Original Article

Assessment of Atrial Function

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This article discusses the traditional and more recent echocardiographic measures that have been employed to evaluate atrial function. Conventional parameters commonly used and reported in the literature include the study of the various phases of atrial activity using atrial volume measurements, the peak A wave velocity, its velocity time integral (VTI) and the fraction of atrial contribution (all obtained from transmitral flow), as also the atrial ejection force. Newer parameters for atrial function assessment include Doppler tissue imaging (DTI) including segmental atrial contractility using colour Doppler tissue imaging (CDTI) and estimates of atrial strain and strain rate.

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Introduction

The left atrium (LA) serves multiple functions, acting as a reservoir during ventricular systole, as a conduit (for blood from the pulmonary veins to the left ventricle) during early diastole, as an active contractile chamber that augments left ventricular filling in late diastole and as a suction source that refills itself in early systole.[1] In total, the atria contribute ~30% of cardiac output.[2,3] This atrial contribution is particularly important in the setting of impaired left ventricular dysfunction when the atrial 'kick' considerably augments left ventricular filling.[2] Conversely, the absence of synchronised atrial contraction can result in symptomatic deterioration as is commonly seen when atrial fibrillation (AF) develops. AF is the commonest arrhythmia, occurring in 0.4% of the general population, increasing to 5% in those over 65 years of age.[4,5] AF is associated with increased morbidity and mortality[6] and with atrial dysfunction. This paper will discuss the non-invasive evaluation of atrial function using echocardiography.

There is currently no widely accepted non-invasive 'gold standard' to evaluate atrial function. In comparison to ventricular function that has been extensively characterised, there is a paucity of literature regarding the evaluation of atrial function. Atrial size is regarded as a surrogate marker of function, with larger atria thought to represent a 'dysfunctioning' atrium.[7,8]

Several echocardiographic parameters have been developed to evaluate atrial function. They include the peak velocity of the A wave and its velocity time integral (VTI) obtained from transmitral Doppler flow,[9,10] the atrial fraction[10,11] and the atrial ejection force.[11,12] More recently, the A′ velocity using Doppler tissue imaging (DTI) has been used to assess global atrial function.[13,14] Segmental atrial function can be evaluated using colour Doppler tissue imaging (CDTI)[13] as well as strain and strain rate imaging.[15,16]

LA Volume: A Surrogate Marker

Left atrial size can be estimated using M-mode echocardiography from the parasternal long axis view.[17] M-mode-derived LA diameter can be used to derive the LA volume assuming that the LA is spherical. However, this technique is less accurate with a tendency for underestimation[18,19] as compared to the ellipsoid model or biplane evaluation of LA volume.

The ellipsoid model represents the LA as a prolate ellipse with a volume = $4\pi/3(L/2)(D1/2)(D2/2)$, where L is the long axis and D1 and D2 orthogonal short axes dimensions. L is measured from the apical four-chamber view, D1 as the antero-posterior diameter from the parasternal long axis view and D2 as the medio-lateral dimension from the parasternal short axis view.[20] The major limitation of this technique is that the volume determined relies on the careful location and direction of the minor axis dimensions. A composite dimension can be derived using long-axis LA areas. Thus, substituting area for length, the LA volume can be derived as $8(A1)(A2)/3\pi(L)$, where A1 and A2 are maximal planimetered areas from the apical four- and two-chamber views, and L = the LA long axis length measured from the apical four-chamber view. To simplify this further, the single plane area–length method was developed where volume = $8(A1)^2/3\pi(L)$,[21,22] with A1 being the maximal planimetered area from the apical four-chamber view.

LA volume, similar to the LV, can be measured using Simpsons rule.[23,24] Simpson's algorithm divides the LA into a series of stacked oval heights (h) whose minor and

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major axes are D1 and D2. Thus, LA volume is derived as = $\pi/4(h)\Sigma(D1)(D2)$. Computerised integration is provided by software packages on most standard commercial systems. Maximal left atrial volume (LAESV) is measured during ventricular systole and the smallest left atrial volume (LAEDV) is measured during ventricular diastole with the pulmonary veins and the mitral valve apparatus excluded from the volume measurement.[22]

More recently, three-dimensional (3D) imaging has been used to evaluate atrial volume. This technique has been validated against magnetic resonance imaging (MRI).[25] Three-dimensional LA volumes by echocardiography have the most favourable test–retest variation with the least inter-/intraobservor variability.[26] Three-dimensional atrial volume demonstrated good correlations to biplane two-dimensional (2D) LA volume estimation with the least underestimation.[26] Thus, it is reasonable to conclude that in routine clinical practice, estimation of biplane LA volume would suffice and that 3D volumes are unlikely to provide an incremental increase in the information obtained. All echocardiographic techniques underestimate LA volume when compared with computed tomography (CT) scans[23] or with MRI,[27] but show excellent correlation to these measures.

Atrial enlargement has been suggested to be a powerful biomarker of ventricular diastolic[28,29] and systolic dysfunction,[30] is a hallmark of valvular disease[31] and atrial fibrillation.[7,8] Increasing LA size is also an independent predictor of cardiovascular events in the elderly[32] and is a risk factor for stroke.[6]

Phasic LA Volumes

The total LA volume represents a composite of three distinct phases of atrial function: the passive emptying volume, the conduit volume and the active emptying volume.[33,34] The former represents the volume of blood that is transported to the left ventricle prior to active atrial contraction. The LA conduit volume represents the volume of blood that passively fills the left ventricle from the pulmonary veins while the mitral valve is open. LA active emptying represents the volume of blood that is actively ejected into the LV during atrial systole. The various LA volumes can be defined as follows:

Left atrial passive emptying volume = LAESV − LA Vol p
Left atrial passive emptying fraction = LAESV − LA Vol p/LAESV
Left atrial conduit volume = LVSV − (LAESV − LAEDV)
Left atrial active emptying volume = LA Vol p − LAEDV
Left atrial active emptying fraction = LA Vol p − LAEDV/LAEDV

Phasic left atrial volume changes have been studied with normal aging,[24] in elite atheletes,[34] as also in disease states.[35] The extent of active, passive and conduit filling by the atrium is strongly influenced by the compliance of the left ventricle. Our studies of a healthy normal cohort demonstrated a decrease in passive atrial filling as well as in conduit volume in the older age group, together with a compensatory increase in active atrial contraction (overall LA volume unchanged) to overcome the normal age-related increases in ventricular diastolic stiffness.[36] When either the extent or duration of LV diastolic abnormalities exceed what is observed with normal 'healthy aging', shifts in the percentage of active and passive LA filling are observed,[35] with a subsequent increase in the total LA volume.

Transmitral Flow: Peak A Wave Velocity, its VTI and the Fraction of Atrial Contribution

In addition to providing information about atrial anatomy, echocardiography is a powerful tool for evaluating atrial mechanical function. Mitral inflow patterns by pulsed wave Doppler examination demonstrate passive ventricular filling in early diastole (E wave) and a late active filling phase representing atrial contraction (A wave). Estimation of the peak A wave velocity is commonly employed in studies that have evaluated atrial function.[10,11] The sample volume is placed at the tips of the mitral leaflets and measurements are made in expiration at a sweep speed of 100 mm/s from the apical four-chamber view. The peak A wave velocity is influenced by heart rate, loading conditions and normal aging.[37] The peak A wave velocity increases with normal aging to overcome the decrease in ventricular diastolic compliance that occurs.[24,38]

The peak A wave velocity has also been employed in the serial follow-up of patients with AF following the restoration of SR by either cardioversion[10] or operative procedures such as the Maze/Star procedure,[14,39,40] and more recently catheter-based ablation techniques.[41] The A wave is absent in the presence of AF, and restoration of sinus rhythm results in its reappearance. The temporal recovery of the A wave velocity was largely dependent on the duration of AF prior to cardioversion. With brief duration of AF (2 days to under 2 weeks), the peak A wave velocity was similar to that of the general population following the restoration of sinus rhythm.[10,42] However, in cases with intermediate duration (2–6 weeks) or prolonged AF (over 6 weeks), the peak A wave velocity was significantly lower than in a normal control cohort despite the restoration and maintenance of SR.[10] Velocities normalised within 1 week in the intermediate duration group and after 4 weeks in the group with prolonged durations of AF. Thus, it was postulated that a period of 'atrial stunning' occurs with the restoration of sinus rhythm that is reversed over a period of 3–4 weeks. The VTI of the A wave is measured as the area under the transmitral A wave[10,11] and demonstrates similar results to that observed with the peak A wave velocity after the restoration of SR in subjects with AF.

Another measure of atrial function is the percentage of atrial systolic contribution to total diastolic filling. Estimation as a percentage of the total diastolic filling would correct to some degree, for the variation in heart rate making the atrial fraction a more robust marker than the peak A wave velocity. The atrial fraction is expressed as a fraction of the total mitral inflow VTI, i.e., atrial fraction = (A wave VTI/total mitral inflow VTI) × 100.[10,11]

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The atrial fraction demonstrates changes similar to that of the peak A wave velocity following the restoration and maintenance of SR.[10,11]

Atrial Ejection Force

The atrial ejection force has been proposed as a measure of atrial function and is based on Newtonian principles. The force exerted by the left atrium during atrial systole is defined as the product of the mass and acceleration of blood from the left atrium.[12] Mass is further defined as the product of the density of blood ($\rho = 1.06\,\text{g/cm}^3$) and the volume of blood passing through the mitral valve orifice. The atrial ejection force is estimated using the following equation:

Atrial ejection force = mass × acceleration; substituting for mass and acceleration, atrial ejection force = $0.5 \times \rho$ (density of blood = $1.06\,\text{g/cm}^3$) × mitral orifice area × (peak A velocity)^2 as previously described.[11,12] The mitral orifice area is assumed to be circular and is estimated from the annulus diameter measured in the apical four-chamber view.

The atrial ejection force has limitations since the peak A velocity and the mitral annular diameter are not measured instantaneously. The peak A velocity is derived from Doppler signals which is angle dependent. Thus, velocity estimations can be underestimated if the Doppler sample is not aligned parallel to the flow of blood.

Doppler Tissue Imaging: A′ Velocity Using Pulsed Doppler

Doppler tissue imaging is a recently developed technique for the quantification of intrinsic myocardial contractility and relaxation.[43,44] A few studies have demonstrated that the peak velocity in late diastole secondary to atrial contraction (A′ velocity) measured using pulsed wave DTI is a rapid and accurate marker of atrial function.[13,45] The pulsed wave DTI sample volume (2 mm axial length) is placed on the atrial side of, or on the mitral annulus at the basal interatrial septum in the apical four-chamber view (Fig. 1). Special attention must be paid to align the Doppler beam parallel to the inter atrial septum to optimise Doppler measurements. Measurements are obtained during end expiration, at a sweep speed of 100 mm/s and an average of three beats is measured. The Nyquist limit is set at a range of 20 to −20 cm/s with minimum gain and low filter settings to optimise the spectral display. Previous studies have demonstrated that there is no significant difference between the basal septal and basal lateral peak A′ velocity, unlike the early diastolic E′ velocity.[46]

We studied the A′ velocity in a normal cohort to determine the effects of normal aging.[13] The A′ velocity was

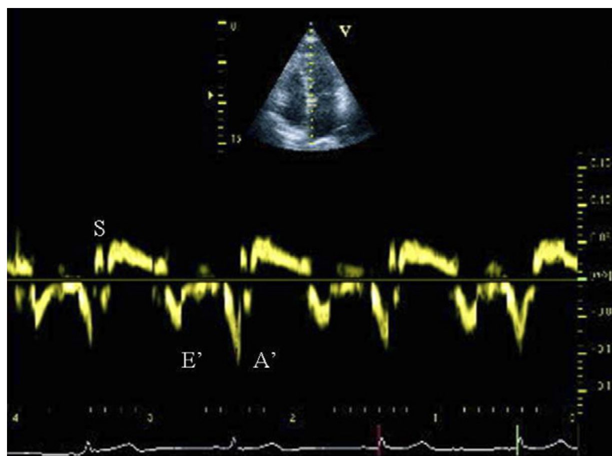

Figure 1. *Pulsed wave Doppler tissue imaging with sample volume placed on the atrial side of the septum. S, systolic velocity; E′, early diastolic left ventricular relaxation; A′, atrial contraction in late diastole.*

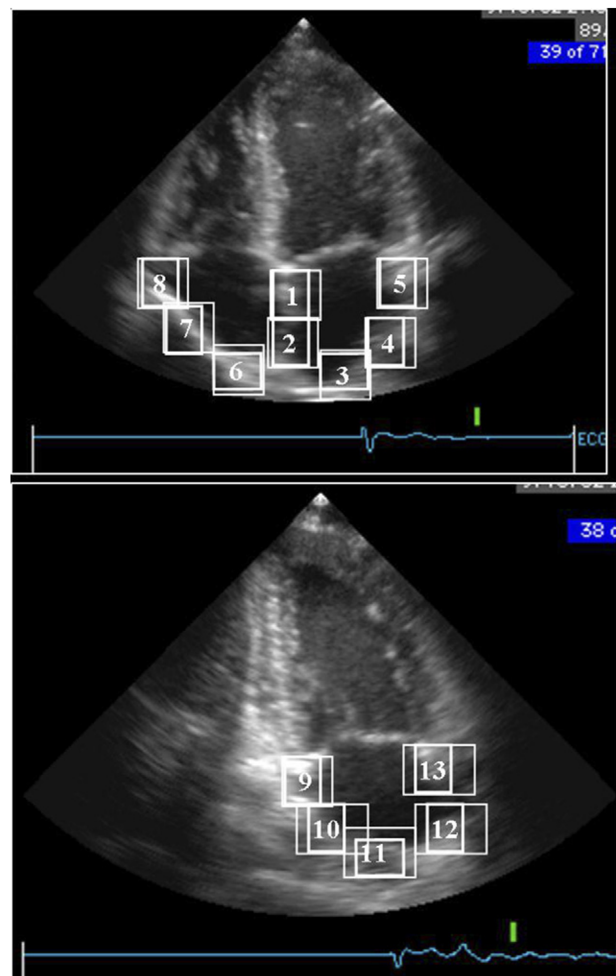

Figure 2. *Atrial segments from the apical four- and two-chamber views. Apical four-chamber view (1, septal annular segment; 2, septal mid-segment; 3, superior segment; 4, lateral mid-segment; 5, lateral annular segment; 6, lateral annular RA segment; 7, lateral mid-RA segment; 8, superior RA segment). Apical two-chamber view (9, posterior annular segment; 10, posterior mid-segment; 11, superior segment; 12, anterior mid-segment; 13, anterior annular segment).*

seen to increase, similar to the peak A wave velocity, with aging. The A′ velocity correlated with other parameters of atrial function, namely, the peak A velocity, atrial fraction and the atrial ejection force. Hesse et al.[45] demonstrated that the A′ velocity correlated with left atrial fractional area and volume change. The A′ velocity is reduced in diseased states associated with atrial dysfunction. We also observed a reduced A′ velocity in subjects with chronic AF restored to SR (unpublished data) and in subjects treated with the operative Star Procedure.[14]

Colour Doppler Tissue Imaging

We recently described atrial segmental function using CDTI.[13] Based on previous studies that estimated segmental ventricular function,[47,48] we divided the atrium into multiple segments at the annular, mid-atrial and superior

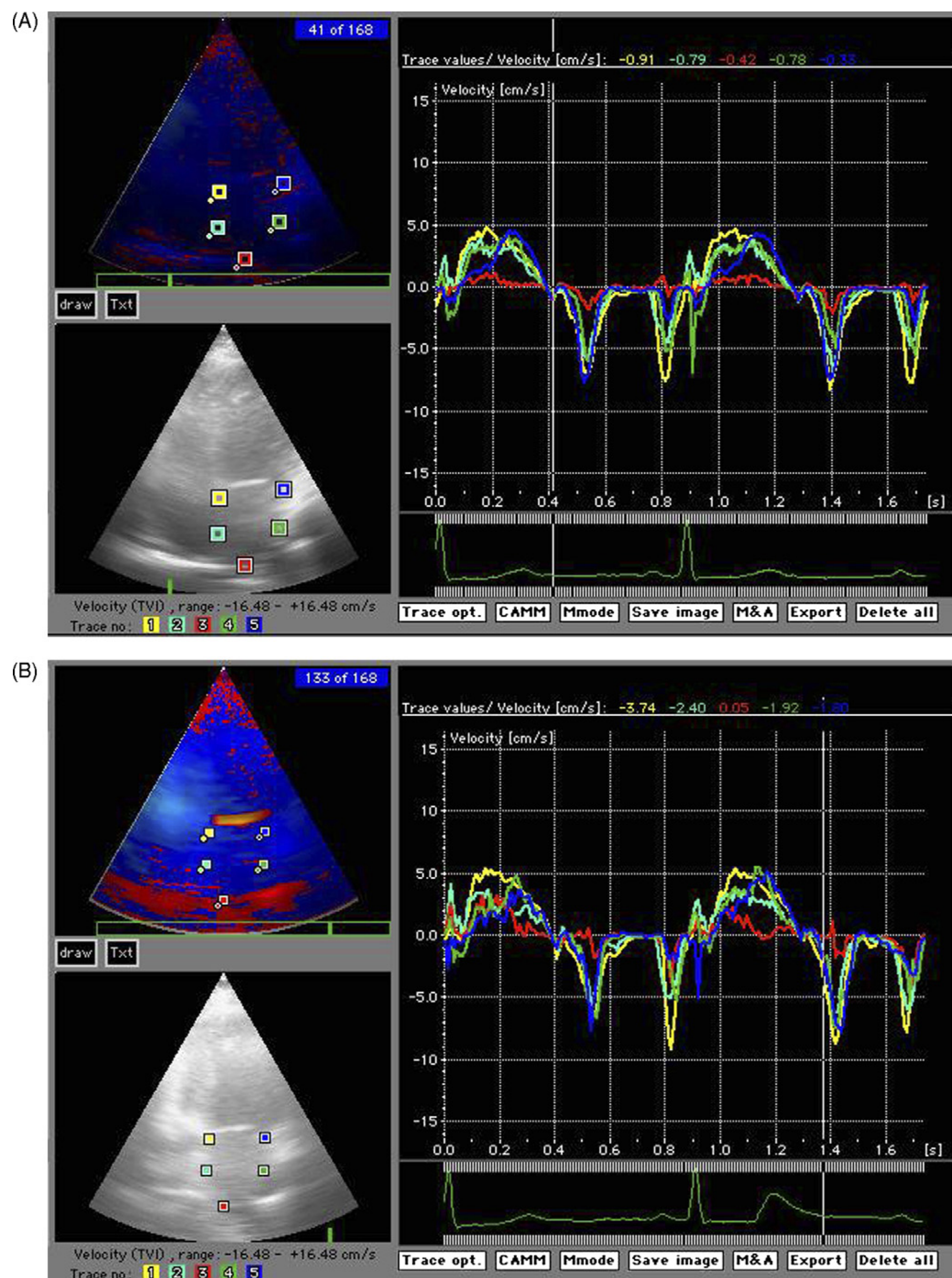

Figure 3. *Segmental longitudinal atrial contraction from the apical four- and two-chamber views. (A) Apical four-chamber view measuring segmental LA velocities. (B) Apical two-chamber view measuring segmental LA velocities.*

Table 1. *Segmental Atrial Contraction Velocities From Apical Four- and Two Chamber in Young (Group Y: <50 Years; n = 47) and Old (Group O: >50 Years; n = 45) "Normal" Subjects*

Apical Four Chamber	Group Y (cm/s)	Group O (cm/s)
Lateral ann-RA	−8.2 ± 2.3	−9.6 ± 2.3
Lateral mid-RA	−4.6 ± 1.8	−5.3 ± 1
Superior RA	−1.3 ± 1	−1.6 ± 1
Septal ann	−6.6 ± 1.6	−7.2 ± 1.3
Septal mid	−4.6 ± 1.6	−5.0 ± 1.6
Superior LA	−1.1 ± 0.7	−1.4 ± 0.8
Lateral mid-LA	−5.9 ± 1.9	−6.8 ± 1.9
Lateral ann-LA	−6.4 ± 2.2	−7.7 ± 2.2
Apical Two Chamber	**Group Y (cm/s)**	**Group O (cm/s)**
Posterior ann-LA	−6.9 ± 1.8	−8.4 ± 1.7
Posterior mid-LA	−4.5 ± 1.9	−5.0 ± 1.6
Superior LA	−1.3 ± 1.1	−1.6 ± 1.0
Anterior mid-LA	−5.6 ± 2.3	−6.6 ± 2.1
Anterior ann-LA	−6.4 ± 2.0	−6.9 ± 2.0

The values in this table have '−' if the movement was away from the transducer and '+' if movement was towards the transducer. Ann, annular; LA, left atrium; RA, right atrium.

level. From the apical four-chamber view, measurements were made from five segments of the left atrium and three segments of the right atrium (Fig. 2). Using the two-chamber view, measurements were made from five segments of the left atrium (Fig. 2). Nine by nine pixel sampling was used and a tissue velocity profile throughout the cardiac cycle was displayed at each location (Fig. 3A and B).

Normal values for segmental atrial contractility are reported in Table 1. An annular-to-superior segment gradient is noted. Thus, the atrium adjacent to the annulus had the maximum movement while the superior segment is relatively fixed. A significantly increased segmental velocity was noted uniformly in the annular and basal segments in the older age group. We further analysed segmental atrial contractility in a cohort of patients cardioverted to SR. This demonstrated a low segmental velocity immediately post-cardioversion with a temporal increase in segmental atrial contractility.[49] Differential recovery of function was noted between the right and left atrium with right atrial function normalising in four weeks. LA function remained subnormal even at six months.

Atrial Strain and Strain Rate Imaging

Strain and strain rate imaging of the ventricular myocardium has been extensively reported in normal[50,51] and in diseased states.[52,53] However, there is a paucity of data on atrial strain and atrial strain rate (A-sr) imaging derived from the atria. Doppler tissue imaging can evaluate regional myocardial function[43,44] and can be used to evaluate global and segmental atrial function.[13] However, a major limitation of DTI is that it cannot distinguish between intrinsic myocardial motion and that produced by passive translatory effects due to tethering. These effects are largely mitigated with the use of strain and strain rate analysis. In fact, with DTI, concordant motion of the atrium to that of the ventricle is observed, presumably reflecting

Table 2. *Strain Rate Measured From the Basal Atrial Segments in the CAF and Normal Groups at Baseline*

	Normals (n = 37)	CAF (n = 37)	p-value
Basal septal E-sr (s^{-1})	−0.99 ± 0.51	−0.63 ± 0.39	0.001
Basal septal A-sr (s^{-1})	−1.6 ± 0.72	−0.53 ± 0.31	0.0001
Septal tA-sr (s)	0.47 ± 0.11	0.55 ± 0.16	0.017
Basal lateral E-sr (s^{-1})	−1.3 ± 0.62	−1.2 ± 0.78	NS
Basal lateral A-sr (s^{-1})	−0.95 ± 0.64	−0.34 ± 0.25	0.0001
Basal inferior E-sr (s^{-1})	−1.5 ± 0.84	−1.1 ± 0.91	NS
Basal inferior A-sr (s^{-1})	−1.8 ± 0.96	−0.6 ± 0.38	0.0001
Inferior tA-sr (s)	0.47 ± 0.13	0.56 ± 0.13	0.005
Basal anterior E-sr (s^{-1})	−0.64 ± 0.4	−0.48 ± 0.3	0.01
Basal anterior A-sr (s^{-1})	−0.90 ± 0.6	−0.63 ± 0.36	0.01

A-sr, peak atrial strain rate in late diastole; tA-sr, duration from aortic valve closure to peak atrial strain rate; E-sr, peak early diastolic strain rate.

the inability of tissue Doppler imaging to distinguish atrial contraction from mitral annular and ventricular motion. In contrast, the longitudinal shortening and lengthening of the atrium are discordant with ventricular longitudinal motion because the atrium fills during ventricular systole and empties during ventricular diastole (Fig. 4A and B). The discordance of atrial versus ventricular motion is recognised because A-sr, unlike DTI-derived A′ velocity, demonstrates a site-specific directional difference.

Images are obtained using a narrow sector (frame rate >110 fps) and attempts are made to align the atrial wall parallel to the Doppler beam.[15] Because of the thin atrial walls, a narrow (10 mm × 2 mm) sample volume is selected and placed in the middle of the basal, septal, inferior, lateral and anterior walls of the atrium in the apical four- and two-chamber views. The image is tracked frame by frame, ensuring in each frame that the sample volume is moved to its original location in the middle of the segment using dedicated software available on an offline measuring station (EchoPac PC, GE-Vingmed, Horten, Norway). Gaussian smoothing (60) is applied prior to the peak strain rate being measured. The values for atrial strain rate in the basal segments in the normal cohort and the chronic AF group is reported in Table 2.

Our study demonstrated a temporal increase in atrial strain rate with the restoration and maintenance of SR. However, unlike the peak A velocity the A-sr did not normalise. This persistent atrial dysfunction could warrant the longer-term use of anti-arrhythmic therapy in the CAF cohort; however, further studies are required before this can be recommended. A recent study also demonstrated that the A-sr and atrial strain following the restoration of sinus rhythm were independent predictors for the maintenance of SR following cardioversion.[16]

Left Atrial Appendage Function

No report of atrial function would be complete without at least briefly alluding to left atrial appendage (LAA) function assessment. However, unlike the previous parameters, transoesophageal echocardiography is required for LAA function assessment.[54] The LAA is usually multilobed (54% bilobed, 80% multilobed).

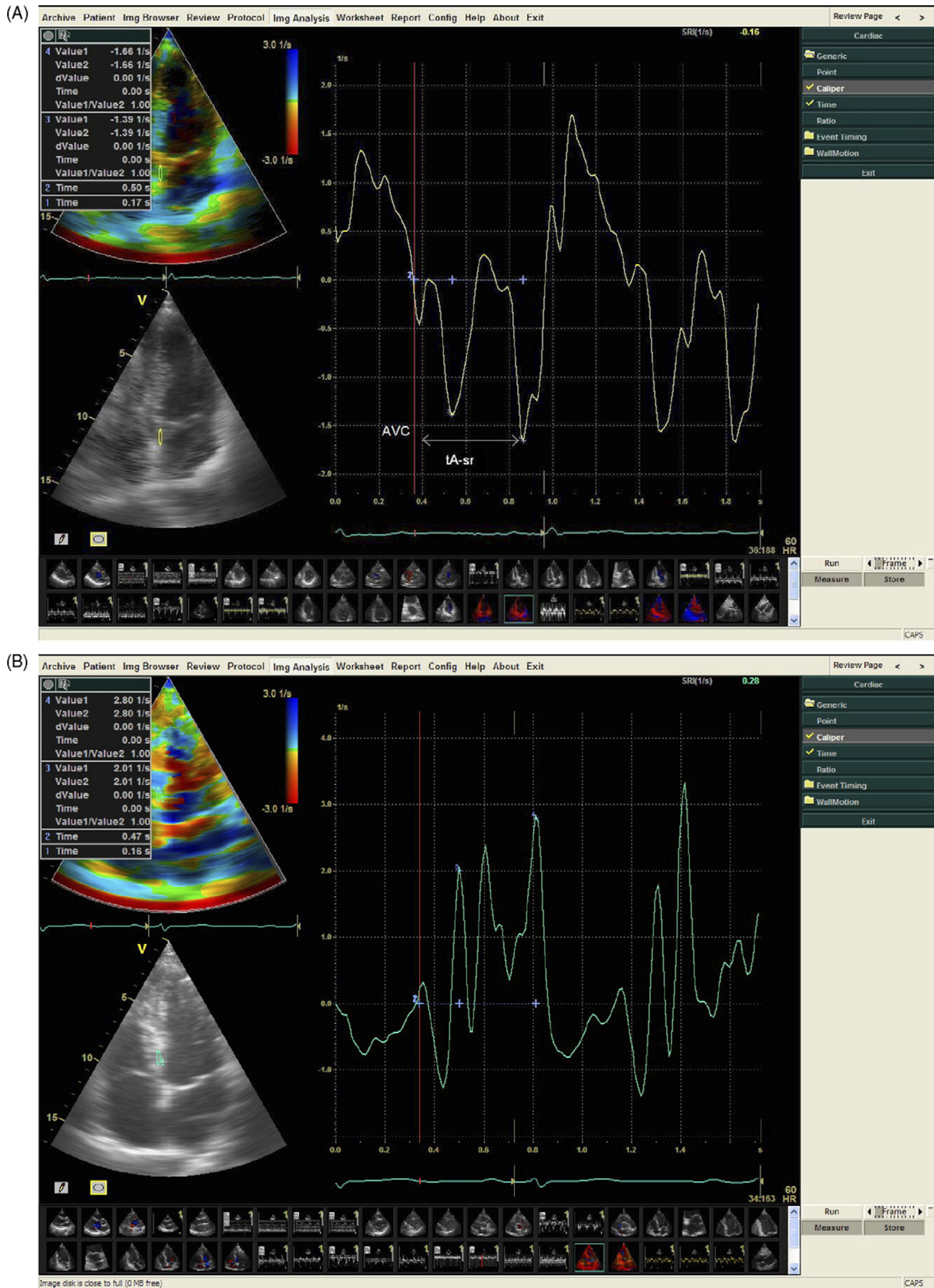

Figure 4. *(A) Atrial strain rate trace obtained with sample volume placed in the basal ventricular septum. The vertical red line denotes aortic valve closure (AVC). The horizontal arrow denotes the time from AVC to peak A-sr (tA-sr). (B) Ventricular strain rate trace obtained with sample volume placed in the basal ventricular septum.*

LAA area and structure have been assessed using 2D echocardiography.[55–57] LAA area has been considered in embolic risk scores in studies such as the SPAF II study. A LAA area >6 cm^2 was a significant risk factor for arterial embolic events.[58]

Pulsed wave Doppler interrogation of the LAA is performed by placing the sample volume at the junction of the atrium and the LAA. Normal LAA flow patterns demonstrate late diastolic contraction, early systolic filling, systolic reflection waves and early diastolic appendage flow. Differing patterns of atrial appendage filling have been described in atrial flutter and in atrial fibrillation.[55]

In addition to Doppler velocity, 'spontaneous echo contrast' (SEC) is also a surrogate marker of LA appendage function.[56] A high grade of SEC has been associated with low LAA contraction velocity.[59,60] The incidence of LA appendage thrombus is associated with low appendage flow (peak velocity <0.22 m/s)[60,61] and increased spontaneous echo contrast.[57,62] The associations of LA appendage dysfunction and thrombus formation are stronger in rheumatic than in non-rheumatic heart disease.[59]

Conclusion

Atrial size is a well-characterised surrogate marker of atrial function. Phasic atrial volumes can be used to further evaluate atrial function in detail. A constellation of new parameters including DTI and strain and strain rate can now be used to quantify atrial function non-invasively. A routine, thorough evaluation of atrial function provides useful adjunctive information for the clinician during cardiac evaluation and is recommended in all individuals undergoing echocardiography.

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