Evaluation of left ventricular mechanical dyssynchrony in patients with heart failure after myocardial infarction by real-time three-dimensional echocardiography

Qing Zhou, MD, Qing Deng, MD, Jia Huang, MM, Jin-Ling Chen, MD, Bo Hu, MD, Rui-Qiang Guo, MD.

ABSTRACT

الأهداف: تقييم مدى انتشار عدم تزامن البطين الأيسر لدى المرضى المصابين بقصور عضلة القلب بعد الاحتشاء وذلك باستخدام تخطيط صدى القلب ثلاثي الأبعاد ذو الوقت الفعلي، واختبار مدى فعالية طريقة التخطيط هذه في تحديد الاستراتيجية السريرية للعلاج.

الطريقة: أُجريت هذه الدراسة المقطعية في مستشفى رينمين التابع لجامعة ووهان، مقاطعة هيوبي، الصين وذلك خلال الفترة من يوليو 2009م إلى ديسمبر 2010م. لقد قمنا بإجراء تخطيط صدى القلب ثلاثي الأبعاد ذو الوقت الفعلي لحوالي 43 مريض مصاب بقصور عضلة القلب المزمن بعد الاحتشاء (مجموعة الدراسة)، و30 شخص سليم (مجموعة الشاهد) وذلك من أجل تحليل حجم البطين الأيسر، والاحتشاء القذفي لهذا البطين، بالإضافة إلى تحليل مؤشر عدم تزامن انقباض البطين الأيسر.

النتائج: لقد كان معدل عدم التزامن لدى مجموع المرضى المصابين باحتشاء عضلة القلب 83.7%، ووصل إلى 100% لدى مجموعة المرضى المصابين بالاختلال الوظيفي الحاد. وكان مؤشر عدم تزامن انقباض البطين الأيسر لدى مجموعة الدراسة أعلى بكثير من مجموعة الشاهد ($p<0.01$)، وكان هذا المؤشر بين المرضى الذين كان معدل الاحتشاء القذفي للبطين الأيسر لديهم أقل من أو يساوي 35% أعلى بكثير من هؤلاء الذين تراوح لديهم معدل الاحتشاء القذفي للبطين الأيسر بين 50%–35%. ولقد كان هنالك علاقة واضحة بين معدل الاحتشاء القذفي للبطين الأيسر ومؤشر عدم تزامن انقباض البطين الأيسر ($r= -0.84, p<0.001$) وذلك لدى المرضى المصابين باحتشاء عضلة القلب. ولوحظ مدى حدة عدم تزامن البطين الأيسر لدى الحالات المصابة باحتشاء عضلة القلب في الجزء الأمامي مقارنةً بمرضى الاحتشاء في الجزء الخلفي.

خاتمة: أظهرت الدراسة كثرة انتشار عدم تزامن البطين الأيسر لدى المرضى المصابين بالاختلال الوظيفي لعضلة القلب بعد حدوث الاحتشاء، كما أن ذلك قد كان مرتبطاً بمعدل انقباض البطين الأيسر. ولهذا يمكن اعتبار طريقة تخطيط صدى القلب ثلاثي الأبعاد ذو الوقت الفعلي فعالة أثناء تقييم عدم تزامن البطين الأيسر.

Objectives: To evaluate the incidence and prevalence of left ventricular (LV) dyssynchrony in patients with heart failure after myocardial infarction (MI) by real-time three-dimensional echocardiography (RT-3DE), and to investigate the clinical application value of using RT-3DE as a method of providing more detailed information for clinical strategy.

Methods: This cross-sectional study was carried out in Renmin Hospital of WuHan University, Hubei Province, China from July 2009 to December 2010. The RT-3DE was performed on 43 patients with chronic heart failure after MI and 30 normal subjects, to analyze LV volume and left ventricular ejection fraction (LVEF), also the LV systolic dyssynchrony index (SDI).

Results: The dyssynchrony rate of the total MI patients was 83.7%, and was 100% in the severe dysfunction group. The SDI of MI patients was significantly higher than the control subjects ($p<0.01$), and the SDI of those whose LVEF ≤35% is obviously higher than the patients whose LVEF ranges from 35-50%. The LVEF was well correlated with SDI ($r = -0.84$, $p<0.001$) in MI patients. The LV dyssynchrony prevalence was more severe in the anterior MI than the inferior MI.

Conclusion: The LV dyssynchrony occurred more often in patients with cardiac dysfunction after MI, and was well related with the LV systolic function. The RT-3DE could be considered as an effective and informative tool for the evaluation of LV dyssynchrony.

Saudi Med J 2012; Vol. 33 (3): 256-261

From the Department of Ultrasound Imaging, Renmin Hospital of WuHan University, Hubei Province, China.

Received 4th October 2011. Accepted 28th December 2011.

Address correspondence and reprint request to: Dr. Rui-Qiang Guo, Department of Ultrasound Imaging, Renmin Hospital of WuHan University, Hubei Province, China. Tel. +86 (189) 86175535. Fax. +86 (027) 88040334. E-mail: Guorq.wh.edu@hotmail.com

After myocardial infarction (MI), the left ventricle (LV) global contraction is asynchronous due to the partial reduction or even the loss of infarct myocardial contractility, which ultimately results in LV global remodeling and dysfunction.[1-3] Furthermore, MI occurring in different segments could bring unequal damage to the LV function and clinical prognosis. However, the incidence of the LV systolic dyssynchrony in patients with chronic heart dysfunction after MI lacks detailed information. Also, the severity of LV dyssynchrony in different parts of MI and its relation with LV systolic function still needs further research. Therefore, utilizing the real time three-dimensional echocardiography (RT-3DE) analysis, this study was designed to quantitatively evaluate LV dyssynchrony and its relation to variant left ventricular ejection fraction (LVEF), and detect the LV dyssynchrony in anterior or inferior infarction, ultimately to try to provide more valuable and detailed information for further assessment of clinical therapy and prognosis.

Methods. This study was carried out in the Department of Ultrasound Imaging, Renmin Hospital of WuHan University, Hubei Province, China from July 2009 to December 2010. The study population consisted of 43 patients with heart failure after MI for 0.5-4 years, and 30 cases of age, gender-matched subjects. The MI group included 43 cases of MI patients (32 males and 11 females, 30 cases of anterior MI, and 13 cases of inferior MI) whose age ranged from 43-79 years, with an average age of 57.8 ± 10.8. The diagnosis of MI was made based on typical ischemic chest pain, typical electrocardiographic changes, and the increase of myocardial enzymes. The inclusion criteria of the patients group were as follows: New York Heart Association (NYHA) cardiac function class II-IV and LVEF <50%; sinus rhythm without evident arrhythmias; endocardial border of 3D image was clear and complete; and only anterior or inferior MI. The exclusion criteria were: patients with multiple wall MI; patients with history and evidence of other cardiac abnormalities, such as pulmonary heart diseases, valvular diseases; patients with arrhythmia; and poor image quality, or unavailable image for analysis. All patients were divided into 2 groups according their LVEF: group A was severe LV systolic dysfunction consisted of those LVEF ≤35% (n=15), and group B was mild-moderate LV systolic dysfunction consisted of those whose LVEF ranges from 35-50% (n=28). The control group consisted of 30 healthy subjects who underwent routine physical examination in Renmin Hospital of WuHan University during the same period, age, gender, and hypertension incidence matched, without evidence of coronary heart disease, and excluded other structured cardiac diseases by echocardiography. The study was carried out according to the principles of Helsinki Declaration. All patients were given oral and written informed consent before participation, and the ethical committee of our hospital approved the study. All subjects were examined using an iE33 echo machine (Philips, Bothell, WA, USA) equipped with a X3-1 matrix array transducer (frequency 1-3 MHz). Echo exam was performed in subjects on the left lateral decubitus position with simultaneous electrocardiogram at rest. The 3DE data of 4 consecutive cardiac cycles were acquired from the apical 4 chamber view, and the image information was stored, and transferred to a separate workstation. The 3D ADV quantitative analysis software (Qlab, version 6.0, Philips Medical Systems, Bothell, WA, USA) was used for off-line analysis. First, 5 points were marked separately at the end-diastolic and end-systolic frame, subsequently the software semi-automatically detected the endocardial border of the LV according to the 5 points marked, afterwards the shape of LV cavity was created following a mathematical model. As defined by the American Society of Echocardiography,[4] the LV cavity was divided into 17 segmental sub-volumes for analysis. Once the 17 segmental time-volume curves were generated, the LV end systolic volume (ESV), end diastolic volume (EDV), and LVEF were obtained automatically. The standard deviation (SD) and the maximum difference of the 16 segments' time from the onset of QRS complex wave in ECG to the minimum systolic volume were calculated as the Tmsv16-SD and Tmsv16-Dif (excluding the apex cap). For convenient comparison among patients with different heart rate, the Tmsv16-SD and Tmsv16-Dif were corrected by the patient's cardiac cycle duration to acquire the Tmsv16-SD% and Tmsv16-Dif%. The Tmsv16-SD% was defined as the systolic dyssynchrony index (SDI).[5,6]

Data were analyzed using Statistical Package for Social Sciences version 13.0 for Windows (SPSS Inc, Chicago, IL, USA). Continuous variable was expressed as mean ± SD, and qualitative variables were expressed as number or percentage. Continuous variable of the 3 groups were analyzed with ANOVA test, and the SN-q test was used for further comparison between the 2

Disclosure. All authors have no conflict of interests, and the work was not supported or funded by any drug company.

Table 1 - Clinical characteristics and left ventricle dyssynchrony parameters of myocardial infract patients with varied LVEF (mean ± SD).

Characteristics	Control Group 1 (n=30)	35%<LVEF<50% Group 2 (n=28)	LVEF≤35% Group 3 (n=15)	*P*-value 3 groups	*P*-value (95% CI) Group 2 versus Group1		*P*-value (95% CI) Group 3 versus Group2	
Age, years	56.9 ± 10.0	57.2 ± 10.3	59.1 ± 11.9	0.584	0.697	(0.21-0.38)	0.326	(1.62-2.18)
Male	18	22	10	0.301	0.127		0.627	
Body surface area (m^2)	1.68 ± 0.13	1.79 ± 0.20	1.76 ± 0.17	0.252	0.167	(0.08-0.14)	0.958	(-0.02 - -0.04)
Hypertension	16	18	12	0.214	0.562		0.471	
Heart rate, beats per minute	68.1 ± 10.9	71.5 ± 10.4	74.2 ± 12.0	0.407	0.326	(-3.05-9.85)	0.458	(-2.57-7.97)
QRS duration, ms	87.2 ± 8.6	103.6 ± 18.7	115.7 ± 17.8	0.000	0.000	(10.50-22.50)	0.024	(2.46-21.73)
LVEDD, mm	46.5 ± 2.3	54.1 ± 3.1	60.8 ± 4.7	0.000	0.000	(4.44-10.76)	0.000	(3.25-10.15)
LVEDV, ml	75.5 ± 11.9	123.6 ± 29.8	196.6 ± 48.6	0.000	0.000	(31.76-65.31)	0.000	(51.14-92.68)
LVESV, ml	28.8 ± 5.8	71.3 ± 20.1	138.8 ± 41.3	0.000	0.000	(33.30-51.68)	0.000	(53.59-81.40)
LVEF, %	64.2 ± 4.2	42.9 ± 4.6	30.9 ± 6.8	0.000	0.000	(-24.30 - -18.30)	0.000	(-10.45 - -13.55)
Tmsv16-SD, ms	17.6 ± 7.1	50.3 ± 34.4	104.3 ± 28.3	0.000	0.000	(24.12-41.28)	0.000	(47.41-60.56)
Tmsv16-Dif, ms	38.8 ± 10.6	192.2 ± 106.6	298.4 ± 99.5	0.000	0.000	(95.92-210.88)	0.000	(63.47-148.93)
Tmsv16-SD%	2.0 ± 0.8	6.0 ± 4.1	12.9 ± 3.5	0.000	0.000	(2.29-5.71)	0.000	(5.01-8.76)
Tmsv16-Dif%	4.4 ± 1.2	22.9 ± 12.7	36.9 ± 12.3	0.000	0.000	(12.12-24.88)	0.000	(9.25-18.71)

LVEF - left ventricular ejection fraction, CI - confidential interval, LVEDD - left ventricular end-diastolic dimension, LVEDV - left ventricular end-diastolic volume, LVESV - left ventricular end-systolic volume, Tmsv - the time from the onset of QRS complex to the minimum systolic volume, SD - standard deviation, Dif - difference

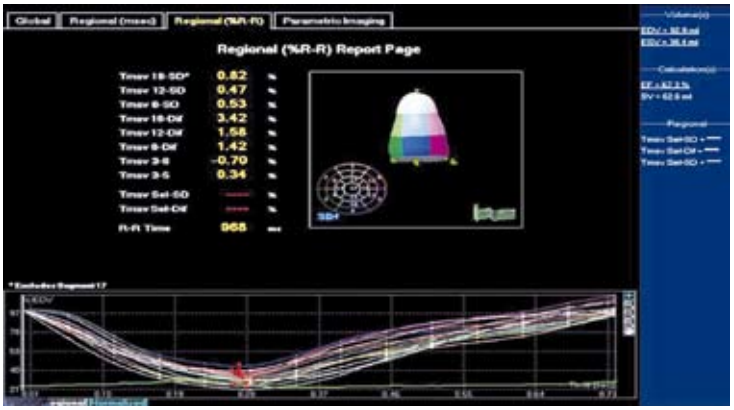

Figure 1 - The 17 segmental time-volume curves of a normal subject. The curve shapes are basically uniform, which indicates a low Tmsv16-SD% (SDI). R-R - R-R interval in electrocardiogram, EDV - left ventricular end-diastolic volume, ESV - left ventricular end-systolic volume, EF - left ventricular ejection fraction, SV - left ventricular stroke volume, Tmsv - the time from the onset of QRS complex to the minimum systolic volume, SD - standard deviation, Dif - difference

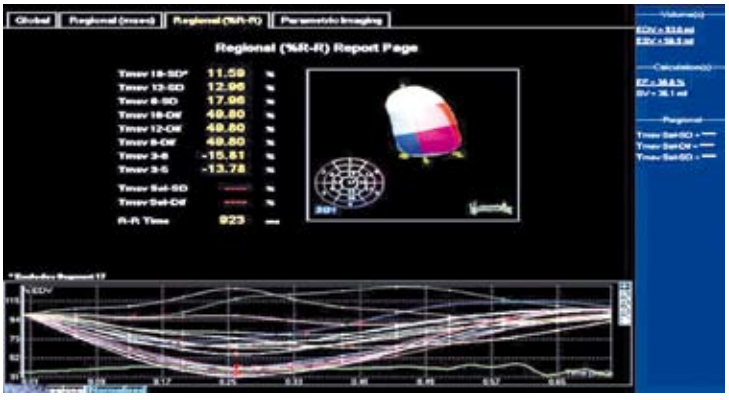

Figure 2 - The 17 segmental time-volume curves of a patient with myocardial infarction. The curve shapes are disorderly, which indicates a high Tmsv16-SD% (SDI). R-R - R-R interval in electrocardiogram, EDV - left ventricular end-diastolic volume, ESV - left ventricular end-systolic volume, EF - left ventricular ejection fraction, SV - left ventricular stroke volume, Tmsv - the time from the onset of QRS complex to the minimum systolic volume, SD - standard deviation, Dif - difference

Table 2 - Clinical characteristics and left ventricle dyssynchrony parameters of different parts of myocardial infraction (MI) (mean ± SD).

Characteristics	Control Group 1 (n=30)	Anterior MI Group 2 (n=30)	Inferior MI Group 3 (n=13)	*P*-value 3 groups	*P*-value (95% CI) Group 2 versus Group1	*P*-value (95% CI) Group 3 versus Group2
Age, years	56.9 ± 10.0	57.5 ± 10.5	58.2 ± 11.4	0.596	0.612 (0.41 - 0.79)	0.526 (0.50 - 0.90)
Male	18	23	9	0.378	0.267	0.894
Body surface area (m^2)	1.68 ± 0.13	1.78 ± 0.19	1.76 ± 0.17	0.252	0.199 (0.08 - 0.12)	0.973 (-0.01 - -0.03)
Hypertension	16	18	9	0.610	0.602	0.817
Heart rate, beats per minute	68.1 ± 10.9	74.7 ± 12.2	72.5 ± 10.2	0.454	0.266 (3.62 - 9.58)	0.696 (-1.17 - -3.23)
QRS duration, ms	87.2 ± 8.6	108.6 ± 16.3	101.3 ± 9.6	0.000	0.000 (13.40 - 29.40)	0.047 (-3.46 - -11.14)
LVEDD, mm	46.5 ± 2.3	58.4 ± 4.2	53.7 ± 3.4	0.000	0.000 (7.44 - 16.36)	0.000 (-2.42 - -7.00)
LVEDV, ml	75.5 ± 11.9	193.6 ± 40.1	108.9 ± 27.5	0.000	0.000 (87.52 - 157.68)	0.000 (-66.20 - -102.00)
LVESV, ml	28.8 ± 5.8	127.4 ± 22.7	64.9 ± 14.6	0.000	0.000 (78.45 - 119.92)	0.000 (-48.76 - -76.24)
LVEF, %	64.2 ± 4.2	34.2 ± 6.0	40.4 ± 4.7	0.000	0.000 (-21.58 - -38.42)	0.000 (3.97 - 8.43)
Tmsv16-SD, ms	17.6 ± 7.1	94.0 ± 26.4	62.1 ± 29.5	0.000	0.000 (58.14 - 94.66)	0.000 (-21.45 - -42.35)
Tmsv16-Dif, ms	38.8 ± 10.6	258.6 ± 95.6	176.2 ± 86.8	0.000	0.000 (199.86 - 239.74)	0.000 (-67.43 - -97.37)
Tmsv16-SD%	2.0 ± 0.8	11.7 ± 3.3	7.5 ± 3.6	0.000	0.000 (6.59 - 12.81)	0.000 (-2.36 - -6.01)
Tmsv16-Dif%	4.4 ± 1.2	32.2 ± 11.9	21.3 ± 10.5	0.000	0.000 (18.96 - 36.64)	0.000 (-6.79 - -15.01)

CI - confidential interval, LVEDD - left ventricular end diastolic dimension, LVEDV - left ventricular end diastolic volume, LVESV - left ventricular end systolic volume, LVEF - left ventricular ejection fraction, Tmsv - the time from the onset of QRS complex to the minimum systolic volume, SD - standard deviation, Dif - difference

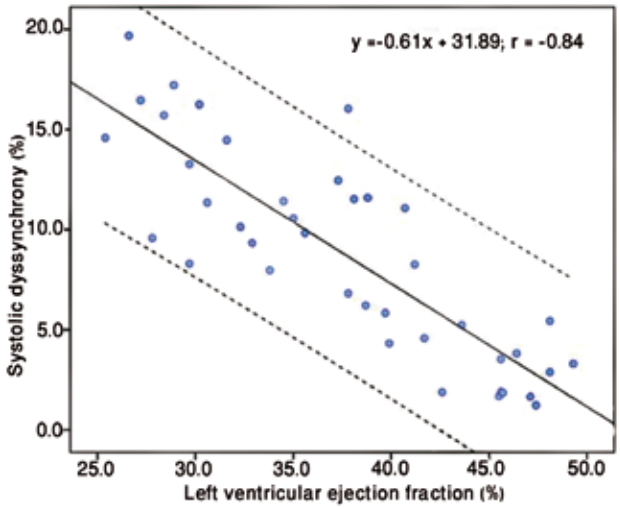

Figure 3 - The relation between SDI and LVEF in MI patients with heart failure. SDI = systolic dyssynchrony index, LVEF = left ventricular ejection fraction, --- shows the 95% confidence interval

groups. Fisher's exact test was computed to assess the differences of the dyssynchrony rate among groups. A $p<0.05$ was considered statistically significant.

Results. The RT-3DE was performed in all participants and reliable echocardiographic data were obtained. The age, gender, and heart rate between MI patients and control group were not different (**Table 1**). Compared with the control group, the EDV and ESV of MI patients were significantly enlarged, while LVEF was lower ($p<0.001$). Among MI patients, compared with inferior MI, EDV, and ESV of anterior MI patients were further increased, while LVEF was decreased statistically ($p<0.001$). Normally, the outline of 17 LV myocardial segments' time-volume curves were uniform, and 17 LV myocardial segments achieved the minimum volume almost at the same time in a cardiac cycle and the amplitude of each curve was very close (**Figure 1**). Whereas, the outline of 17 segment time-volume curves were in disorder in MI patients, and the reduction of motion amplitude were commonly presented, or even reverse movement occurred. The time point of each segment reaching the minimum systolic volume were varied, and were more severe in those whose LVEF <35% (**Table 1**, **Figure 2**). The Tmsv16-SD, Tmsv16-Dif, Tmsv16-SD%, Tmsv16-Dif% of MI patients were all obviously enhanced than controls ($p<0.01$), and were more severe in patients whose LVEF<35% compared with the patients whose LVEF ranged from 35-50% (SDI 12.9 ± 3.5 versus 6.0 ± 4.1%, $p<0.01$). The SDI of anterior MI patients was much higher than that of inferior patients (11.7 ± 3.3 versus 7.5 ± 3.6%, $p<0.01$) (**Table 2**). According to the statistical principle of setting normal reference range, the cut-off value of dyssynchrony was defined as the mean SDI +2.33 SD of the normal population, which means SDI>3.86% was defined as systolic dyssynchrony in this study. Among all the MI patients with heart failure, the dyssynchrony rate was 83.7% (36/43), and was 75% in LVEF ranged from 35-50% group (21/28), and 100% in LVEF≤35% group (15/15) ($p=0.037$). The LV dyssynchrony occurrence

in anterior MI patients was higher than that of the inferior MI (93% versus 61%, p=0.019). A significant correlation was identified between SDI and LVEF (r = -0.84, p<0.001, **Figure 3**) in MI patients with heart failure. However, there was no significant correlation between the SDI and LVEF in normal controls because the LVEFs were clustered in a narrow range.

Discussion. The LV systolic function failure is a severe complication after MI both in developing and developed countries. As for clinical therapy, accurate and detailed assessment of LV remodeling and systolic dyssynchrony occurrence is of great implication for guiding clinical treatment and more accurate evaluation of prognosis. The left ventricular dyssynchrony included myocardial mechanical motion dyssynchrony and electrical dyssynchrony, and the mechanical motion dyssynchrony was commonly accepted as a direct parameter to reflect the LV systolic dyssynchrony.[6,7] Mollema et al[8] identified that dyssynchronized contraction would result in LV enlargement, and reduction of contractive function within a relatively short period after MI. A recent research from Ng et al[9] indicated that if LV systolic dyssynchrony existed, the left ventricular systolic function of most MI patients would have more significant reduction even in those non-transmural MI patients without cardiac dysfunction. Therefore, the detection of the existence of LV dyssynchrony would provide valuable information for predicting clinical prognosis after MI.

This study indicated that the occurrence of LV dyssynchrony was 83.7% among the patients with chronic heart failure caused by MI, and the loss of LV synchronous contraction (expressed as SDI) was significantly related to the impaired LV systolic function (expressed as LVEF), which was in line with previous observations.[10] The reason would be considered as that infarction resulting in regional segment contraction weakened or even loss, while the non-infarcted segments contracted normally or even compensatively enhanced, ultimately resulted in global LV dyssynchrony.[11] On one hand, LV dyssynchrony might cause LV systolic dysfunction, which was a predictor for poor prognosis in patients with chronic heart failure as it resulted in disordered hemodynamics and inefficient circulation in the LV. This would increase the myocardial oxygen consumption and undermine the residual viable myocardium, which ultimately impair LV systolic function.[12] On the other hand, the impairment of the residual viable myocardium, and the LV remodeling after MI would aggravate the LV systolic dyssynchrony in turn.[13-15] Thus, a vicious circle that progressively harmed the LV systolic function has been formed. As the anterior and inferior MI were most common, it was necessary to discuss the LV dyssynchrony in these 2 parts of MI. This study showed that compared with inferior MI, the incidence and severity of LV dyssynchrony in anterior MI was more serious for the reason that the extent of abnormal contractive segments was wider, ultimately resulted in the increased variation of the dispersion of 17 segment time-volume curve. Besides that, the contribution of anterior wall to the overall LV function was greater than the inferior wall, therefore, the global LVEF of patients with anterior MI was reduced more than that of the inferior MI. These 2 factors led to a worse LV dyssynchrony of anterior MI, which was supported by the study of Zhang et al[16] as well.

In recent years, the main technique for LV mechanical dyssynchrony evaluation was tissue Doppler imaging (TDI). As known, its main limitation, angle-dependency is an obstacle for precise analyses of the 4 apical segmental motion that resulted in inaccurate assessment of LV global dyssynchrony, however, the apex had an essential impact on LV global contractive synchrony in anterior MI. Besides, TDI is unavailable to detect the movement of all LV segments in one single cardiac cycle, thus, it was necessary to develop a more efficiency technique to evaluate LV dyssynchrony.[17,18] To overcome the weak points above, RT-3DE offers a complete assessment of the entire LV in the same cardiac cycle, and comprehensive evaluation of the movement of all LV segments in different direction without angle-dependency. Furthermore, RT-3DE can simultaneously calculate the LV function parameters such as EDV, ESV, and LVEF which are not influenced by the deformation of LV after MI, and are more accurate than the data produced from the 2D Simpson's method.[19-21]

The current study has the following limitations: a relative small number of normal subjects in this study may prohibit the definition of a cut-off value for dyssynchrony, so results should be interpreted with caution, and further study in a large number of subjects are warranted; and there is still a lack of a widely accepted standard to assess the LV systolic dyssynchrony at present, which makes it incapable to compare RT-3DE data of our study with the "standard".

In conclusion, the incidence of LV dyssynchrony is relatively high in patients with chronic heart failure caused by MI, especially in anterior MI, and those with a more severe dysfunction. The SDI derived from RT-3DE could precisely assess LV mechanical dyssynchrony and systolic function within one single heartbeat, and be more helpful to provide effective and detailed information for determining clinical treatment strategies.

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