Correlation between Left Atrial Volume Index and Pulmonary Artery Pressure in Patients with Chronic Severe Mitral Regurgitation

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Background: *Left atrial dilatation is a response to volume overload in chronic mitral regurgitation (MR). Left atrium volume index (LAVI) was associated with mortality, heart failure and atrial fibrillation in patients with MR. The authors do not know the association between LAVI and pulmonary artery pressure in patients with chronic severe primary MR.*

Material and Method: *The authors retrospectively studied patients with chronic severe MR (either one or both echocardiographic criteria of effective regurgitant orifice area $\geq$ 40 mm^2 or regurgitant volume $\geq$ 60 ml by proximal isovelocity surface area method) who underwent transthoracic echocardiography at Siriraj Hospital between January 2005 and December 2009.*

Results: *A total of 181 patients (age 53.1 $\pm$ 17.7 years, 53.6% male) were enrolled. Right ventricular systolic pressure (RVSP) tended to increase when LAVI increased (r_s = 0.32, p < 0.001). The mean RVSP in 4 different quartiles of LAVI ($\leq$ 48.80 ml/m^2, 48.81-66.00 ml/m^2, 66.01-97.40 ml/m^2, > 97.40 ml/m^2) were 41 $\pm$ 14, 42 $\pm$ 16, 44 $\pm$ 16 and 56 $\pm$ 18 mmHg, respectively. RVSP in patients with LAVI > 97.40 ml/m^2 was significantly higher than those of the other 3 quartiles (p = 0.004). LAVI in patients with RVSP $\leq$ 50 and > 50 mmHg were 74 $\pm$ 53 and 116 $\pm$ 82 ml/m^2, respectively (p = 0.001).*

Conclusion: *In chronic severe primary MR, RVSP tends to increase when LAVI increases.*

Keywords: *Echocardiography, Left atrium volume, Chronic mitral regurgitation, Pulmonary hypertension*

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Left atrial (LA) dilatation may be a consequence of atrial fibrillation (AF), LA pressure overload (*e.g.* left ventricular (LV) diastolic dysfunction, mitral stenosis), or LA volume overload (*e.g.* mitral regurgitation [MR]). In case of LA pressure or volume overload, dilatation of LA is a compensatory mechanism to maintain normal LA pressure. At present, there are various different methods to measure LA volume (LAV) or size. LAV as determined by echocardiography provides a more accurate assessment of LA than a conventional LA dimension by M-mode technique[1]. LAV index (LAVI) is a more accurate cardiovascular risk marker than LA area or diameter in patients with sinus rhythm[2]. A high LAVI not only represents chronicity and severity of underlying pathologic process but also has a prognostic implication. LAVI predicts a new-onset AF[3-6], stroke[7,8], congestive heart failure (CHF)[9,10] and cardiovascular death[7,11-14].

LA enlargement is an important compensatory mechanism in chronic MR. The adaptation of LA in patients with chronic MR is essential to maintain normal pulmonary artery pressure. Pulmonary hypertension and LA dilatation are common in patients with mitral valve (MV) disease. The importance of LA (volume/diameter) assessment by echocardiography should be emphasized in patients with MR[15-17]. In patients with organic MR who were in sinus rhythm, LAVI at the diagnosis of MR was related to survival and cardiac events (AF, CHF or cardiac death)[18]. Pulmonary hypertension due to left heart disease (including MR) associated with adverse prognosis[19]. Furthermore, in asymptomatic patients with chronic severe MR, pulmonary hypertension (pulmonary artery systolic pressure > 50 mmHg at rest or > 60 mmHg with exercise) is an indication for mitral valve surgery[15]. However, the authors do not know association between LAVI

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and pulmonary artery pressure in patients with chronic severe primary MR.

Material and Method

Study design

The present study was a retrospective study conducted in a large university medical center (Siriraj Hospital, Mahidol University, Bangkok, Thailand). The present study protocol was approved by Siriraj Ethics Committee.

Study population

The Siriraj echocardiographic database was searched for patients with severe MR during the period from January 2005 to December 2009. Severe MR was diagnosed by transthoracic echocardiography (TTE) based on either one or both criteria of proximal isovelocity surface area (PISA) method with effective regurgitant orifice area (ERO) $\geq$ 40 mm^2 or regurgitant volume (20) (RVol) $\geq$ 60 ml. The inclusion criteria were patients of age $\geq$ 15 years who had chronic severe MR. The patients were excluded if they had functional or ischemic MR, significant left-sided valvular stenosis or regurgitation other than isolated MR, congenital heart disease, prior cardiac surgery including valvular surgery, coronary artery bypass graft surgery or correction of congenital heart defect, prior percutaneous balloon valvulotomy, prior intra-cardiac device implantation (permanent pacemaker, implantable cardioverter-defibrillator, cardiac resynchronization therapy), pulmonary hypertension from causes other than MR, or incomplete echocardiographic data.

Clinical data

Medical history, results of investigation, electrocardiography and chest x-ray were comprehensively reviewed from both outpatient medical records and online medical record data. Age, gender, height, weight, blood pressure, heart rate, cardiac rhythm, symptoms, NYHA functional classification, history of comorbid conditions, history of cardiac surgery or intervention and serum creatinine were retrieved from medical history.

Echocardiographic data

Transesophageal echocardiographic (TEE) report, video record or electronic record (Xcelera® and EchoPAC®) were reviewed and analyzed. Echocardiographic data from TTE including LV dimension, wall thickness and ejection fraction (LVEF), LA diameter (M-mode, parasternal short axis view), LAV and LAVI by biplane area-length method, RVSP, etiology and severity of MR were collected. Pulmonary hypertension in patients with chronic severe MR was defined as RVSP > 50 mmHg[15]. LAV was measured by the biplane area-length method as previously described[20,21]. LA area (A) and length (L) were measured from apical 4-chamber (A1 and L1) and apical 2-chamber view (A2 and L2). LAV was calculated by the formula: (0.85 x A1 x A2)/L, when L was LA length as the average of L1 and L2. LAVI was equal to LAV divided by body surface area. Etiology of MR was based on data from TTE, TEE, or operative findings in patients who underwent MV surgery.

Statistical analysis

PASW version 18.0 was used for statistical analysis. Clinical and echocardiographic data were described using descriptive statistics, including means, standard deviation (SD), minimum, maximum, frequencies and percentage. The normality of distribution of the variables was examined with the Kolmogorov-Smirnov test. The Student's t-test and Mann-Whitney U test were used as appropriate to compare continuous variable differences between patients with sinus rhythm and AF. Comparison of categorical variables was performed using the Chi-square test or the Fisher exact test. Spearman's rank correlation coefficient and Pearson's correlation coefficient were used to measure the correlation between LAVI and RVSP. One-Way ANOVA and Gabriel multiple comparisons were used to compare 4 different quartiles of LAVI. Student's t-test was used to compare LAVI between patients with and without pulmonary hypertension. A p-value of $\leq$ 0.05 was considered statistically significant.

Results

According to Siriraj echocardiographic database, 570 patients were diagnosed with severe MR during January 2005 to December 2009 (Fig. 1) and 389 patients met the exclusion criteria (*e.g.* no LAVI data (47%), left-sided valvular heart disease other than isolated MR (25%), prior cardiac surgery or intervention (15.7%), functional or ischemic MR (6.2%), congenital heart disease (2%), equivocal severity of MR (1.8%)). TEE was performed in 68 patients (37.6%).

Baseline characteristics

The present study population consisted of 181 patients with chronic severe primary MR. Baseline clinical characteristics are shown in Table 1. The mean

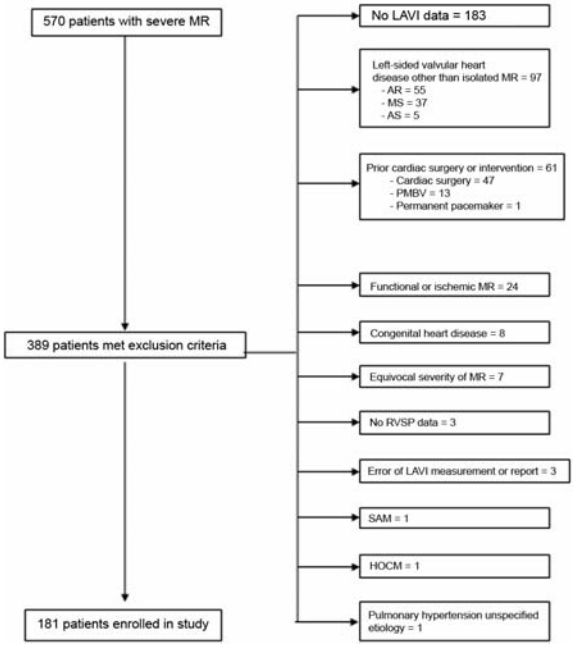

Fig. 1 Flow chart of patient enrollment, AS = aortic stenosis; AR = aortic regurgitation; HOCM = hypertrophic obstructive cardiomyopathy; LAVI = left atrial volume index; MR = mitral regurgitation; MS = mitral stenosis; PMBV = percutaneous mitral balloon valvotomy; RVSP = right ventricular systolic pressure; SAM = systolic anterior motion of mitral valve leaflet

age was 53.1 ± 17.7 years and 53.6% were male. Sinus rhythm was reported in 70.7% and AF in 29.3% of patients. Among patients with AF, 71.7% of patients had persistent or permanent AF. Patients were symptomatic in 52.5% and the majority was in NYHA class II. History of CHF, infective endocarditis and myocardial infarction were reported in 21.5%, 6% and 3.9% of patients, respectively. Etiologies of MR were ruptured chordae tendineae, mitral prolapse and rheumatic process in 53.6%, 33.7% and 7.2% of patients, respectively.

Patients were then classified into 2 groups according to cardiac rhythm; sinus rhythm in 128 (70.7%) and AF in 53 (29.3%) patients. Gender, body surface area, body mass index, heart rate and blood pressure were not significantly different between patients with sinus rhythm and AF. Patients with AF were significantly older, more symptomatic and more likely to have the history of stroke or transient ischemic attack, CHF and rheumatic MR than those in sinus rhythm (Table 1).

Echocardiographic data

Echocardiographic data were demonstrated in Table 2. Severe MR was quantitatively assessed by PISA method; ERO 81 ± 44 mm^2, RVol 124 ± 68 ml. Mean LVEF by modified Simpson's method was 71 ± 6%. LAV and LAVI were 134 ± 91 ml and 86 ± 65 ml/m^2, respectively. There were no statistically significant

Table 1. Baseline clinical characteristics of the entire patient population and groups stratified by cardiac rhythm

Variables	All patients (n = 181)	Sinus rhythm (n = 128)	Atrial fibrillation (n = 53)	p-value
Age (years)	53.1 ± 17.7	50.6 ± 18	59.0 ± 15.5	0.004
Male gender	97 (53.6)	72 (56.3)	25 (47.2)	0.27
Symptomatic MR	95 (52.5)	57 (44.5)	38 (71.7)	0.001
Medical history				
Dyslipidemia	54 (29.8)	39 (30.5)	15 (28.3)	0.77
Hypertension	46 (25.4)	35 (27.3)	11 (20.8)	0.35
Current smoking	11 (6.1)	7 (5.5)	4 (7.5)	0.73
Diabetes mellitus	10 (5.5)	6 (4.7)	4 (7.5)	0.48
Stroke or TIA	11 (6.0)	4 (3.1)	7 (13.2)	0.007
Heart failure	39 (21.5)	14 (10.9)	25 (47.2)	< 0.001
Etiology of MR				0.002
Ruptured chord	97 (53.6)	70 (54.7)	27 (50.9)	-
Prolapse valve	61 (33.7)	49 (38.3)	12 (22.6)	-
Rheumatic	13 (7.2)	5 (3.9)	8 (15.1)	-

Data are expressed as number (%) and mean ± standard deviation
MR = mitral regurgitation; TIA = transient ischemic attack

Table 2. Echocardiographic data of the entire patient population and groups stratified by cardiac rhythm

Variables	All patients (n= 181)	Sinus rhythm (n= 128)	Atrial fibrillation (n= 53)	p-value
ERO, mm^2	81 ± 44	84 ± 48	74 ± 31	0.52
RVol, mL/beat	124 ± 68	130 ± 76	110 ± 44	0.22
LA diameter, mm	53 ± 10	50 ± 9	61 ± 9	< 0.001
LA volume, mL	134 ± 91	108 ± 58	198 ± 123	< 0.001
LA volume index, mL/m^2	86 ± 65	68 ± 40	129 ± 90	< 0.001
RVSP, mmHg	46 ± 17	43 ± 15	53 ± 19	0.001
PAEDP, mmHg	15 ± 6	14 ± 5	20 ± 7	< 0.001
Moderate to severe TR	44 (24.3)	14 (10.9)	30 (56.6)	< 0.001
Moderate to severe PR	31 (17.1)	13 (10.2)	18 (34.0)	0.001

Data are expressed as number (%) and mean ± standard deviation
ERO = effective regurgitant orifice; LA = left atrium; PAEDP = pulmonary artery end-diastolic pressure; PR = pulmonic regurgitation; RVol = regurgitant volume; RVSP = right ventricular systolic pressure; TR = tricuspid regurgitation

difference in ERO, RVol, LVEF and LV dimension between patients with sinus rhythm and AF. Patients with AF had higher LA diameter, LAV, LAVI, pulmonary artery pressure, and were more frequently associated with significant tricuspid regurgitation and pulmonic regurgitation than patients in sinus rhythm.

Correlations between LA size and pulmonary artery pressure

There was a positive correlation and fair degree of relationship between LAVI and RVSP. RVSP tends to increase when LAVI increases (Spearman's correlation coefficient = 0.32, $p < 0.001$). Furthermore, LAVI was divided into 4 quartiles: ≤ 48.80 ml/m^2 (n = 44), 48.81-66.00 ml/m^2 (n = 46), 66.01-97.40 ml/m^2 (n = 46), > 97.40 ml/m^2 (n = 45). The mean RVSP in 4 quartiles were 41 ± 14, 42 ± 16, 44 ± 16 and 56 ± 18 mmHg, respectively. The differences of RVSP among 4 quartiles of LAVI were statistically significant as determined by One-Way ANOVA ($p < 0.001$). Multiple comparisons between each quartile of LAVI demonstrated RVSP of 4^{th} quartile was significantly higher than that of other 3 quartiles ($p = 0.004$) (Fig. 2). Regarding the prognostic and therapeutic implication of pulmonary hypertension in patients with chronic severe MR, patients were divided into 2 groups with and without pulmonary hypertension (n = 52 and 129, respectively). LAVI in patients with and without pulmonary hypertension were 116 ± 82 and 74 ± 53 ml/m^2, respectively. LAVI was significantly different between 2 groups of RVSP ($p = 0.001$) (Fig. 3).

When the patients were grouped according to cardiac rhythm, those with sinus rhythm tended to have a higher valve of RVSP when LAVI increased (Pearson's correlation coefficient = 0.27, $p = 0.002$). However, there was no significant correlation between LAVI and RVSP in patients with AF (Pearson's correlation coefficient = 0.06, $p = 0.68$).

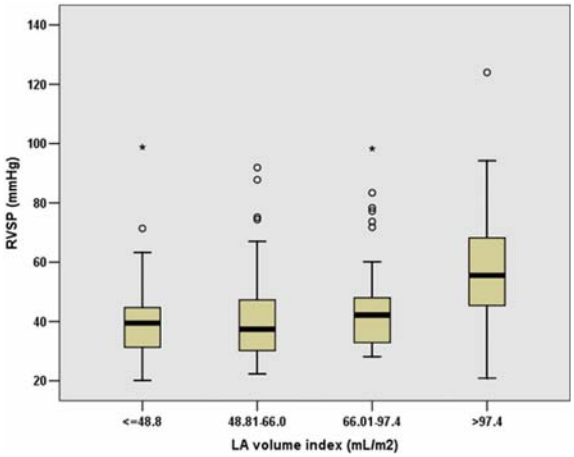

Fig. 2 RVSP and 4 quartiles of LA volume index in patients with chronic severe mitral regurgitation
LA = left atrium; RVSP = right ventricular systolic pressure

Discussion

In the present study of patients with chronic severe MR, the most common causes of MR were ruptured chordae tendineae and mitral valve prolapse. Most patients were symptomatic. The study showed that LAVI by biplane area-length method was minimally correlated with RVSP. Patients with higher LAVI (especially, those with LAVI > 97.40 ml/m^2) had

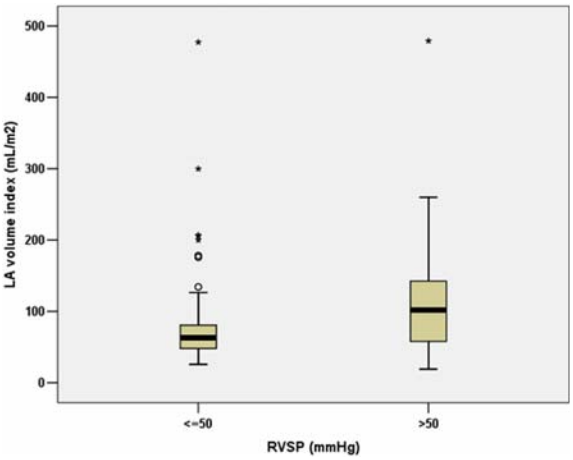

Fig. 3 LA volume index and the presence of pulmonary hypertension
LA = left atrium; RVSP = right ventricular systolic pressure

significantly higher RVSP than patients with lower LAVI. Patients with pulmonary hypertension had higher LAVI than those without. Regarding cardiac rhythm, LAVI was still significantly correlated with RVSP in patients with sinus rhythm. However, the correlation was not demonstrated in AF group.

LA dilatation is a consequence of volume or pressure overload to LA or loss of LA contraction such as AF. LAV is a marker of chronicity of LV diastolic dysfunction in patients without significant MV diseases and indicates adverse cardiovascular outcomes[2,8,9,12-14]. In patients with chronic MR, the degree of LAVI at the diagnosis of MR was correlated to future cardiovascular events[18] and further enlargement of LA was associated with the elevation of pulmonary artery pressure[18] and a new-onset AF[21]. Chronic MR leads to LA dilatation as the earlier stage of LA adaptation to chronic volume overload and the preservation of pulmonary artery pressure. In an advance stage of chronic volume overload of MR, AF develops as a consequence of progressive LA dilatation and further LA dilatation is inadequate to maintain normal pulmonary artery pressure. Therefore, pulmonary hypertension develops later on during the course of chronic MR.

The present study of patients with chronic severe MR demonstrated that LAVI correlated with RVSP when cardiac rhythm was sinus rhythm, but the correlation between LAVI and RVSP disappeared as AF developed. However, patients with AF had a significantly higher RVSP than patients in sinus rhythm. Therefore, the occurrence of AF in chronic MR represents an advance stage of disease process and the impairment of the compensatory mechanism of LA and indicates the timing for mitral valve surgery.

AF associates with adverse cardiovascular events both in patients with and without MR[7,9,12,18,19]. The presence of AF indicates the more advanced disease severity of MR, but the degree of LAVI in patients with AF was poorly correlated to the adverse cardiovascular events and pulmonary artery pressure. The present study confirmed that LAVI by biplane area-length method correlated with RVSP, especially in patients with sinus rhythm. While patients with AF had larger LAVI, higher pulmonary artery pressure and more advanced disease (such as more symptoms, CHF events or NYHA functional class) as compared to those with sinus rhythm, there was no statistically significant correlation between LAVI and RVSP in patients with chronic severe MR in AF rhythm. At present, the authors do not have a cut-off value for LAVI to indicate the optimal timing for MV surgery in patients with chronic severe MR. The additional prognostic roles of LAVI in patients with chronic severe MR will be further determined by larger prospective studies.

Limitations of the present study are due to the retrospective design, the mixed population between patients with sinus rhythm and AF, the small number of study population and the questionable accuracy and validity of the echocardiographic measurement of LAVI, using the biplane area-length method, in patients with chronic severe MR who usually have huge LA.

Conclusion

In chronic severe primary MR, RVSP tends to increase when LAVI increases.

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Potential conflicts of interest

None.

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ความสัมพันธ์ระหว่างปริมาตรหัวใจห้องบนซ้ายและความดันหลอดเลือดแดงปอดจากการตรวจคลื่นเสียงสะท้อนหัวใจในผู้ป่วยลิ้นหัวใจไมตรัลรั่วเรื้อรังชนิดรุนแรง

ยุทธชัย มาตระกูล, นิธิมา เชาวลิต

ภูมิหลัง: *การขยายตัวของหัวใจห้องบนซ้ายเป็นผลจากภาวะลิ้นหัวใจไมตรัลรั่ว ปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกายในผู้ป่วยลิ้นหัวใจไมตรัลรั่วมีความสัมพันธ์กับอัตราการเสียชีวิต ภาวะหัวใจเต้นผิดปรกติชนิด atrial fibrillation และภาวะหัวใจล้มเหลว*

วัตถุประสงค์: *เพื่อหาความสัมพันธ์ระหว่างปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกายและความดันหลอดเลือดแดงปอดจากการตรวจคลื่นเสียงสะท้อนหัวใจในผู้ป่วยลิ้นหัวใจไมตรัลรั่วเรื้อรังชนิดรุนแรง*

วัสดุและวิธีการ: *ศึกษาข้อมูลย้อนหลังในโรงพยาบาลศิริราช ระหว่างเดือนมกราคม พ.ศ. 2548 ถึง เดือนธันวาคม พ.ศ. 2552 ในผู้ป่วยลิ้นหัวใจไมตรัลรั่วเรื้อรังชนิดรุนแรงซึ่งวินิจฉัยด้วยวิธี proximal isovelocity surface area ได้แก่ effective regurgitant orifice area $\geq$ 40 mm^2 หรือ regurgitant volume $\geq$ 60 ml*

ผลการศึกษา: *ผู้ป่วยภาวะหัวใจไมตรัลรั่วเรื้อรังชนิดรุนแรง จำนวน 181 คน อายุเฉลี่ย 53.1 $\pm$ 17.7 ปี เป็นเพศชายร้อยละ 53.6 ความดันหัวใจห้องขวาล่างขณะหัวใจบีบตัวมีแนวโน้มสูงมากขึ้น เมื่อปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกาย เพิ่มมากขึ้น (r_s = 0.317, p < 0.001) เมื่อแบ่งผู้ป่วยเป็น 4 กลุ่มตามปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกายได้แก่ $\leq$ 48.80 มล./ตร.ม., 48.81-66.00 มล./ตร.ม., 66.01-97.40 มล./ตร.ม., > 97.40 มล./ตร.ม. ได้ค่าเฉลี่ยของความดันหัวใจห้องขวาล่าง ขณะหัวใจบีบตัวเรียงตามลำดับคือ 41 $\pm$ 14, 42 $\pm$ 16, 44 $\pm$ 16, 56 $\pm$ 18 มิลลิเมตรปรอท กลุ่มที่มีปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกายมากกว่า 97.40 มล./ตร.ม. มีความดันหัวใจห้องขวาล่างขณะหัวใจบีบตัวสูงมากกว่ากลุ่ม ที่มีปริมาตรต่ำกว่า 97.40 มล./ตร.ม. อย่างมีนัยสำคัญ (p = 0.004) ผู้ป่วยที่มีความดันหัวใจห้องขวาล่างขณะหัวใจบีบตัวมากกว่า 50 มิลลิเมตรปรอท มีค่าเฉลี่ยของปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกายสูงมากกว่ากลุ่มที่มีค่าต่ำกว่าหรือเท่ากับ 50 มิลลิเมตรปรอท อย่างมีนัยสำคัญ (74 $\pm$ 53 มล./ตร.ม. ต่อ 116 $\pm$ 82 มล./ตร.ม.; p = 0.001)*

สรุป: *ในผู้ป่วยลิ้นหัวใจไมตรัลรั่วเรื้อรังชนิดรุนแรง ค่าความดันหัวใจห้องขวาล่างขณะหัวใจบีบตัวมีแนวโน้มสูงมากขึ้น เมื่อปริมาตรหัวใจห้องบนซ้ายต่อพื้นที่ผิวกายเพิ่มมากขึ้น*