19-4.96), p = 0.019
TV (ml)	483.9±106.6	390.0±91.3	0.003	0.27 (0.09-0.8), p = 0.018
PaO <sub>2</sub> / FiO <sub>2</sub>	263.0±174.9	172.8±95.1	0.01	0.97 (0.64-1.36), p = 0.87
RRT (%)	34.0	61.9	0.03	0.34 (0.06-1.81), p = 0.21

BMI = body mass index, PIP = peak inspire pressure, TV = tidal volume, RRT = renal replacement therapy

**Table 5.** Mortality of patients with right ventricular dysfunction and without.

Mortality	Overall (n=118)	No RVD (n=97)	RVD (n=21)	p
ICU mortality	53.4%	50.5%	66.7%	0.14
28 days mortality	50.8%	48.5%	61.9%	0.19
Hospital mortality	64.4%	60.8%	81.0%	0.06

showed trends toward higher ICU mortality, 28 days mortality and hospital mortality among patient with RVD than non-RVD group.

### Discussion

The above data can be summarized that, in our review, the incidence of right ventricular dysfunction (RVD) in septic shock patients was substantial (17.8%). According to our diagnostic criteria, the right ventricular dysfunction secondary to left ventricular failure was excluded by the low level of pulmonary occlusive pressure. When compared with those without RVD, the septic shock patients with RVD had higher body mass index and tended to be older. Although the same proportion of both groups underwent respiratory support, those with RVD received lower tidal volume despite higher peak airway pressures and they also had lower PaO<sub>2</sub>/FiO<sub>2</sub> ratio. Patients with RVD had lower cardiac output while they tended to received more inotropic and vasopressor support. Higher proportion of them underwent renal replacement therapy. The hospital mortality seemed to be high in the RVD group and the significant factors associated with RVD were higher peak airway pressure and lower tidal volume.

These findings paralleled with works of others.

Zapol<sup>(4)</sup>, in 1977, first reported the sequential pulmonary hemodynamic data derived from pulmonary artery catheters in ARDS patients. High pulmonary vascular pressures were noted, which later, proved to be from diffuse obstruction of the pulmonary capillary<sup>(20)</sup>. Survivors in the study had progressive decreases of pulmonary vascular resistance with time, while non-survivors tended to maintain or increase pulmonary vascular resistance. It was concluded that work load imposed upon the right ventricle by elevation of pulmonary vascular resistance might be a factor limiting survival in severe acute respiratory failure. Subsequent studies using radionuclide study<sup>(21)</sup> and echocardiography<sup>(6-9)</sup> also disclosed similar findings. The patient population in our review consisted mostly of sepsis patients which were not identical to the reports above, however, acute lung injury in sepsis shares the same or overlapping pathology as ARDS. Patients with right ventricular dysfunction had more lung parenchymal pathology as judged by poorer compliance and more severe gas exchange alteration.

When compared with others, RVD incidence in our study (17.8%) was considerably lower (previous studies 25% - 92.2%)<sup>(7,8,21)</sup>. These can be explained by the difference in patient population as mentioned above. The acute respiratory distress syndrome was the leading diagnosis in most studies whereas all of ours were sepsis patients and only 21% of them met the criteria of ARDS. Although more than 80% of patients in our study need mechanical ventilation, the main indication was to support vital organ during treatment of shock, especially septic shock which occurred in 70% of our patients.

Additional information from our review was that the patients with RVD had lower cardiac output and more of them underwent renal replacement therapy, indicating more organ failure. Whether these

derangements were the causes or the effects of RVD or all of them occurred during the common pathophysiologic process, preventing the patients with sepsis from developing multiple organ failure by using bundles of evidence based information and early goal directed concept is necessary. As noted from Rivers's study<sup>(22)</sup> as well as our previous report<sup>(23)</sup>, septic shock patients who were adequately resuscitated within 6 hours had lower mortality and less organ failure. Methods to prevent high airway pressure, for example protective ventilator strategies should be emphasized in all patients with acute lung injury as well as other causes. Preventive measures for ventilator associated pneumonia or hospital acquired infection must be strictly employed. Patients with RVD should be investigated for possible reversible causes, for example high airway pressure, thromboembolism and sepsis.

There are some limitations in this study. Firstly, hemodynamic criteria for diagnosis of RVD, using in this study, is base on the postulation that increase right ventricular afterload accompanying with impaired right ventricular systolic function plus rigorous fluid loading were the main pathophysiology of RVD associated with septic shock. Hence, isolated right ventricular dysfunction unrelated with increasing of right ventricular afterload, such as in right ventricular infarction, was not included in RVD group by these criteria. Secondly, the population of this study was hemodynamically unstable patient, admitted in the medical intensive care unit and need for invasive hemodynamic monitoring. These patients generally received high volume of fluid loading and all of them required at least one of moderate dose of vasopressive agents. The incidence of RVD associated with septic shock from our study may not reflect the incidence of RVD among the severe sepsis or septic shock patient who responds with fluid management and low dose of vasopressive agent. Thirdly, hemodynamic data achieved from the first time after pulmonary artery catheter insertion might not represent the entire course of disease. It is possible that RVD may develop later due to increasing of volume resuscitation and changing of vasopressors dosages. Although, the proportion of patient with RVD can increase along the disease progression, the first hemodynamic parameters measurement gives the idea of RVD that cause hemodynamic disturbance.

### Conclusion

The incidence of RVD in patients with sepsis shock is substantial. Patients with RVD had lower tidal

volume, had higher peak airway pressure. There was tendency towards increased mortality in such patients. Measures to prevent RVD must be employed on order to improve patients' outcomes.

### Potential conflicts of interest

None.

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## การทำงานผิดปกติของหัวใจห้องล่างขวา ในผู้ป่วยภาวะช็อกจากการติดเชื้อ

สุรัตน์ ทองอยู่, ไชยรัตน์ เพิ่มพิกุล, สุภาพร เลิศสว่างวงศ์, เอกรินทร์ ภูมิพิเชษฐ, อารุง เลี้ยวรักษ์โอฬาร, วรการ วิไลชนม์, รณิษฐา รัตนะรัต

**ภูมิหลัง:** ภาวะหัวใจห้องล่างขวาทำงานผิดปกติเป็นสาเหตุหนึ่งของความผิดปกติในระบบไหลเวียนโลหิตที่พบในผู้ป่วยภาวะช็อกจากการติดเชื้อและอาจเพิ่มอัตราการเสียชีวิตในผู้ป่วยกลุ่มนี้ ความเข้าใจในปัจจัยเสี่ยงและการดำเนินโรคของภาวะหัวใจห้องล่างขวา ทำงานผิดปกติอาจช่วยให้การรักษาผู้ป่วยภาวะช็อกจากการติดเชื้อมีประสิทธิภาพมากขึ้น

**วัตถุประสงค์:** เพื่อศึกษาปัจจัยเสี่ยงและการพยากรณ์โรคของภาวะหัวใจห้องล่างขวาทำงานผิดปกติที่เกิดขึ้นในผู้ป่วยภาวะช็อกจากการติดเชื้อ

**วัสดุและวิธีการ:** ศึกษาข้อมูลจากการบันทึกการรายงานผู้ป่วยภาวะช็อกจากการติดเชื้อที่ได้รับการใส่สายสวนหลอดเลือดแดงของปอดในหออภิบาลผู้ป่วยวิกฤตระหว่าง มกราคม พ.ศ. 2550 ถึง ตุลาคม พ.ศ. 2552

**ผลการศึกษา:** ผู้ป่วยที่เข้าเกณฑ์การศึกษามีทั้งหมด 118 คน อายุเฉลี่ย  $58.0 \pm 18.5$  ปี เป็นเพศชาย ร้อยละ 71 มีขนาดตัวเฉลี่ย  $26.0 \pm 6.6$  กิโลกรัมต่อตารางเมตรและมีค่าเฉลี่ยของ APACHE II score  $26.1 \pm 7.7$  ภาวะช็อกจากการติดเชื้อมีสาเหตุหลักจากการติดเชื้อในปอดร้อยละ 38 พบผู้ป่วยที่มีภาวะ acute respiratory distress syndrome ร้อยละ 21 พบผู้ป่วยตรงตามเกณฑ์การวินิจฉัยภาวะหัวใจห้องล่างขวาทำงานผิดปกติ 21 ราย คิดเป็นร้อยละ 17.8 ผู้ป่วยเหล่านี้มีอัตราการเสียชีวิตในโรงพยาบาลสูงกว่าผู้ที่ไม่มีความผิดปกติ (ร้อยละ 81 เทียบกับ ร้อยละ 60.8,  $p = 0.06$ ) การมีความดันในทางเดินหายใจสูงและการมีปริมาตรอากาศที่หายใจเข้าในแต่ละครั้งต่ำ เป็นปัจจัยเสี่ยงที่สัมพันธ์กับภาวะหัวใจห้องล่างขวาทำงานผิดปกติ

**สรุป:** จากการศึกษาพบว่าผู้ป่วยภาวะช็อกจากการติดเชื้อส่วนหนึ่งมีหัวใจห้องล่างขวาทำงานผิดปกติร่วมด้วยเนื่องจากปัจจัยเสี่ยงที่สัมพันธ์กับภาวะดังกล่าวได้แก่ การมีความดันในทางเดินหายใจเพิ่มขึ้น และปริมาตรอากาศที่หายใจเข้าต่ำ ซึ่งบ่งบอกว่าปอดมีความสามารถในการขยายตัวต่ำ ดังนั้นการรักษาผู้ป่วยโดยพยายามลดสาเหตุที่อาจทำให้ความสามารถในการขยายตัวของปอดลดลงอาจช่วยป้องกันภาวะหัวใจห้องล่างขวาทำงานผิดปกติและช่วยลดอัตราการเสียชีวิตของผู้ป่วยช็อกจากการติดเชื้อได้

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