Atrial deformation: the key to maintenance of sinus rhythm in patients with atrial fibrillation?

Hisham Dokainish1* and Stuart J. Connolly2

1Department of Medicine, Section of Cardiology, Baylor College of Medicine, Houston, TX 77030, USA; and 2McMaster University Health Sciences Centre, Hamilton, Ontario, Canada

This editorial refers to ‘Strain rate imaging for functional quantification of the left atrium: atrial deformation predicts the maintenance of sinus rhythm after catheter ablation of atrial fibrillation’† by C. Schneider et al., on page 1397 (Volume 29 number 11 June 2008)

Atrial fibrillation (AF) is the most common serious arrhythmia encountered in clinical practice, and accounts for approximately one-third of all hospitalizations for cardiac rhythm disturbances.1 Consequently, effective therapies for conversion of AF to, and maintenance of, sinus rhythm (SR) have been actively sought. Currently, catheter-based ablation techniques have been shown to be promising in the elimination of AF. However, a significant percentage of patients undergoing catheter ablation have recurrence of AF; in one recent study, only 64% of patients with paroxysmal and 45% of patients with persistent AF were AF-free at 12 months follow-up.2 Other groups have shown that with complete pulmonary vein and posterior left atrial (LA) isolation, up to 63% of chronic AF patients maintained SR at 2 years3. Therefore, there is a great interest in markers, either clinical, biological, or imaging-based, that can predict which patients will maintain SR after catheter ablation and which will revert back to AF.

Schneider and colleagues measured tissue Doppler-based strain and strain rate—measures of the degree and rate of LA deformation—are readily obtainable and reproducible; in this study, normal myocardial tissue is often replaced with fibrosis has been shown to occur in patients with AF, and it has been previously been shown that AF patients have significantly lower LA systolic and diastolic strain and strain rate than controls, and that these variables were correlated to age and LA dimension.5 It has also been previously demonstrated that such measures of atrial deformation are not necessary static. It has been shown that LA strain rate was lowest immediately post-electrical cardioversion, and increased thereafter, achieving a maximal value at 4 weeks post-cardioversion; such improvement has been associated with the process of reverse atrial remodelling.6 In patients with lone AF, it has also been shown that the only multivariate echocardiographic predictors of SR after electrical cardioversion—which also assessed transoesophageal echocardiographic parameters of the LA appendage—were LA systolic strain and strain rate.7

What is the biological basis of these findings? Atrial remodelling has been shown to occur in patients with AF, and it has been shown that normal myocardial tissue is often replaced with fibrosis in AF patients. It has been demonstrated that LA protein concentrations were increased in AF patients compared with SR patients; deposition of collagen I and III and fibronectin was seen to be increased in AF patients vs controls.8 In addition, other investigators have shown that, in LA tissue from explanted hearts in patients with cardiomyopathy undergoing heart transplantation,
atrial extracellular matrix remodelling manifested by downregulation of tissue inhibitor of metalloproteinase-2 along with upregulation of atrial metalloproteinase-2 and type I collagen volume fraction is associated with sustained AF. Thus, replacement of normal atrial tissue with fibrosis, at the structural and ultrastructural levels, can be postulated to be associated with a lower degree and rate of deformation (strain and strain rate, respectively) in patients with persistent AF compared with patients with SR; note should be made that not all of these structural changes, or to such a degree, are seen in patients with lone AF. In addition, from a biological standpoint, it is important to note that no invasive haemodynamic measures of LA pressure were made during the echocardiographic acquisition in the study by Schneider and colleagues—which may be feasible, moreover, in the electrophysiology laboratory during the ablation procedure; thus, the correlation of atrial strain and strain rate with LA pressure needs to be elucidated, as it has been done for left ventricular strain and strain rate with left ventricular pressures.

There are some caveats that should be taken into consideration when interpreting the data presented by Schneider and colleagues. First, 2500 patients with AF were screened before 118 were included for analysis in this study; therefore, the generalizability of these findings to a broad spectrum of patients with AF—particularly patients with left ventricular ejection fraction <50%, ‘severe’ coronary artery disease, or mitral valve disease, all of whom were excluded in this study—is still unclear. Secondly, that the pre-ablation atrial strain and strain rate data were less predictive of AF recurrence than post-ablation data underlines the challenges of obtaining reproducible Doppler-based data in patients during AF, and the effect of the loss of late atrial activity—and hence deformation—in AF during diastole. Thirdly, it is unknown whether the ablation procedure itself could affect LA strain and strain rate values, owing to post-ablation LA oedema and scarring. Finally, these were tissue Doppler-based strain and strain data which, like all Doppler techniques, are prone to angulation error; therefore, atrial strain and strain rate data using non-Doppler speckle-based echocardiographic imaging, which are angle-independent, would be of interest in future research.

What is the clinical relevance of these findings? That pre-ablation atrial strain parameters were predictive of maintenance of SR post-ablation raises the possibility that patients with AF could potentially be screened before considering ablation, and patients with very low values may be considered as suboptimal candidates for ablation. It also seems reasonable, as the authors state, that patients with impaired left atrial deformation properties after catheter ablation should be followed up closely and over a longer period. Furthermore, patients with low strain and strain rate values may be considered for long-term anticoagulation therapy given their apparently higher likelihood of reverting to AF after ablation. Although these data are thought-provoking and exciting, larger, prospective studies across a broad spectrum of AF patients are needed to validate these findings, before such echocardiographic indices can potentially be used to screen patients for appropriateness for catheter ablation.

Conflict of interest: none declared.

References