Left atrial and left atrial appendage systolic function in patients with post-myocardial distal blocks

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Abstract

Introduction: The study aimed to evaluate function of the left atrium (LA) and of the left atrial appendage (LAA) after myocardial infarction (MI) complicated by intracardiac conduction disturbances.
Material and methods: The study comprised 59 patients with persistent post-myocardial distal blocks, who were allocated to one of the three following subgroups: study group I – 20 patients with left bundle branch block (LBBB); study group II – 20 patients with right bundle branch block (RBBB), and study group III –19 pts with left anterior hemiblock (LAHB). The control groups included patients with MI in their history and no BBBs (19 pts – group IV) and clinically healthy people (16 patients – group V). The parameters of LA and LAA systolic function were determined by means of transthoracic (TTE) and transoesophageal echocardiography (TOE).
Results: We showed that patients who experienced myocardial infarction not complicated with conduction disturbances expressed compensatory LA systolic function enhancement. In patients with post-myocardial RBBB and LAHB significant enhancement of LA systolic function was observed as well but it was expressed to a lesser degree. There was also a tendency towards deterioration of LA systolic function in patients with post-myocardial LBBB. LBBB did not affect LAA systolic function negatively.
Conclusions: Parameters of LAA systolic function showed its enhancement in all patients after myocardial infarction irrespective of whether it was complicated by conduction disturbances.

Key words: left atrium, left atrial appendage, echocardiography, distal blocks

Introduction

The cardiac conduction system is responsible for generation and conduction of synchronized bioelectric stimuli in the heart, which results in coordinated physiological and effective heart work. Defects of this system affect and deteriorate left and right ventricular as well as atrial function [1-3].

Bundle branch blocks (BBBs) are known to be associated with higher mortality in patients with acute myocardial infarction (MI) and with congestive heart failure (CHF) [4-8]. There are limited data on the influence of BBBs on prognosis in ischaemic heart disease. BBBs are the cause of

left and right ventricular systolic and diastolic function deterioration [9, 10]. Little is known about left atrial function and about left atrial appendage function in patients with post-myocardial BBBs.

It is known that left atrial (LA) systolic function is enhanced in hypertension [11], in CHF [12, 13], and post MI [14]. LA and left atrial appendage (LAA) function may be a useful indicator of the degree of left ventricular (LV) impairment and may determine the prognosis [15-22].

Poor physical efficiency, impaired LV function and intraventricular conduction prolongation are classical, well established indications for cardiac resynchronised therapy (CRT) [23]. But better, more efficient criteria for CRT are still being sought.

The degree of LA and LAA function impairment might become in future useful clues to qualify patients with CHF for CRT.

As we know there have been no clinical studies on the mutual interaction between BBBs and LA and LAA function. That is why we assessed and compared LA and LAA systolic function in patients with post-myocardial left bundle branch block (LBBB), right bundle branch block (RBBB) and left anterior hemiblock (LAHB), and compared the data to those of patients after MI with no conduction disturbances as well as to healthy subjects.

Material and methods

Study population

The study group comprised 59 patients with persistent post-myocardial distal blocks. They were allocated to one of the three following subgroups: study group I – 20 patients with LBBB; study group II – 20 patients with RBBB; study group III – 19 patients with LAHB. Control groups consisted of patients with MI in their history and no intracardiac conduction disturbances (19 patients – group IV) and clinically healthy people (16 patients – group V).

Patients with BBBs other than post-myocardial aetiology were not entered into the study. The patients were included only if they had had ECG records done within 6 months before MI and had no BBBs. That made it highly probable that conduction disturbances were the effects of MI. Patients with congenital and significant valvular anomalies, with heart rhythm disturbances, with acute and chronic pulmonary disease, with anaemia, with hypo- and hyperthyreosis and with neoplastic diseases were excluded from the study. Patients with echocardiographically documented ventricular hypertrophy were not admitted to the study either.

The study protocol was approved by the Ethics Committee of the Medical University of Lodz, Poland. All patients signed informed consent prior to inclusion in the study.

Echocardiography

All enrolled patients underwent transthoracic and transoesophageal echocardiography. Investigations were performed with a 1.7-3.5 MHz transducer (Acuson) connected to an Acuson Sequoia 512 and with a multiplane transducer 5.5 MHz connected to the same echocardiography system. Each parameter was calculated twice and the averaged values were analysed.

Transthoracic echocardiography

The routine parasternal long and short axis views as well as four-chamber and two-chamber views were used to measure the parameters. Simultaneously to echocardiography one ECG lead record was performed.

M-mode echocardiography (left atrial parameters)

From M-mode in parasternal long axis view the following parameters of the left atrium were measured: (1) LA maximum dimension ($LA_{max.}$), measured just after the T wave, (2) LA minimum dimension ($LA_{min.}$), measured just before the QRS complex, (3) LA pre-systolic dimension (LA_a), measured at the peak of the *P* wave of the simultaneously recorded ECG [24].

On the basis of these three parameters the following four LA indexes describing its haemodynamic function were calculated [24, 25].

Active emptying fraction of LA (FAE LA):

$$\text{FAE LA} = \frac{LA_a - LA_{min.}}{LA_a}$$

Passive emptying fraction of LA (FPE LA):

$$\text{FPE LA} = \frac{LA_{max.} - LA_a}{LA_{max.}}$$

Total emptying fraction of LA (FT LA):

$$\text{FTE LA} = \frac{LA_{max.} - LA_{min.}}{LA_{max.}}$$

Echocardiographic parameters of LA:

2D LA parameters: $LA_{infero\text{-}posterior}$, $LA_{medio\text{-}lateral}$ – dimensions measured in four-chamber apical view at the end of the LA systole.

Multiplying the latter two dimensions by each other ($LA_{infero\text{-}posterior} \times LA_{medio\text{-}lateral}$) LA area was calculated (LA_{area}).

Doppler parameters of LA function:

From trans-mitral flow the following parameters were determined by means of pulsed Doppler method:

PEP LA – pre-ejection period of LA – time interval measured from the beginning of the P-wave in

simultaneously recorded ECG to the beginning of the A (atrial) wave in pulsed Doppler spectrum power of transmitral flow.

ET LA ejection time of LA – time interval between the beginning and the end of the A (atrial) wave in pulsed Doppler spectrum power of transmitral flow.

PEP/ET LA index – calculated as PEP LA/ET LA [25-26].

Pulmonary venous flow parameters

The flow in the upper right pulmonary vein was analysed. The pulsed Doppler sample volume was placed 0.5 to 1.0 cm in the orifice of the upper right pulmonary vein into the left atrium. The vein was visualized by a slightly cephalic elevation of the interrogation plane from a standard four-chamber view. We analysed peak velocity of retrograde (PVR) (atrial) pulmonary venous flow [29, 30] as the parameter describing LA systolic function.

Transthoracic echocardiography 2D left ventricular parameters: LVEDd – left ventricular end-diastolic volume, LVESd – left ventricular end-systolic volume, LVEF – left ventricular ejection fraction, measured according to Simpson's formula.

Transthoracic Doppler echocardiography of mitral flow

Mitral flow was recorded between the mitral leaflets in the four-chamber view. The following parameters were analysed from the velocity tracings: E – early mitral flow peak velocity, A – atrial flow peak velocity, DT – deceleration time of E wave, E/A – early mitral/atrial flow peak velocities index [31], IVRT – isovolumic relaxation time of left ventricle.

Transoesophageal echocardiography left atrial appendage parameters

The following LAA parameters were analysed:

– LAA transversal dimension ($LAA_{trans.}$) i.e.
– LAA longitudinal dimension ($LAA_{long.}$) i.e.
– LAA maximal ($LAA_{area\ max.}$) i.e. diastolic area – measured at the onset of P wave of ECG

LAA minimal ($LAA_{area\ min.}$) i.e. systolic area – measured just after R wave of QRS complex of ECG [32].

The LAA ejection fraction (EF_{LAA}) was calculated as maximal area minus minimal area divided by the maximal area:

$$EF_{LAA} = \frac{LAA_{\ max.\ area} - LAA_{\ min.\ area}}{LAA_{\ max.\ area}}$$

The LAA blood flow velocity was obtained by placing the pulsed Doppler sample volume into the outlet of the appendage blood cavity > 1 cm away from the left atrial cavity. The peak LAA emptying (LAAE) and the peak LAA filling (LAAF) velocities were recorded as described previously [33-35]. LAAE [m/s]-left atrial appendage (LAA) peak emptying velocity was obtained in the left atrial appendage long-axis view with the sample volume placed 0.5-1.0 cm within the appendage outlet.

LAAF [m/s]-LAA peak filling velocity – measured in the same way as LAAE.

Statistical analysis

Data are reported as mean ± SD. A p-value < 0.05 was considered significant. The following tests for statistical significance were used: (1) for dependent variables: Student's t-test if the distribution was normal, Wilcoxon's test if the distribution of at least one of the characters differed from normal, (2) for independent variables: Student's t-test if the variances were equal and the distribution of the character was normal, Cochran's test if the variances were not equal and the distribution of the character was normal, Mann-Whitney test or Kolmogorov-Smirnov test if the distribution of at least one of the characters differed from normal. The Kolmogorov-Smirnov test was used to assess whether the distribution of the analysed character was normal.

Results

Study group I – comprised 13 women and 7 men aged from 53 to 77 years (mean 67.6 ±9.96). Study group II: 9 women and 11 men aged from 52 to 85 years (mean 71.4 ±8.88). Study group III: 4 women and 15 men aged from 54 to 79 years (mean 68.95 ±9.24). Every patient experienced MI at least 6 weeks before inclusion in the study and had the proper level of myocardial necrosis markers (creatine kinase-MB fraction [CK-MB] and troponins) at the time of inclusion. In group I 15 patients had anterior, 3 infero-lateral MI. In group II 12 patients had anterior; 2 antero-lateral; 4 lateral; 2 inferior MI. In group III 8 patients had anterior; 9 inferior and 3 lateral MI. All patients had sinus rhythm in ECG Holter monitoring. Sinus rhythm was necessary to be included in the study.

Forty-two patients had hyperlipidaemia, 36 hypertension, 27 were addicted smokers, 22 had positive family history, 3 diabetes mellitus (DM) type 2 and 4 obesity. A precise description of the risk factors in particular groups is presented in Table I.

Groups IV and V constituted control groups. Patients of group IV (16) experienced MI at least 6 weeks before inclusion in the study and had no His-Purkinje system disturbances. All the individuals had q-wave MI; 7 anterior; 5 inferior; 3 lateral; 1 apical MI. There were 10 men and 6 women in the group aged from 45 to 96 years (mean 63.94 ±12.75). Group V comprised 14 clinically healthy people: 8 men and 6 women, aged from 24 to 69

Table I. Clinical characteristics of study groups

	Group I	Group II	Group III	Group IV	Group V	All
Dyslipidaemia	12	11	5	11	3	42
Hypertension	13	10	6	7	0	36
Smoking	8	9	4	5	1	27
Positive family history	7	8	4	2	1	22
Obesity	1	1	2	0	0	4
Diabetes mellitus	1	1	0	1	0	3

(medium 43.79 ±12.23). They had no significant changes in the coronary arteries.

LAmax., LAmin., and LAa were significantly higher in groups I-IV than in group V. But they did not differ significantly between groups I-IV. There were no significant differences as to FAE LA and FPE LA between groups I-V. The values of FTE LA in groups II and III were significantly higher than in group V. All the results are presented in Tables II-IV.

E was significantly higher in group I than in groups II, III, IV, did not differ from group V, while the values in groups II, III, IV were significantly lower than in group V. A was significantly higher in groups II, III, IV than in group V. A in group I did not significantly differ from other groups. E/A ratio was significantly lower in groups II and IV than in group V. DT was significantly shorter in groups II and III than in group IV, and significantly longer in group IV than in group V.

PEP LA was significantly longer in group I than in groups III and IV while it was significantly shorter in groups III and IV than in group V. ET LA was significantly shorter in group IV and it was significantly longer in groups III and IV than in group V. In group II ET was significantly longer than in group V, significantly shorter than in group IV, and did not differ from the other groups. PEP/ET LA index was significantly higher in group I than in groups III and IV, and in group II as compared with IV. It was significantly lower in groups II, III, IV than in group V.

PVR was significantly higher in groups I, II, III, IV than in group V.

Parameters of LAA systolic function

$LAA_{trans.}$ was significantly higher in group I than in group V. $LAA_{long.}$ was significantly higher in group I than in groups II, III, V and it was significantly

Table II. Echocardiographic parameters of left atrium

	Group I	Group II	Group III	Group IV	Group V
$LA_{max.}$ [cm]	3.85 ±0.64^{V}	3.74 ±0.44^{V}	3.76 ±0.42^{V}	3.77 ±0.44^{V}	3.13 ±0.40
$LA_{min.}$ [cm]	2.69 ±0.78^{V}	2.65 ±0.44^{V}	2.70 ±0.58^{V}	2.50 ±0.62^{V}	2.05 ±0.31
LA_{a} [cm]	3.25 ±0.72^{V}	3.12 ±0.49^{V}	3.26 ±0.47^{V}	3.06 ±0.52^{V}	2.54 ±0.32
FAE	0.18 ±0.11	0.17 ±0.09	0.15 ±0.08	0.19 ±0.09	0.19 ±0.07
FPE	0.16 ±0.08	0.17 ±0.08	0.14 ±0.05$^{IV. V}$	0.19 ±0.09	0.19 ±0.05
FTE	0.31 ±0.11	0.29 ±0.09^{V}	0.28 ±0.10^{V}	0.35 ±0.10	0.35 ±0.06
E [m/s]	0.82 ±0.26$^{II .III. IV}$	0.62 ±0.20^{V}	0.65 ±0.17^{V}	0.66 ±0.17^{V}	0.80 ±0.12
A [m/s]	0.76 ±0.32	0.78 ±0.22^{V}	0.80 ±0.27^{V}	0.77 ±0.26^{V}	0.57 ±0.14
E/A	1.28 ±0.75	0.90 ±0.49^{V}	1.03 ±0.90	0.93 ±0.35^{V}	1.45 ±0.33
DT [ms]	177.75 ± 78.54	157.52 ± 69.67^{IV}	159.53 ±86.93^{IV}	221.69 ±74.31^{V}	162.07 ±12.48
IVRT [ms]	103.35 ± 39.51	98.30 ± 29.96	107.47 ±34.51	101.13 ±27.92	90.21 ±16.08
PEPLA [ms]	88.40 ± 46.97$^{III. IV}$	71.60 ± 28.49	62.88 ±14.54^{V}	62.20 ±15.09^{V}	76.57 ±12.04
ET [ms]	145.90 ±49.57^{IV}	154.35 ±33.69$^{IV. V}$	173.32 ±67.69^{V}	189.94 ±48.50^{V}	128.43 ±15.93
PEP/ET	0.66 ±0.44$^{III. IV}$	0.48 ±0.19$^{IV. V}$	0.41 ±0.18^{V}	0.35 ±0.12^{V}	0.60 ±0.11
PVR [m/s]	0.33 ±0.14^{V}	0.35 ±0.13^{V}	0.35 ±0.11^{V}	0.31 ±0.09^{V}	0.19 ±0.02

$LA_{max.}$ – LA maximum dimension, $LA_{min.}$ – LA minimum dimension, LA_{a} – LA pre-systolic dimension, FAE – active emptying fraction of LA, FPE – passive emptying fraction of LA, FTE – total emptying fraction of LA, E – early mitral flow peak velocity, A – atrial flow peak velocity DT – deceleration time, IVRT – isovolumic relaxation time of left ventricle, PEPLA – pre-ejection period of LA, ET – ejection time of LA, PEP/ET – PEP/ET index, PVR – peak velocity of retrograde (atrial) pulmonary venous flow

$^{II, III, IV, V}$ – Group number under a parameter value indicates significant difference with that group. Statistical significance $p < 0.05$

Table III. Echocardiographic parameters of left atrial appendage

	Group I	Group II	Group III	Group IV	Group V
LAA_{long} [cm]	4.37 ±0.72^{V}	3.75 ±0.69	3.63 ±1.03	3.60 ±1.01	3.11 ±0.69
LAA_{trans} [cm]	1.59 ±0.39$^{II. III. V}$	1.53 ±0.53^{V}	1.56 ±0.38	1.6 ±0.26	1.32 ±0.25
$LAA_{area\ max.}$ [cm^2]	4.41 ±1.67^{V}	3.8 ±1.43	3.43 ±1.91	3.58 ±2.56	3.1 ±0.87
$LAA_{area\ min.}$ [cm^2]	1.79 ±1.08	1.3 ±0.94	1.34 ±1.27	1.54 ±1.35	2.08 ±0.68
EFLAA [%]	47.37 ±17.91$^{II. V}$	33.7 ±13.67^{V}	35.99 ±17.91^{V}	39.45 ±9.55^{V}	33.29 ±8.28
LAAE [m/s]	0.74 ±0.22$^{II. V}$	0.53 ±0.23	0.57 ±0.35	0.81 ±0.06^{V}	0.41 ±0.04
LAAF [m/s]	0.61 ±0.18$^{II. III. V}$	0.44 ±0.12	0.44 ±0.21	0.49 ±0.06	0.42 ±0.05

$LAA_{long.}$ – left atrial appendage longitudinal dimension, LAA_{trans} – left atrial appendage transversal dimension, $LAA_{area\ max.}$ – left atrial appendage maximal area, $LAA_{area\ min.}$ – left atrial appendage minimal area, EFLAA – left atrial appendage ejection fraction, LAAE – left atrial appendage maximal empting velocity, LAAF – left atrial appendage maximal filling velocity

$^{II, III, IV, V}$ – Group number under a parameter value indicates significant difference with that group. Statistical significance $p < 0.05$

Table IV. Left ventricular parameters

	Group I	Group II	Group III	Group IV	Group V
LVEDd [cm]	5.56 ±0.82^{V}	5.54 ±0.95^{V}	5.44 ±0.89	5.47 ±0.67^{V}	4.92 ±0.43
LVESd [cm]	4.26 ±0.80$^{IV. V}$	4.06 ±1.29^{V}	3.7 ±1.05^{V}	3.57 ±0.72^{V}	2.76 ±0.43
LVEF [%]	34.75 ±11.59$^{IV. V}$	40.3 ±9.58$^{IV. V}$	37.53 ±8.85$^{IV. V}$	47.25 ±9.66^{V}	58.83 ±4.85

LVEDd – left ventricular end-diastolic dimension, LVESd – left ventricle end-systolic dimension, LVEF – left ventricular ejection fraction

$^{IV, V}$ – Group number under a parameter value indicates significant difference with that group. Statistical significance $p < 0.05$

higher in group II than in group V. $LAA_{area\ max.}$ was significantly higher in group I than in group V. $LAA_{area\ min.}$ was significantly lower in group III than in V.

EF_{LAA} was significantly higher in groups I, II, III, IV than in group V and it was significantly higher in group I than in group II.

LAAE was significantly higher in group I than in groups II and V as well as in group IV than in group V. LAAF was significantly higher in group I than in groups II, III, V.

LVEDd was significantly higher in groups I, II, IV than in group V. LVESd was significantly higher in groups I, II, III, IV than in group V; it was also significantly higher in group I than in group IV. EF was significantly lower in groups I, II, III, IV than in group V. In groups I, II, III EF was significantly lower than in group IV.

Discussion

Echocardiography is a widely used and accepted method for LA function evaluation. In the study we assessed systolic LA and LAA function in patients after MI complicated with BBBs.

As far as we know, nobody has investigated LA and LAA systolic function in post-MI BBBs in a clinical context until now.

Our findings indicate that LA compensates LV deterioration after MI, but the presence of post-myocardial intra-ventricular conduction disturbances impairs the compensatory mechanisms. The more advanced conduction disturbances were observed, the more was compensation impaired. In patients who had MI with no conduction disturbances in the His-Purkinje system the LA systolic function enhancement was most expressed. The compensatory mechanism was also observed in patients with post-myocardial LAHB and RBBB but to a far lesser degree. Analysis of echocardiographic parameters showed no enhancement but rather a tendency towards deterioration of LA systolic function in patients with post-myocardial LBBB (group I) as compared to healthy individuals (group V).

It is widely known that the LA contributes as a pump in the LV filling increase after MI and this is a compensatory mechanism to LV function deterioration due to post-infarction myocardial damage [36-38]. LA and LV function are strictly associated with each other – they cooperate mutually. There are observations that LV function deterioration results in LA systolic function enhancement [37]. It happens in congestive heart failure as after MI [39] and in congestive cardiomyopathies. The LA works as a "booster pump", compensating LV systolic function loss to support cardiac output [38].

The enhancement of LA systolic function was observed in patients with post-myocardial RBBB and LAHB (group II and group III), as compared to healthy individuals (group V).

There is evidence that LV function is impaired in patients with LBBB as compared with individuals

with no conduction disturbances [41]. LBBB is associated with higher LVEDd, lower EF, and longer IVRT [1, 41]. These indicate that LBBB affects systolic as well as diastolic function. In our study the parameters of LV systolic function (LVEDV, LVESd, EF) showed significantly worse LV systolic function in patients with BBBs as compared with subjects after MI and no conduction disturbances (group IV) as well as compared with healthy subjects (group V). But there were no significant differences in LV systolic function parameters between groups of patients with different conduction disturbances. Meanwhile, most parameters describing LA systolic function showed significant differences between analysed groups of patients. Generally speaking, patients with post-MI RBBB and LAHB had more enhanced LA systolic function than patients with post-MI LBBB.

We conclude that LA systolic function and the parameters describing it are probably more sensitive than LV systolic function and its parameters to heart overload. That is why despite no significant differences among LV function parameters in patients with RBBB and LAH, the parameters of LA presented less enhanced systolic function in patients with RBBB compared with LAH.

According to the results of our study the compensatory mechanism of LA enhancement that occurs in response to LV function impairment seems to be reduced by intracardiac conduction disturbances. The more severely conductance was impaired the more the effect of diminishing of the LA compensation in LA systolic parameters was visible. In patients with post-MI LBBB LA systolic function was observed not to be enhanced but it showed even a tendency to be deteriorated as compared with patients who had MI with no conductance disturbances. In patients with post-MI RBBB and LAHB enhancement of LA systolic function seemed to have been less expressed than in patients with MI and no BBBs. RBBB and LAHB reduced LA systolic compensation but definitely to a lesser degree than LBBB.

Conductance disturbances in the His-Purkinje system resulting from MI are additional factors, together with post-myocardial scar, that impair left ventricular function. In our study, patients with post-MI LBBB had the worst LV systolic function (the lowest EF). In these patients LA systolic function could have been expected to be mostly enhanced. However, we observed the contrary phenomenon.

LV impairment causes LV end diastolic pressure to increase. The pressure in the LA chamber increases as well. The LA like the LV works according to Frank-Starling's law. LA contraction is determined by the length of the atrial fibres, i.e. by the pressure existing in the LA chamber just prior to its systole [42-44]. During ventricular diastole, the LA is directly exposed to LV pressures through the open mitral valve [45]. The higher the LA pressure, the stronger the LA muscle fibre contractions and the greater the volume expelled from the LA to the LV chamber. However, this compensatory mechanism is limited to some degree. If the LA pressure exceeds the higher limit of Frank-Starling's law the compensatory mechanisms fail [24, 46]. In patients with LBBB LV function was impaired the most severely, which resulted in the highest LV end diastolic and LA pressure. The values of LA pressure might have exceeded the upper limit within which Frank-Starling's law operates.

Another explanation of the observed reduction of LA compensation in post-MI LBBB might be the fact that BBBs disrupt LA and LV mutual haemodynamic cooperation. The effectiveness of atrial systole in augmenting ventricular output depends on the time relationship between atrial and ventricular systole.

The more the intracardiac conductance was impaired, the more were disintegration of LV systole and visible reduction of LA compensation observed.

The presence of LBBB was not associated with any LAA systolic function deterioration (in contrast to the LA systolic function). In group I LAA planimetric parameters describing its size had the highest values among the study groups: $LAA_{long.}$, $LAA_{area\ max.}$, $LAA_{area\ min.}$, EF_{LAA}. Also Doppler parameters had the highest values in group I. LAAE was higher only in the patients in group IV – individuals who had MI with no conduction disturbances.

There was not observed any harmful haemodynamic effect of BBBs on LAA systolic function. All LAA parameters indicated LAA systolic function enhancement; this was the case also in patients with post-MI LBBB. In this group the LAA systolic function was the most enhanced.

The LAA is a highly autonomic chamber that differs in many aspects from the LA. The LAA has quite distinct anatomy and physiology from the LA [47, 49]. The LAA contractile pattern differs from that of the main LA body [50]. The LAA shortens to a greater extent and it is also more distensible than the body of the LA [51-54]. This autonomy and the physiological differences between the LAA and the LA might explain why the LAA reacts differently than the LA to BBBs.

Our study tries to explain the role that the LA and LAA plays in the pathophysiology of the heart. The results of our study suggest how the cooperation between the LV, LA and LAA occurs after MI complicated with BBBs. The pattern of cooperation between the LV, LA and LAA might have an important impact on numerous clinical aspects such as physical activity, individual response to CRT, heart remodelling, and post-myocardial prognosis.

LA remodelling after MI is associated with worse prognosis [55, 56]. Improvement of left atrial systolic function after CRT led to reverse LA remodelling expressed by reduction of LA size [57]. LA and LAA systolic function changes may help qualify patients for CRT.

Better and more precise description of the mutual interactions between heart chambers needs further studies including larger populations. Clinical trials are also necessary to define the exact role the LA and LAA play in heart pathology [58-61].

In conclusion, in patients who experienced MI, compensatory enhancement of systolic left atrial function is observed. Post-myocardial BBBs (mainly LBBB) are elements that reduce systolic left atrial function enhancement – the phenomenon that compensates post-myocardial left ventricular impairment. LA systolic function parameters seem to be more sensitive than LV systolic function parameters to heart overload. Post-myocardial LBBB do not affect systolic left atrial appendage function, in contrast to systolic left atrial function.

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