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Editorial

Left Atrial Dyssynchrony in Dilated Cardiomyopathy: Diastolic Dysfunction Matters but Left Bundle Branch Block Does Not

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▸ See the article "Impact of Left Bundle Branch Block on Left Atrial Dyssynchrony and Its Relationship to Left Ventricular Diastolic Function in Patients with Heart Failure and Dilated Cardiomyopathy" in volume 1 on page 42.

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Left bundle branch block (LBBB) is known to have a negative impact on left ventricular (LV) function.

The mechanism of systolic dysfunction in LBBB is incoordinated ventricular contraction. Primary effect of LBBB on mechanical dyssynchrony is preejection septal contraction accompanied with passive prestretch in not-yet-activated lateral wall. Late activated lateral wall pushes the blood back against the septal wall and causes septal rebound stretch.[1] Due to early activation, septum terminates contraction prematurely, whereas the lateral wall still continues contraction.[2] There is also post-systolic shortening in septum due to recoil or imbalance of contraction force during isovolumic relaxation time.

Intraventricular dyssynchrony has been widely studied in patients with heart failure (HF) with reduced ejection fraction (HFrEF) to predict the effect of cardiac resynchronization therapy (CRT). Since the main mechanism of CRT is the correction of intraventricular dyssynchrony, various intraventricular dyssynchrony indices based on tissue velocity or strain imaging were proposed to be predictive for the effect of CRT,[3][4] although recent guidelines do not recommend to select the candidate for CRT based on mechanical dyssynchrony.

Similarly to the intra-LV dyssynchrony, intra-left atrial (LA) mechanical dyssynchrony has also been validated its clinical utility. Previous studies demonstrated that LA dyssynchrony predicted recurrence in patients after catheter ablation of atrial fibrillation,[5-9] occurrence of atrial fibrillation in HF,[10] and associated with the history of ischemic stroke in atrial fibrillation.[11] Thus far, the usefulness of LA dyssynchrony has been mainly studied in the population of paroxysmal atrial fibrillation and/or HF with preserved ejection fraction (HFpEF). However, knowledge about LA dyssynchrony in HFrEF is scarce and the link between intraventricular conduction delay and LA dyssynchrony has not been elucidated.

In this issue of *International Journal of Heart Failure*, Park et al.[12] investigate the impact of LBBB on LA mechanical dyssynchrony in 80 patients with non-ischemic dilated cardiomyopathy (DCM). LA dyssynchrony was measured as the standard deviation (SD) of time intervals from P wave onset to peak atrial velocity by tissue Doppler imaging among 4 LA walls.

They also measured LV systolic dyssynchrony and diastolic dyssynchrony as the SD of time to peak systolic velocity and early diastolic velocity. They compared these dyssynchrony indices between in patients with LBBB and without LBBB. Furthermore, they examined the relationship between dyssynchrony indices and echocardiographic parameters of systolic/diastolic function, then investigated the determinant of LA mechanical dyssynchrony.

Authors found that LA dyssynchrony did not differ in patients with LBBB compared to without LBBB (20.9±11.5 ms vs. 22.5±14.5 ms, p=0.60), although there was a significant difference in LV systolic (68.2±38.2 ms vs. 48.5±32.0 ms, p=0.014) and diastolic dyssynchrony (59.2±36.5 ms vs. 45.3±24.1 ms, p=0.045) between in LBBB and no LBBB patients. Interestingly, the difference in diastolic dyssynchrony between groups was not as apparent as in systolic dyssynchrony. LA dyssynchrony index did not show significant relationship either with LV systolic dyssynchrony (p=0.465) or diastolic dyssynchrony (p=0.499). The factors significantly related to LA mechanical dyssynchrony were LV diastolic function parameters, such as LA volume (r=0.242, p=0.031), deceleration time of mitral E velocity (r=−0.249, p=0.029), and E/e′. (r=0.344, p=0.002). E/e′ showed the strongest correlation with LA dyssynchrony among these diastolic parameters.

The findings reported by Park et al.[12] provide deep insight about the mechanism of LA dyssynchrony. Determinant of LA mechanical dyssynchrony includes both LA electrical conduction delay and LA remodeling. Lack of link between LBBB and LA dyssynchrony in present study is reasonable because LBBB alters neither LA electrical conduction nor the timing of LA active contraction directly.

Dyssynchronous septal and lateral shortening and shuffle motion during systole in LBBB may alter the segmental timing of LA stretching during reservoir phase through decent of the LV base, because LA and LV are connected each other at mitral annulus. However, in present study, Park et al.[12] measured dyssynchrony based on the timing of LA active contraction and not of reservoir phase. Therefore, it is presumed that the passive effect of LV dyssynchronous contraction on LA dyssynchrony is limited, as Park et al.[12] showed no significant correlation between LA dyssynchrony and LV systolic dyssynchrony in this article.

Dyssynchrony in LBBB affects not only LV contraction but also relaxation phase. Abnormal septal-lateral interaction during systole prolongs LV pressure decay and reduces rate of pressure fall during early diastole.[13] This leads to prolongation of the isovolumic relaxation time and therefore causes shortening of filling time.[14] One study suggest that LBBB patients show higher LV end-diastolic pressure and more severe diastolic function than patients with normal conduction,[15] but this is not the case in present study considering similar E/e′ in both groups.[12] Consequence of LBBB on diastolic function is not as direct as on early systole and can be modified by other factors such as loading condition. So far, there is no evidence that LBBB directly modifies LA active contraction. Lack of relationship between LV diastolic dyssynchrony and LA dyssynchrony in present study confirms that impaired relaxation itself due to LBBB does not alter LA dyssynchrony.[12]

Park et al.[12] illustrates that LV diastolic dysfunction and LA remodeling is more important on the severity of LA dyssynchrony in DCM patients rather than the presence of LBBB. LA afterload is determined by downstream pressure and increases with more severe diastolic dysfunction and elevated filling pressure. Continuous pressure overload leads to LA remodeling with progressive interstitial fibrosis. LA dilatation is an apparent manifestation

of LA remodeling.[16] Regional fibrosis cause the heterogeneity of LA compliance and lead to the local conduction delay in LA.[17] These mechanical and electrical heterogeneity can be a source of intra-atrial mechanical dyssynchrony.

Before applying intra-LA dyssynchrony into clinical practice, one thing to note is that there are various definitions intra-LA dyssynchrony depending on literatures. They are divided into 2 types roughly: one is dyssynchrony in atrial contraction phase and the other is in reservoir phase. Intra-LA dyssynchrony in atrial contraction phase, as Park et al.[12] reported in this article, is based on timing measurement from P wave to tissue velocity peak (or onset of) A wave,[5][6] or time to atrial contraction in strain imaging,[7][8] Intra-LA dyssynchrony in reservoir phase is calculated as a dispersion of time from R wave to peak maximum LA strain derived from speckle tracking echocardiography[10] or MRI tissue tracking.[9][11] Atrial contraction is expected to be more dependent on intra-atrial electrical conduction delay than reservoir timing is, and reservoir function depends more on LA compliance and LV longitudinal contraction than atrial contraction does. LA remodeling is related to both atrial contraction and reservoir function to some extent. Therefore, dyssynchrony indices in both phase are interrelated each other and both parameters seem to be useful in similar patient population. Which one should be used is dependent on the clinical question and technical feasibility. It is important not to lump different dyssynchrony indices all together when interpreting previous literatures.

Considering that LA dyssynchrony is closely related to LA remodeling, it is plausible that LA dyssynchrony can be utilized for the management of atrial fibrillation.[5-11] Next step should be if we can use LA dyssynchrony as a target of therapy in patient with HF. Preliminary data suggest that atrial resynchronization therapy by dual-site right atrial or LA pacing is effective to maintain sinus rhythm in HF with atrial fibrillation,[18] or showed preferable acute hemodynamic effect in HFpEF patients with considerable atrial electromechanical delay.[19] Pacemaker does not reverse myopathic process directly, whereas it can reduce electrical dyssynchrony. It is important to sort out if dyssynchrony is a result or a cause of the disease and if mechanical dyssynchrony can be treated electrically or not, as we learned from rethinQ trial in which CRT failed to improve HF patients with narrow QRS with significant LV mechanical dyssynchrony.[20]

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