Left atrial appendage—beyond being a thrombus-spreading tiger

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Received: 11 June 2018 / Accepted: 7 September 2018 / Published online: 17 September 2018
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In the last decade, the left atrial appendage has raised attention being a key driver for thromboembolism among patients suffering from atrial fibrillation (AF). Because of the enormous health economic effects of atrial fibrillation and its complications, diagnostic tools like implantable loop recorders for detection of silent AF after embolic stroke of unknown source were forced [1, 2] as well as the stringency of anticoagulative or interventional treatment of patients with AF being at risk for thromboembolic events according to their bleeding risk [3, 4].

Beyond the thromboembolic risk, the LAA has different other functions which are scientifically underappreciated.

The paper of Asmarats and colleagues published in this issue of the Journal of Interventional Cardiac Electrophysiology [5] is focusing on hemodynamic effects of LAA occlusion. LAA contributes up to 8–9% of the left atrial volume [6, 7] and is highly variable in its morphology [8].

Until now, there is one publication of Tabata and colleagues from 1998 [9] concluding that LAA occlusion is thought to increase LA systolic function by increasing mechanic function via the Frank-Starling mechanism by decreasing distribution volume.

In this surgical analysis, LAA showed a higher compliance compared to the LA chamber itself and is thought to play an important reservoir function especially when LA pressure is high or a volume overload is present. This presumed reservoir function is questioned by the observation that LAA/LA-volume ratio seems not be affected by cardiomyopathies or severe mitral regurgitation [7].

Asmarats and colleagues present echocardiographic data before and after endocardial LAA occlusion among patients with sinus rhythm with regard to LA phasic functions and left

ventricular systolic and diastolic function parameters. The population is restricted to patients with paroxysmal atrial fibrillation as left atrial morphological changes are more pronounced among patients with persistent compared to those with paroxysmal atrial fibrillation [10] that may affect LA hemodynamics.

The results of Asmarats and colleagues should be interpreted as hypothesis generating as the sample size is small and different anatomic variants of LAA morphology were not sub-differentiated.

Moreover, Asmarats and colleagues used only endocardial LAA occlusion systems that do not eliminate the endocrine function of the LAA. LAA is known to have an important impact on release of atrial natriuretic peptides (ANP) regulating the fluid balance [11]. Despite the fact that LAA contains less ANP compared to the right atrial appendage [12], water intake behavior is severely changed in an animal model with acute hypovolemia when LAA is removed [13]. In humans, acute disturbances of fluid balance have not been reported after surgical removal of LAA but less decrease in ANP levels post surgery when the right atrial appendage was left [14].

In contrast to endocardial LAA occlusion, epicardial LAA occlusion showed a significant and persistent reduction in systolic blood pressure and an acute but not persistent change in sodium levels [15] although ANP and brain natriuretic peptide (BNP) levels did not change after epicardial and endocardial occlusion [16]. Regarding endocardial LAA occlusion, there is only one trial that showed a decrease in BNP levels in a short-term follow-up [17].

In the early 2018, the non-randomized, observational LAA HOMEOSTASIS Study did not show any significant differences between BNP levels before LAA occlusion and at 3 months follow-up with an endocardial or epicardial approach [18]. The decrease of blood pressure was only observed in the epicardial group but not in the endocardial group suggesting to be caused by endocrine changes rather than due to mechanical/hemodynamic reasons.

Therefore, using epicardial LAA occlusion systems may have a significant effect on LA and LV hemodynamics. But

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epicardial LAA occlusion systems have not been the aim of the work of Asmarats and colleagues.

Nevertheless, the manuscript of Asmarats and colleagues has a strong impact on the field of research of LAA function and LAA occlusion and should be understood as a signal of reinforcement for sophisticated research of hemodynamics and in future of endocrine function after LAA occlusion with endocardial and epicardial systems. The growing number of publications beyond describing LAA as thrombus-spreading structure illustrates the profundity of this small appendage.

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