Transthoracic Echocardiogram for the Diagnosis of Right Ventricular Dysfunction in Critically Ill Patients

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Background: *Acute right ventricular dysfunction (RVD) is one of the hemodynamic alterations in patients with septic shock, pulmonary embolism and ARDS. This condition had previously been diagnosed by pulmonary artery catheters (PAC). This report is on the use of transthoracic echocardiography (TTE) to diagnose RVD in critically ill patients.*
Objective: *To evaluate the use of TTE for the diagnosis of RVD.*
Material and Method: *A single center, cohort study, was performed in a 12-bed medical ICU. All patients who had had PAC insertions during the period from August 2009 to October 2010 were included in this study. TTE was performed by an investigator (WS. or ST.) who was not aware of the patients' diagnoses. The hemodynamic parameters were measured within the hour prior to performing a TTE. The RVD was diagnosed according to the following criteria: Right atrial (RA) pressure $\geq$12 mmHg, pulmonary artery occlusion pressure (PAOP) <18 mmHg, mean pulmonary artery pressure (PAP) >25 mmHg, and pulmonary vascular resistance (PVR) >250 dyne*sec*cm^{-5}.*
Results: *The PACs were inserted in 59 patients. Of these, 15 had been diagnosed with RVD. A total of 83 TTE examinations, in comparison with hemodynamic parameters measured from PACs, were studied. The TTE parameters; left ventricular (LV) D-shape (sensitivity 61.1%, specificity 84.6%), loss of right ventricular (RV) apical triangle (sensitivity 44.4%, specificity 80%), RV systolic pressure >40 mmHg (sensitivity 77.8%, specificity 60%) and right ventricular end systolic areas: left ventricular end systolic areas (RVESA:LVESA) >0.65 (sensitivity 94.4%, specificity 39.1%) were consistent with RVD. The presence of at least 2 out of 4 echocardiograph findings correlated with RVD, with the area under the ROC curve at 0.79, with a sensitivity of 77.8% and a specificity of 67.7%.*
Conclusion: *TTE is an accurate tool for the diagnosis RVD in critically ill patients with acceptable sensitivities and specificities.*

Keywords: *Right ventricular dysfunction, Echocardiography, LV D-shape, Loss of RV apical triangle*

J Med Assoc Thai 2014; 97 (Suppl. 1): S84-S92
Full text. e-Journal: *http://www.jmatonline.com*

Acute right ventricular dysfunction (RVD) is one of the important causes of hemodynamic instability in critically ill patients. The reported incidence varies from 17% to 92% of patients in the ICU. This is dependent on the diagnostic methods and the patients' population[1]. The conditions associated with increased pulmonary vascular resistance; acute pulmonary emboli[2], acute respiratory distress syndrome (ARDS)[3,4] and severe sepsis/septic shock are known to be predisposing factors of RVD[5-8].

The diagnosis of RVD is obtained from the hemodynamic parameters as measured by pulmonary artery catheters (PAC)[9,10]. Currently, this procedure's use is decreasing due to its invasive nature and the possible complications. Various echocardiographic findings such as elevation of estimated right ventricular (RV) systolic pressure, left ventricular D-shape (LV D-shape), interventricular septal dyskinesia, increased RV size and the loss of the RV apical triangle have been reported as the diagnostic criteria for RVD[11-13]. However, the accuracy of the individual findings or a combination of these findings has not been identified. The authors performed this study to examine the accuracy of transthoracic echocardiogram for the diagnosis of RVD in the critical care setting.

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Material and Method

Patients

The present study was a single center, prospective cohort, conducted in a 12 bed medical, non-coronary, intensive care unit at Siriraj Hospital, Bangkok, Thailand, between August 2009 and

November 2010. The authors included all critically ill patients, over 18 years of age, who had indications for PAC insertion and had agreed to participate in the present study either by themselves or with the permission of their families. Patients with severe subcutaneous emphysema, pneumothorax, and significant valvular lesions were excluded from the present study. The patients' information: age, sex, body weight, height, body mass index and their APACHE II scores for the first 24 hours were recorded. The patient's underlying condition, the diagnoses of acute events and mechanical ventilator settings were also noted. Hemodynamic parameters and the dosages of vasoactive agents were recorded after placement of the PAC. Transthoracic echocardiogram was performed by one of the operators (ST or WS), who were unaware of the patient's hemodynamic status and diagnosis.

Hemodynamic parameters measurement from PAC

The indication for PAC insertion was persistent inadequate tissue perfusion specified by at least one of the following criteria; an alteration in the level of consciousness, urine output less than 0.5 ml/kg/hours, central venous oxygen saturation ($SCVO_2$) less than 70% or an elevation of serum lactate more than 4 mmol/l despite receiving at least 2,000 ml of isotonic crystalloid solution and a moderate dose of dopamine or norepinephrine. The pulmonary artery catheter used in our intensive care unit was a 7Fr, Thermodilution Swan-Ganz catheter; Edwards Lifesciences LLC, Irvine, USA. The pressure transducer was connected to the monitoring system (Phillips Medizin system, IntelliVue MP70, Boellingen, Germany). During hemodynamic measurements, the patients were in a supine position with pressure transducers leveled at the mid-axillary area prior to zeroing the monitor. All intracardiac pressures were measured at the end of expiration. Systemic blood pressures were measured via arterial lines when indicated; otherwise, a non-invasive sphygmomanometer was used. Cardiac output (CO) was measured by the thermodilution technique using 3 to 5 injections of 10 ml of cold, 0-4°C, normal saline solution. The values beyond 15 percent of the average or the highest and lowest values were eliminated before the final calculation of the values. In accordance to the criteria established by the World Health Organization Symposium on Primary Pulmonary Hypertension, RVD is diagnosed if the hemodynamic parameters show evidence of increasing right ventricular afterload accompanied by the elevation of right atrial pressure (RAP) or central venous pressure (CVP) which reflects the accumulation of RV end diastolic volume after decompensation of the RV systolic function. In this study, the presence of RVD was considered if the hemodynamic parameters measured via the PAC met the following criteria[8,14,15]: 1) RAP >12 mmHg, 2) Mean pulmonary artery pressure (PAP) >30 mmHg, 3) Pulmonary vascular resistance >250 dyne/sec/cm^{-5}, and 4) Pulmonary artery occlusive pressure <18 mmHg.

Echocardiography

Every echocardiogram study was performed within 1 hour of the measurement of the hemodynamic parameters taken by the PAC. A Phillips HP15 echo-machine and a S5-2 probe was used to performed the echocardiograms. The most basic examination was done according to the following protocol:

Subcostal view

The echocardiograph probe was placed at the subxiphoid area with the probe's marker pointing toward the patient's head. After identification of the inferior vena cava (IVC), motion mode (M-mode) was used. The inferior vena cava (IVC) diameter was measured during expiration and inspiration.

Parasternal short axis view

The echocardiograph probe was placed at the 3rd or 4th left intercostal space with the marker pointing toward the patient's left shoulder. The morphology of the left ventricle (LV) and the right ventricle (RV) was recorded. Normally, the cross sectional morphology of the LV at the mid level appears as a circular shape. The RV free wall covers at the anteromedial portion and the RV appears as a crescentic space (Fig. 1A). The right ventricular dysfunction was suspected if there was evidence of RV dilatation and the cross sectional morphology of the LV appeared as a D-shape due to the shifting of the interventricular septum in the direction of the left ventricle (Fig. 1B). Then the M-mode across the mid LV was used. The RV free wall, RV diameter, interventricular septum, LV diameter and LV posterior wall were then measured. Dyskinesia of the interventricular septum was present when delay of the interventricular septal contraction compared with the motion of the LV posterior wall, was noted.

Apical 4-chamber view

The echocardiograph probe was placed at the LV apex with the marker pointing towards the right side of the patient. The normal RV was smaller than LV and the RV apex appears at an acute angle and finishes before joining the LV apex. RV dysfunction was suspected when there was an increase of the RV's area in comparison with the LV area and loss of the RV apical triangle (Fig. 1C, D).

Modified 4-chamber view

From the apical 4-chamber view, the echocardiograph probe was shifted medially and slightly upward to identify the tricuspid valve. The tricuspid valve's regurgitation peak velocity was measured. The RV systolic pressure (RVSP) was calculated, base on the modified Bernoulli equation. The calculated RVSP was the summation of the gradient across the tricuspid valve and the right atrial pressure. The trans-tricuspid valve gradient is $4v^2$, when v (meter/second) is the peak tricuspid valve regurgitation velocity. The predicted right atrial pressure (RAP) was estimated to be 5, 10, 15 or 20 mmHg, based on the IVC's diameter and respiratory variation. If the maximal IVC diameter <15 mm, with a diameter variation of more than 50%, the predicted RAP was 5 mmHg. For the maximum IVC diameter of 15-20 mm with diameter variation of more than 50%, the predicted RAP was 10 mmHg. When maximum IVC diameter was at 15-20, the predicted RAP was 15 mmHg if the diameter variation <50%, and the predicted RAP was considered to be 20 mmHg if there was no IVC diameter variation.

To determine the intra-observer and the inter-observer variation, the echocardiograph examinations were performed separately by the same operator and by both operators in 15 randomly selected cases.

Statistical analysis

The continuous variables were presented as a mean $\pm$ standard deviation (SD), while the categorical variables were presented in percentages. The comparisons of continuous variables were assessed by an unpaired student's t-test. The Fisher's exact test and Chi-square test were used to compare the categorical variables between the groups. Logistic regression analysis was used to describe the correlation between the different methods for diagnosing RVD. The predictive accuracy for the diagnosis of RVD by echocardiography as compared with the diagnosis by PACs was assessed from a receiver operating characteristic curve (ROC). To evaluate the agreement of echocardiograph parameters measured by the same operator and by different operators, the pairs of echocardiograph parameters measured from the same operator in separate examinations or from different operators were used. The inter-rater agreement and the kappa index were used to assess the accuracy for the continuous and the categorical variables, sequentially. All statistical analysis used the software package for windows, version 13.0 (SPSS V.13.0, SPSS Inc. Chicago, IL, USA). A p-value <0.05 was considered as statistically significant.

Ethical consideration

This study was reviewed and approved by the Siriraj Institutional Review Board using the Declaration of Helsinki. For all data, witnessed consent was obtained from a relative or from the patients' themselves.

Results

Among the 450 patients admitted to the ICU during the study period, 63 had pulmonary artery catheters inserted. Cardiac output measurements by the thermodilution technique were performed in 61 patients. One patient declined the informed consent, and one patient died before an echocardiogram could have been performed. Therefore, 59 patients were included in the present study, 15 patients were diagnosed with RVD while a total of 83 pairs of hemodynamic variables were recorded. Eighteen of the patients' met the criteria for the diagnosis of RVD. The patients' baseline characteristics are shown in the Table 1. There were no significant differences in mean ages, body sizes, sex, APACHE II scores and underlying conditions between the non-RVD and the RVD group. Severe sepsis and septic shock were the leading ICU admission diagnoses, followed by a diagnosis of severe pneumonia. The 28 day mortality rates and the hospital mortality rates were slightly higher among the patients with RVD. The patients' hemodynamic parameters, use of vasopressors, and respiratory parameters are shown in the Table 2. The RVD patients had significantly lower diastolic blood pressures. There were trends toward lower systemic blood pressures, pulmonary occlusive pressures and cardiac indexes in the RVD patients. The vasopressors; norepinephrine, dopamine, adrenaline and dobutamine were used in a larger proportion in the patients with RVD than those in the non-RVD group. There were no significant differences in the respiratory parameters between the groups.

Echocardiograph findings

Table 3 illustrates the echocardiograph parameters. It was noted that the RVD patients had smaller left ventricular end systolic and end diastolic areas, lower left ventricular stroke volumes, calculated

Table 1. Patients' baseline characteristics comparing the groups with and without right ventricular dysfunction

Clinical variables	Non RVD (n = 65)	RVD (n = 18)	p-value
Age (years)	56.9±21.1	56.4±13.8	0.92
Sex (% of male)	49.2%	38.9%	0.60
Height (cm)	161.1±11.2	158.8±11.2	0.44
Body weight (kg)	67.0±22.5	60.2±29.7	0.38
Body mass index (kg/m^2)	25.5±6.7	23.6±9.6	0.43
APACHE II score	21.4±8.4	23.5±9.4	0.40
Underlying conditions			
Hypertension	38.5%	38.9%	1.00
Chronic renal insufficiency	36.9%	22.2%	0.28
Diabetes mellitus	26.2%	27.8%	1.00
Coronary artery disease	18.5%	22.2%	0.74
Systemic lupus erythematosus	13.8%	22.2%	0.47
Malignancy	12.3%	16.7%	0.70
Congestive heart failure	9.2%	11.1%	1.00
Chronic lung disease	9.2%	5.6%	0.33
Diagnosis			
Severe sepsis/septic shock	63.1%	83.3%	0.16
Pneumonia	26.2%	11.1%	0.22
Others	10.7%	5.6%	0.36
Hospital mortality	53.8%	77.8%	0.17
28-days mortality	46.2%	72.2%	0.13
60-days mortality	58.5%	77.8%	0.20

Table 2. Patients' hemodynamic and mechanical ventilator parameters

Clinical parameters	Non RVD (n = 65)	RVD (n = 18)	p-value
Hemodynamic parameters			
Heart rate (beat per min)	101.3±20.7	104.8±26.7	0.600
Systolic blood pressure (mmHg)	120.5±21.0	111.1±21.8	0.110
Diastolic blood pressure (mmHg)	70.0±15.3	58.7±13.5	0.005
Mean systemic blood pressure (mmHg)	82.7±15.3	76.1±15.2	0.110
Pulmonary systolic pressure (mmHg)	44.4±12.9	49.5±14.7	0.190
Pulmonary diastolic pressure (mmHg)	25.7±8.4	26.8±6.3	0.570
Mean pulmonary artery pressure (mmHg)	31.8±8.7	34.8±8.7	0.200
Central venous pressure (mmHg)	14.3±6.2	16.6±5.9	0.160
Pulmonary occlusive pressure (mmHg)	18.5±6.7	16.0±2.6	0.100
Cardiac index (l/min/sq.m.)	3.6±1.6	3.0±1.2	0.100
Systemic vascular resistance (dyne/sec/cm^{-5})	1,136.9±665.7	1,134.2±343.3	0.980
Pulmonary vascular resistance (dyne/sec/cm^{-5})	214.3±150.8	362.1±147.9	0.001
Vasoactive agents			
Dopamine	18.5%	44.4%	0.004
Norepinephrine	56.9%	61.1%	0.130
Adrenaline	6.1%	11.1%	0.600
Dobutamine	21.5%	27.8%	0.550
Respiratory parameters			
Mechanical ventilator support	95.4%	100%	1.000
Peak airway pressure (cmH_2O)	25.4±5.8	25.6±7.5	0.940
Positive end expiratory pressure (cmH_2O)	7.0±3.2	6.9±2.5	0.200

Table 3. Echocardiogram findings

Echocardiograph parameters	Non-RVD (n = 65)	RVD (n = 18)	p-value
Inferior vena cava minimal diameter (cm)	1.67+0.52	1.67+0.60	0.970
Inferior vena cava maximal diameter (cm)	2.03+0.55	1.98+0.48	0.710
Estimated RV systolic pressure (mmHg)	39.40+12.70	46.50+12.00	0.040
Estimated RV systolic pressure ≥40 mmHg	40.0%	77.8%	0.005
Calculated stroke volume (ml)	53.50+25.80	41.80+17.00	0.080
Calculated cardiac output (L/min)	5.30+2.60	4.10+1.50	0.040
LV D-shape	15.4%	61.1%	<0.001
Loss of RV apical triangle	20.0%	44.4%	0.040
Septal dyskinesia	30.8%	50.0%	0.170
RV end systolic area (cm)	14.80+6.40	16.50+6.90	0.360
RV end diastolic area (cm)	19.80+7.90	20.20+6.50	0.830
LV end systolic area (cm)	20.50+9.10	14.60+7.80	0.010
LV end diastolic area (cm)	29.00+12.90	21.50+8.00	0.020
Left ventricular ejection fraction (%)	48.00+17.00	52.00+17.00	0.400
RV:LV end systolic area ratio	0.80+0.40	1.70+1.70	<0.001
RV:LV end diastolic area ratio	0.70+0.30	1.20+0.80	0.020
RV:LV end systolic ratio ≥0.65	60.9%	94.4%	0.008

cardiac outputs, and higher calculated right ventricular systolic pressures than the non-RVD patients. There was no significant differences among inferior vena cava diameters, right ventricular end systolic and end diastolic areas and left ventricular ejection fractions. The previously reported morphological findings associated with RVD, including LV D-shape and loss of the RV apical triangle, were identified in a significantly higher proportion of the RVD patients. Although a higher proportion of septal dyskinesia was detected in the RVD group, there was no statistical significance. The inter-observer analysis showed agreement in the detection of LV D shape, loss of right ventricular triangle, septal dyskinesia, right ventricular diastolic and systolic areas, left ventricular diastolic and systolic areas, and right ventricular systolic pressures with a keppa index at 0.9-1.0. As for the calculated values, the RV:LV end systolic and end diastolic area ratios were higher in the RVD group. The receiving operative curve (ROC) was performed to evaluate the cut-off value for echocardiogram diagnosis of RVD by using right ventricular systolic pressures (RVSP), RV:LV end systolic area ratios and RV:LV end diastolic area ratios. The results are shown in Fig. 2a. The cut-off value of RVSP for the diagnosis of RVD was 40 mmHg with an area under the curve (AUC) = 0.65 and p-value = 0.05. The RV:LV end systolic area ratio >0.65 and RV;LV end diastolic area ration >0.72 were identified as the cut-off points for RVD diagnosis with AUCs of 0.72 and 0.66 and p-values of 0.005 and 0.04, respectively. In accordance with the higher AUCs, the RV:LVESA ratio >0.65, the RVSP >40 mmHg, the LV D-shape and loss of RV apical triangle were identified as the echocardiograph findings associated with right ventricular dysfunction. The additional benefits for RVD diagnosis with the combination of these 4 echocardiograph parameters was evaluated by the ROC curve. The detection of at least 2 out of 4 parameters was a good predictor for the diagnosis of RVD with the AUC of 0.79 and p-value <0.001. Table 4 shows the sensitivity, specificity, the positive and the negative predictive values of the individual echocardiograph findings and the combination of 2 out of 4 parameters for the diagnosis of RVD.

Discussion

Our results can be summarized in that the occurrence of right ventricular dysfunction in our critically ill patients was substantial. The majority of these patients had the diagnosis of sepsis or septic shock. The patients with RVD tend to have a high mortality rate. The diagnostic accuracy of echocardiography for this condition, primarily the LV-D shape, loss of the LV apical triangle, RV systolic pressures >40 mmHg and RV:LV end diastolic areas (RVEDA/ LVEDA ratio) >0.65 were acceptable. The latter was the most sensitive and had a high negative predictive value. In addition, the presence of two or more of the above

Table 4. The efficacy of echocardiograph parameters for the diagnosis of right ventricular dysfunction

Echocardiograph parameters	Sensitivity	Specificity	Positive predictive value	Negative predictive value	p-value
LV D-shape	61.1%	84.6%	52.4%	88.7%	0.001
Loss of RV apical triangle	44.4%	80.0%	38.1%	83.9%	0.040
RV systolic pressure $\geq$40 mmHg	77.8%	60.0%	35.4%	90.7%	0.005
RV:LV end systolic area $\geq$0.65	94.4%	39.1%	30.4%	96.2%	0.007
Present of echo-finding $\geq$2	77.8%	67.7%	40.0%	91.7%	0.001

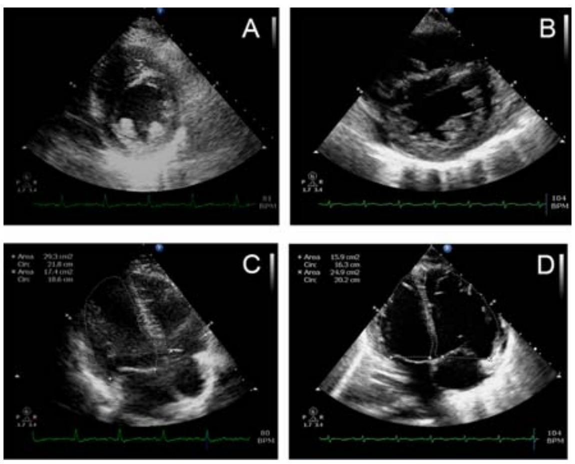

Fig. 1 The morphology of the LV and the RV: A) Parasternal short axis view showed cross sectional views of a normal LV. The RV appears as a crescentic structure, covering the left upper quadrant of the LV. B) Parasternal short axis view of a patient with RV dysfunction shows LV D-shape. C) Apical 4-chamber view shows LV on the left side and RV on the right side of picture. Normal RVEDA and RVESA are smaller than LVEDA and LVESA. The RV apex appears as a triangular shape and finishes before the LV apex. D) Apical 4-chamber view of a patient with RV dysfunction shows dilation of the RV with loss of the RV apical triangle.

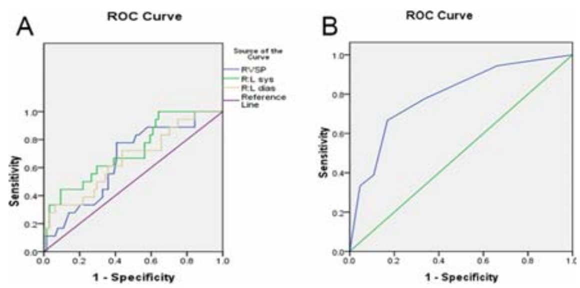

Fig. 2 The receiving operative curve (ROC) of RVSP (AUC = 0.65, p = 0.05), RV: LVEDA ratio (AUC = 0.66, p = 0.04) and RV: LVESA ratio (AUC = 0.72, p = 0.005) are illustrated in fig. 2a. The ROC of the combination of 4 echocardiograph findings for the diagnosis of RV dysfunction as is shown in fig. 2b (AUC = 0.79, p<0.001).

criteria enhances the likelihood of this condition.

The significance of right ventricular dysfunction in critically ill patients had been reported in 1978 by Sibbald et al[9]. With the use of pulmonary catheters, pulmonary hypertension was identified in 59% of sepsis patients and this was associated with a high mortality rate. In 1977, Zapol[10] reported on pulmonary arterial hypertension in mechanically ventilated patients. Although a direct association with mortality was not identified, the survivors had progressively decreased pulmonary vascular resistance while patients who failed to survive did not. Since then, there has been accumulated information regarding the incidences and various diagnostic tests[16,17]. Our findings disclosed parallel evidences. While including the patients with a main diagnosis of sepsis, no differences in mortality rates between the RVD patients and the non-RVD patients were noted. This is possibly from the small population of the patients included in the presentstudy.

As for the echocardiograph findings, the present study clearly demonstrates the diagnostic capability of the test for the condition of right ventricular dysfunction. This is significant since pulmonary artery catheterization is invasive and poses risks of complications. Knowledge of the proper use of the, minimally invasive echocardiogram, is essential for clinicians to diagnose the condition correctly. Up to now, the diagnostic criteria of RVD have been unclear[18,19] and the identification of this condition appears to be subjective. Some diagnose this condition by using the RVEDA/LVEDA ratio in the long axis, >0.6, as associated with septal dyskinesia and in the short axis during transesophageal echocardiography (TEE) examination[16]. Others have defined RV dysfunction when the RVEDA/LVEDA ratio is >0.6 and RV failure when RVEDA/LVEDA ratio is >0.9[17,20]. Of the parameters used in our study, the RV:LV end systolic area ratio of >0.65 was deemed sensitive and had a high negative predictive value. This means that this parameter is the perfect screening tool for RVD. The elevation of RVSP measured by echocardiogram is an acceptably sensitive test in the detection of RVD. Although the sensitivity of the LV D-shape and loss of RV apical triangle in diagnosing RVD are relatively low, its specificity and negative predictive value are significant. To enhance the diagnostic accuracy, we examined the effect of adding more echo studies. The combination of high sensitivity tests, especially the RV:LV end systolic area ratio >0.65 with the addition of one of the other, more specific echocardiograph criteria (LV-D shape, loss of LV apical triangle or RV systolic pressure of >40 mmHg), slightly improved the specificity to 67.7% (AUC 0.79, $p<0.001$). Hence, currently, echocardiographs are an important tool in screening for RVD in critically ill patients.

The limitations of our study, as noted above, included: 1) The limited number of patients (especially those with RVD) in our population, and 2) The heterogeneity of the study's population. Having a greater number of patients with the same conditions and in a well-designed prospective study would provide a greater amount of informative data. This will be included in our future plan to validate the criteria for echocardiograph diagnosis.

Right ventricular dysfunction is an important condition of which the etiologies are heterogeneous. The disorders of contractile function resulting from coronary artery disease, acute and chronic pulmonary hypertension, sepsis and the side effects of certain drugs can contribute to developing RVD while disorders in pulmonary circulation contribute to high afterload and can impair RV function. The pathophysiology of each condition is diverse. Some diseases are reversible while others are not. It is crucial that intensivists realize the significance of RVD, routinely screen for the risk factors and the occurrences of this condition. Bedside echocardiography is an important screening tool, especially with the application of the above criteria. If RVD is found, the possible causes must be alleviated. Those who have acute respiratory distress syndrome need to be supported by protective strategies. The possible sources of sepsis must be well controlled.

In conclusion, we report here the occurrences of right ventricular dysfunction in our unit. Also, the authors demonstrate the use of bedside echocardiography as a non-invasive tool for diagnosis. The echocardiograph parameters of RV:LV end systolic area ratios of >0.65 have proven to be sensitive and have a high negative predictive value. This may be applied as a screening tool for this condition. Further studies are needed to validate this tool and to better understand RVD in specific settings.

Potential conflicts of interest

None.

References

1. Piazza G, Goldhaber SZ. The acutely decompensated right ventricle: pathways for diagnosis and management. Chest 2005; 128: 1836-52.
2. Kreit JW. The impact of right ventricular dysfunction on the prognosis and therapy of normotensive patients with pulmonary embolism. Chest 2004; 125: 1539-45.
3. Vieillard-Baron A, Schmitt JM, Augarde R, Fellahi JL, Prin S, Page B, et al. Acute cor pulmonale in acute respiratory distress syndrome submitted to protective ventilation: incidence, clinical implications, and prognosis. Crit Care Med 2001; 29: 1551-5.
4. Moloney ED, Evans TW. Pathophysiology and pharmacological treatment of pulmonary hypertension in acute respiratory distress syndrome. Eur Respir J 2003; 21: 720-7.
5. Mitsuo T, Shimazaki S, Matsuda H. Right ventricular dysfunction in septic patients. Crit Care Med 1992; 20: 630-4.
6. Dhainaut JF, Pinsky MR, Nouria S, Slomka F, Brunet F. Right ventricular function in human sepsis: a thermodilution study. Chest 1997; 112: 1043-9.
7. Chan CM, Klinger JR. The right ventricle in sepsis. Clin Chest Med 2008; 29: 661-76.
8. Tongyoo S, Permpikul C, Lertsawangwong S, Poompichet A, Leawruxoran A, Vilaichone W, et al. Right ventricular dysfunction in septic shock. J Med Assoc Thai 2011; 94 (Suppl 1): S188-95.
9. Sibbald WJ, Paterson NA, Holliday RL, Anderson RA, Lobb TR, Duff JH. Pulmonary hypertension in sepsis: measurement by the pulmonary arterial diastolic-pulmonary wedge pressure gradient and the influence of passive and active factors. Chest 1978; 73: 583-91.
10. Zapol WM, Snider MT. Pulmonary hypertension

in severe acute respiratory failure. N Engl J Med 1977; 296: 476-80.
11. Jardin F, Brun-Ney D, Hardy A, Aegerter P, Beauchet A, Bourdarias JP. Combined thermodilution and two-dimensional echocardiographic evaluation of right ventricular function during respiratory support with PEEP. Chest 1991; 99: 162-8.
12. Vieillard-Baron A. Assessment of right ventricular function. Curr Opin Crit Care 2009; 15: 254-60.
13. Jardin F, Vieillard-Baron A. Monitoring of right-sided heart function. Curr Opin Crit Care 2005; 11: 271-9.
14. Greyson CR. Pathophysiology of right ventricular failure. Crit Care Med 2008; 36: S57-S65.
15. Mebazaa A, Karpati P, Renaud E, Algotsson L. Acute right ventricular failure—from pathophysiology to new treatments. Intensive Care Med 2004; 30: 185-96.
16. Vieillard-Baron A, Schmitt JM, Augarde R, Fellahi JL, Prin S, Page B, et al. Acute cor pulmonale in acute respiratory distress syndrome submitted to protective ventilation: incidence, clinical implications, and prognosis. Crit Care Med 2001; 29: 1551-5.
17. Guervilly C, Forel JM, Hraiech S, Demory D, Allardet-Servent J, Adda M, et al. Right ventricular function during high-frequency oscillatory ventilation in adults with acute respiratory distress syndrome. Crit Care Med 2012; 40: 1539-45.
18. Beaulieu Y. Bedside echocardiography in the assessment of the critically ill. Crit Care Med 2007; 35 (5 Suppl): S235-49.
19. Woods J, Monteiro P, Rhodes A. Right ventricular dysfunction. Curr Opin Crit Care 2007; 13: 532-40.
20. Jardin F, Vieillard-Baron A. Is there a safe plateau pressure in ARDS? The right heart only knows. Intensive Care Med 2007; 33: 444-7.

การวินิจฉัยภาวะหัวใจห้องล่างขวาทำงานผิดปกติในผู้ป่วยวิกฤตทางอายุรกรรมด้วยเครื่องตรวจคลื่นเสียงสะท้อนหัวใจ

สุรัตน์ ทองอยู่, วิมลรัตน์ แสงนพคุณศรี, ไชยรัตน์ เพิ่มพิกุล

ภูมิหลัง: ภาวะหัวใจห้องล่างขวาทำงานผิดปกติ เป็นภาวะแทรกซ้อนในผู้ป่วยภาวะช็อกจากการติดเชื้อ ผู้ป่วยลิ่มเลือดอุดตันในหลอดเลือดแดงของปอด และผู้ป่วยปอดอักเสบอย่างรุนแรง การวินิจฉัยภาวะนี้อาศัยการวัดความดันโลหิตผ่านสายสวนหลอดเลือดแดงปอด ซึ่งเป็นหัตถการที่อาจก่อให้เกิดภาวะแทรกซ้อนที่รุนแรงได้ การตรวจหัวใจด้วยคลื่นเสียงสะท้อนความถี่สูงอาจให้การวินิจฉัยภาวะหัวใจห้องล่างขวาทำงานผิดปกติได้โดยไม่ต้องใส่สายสวนหลอดเลือดแดงปอด

วัตถุประสงค์: เพื่อศึกษาความถูกต้องของการใช้เครื่องตรวจคลื่นเสียงสะท้อนความถี่สูงในการวินิจฉัย ภาวะหัวใจห้องล่างขวาทำงานผิดปกติในผู้ป่วยวิกฤต

วัสดุและวิธีการ: ศึกษาข้อมูลผู้ป่วยที่รับไว้ในหออภิบาลผู้ป่วยวิกฤตทางอายุรกรรมของโรงพยาบาลศิริราช ระหว่างวันที่ 1 สิงหาคม พ.ศ. 2552 ถึง 31 ตุลาคม พ.ศ. 2553 ผู้ป่วยที่ได้รับการใส่สายสวนหลอดเลือดแดงปอด จะได้รับการตรวจการทำงานของหัวใจด้วยเครื่องคลื่นเสียงสะท้อนความถี่สูงเปรียบเทียบกับการวินิจฉัย ด้วยสายสวนหลอดเลือดแดงปอด โดยมีเกณฑ์การวินิจฉัยภาวะหัวใจห้องล่างขวาผิดปกติดังนี้ 1) ความดันในหัวใจห้องบนขวา $\geq$12 มิลลิเมตรปรอท 2) ความดัน pulmonary occlusive pressure <18 มิลลิเมตรปรอท 3) ความดันหลอดเลือดแดงปอดเฉลี่ย $\geq$25 มิลิเมตรปรอท และค่าแรงต้านทานของหลอดเลือดแดงปอด $\geq$250 dyne*sec*cm^{-5}

ผลการศึกษา: ผู้ป่วยเข้าร่วมการศึกษาจำนวน 59 ราย ได้รับการวินิจฉัยภาวะหัวใจห้องล่างขวาผิดปกติจำนวน 15 ราย มีการประเมินการไหลเวียนโลหิตทั้งหมด 83 ครั้ง พบว่าการตรวจพบหัวใจห้องล่างซ้ายมีลักษณะเป็นรูปอักษร D (ความไวร้อยละ 61.1, ความจำเพาะร้อยละ 84.6) ส่วนปลายของหัวใจห้องล่างขวามีลักษณะเป็นมุมป้าน (ความไวร้อยละ 44.4, ความจำเพาะร้อยละ 80) ค่าความดันซิสโตลิกในหัวใจห้องล่างขวา $\geq$40 มิลลิเมตรปรอท (ความไวร้อยละ 77.8, ความจำเพาะร้อยละ 60) และอัตราส่วนของพื้นที่หน้าตัดในแนวยาวของหัวใจห้องล่างขวา ต่อหัวใจห้องล่างซ้ายในช่วงสิ้นสุดการบีบตัว $\geq$0.65 (ความไวร้อยละ 94.4, ความจำเพาะร้อยละ 39.1) สัมพันธ์กับการมีภาวะหัวใจห้องล่างขวาทำงานผิดปกติ การตรวจพบดังกล่าว 2 ใน 4 ข้อ ช่วยเพิ่มความจำเพาะ ในการวินิจฉัยภาวะหัวใจห้องล่างขวาผิดปกติโดยใช้เครื่องตรวจคลื่นเสียงสะท้อนความถี่สูงได้

สรุป: การตรวจหัวใจด้วยเครื่องคลื่นเสียงสะท้อนความถี่สูงสามารถใช้ในการวินิจฉัยภาวะหัวใจห้องล่างขวาทำงานผิดปกติได้ในผู้ป่วยวิกฤต