Echocardiographic Assessment of the Left Atrial Appendage

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The left atrial (LA) appendage is a common source of cardiac thrombus formation associated with systemic embolism. Transesophageal echocardiography allows a detailed evaluation of the structure and function of the appendage by two-dimensional imaging and Doppler interrogation of appendage flow. Specific flow patterns, reflecting appendage function, have been characterized for normal sinus rhythm and various abnormal cardiac rhythms. Appendage dysfunction has been associated with LA appendage spontaneous echocardiographic contrast, thrombus formation and thromboembolism. These associations have been studied extensively in patients with atrial fibrillation or atrial flutter, in patients undergoing cardioversion of atrial arrhythmias and in patients with mitral valve disease. The present review summarizes the literature on the echocardiographic assessment of LA appendage structure, function and dysfunction, which has become an integral part of the routine clinical transesophageal echocardiographic examination. (J Am Coll Cardiol 1999;34:1867–77) © 1999 by the American College of Cardiology

Cardiogenic embolism accounts for $\geq 15\%$ of ischemic strokes (1). Left atrial appendage (LAA) thrombi are believed to be the source of embolism in a substantial number of these patients, primarily in association with atrial fibrillation (AF) or rheumatic mitral valve disease (or both). Despite the blind cul-de-sac and multilobed anatomic structure of the LAA (2), thrombus formation is normally prevented by vigorous blood flow in the appendage cavity. Nevertheless, LA dysfunction in various pathophysiologic states may predispose to local thrombosis and systemic embolization.

Transesophageal echocardiography (TEE) has been used extensively to characterize LAA structure and function in numerous recent studies. Echocardiographic assessment of LAA function was initially described by Suetsugu et al. (3) and by Pollick and Taylor (4) and has become an integral part of the routine TEE examination. The present review summarizes the literature on the anatomy and function of the LAA, as assessed by echocardiography, and the clinical implications of LAA dysfunction.

**ECHOCARDIOGRAPHIC ASPECTS**

The LAA has been imaged primarily in two basic biplane TEE views: 1) the horizontal short-axis view at the base of the heart (5) and 2) the two-chamber longitudinal view of the left atrium (LA) and ventricle (LV) (6). Multiplane TEE (7) allows greater versatility in obtaining these views and enables visualization of the appendage in a continuum of intermediate planes (8). Although the LAA generally is visualized satisfactorily, attempts have been made to improve echocardiographic images and Doppler recordings by injection of transpulmonary contrast agents (9). Transesophageal imaging is necessary for adequate visualization of the LAA, a posterior cardiac structure in proximity to the esophagus. Although the LAA may, at times, be imaged by transthoracic echocardiography (parasternal short-axis view of the base of the heart or apical two-chamber view), the transesophageal approach is mandatory for consistent and precise delineation of the LAA, particularly in the adult population. A recent report has suggested that Doppler interrogation of the LAA is feasible and accurate with transthoracic imaging (10), although this finding needs to be validated in additional studies.

Complete structural and functional assessment of the LAA should consist of two-dimensional imaging of LAA size, morphology and contraction. In addition, LAA function is assessed quantitatively by pulsed-Doppler interrogation of LAA flow. These data are integrated with those from the complete echocardiographic examination, including assessment of LA size, LV systolic and diastolic function and associated valvular disease, primarily mitral valve disease (11).

**Two-Dimensional Imaging of LAA**

LAA area and ejection fraction have been assessed and reported in numerous studies of LAA function (4,12–20). However, measurements of LAA cross-sectional areas are inherently prone to substantial interobserver variability, during both data acquisition and off-line analysis (4,20), primarily because of the complex three-dimensional anatomy of the LAA, which limits accurate definitions of
standard tomographic imaging planes of this structure. In our clinical experience, attempts to quantitate LAA size and function by planimetric methods are time-consuming and do not seem to offer any advantage over gross visual estimation of LAA size and function. In contrast, assessment of LAA function by Doppler echocardiography, the main focus of this review, is easily performed, reproducible and highly relevant clinically.

In addition to assessing LAA size and function, two-dimensional imaging is used to determine the presence of LAA spontaneous echocardiographic contrast (SEC) (21), semiquantitative grading of SEC (22), and defining the presence, size and mobility of LAA thrombi (23). The LAA is usually a multilobed structure (2) (Fig. 1). In an autopsy study of 500 normal human hearts, the LAA was bilobed in 54% and multilobed (=2 lobes) in 80% of hearts (2). Thus, the LAA should be scanned meticulously in multiple echocardiographic planes, most precisely by multiplane TEE, and the number of lobes determined. A detailed examination of all lobes is necessary for exclusion of LAA thrombi. Owing to the complex structural features of the LAA, the diagnosis of LAA thrombi by TEE is prone to misdiagnosis, both overdiagnosis (false interpretation of prominent pectinate muscles) (2,24) and underdiagnosis (occult thrombi in multilobed appendages) (25).

**Doppler Evaluation of LAA Function**

LAA flow is evaluated by pulsed-wave Doppler interrogation of the LAA cavity. The following technical aspects need to be emphasized:

1) **LAA view**—The view with the optimal alignment of Doppler with LAA flow, as determined by color flow imaging, should be selected. No differences in Doppler velocities have been noted using various LAA views (8,26).

2) **Sample volume location**—Currently, there is no standard sampling site in the appendage (i.e., sampling at the LAA-LA junction versus sampling at different sites within the LAA cavity). It is unclear whether variations in sampling site location produce significant changes in measured velocities, as demonstrated for other Doppler measurements such as mitral inflow velocities (27). In a recent report, a small trend was observed for lower velocities at the wider orifice of the LAA, in comparison with velocities obtained at the narrower middle portion of the appendage (10). LAA flow should be sampled at the site of maximal flow velocities (determined by color flow imaging), while avoiding wall motion Doppler artifacts, which are commonly observed in the more distal (narrow) portions of the appendage. In practice, technically adequate tracings of maximal LAA flow velocities are commonly recorded within the proximal third of the appendage.

3) **Doppler sample size and machine gains**—These are set to visualize a spectral Doppler signal with a clear envelope, typical of the normal laminar appendage flow. Initially, filters are set at low values to enable visualization of low velocity flow, characteristic of early diastolic LAA flow in sinus rhythm and LAA flow in a subset of patients with AF.

**LAA Flow Patterns in Various Cardiac Rhythms**

Distinct LAA flow patterns have been observed in sinus rhythm, AF, atrial flutter (AFL) and cardiac pacing. Previously published values of LAA flow velocities in small groups of volunteers and patients without cardiac disease are presented in Table 1 (15,26,28). Values of LAA velocities in a large population-based study have been reported recently (29).

**Sinus Rhythm**

Discrete Doppler flow signals have been described in patients in sinus rhythm (Fig. 2).

1) **LAA contraction**, resulting in a late diastolic, positive (i.e., toward the TEE transducer) Doppler outflow signal, shortly following the onset of the ECG P wave. This signal coincides with two-dimensional and color flow imaging of LAA contraction and outflow (4) and is related temporally to late diastolic mitral flow (mitral A wave) (30). Measurements of contraction velocities, which are easily reproducible, correlate with two-dimensional measurements of LAA ejection fraction (15).

2) **LAA filling**, an early systolic, negative (i.e., away from the TEE transducer) Doppler inflow signal, immediately following LA contraction (4,28). The physiologic processes underlying LAA filling have not been studied in detail as have their counterpart ventricular processes, and the relative roles of active (LAA relaxation) versus passive (e.g., elastic recoil) processes resulting in LAA filling are poorly defined (hence, the preferred general term “LAA filling”). Although not studied systematically in various patient populations, there is generally a gross correlation between LAA contraction and filling velocities.

3) **Systolic reflection waves**, following LAA contraction and filling. A variable number of alternating LAA outflow
Figure 1. Pathologic specimens of the LAA displaying its complex and extremely variable configuration, thus emphasizing the need for routine meticulous echocardiographic scanning of the appendage in multiple planes. A, Bilobed LAA (LAA is on right side of picture). B, Single-lobed LAA with an additional "appendix" at its distal end (outside the plane of main LAA body). C, Multilobed LAA (multiple small lobes). D, Multiplane transesophageal echocardiographic demonstration of a multilobed LAA (90° and 135° scanning in D-1 and D-2, respectively). The orifice of the LAA (LA–LAA junction) is marked by a pair of arrows in A to C. (A to C, Courtesy of Dr. W. B. Edwards, Department of Anatomic Pathology, Mayo Clinic.)
and inflow signals of diminishing amplitude are commonly recorded (14,31), resulting from passive outward and inward flow waves following the initial high velocity flows of appendage contraction and filling. The velocities of these reflection waves correlate with the preceding LAA contraction and filling velocities (14) and usually are observed in subjects with high LAA contraction velocities (31).

4) Early diastolic LAA flow, a low velocity outflow signal following early diastolic mitral flow (mitral E wave) and pulmonary venous diastolic flow signals (28,32). Initially, it was proposed that early diastolic LAA flow results from compression of the LAA medial wall by the superior motion of the base of the LV during diastole (4). However, a more plausible explanation is passive emptying of the appendage, paralleling LA emptying during rapid ventricular filling in early diastole. Following early diastolic LAA outflow, a low velocity LAA filling signal is infrequently detected (Fig. 2B) and is related to continuous filling of the LA and LAA from pulmonary venous flow during mid-diastole. LAA diastolic filling flow may be observed more commonly in subjects with relatively slow heart rates (28). As shown in Table 1, early LAA diastolic velocities are significantly lower than velocities during active LAA contraction and are of undetermined significance. However, it is possible that passive early diastolic LAA flow may gain significance in the presence of LAA contractile dysfunction (33).

Effect of heart rate. The effect of heart rate on LAA has not been studied sufficiently in patients with sinus rhythm. Fusion of LAA early diastolic and contraction velocities, resulting in higher total velocities, has been observed in young healthy volunteers with sinus tachycardia (28).

Atrial Fibrillation

Active LAA flow is commonly observed in patients with AF, with alternating positive and negative sawtooth-appearing flow signals of variable amplitude and regularity (Fig. 3A). Mean LAA flow velocities have greater physiologic significance than peak velocities. These should be averaged for each cardiac cycle and then averaged for several cycles. Characteristically, flow signals have lower velocities during ventricular systole (LAA contraction in the presence of a closed mitral valve) than during diastole (34). In addition to flow resulting from active appendage contraction, a discrete early diastolic outflow signal, similar to that in sinus rhythm, is observed occasionally (22), although commonly it is difficult to identify this signal clearly and to differentiate it from fibrillatory flow signals.

Generally, flow velocities during AF are lower than those during sinus rhythm (12,32,35). However, flow velocities in patients with AF are highly variable, with high velocity flows on one end of the spectrum (velocities similar to, or even exceeding, those observed in sinus rhythm), and minimal to absent flow on the other end (12). This represents the wide continuum of LAA contractile dysfunction in patients with AF, from relatively preserved contraction to complete paralysis of the appendage.

Effect of heart rate. The effect of ventricular response rate in AF on LAA flow velocities has been studied by comparing cardiac cycles with long and short RR intervals. Because LAA flow velocities in AF are higher in diastole than in systole, longer cardiac cycles are associated with higher mean LAA velocities because of prolongation of diastole (34). Similarly, an inverse correlation has been observed between LAA ejection fraction and ventricular response rate during AF (36). Therefore, high ventricular response rates during AF may be associated with further impairment of LAA flow.

ECG correlation. Generally, the correlation between ECG fibrillatory wave amplitude in nonrheumatic AF and LAA velocities is not clear. A lack of any significant correlation has been reported in two studies (35,37). In a single study, an association has been noted between coarse AF (greatest amplitude of fibrillatory waves in ECG lead V1 ≥ 1 mm) and low LAA contraction velocities and LAA

<table>
<thead>
<tr>
<th>Study</th>
<th>Study Group</th>
<th>Contraction Velocities, cm/s</th>
<th>Filling Velocities, cm/s</th>
<th>Early Diastolic Forward Velocities, cm/s</th>
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<tr>
<td>Kortz et al. (28)</td>
<td>46 healthy volunteers; age, 22–41 yr</td>
<td>64 ± 19</td>
<td>46 ± 12</td>
<td>38 ± 11</td>
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<tr>
<td>Mügge et al. (26)</td>
<td>30 patients with structurally normal hearts (clinically indicated TEE)</td>
<td>Transverse view, 50 ± 6</td>
<td>Transverse view, 52 ± 13</td>
<td>NA*</td>
</tr>
<tr>
<td>Tabata et al. (15)</td>
<td>50 patients with structurally normal hearts (clinically indicated TEE)</td>
<td>Longitudinal view, 52 ± 15</td>
<td>Longitudinal view, 58 ± 18</td>
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*NA = data not available. TEE = transesophageal echocardiography.
ejection fraction (38). These nonconclusive findings probably result from the multiple factors associated with the ECG manifestations of AF, and thus explain the lack of association between ECG AF pattern and the risk of embolic events (37).

Atrial Flutter

In patients with AFL, a more consistent high velocity, relatively regular sawtooth flow pattern is the rule (39–41) (Fig. 3B). The rate of LAA contraction typically is slower in AFL than in AF (40). Flow velocities generally are approximately twice the velocities observed in AF (39,41), although there is significant overlap in the velocities of these arrhythmias.

**ECG correlation.** An intermediate ECG pattern of atrial activity, atrial “fibrillation-flutter,” is associated with a velocity profile resembling that of AF, with lower velocities than those observed in pure AFL (39). Similarly, LAA flow velocities are higher in patients with pure AFL than in those with AFL and intermittent episodes of AF (42).

**Cardiac Pacing**

The effect of atrioventricular asynchrony on LAA flow pattern has been studied in patients with implanted cardiac pacemakers. In the absence of associated AF, ventricular-demand pacing (VVI pacing mode) is accompanied by atrioventricular dissociation or retrograde ventriculoatrial conduction (43). In patients with atrioventricular dissociation, LAA contraction occurs intermittently during ventricular diastole, resulting in relatively high LAA flow velocities, whereas LAA contraction against a closed mitral valve, during ventricular systole, is associated with low velocities (44). In the presence of ventriculoatrial conduction, LAA contraction uniformly occurs during ventricular systole, resulting in low velocity LAA flow (45). Restoration of atrioventricular synchrony by dual-chamber pacing (DDD pacing mode) is associated with higher LAA flow velocities (46).

**PATHOPHYSIOLOGY OF LAA FUNCTION**

**General Aspects**

**Clinical associations.** Age-associated changes in LAA velocities have been described in 50 subjects with normal
hearts (15). Aging was associated with a progressive linear decline in all LAA flow variables (LAA contraction and filling velocities and early diastolic LAA flow) (15). A possible effect of various systemic diseases (e.g., hypertension) on LAA function has not been determined.

**Echocardiographic correlates.** 1) LA/LAA size: in patients with sinus rhythm, there is no clear association between LA size and LAA flow velocities (4). However, concomitant LAA enlargement and low LAA contraction velocities are common in patients with mitral stenosis who are in sinus rhythm (13). In patients with AF, larger LA and LAA sizes commonly are associated with lower LAA flow velocities (14,22,47), both in patients with (13) and in those without (48) associated rheumatic mitral disease. This is most pronounced in patients with near absence of LAA flow, in whom significant dilation of the LA and LAA is the rule. Of note, significant LAA dilation resulting from any cause may potentially further impair LAA function by increasing LAA wall stress.

2) LAA flow versus mitral and pulmonary venous flow: the following correlations were observed between LAA flow velocities and mitral inflow and pulmonary venous flow velocities in the presence of sinus rhythm: a) lack of correlation (14,32) or even a negative correlation (15) between LAA contraction velocities and mitral A velocities, b) lack of correlation between LAA and pulmonary venous flow velocities (15), and c) significant positive correlation between early diastolic LAA outflow and both mitral E velocities and pulmonary venous diastolic velocities, all of which decline progressively with aging due to age-related slowing of LV relaxation (15).

**LAA function versus global LA function.** Various methods developed to evaluate LA contractile function are time-consuming and, thus, not routinely used in clinical practice. Hence, it is tempting to apply LAA flow variables as clinically applicable surrogates for global LA function (20,49), although the validity of such an approach is questionable. The LA and main LA cavity are derived embryologically from different sources. The trabecular LAA is a remnant of the embryonic LA, whereas the smooth LA cavity is derived from an outgrowth of the pulmonary veins (50). Therefore, it is conceivable that LAA function may dissociate from global LA function. The following arguments support this:

1) Dissociation of LAA and LA function has been anecdotally reported during sinus rhythm in patients with preserved global LA function (manifested as normal mitral inflow A velocities), but low or absent LAA flow velocities, in association with LAA thrombus formation, spontaneous echocardiographic contrast and clinical embolic events (35). Dissociation of LA and LAA mechanical activity has been described recently in patients after cardioversion, in whom organized LA mechanical activity may be present along with disorganized LAA contraction (51). A contrasting phenomenon has been described in patients in whom effective LAA contraction was restored early after cardioversion, in the absence of effective global LA activity (absent mitral A waves) (52).

2) As noted above, a lack of correlation (14,32) or even a negative correlation (15) between LAA contraction velocities and mitral inflow A velocities has been observed in patients with sinus rhythm. However, this apparent dissociation between LA and LAA function may result from the multiple determinants of mitral A velocities, which are independent of LA contractility (LV diastolic characteristics and loading conditions), rather than a true functional dissociation between LA and LAA function.

3) Relatively preserved LAA function with high velocity LAA flow is commonly observed during AF in patients without evidence of atrial activity in mitral inflow. Although functional dissociation between the LA and LAA is a possible explanation, it is more likely that for a similar degree of residual contractility, the small-sized appendage is able to generate significant flow during AF, whereas the larger main LA cavity is not. Despite the above observations, clinical experience suggests that evaluation of LAA function may serve as a clinically applicable surrogate of overall LA function.

**LAA FUNCTION AND CARDIAC HEMODYNAMICS**

**LAA Contractility**

LAA contractility is the primary determinant of LAA end-diastolic outflow. However, similar to ventricular physiology, loading conditions may affect LAA function and flow. These interactions were assessed by observations of the effects of LA (and, hence, LAA) pressures, concomitant mitral valve disease and LV hemodynamics on LAA function.

**LA Pressure**

LA pressure is a major determinant of LAA flow. In a combined Doppler and catheterization study of 31 patients with various myocardial (nonvalvular) diseases, Tabata et al. (16) demonstrated an inverse correlation between LAA contraction velocities and mean pulmonary capillary wedge pressure. Similarly, treatment of heart failure, resulting in a decrease in LA filling pressures, is accompanied by an improvement in LAA function (19). In a canine model, preload elevation by volume infusion, resulting in an increase in LA pressures in the normal physiologic range, was associated with subtle increases in LAA contraction velocities (53). Hence, mildly increasing LA pressures may enhance LAA function, as a possible reflection of LAA preload reserve, whereas more significant increases in LA pressures may result in LAA contractile dysfunction due to afterload elevation.

Of interest, recent studies have suggested that the LAA plays a role as a beneficial modulator of LA pressure. The LAA is more compliant than the main LA cavity (54).
Accordingly, surgical LAA clamping (55) or appendectomy (56) decreases overall LA compliance, resulting in a shift of the LA pressure-volume relation upward and to the left.

**Mitral Valve Disease**

Hemodynamically significant mitral stenosis increases the resistance to both active and passive LAA emptying, resulting in an overall lowering of LAA flow velocities, regardless of the specific rhythm. Hwang et al. (13) have demonstrated a marked decrease in LAA contraction velocities in patients with mitral stenosis, compared with patients without rheumatic heart disease, both in sinus rhythm and in AF. In addition to overall impairment of LAA function, mitral stenosis specifically limits the normal augmentation of LAA flow during diastole in patients with AF, a phenomenon related to the severity of the stenosis (57). In the presence of AF, patients with severe mitral stenosis typically demonstrate low-to-absent LAA velocities. This is in contrast to nonrheumatic AF, which is associated with a wide spectrum of flow velocities, with both “high velocity” (velocities $\geq 25$ cm/s) and “low velocity” (velocities $<25$ cm/s, i.e., a flow profile similar to flow in rheumatic AF) flow variants (26). These effects of mitral stenosis on LAA function are probably the result of the severe hemodynamic impairment in patients with mitral stenosis (significant elevation of LA pressures). Direct LA and LAA involvement in the rheumatic inflammatory process, and an atrial myopathy resulting from chronic LA pressure elevation, are additional possibilities. Similar changes in LAA function have been observed in the presence of mitral valve prostheses (58), resulting from the prolonged effects of the presurgical valvular disease on LAA function as well as inherent prosthetic valve stenosis.

The hemodynamic effects of mitral stenosis on LAA function are further understood by analyzing the effect of percutaneous balloon mitral valvotomy on LAA function. TEE studies early after percutaneous balloon mitral valvotomy have demonstrated recovery of LAA function within 24 to 72 h after the procedure, both in patients with sinus rhythm and those with AF (17,59,60). The augmentation of LAA velocities following balloon valvotomy is related directly to the hemodynamic relief of the valvular stenosis (i.e., the decrease in transmitral pressure gradient) (17).

Few data are available about the effects of mitral regurgitation on LAA function. Hemodynamically significant mitral regurgitation is predicted to impair LAA function via LA and LAA dilatation and increased filling pressures. This finding has been observed (55), although inconsistently (32), in small-sized studies. Despite a possible deleterious effect of mitral regurgitation on LAA function, mitral regurgitation has an overall protective effect against thromboembolism (61) by prevention of LA stasis.

**LV Function**

LV systolic (62–64) and/or diastolic dysfunction may affect LAA contraction, primarily through its effect on LAA filling pressures (19). Thus, in addition to the potential for LV thrombi, LV dysfunction may predispose to LAA thrombus formation. Furthermore, impaired LV relaxation is associated with a concomitant decline in both mitral inflow E waves and LAA early diastolic flow. This has been observed in relation to aging (14,15) and LV hypertrophy (14). Similarly, in an animal study, early diastolic LAA velocities were determined independently by the time constant of LV relaxation (tau) (53). The possible clinical role of LV dysfunction as a determinant of LAA function is suggested by clinical and echocardiographic data from the Stroke Prevention in Atrial Fibrillation (SPAF) study, in which clinical heart failure and echocardiographic evidence of LV dysfunction (65) were independent predictors of thromboembolic events.

**CLINICAL IMPLICATIONS OF LAA DYSFUNCTION**

Many studies have suggested that determination of LAA function by echocardiography allows the identification of patients with AF or AFL who are at high risk for the development of LA or LAA thrombi and thromboembolic complications. Assessment of LAA function has also revealed the phenomenon of postcardioversion LAA dysfunction (“stunning”), responsible for the prothrombotic state following cardioversion. In addition, a few recent studies have suggested a role for LAA function assessment in predicting the immediate and, possibly, the long-term success of conversion of AF.

**AF AND AFL**

**LAA Dysfunction and LAA**

**SEC, Thrombi and Embolic Events**

LA and LAA SEC, resulting from local blood stasis, is associated with a high incidence of thrombus formation and thromboembolic events (21,22,66). LAA dysfunction, associated with AF of various causes, is commonly accompanied by SEC (26,47,48,66,67). In the SPAF III TEE substudy of high-risk AF patients, LAA contraction velocities of $\leq 20$ cm/s were associated with SEC in 75% of patients, significantly less than the 58% frequency noted in the higher velocity group. Similarly, Mügge et al. (26) demonstrated SEC in a significantly higher proportion of patients with LAA velocities $<25$ cm/s. By semiquantitative analysis, the degree of LAA SEC is negatively associated with LAA velocities (22).

Significant LAA dysfunction is similarly associated with LAA thrombus formation (22,47), although anticoagulation therapy may weaken this association. LAA velocities are significant predictors of thrombus formation, independent of various hemostatic variables indicating platelet or coagulation activation (68). Almost uniformly, the presence of LAA thrombus is accompanied by extreme LAA dysfunction, manifested as a low-to-absent LAA flow velocity profile (47,48). In SPAF III, LAA thrombi were more
prevalent in the low (≤20 cm/s) than in the higher LAA velocity groups (17% vs. 5%, respectively) (66). From a practical echocardiographic standpoint, assessment of LAA function may assist in the accurate diagnosis of intra-appendage masses and "pseudomasmes" (24). Significant impairment of LAA function supports a diagnosis of LAA thrombus, whereas normal function suggests an alternative diagnosis.

Many studies have shown retrospectively an association between LAA dysfunction and previous systemic embolic events, primarily cerebral embolism (26,69–72). Recently, the SPAF III trial has demonstrated prospectively the role of LAA dysfunction as a risk factor for future embolic events (66). The relative risk of ischemic stroke was 2.6 times greater in patients with LAA flow velocities <20 cm/s than in those with higher LAA velocities. Furthermore, LAA flow velocities progressively decline with increasing clinically defined risk of thromboembolism (73). In general, the above associations of LAA dysfunction and SEC, thrombus formation and thromboembolism are stronger in rheumatic than in nonrheumatic AF (13,26), and in AF than in AFL (39,41), relating to greater degrees of LAA dysfunction in rheumatic heart disease and in AF, respectively.

The clinical implications of the preceding observations relating LAA dysfunction to thromboembolic risk have not been defined clearly. Although patients with AF can be stratified according to thromboembolic risk by assessment of LAA function, the clinical impact of risk stratification on patient therapy has not been determined.

**Effect of Cardioversion on LAA Function**

Initially, it was proposed that resumption of LA mechanical activity after cardioversion may result in systemic embolization of preformed LA thrombi, thus explaining the occurrence of thromboembolic events in the immediate postcardioversion period. However, several recent studies have supported an opposing mechanism, in which cardioversion of AF produces transient worsening of LA mechanical function ("stunning") (74), thus predisposing to thromboembolism. This observation has been extended to the LAA by numerous studies that uniformly have demonstrated the phenomenon of postcardioversion LAA stunning (18,41,52,75–82). LAA stunning is manifested echocardiographically as a “paradoxical” reduction in LAA flow velocities, compared with the precardioversion LAA velocities in AF, despite the reversion to a regular sinus rhythm LAA flow pattern. LAA dysfunction after cardioversion is commonly associated with new or worsening SEC (52,75,77,78), thus predisposing to thromboembolism (75).

Initially described in association with direct-current external cardioversion (52,75), post-AF LAA stunning has also been observed in spontaneous cardioversion (76), pharmacologic cardioversion (77) and low energy internal atrial defibrillation (78). This supports the hypothesis that conversion to sinus rhythm itself, rather than the method of cardioversion, is responsible for LAA stunning. The lack of LAA dysfunction after direct-current endocardial or external shocks in patients with sinus rhythm (80) and the absence of correlation between the electrical energy used for cardioversion and the degree of LAA stunning (82) further support the above observations. Although well-described echocardiographically, the pathophysiologic basis of LAA stunning has not been defined.

Postcardioversion LAA stunning is also observed after cardioversion of AFL (41). Because of higher initial LAA flow velocities in AFL, postcardioversion LAA velocities are greater following cardioversion of AFL than of AF despite a similar absolute reduction in LAA velocities (41). Therefore, new or worsening SEC after cardioversion is less common in AFL (41), explaining the relatively low incidence of embolic episodes following cardioversion of AFL. LAA stunning has been observed following radiofrequency ablation of chronic AFL (79), further emphasizing the role of cardioversion itself rather than the technique in the pathogenesis of LAA stunning.

The time course of resolution of LAA stunning is variable and dependent on several patient, cardiac and arrhythmia characteristics, as suggested for overall LA function assessed by transthoracic echocardiography (74). Because TEE is an invasive procedure, sequential TEE data assessing recovery of LAA function are limited. Significant improvement has been observed in LAA function within 7 to 30 days (18,78,79,81) after cardioversion, although the determinants of the time-course of LAA recovery are incompletely defined. The clinical implication of postcardioversion LAA stunning is straightforward, emphasizing the need for well-controlled anticoagulation therapy in the postcardioversion period in the absence of demonstrable LA or LAA thrombi before cardioversion. The possible role of TEE assessment of LAA function, as a guide to the duration of postcardioversion anticoagulation, has not been determined to date (to our knowledge).

**Prediction of Cardioversion Success**

Recent studies have suggested that the success of cardioversion of AF to sinus rhythm (spontaneous, drug-induced or electrical) may be predicted by assessing LAA function before cardioversion. LAA velocities during AF may predict both the short-term success of cardioversion (18,70,83,84) and the long-term maintenance of sinus rhythm (85), although this is not a uniform short-term (64,85) or long-term (64,83) observation. Velocities greater than approximately 20 cm/s are associated with a higher rate of short-term success in converting to sinus rhythm (83,84). The association between LAA velocities and successful cardioversion is probably related to the association of LAA velocities and LA and/or LAA size (70,84,85), LA filling pressures and the duration of AF (67,70,84,85), all of which are predictors of cardioversion success.
A few studies have considered the effects of LAA dysfunction in patients with rheumatic mitral disease in sinus rhythm in whom LA and LAA SEC is associated with low LAA flow velocities (13,86). However, the role of LAA dysfunction in patients with sinus rhythm and no associated rheumatic valvular disease is not clear and has been reported only anecdotally (35,87). The role of LAA dysfunction in sinus rhythm as a predictor of future embolic events, and possibly the future development of AF, needs to be addressed in large prospective trials.

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