**Title**

**Right atrial myocardial deformation by two-dimensional speckle tracking echocardiography predicts recurrence in paroxysmal atrial fibrillation**

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**Keywords**

Atrial fibrillation; right atrial strain; right ventricular strain; two-dimensional speckle tracking echocardiography.

**Abstract**

**Background:** Atrial fibrillation (AF) is a bi-atrial disease yet little attention has been given to right heart function in AF. We propose that assessment of right atrial (RA) and right ventricular function (RV) using two-dimensional speckle tracking echocardiography (2D-STE) could be valuable in predicting AF recurrence in patients with paroxysmal AF (PAF).

**Methods:** Thirty patients with PAF were prospectively recruited from a dedicated AF clinic. Right atrial size, volume and area and RV dimensions were analysed along with RA and RV strain derived from 2D-STE at baseline, 3 and 12 months.

**Results:** Higher RA booster strain independently predicted sinus rhythm (SR) maintenance for up to 1 year, (p=0.001). RV strain was impaired in patients with recurrent AF compared to those in SR (p<0.05) but did not predict AF recurrence. Two-dimensional STE for RA and RV function was simple to perform with excellent reproducibility (adjusted R2 0.92-0.99).

**Conclusions**: Two-dimensional STE is useful and highly reproducible in assessing right heart function in AF patients. RA booster strain function was predictive of sinus rhythm maintenance for up to 1 year.

**Introduction**

Research efforts in atrial fibrillation (AF) have largely been dedicated to the study of structural and electrical remodelling of the left atrium (LA). Clinical research has focused on imaging assessment of LA size, function and myocardial mechanics as clinical predictors of outcome and sinus rhythm (SR) maintenance. Therapeutic interventions too, have focused on LA linear ablation and pulmonary vein isolation which have cured or reduced AF burden [1,2 ]. However, the role of the right atrium (RA) in AF has not been investigated, though AF is indeed a bi-atrial disease.

Histological studies of RA myocardium in AF show the same substrate of patchy fibrosis, inflammatory cell infiltrate, necrosis and vascular degeneration [3,4] as seen in the LA. Similar electrical remodelling with downregulation of the L-type calcium currents (Ical) [5] and Ca(2+)-ATPase [6] is also seen in *both* atria of patients with paroxysmal and persistent AF[7]. Volume assessments based on guidelines [8,9] may not be capable to detect such microscopic changes.

Two-dimensional speckle tracking echocardiography ( 2D STE) is a robust tool [10,11] that has been used to detect changes in LA myocardial deformation in AF patients [12-16] with higher LA strain percentage corresponding to SR maintenance in paroxysmal AF [17]. Higher burden of LA fibrosis was negatively correlated with LA strain percentage [18]. Global LA strain has also been shown to be a reproducible marker of dynamic LA function and predictor of stroke in AF[19]. Two-dimensional STE has been used to define normative values of RA in healthy volunteers [20] but no studies have assessed RA myocardial mechanics in the AF.

The principal aim of this study was to assess right heart function, particularly RA mechanical function in patients with paroxysmal AF, as we have published the impact of AF on the left sided heart.

**Methods**

**Study Population**

Thirty patients with documented non-valvular paroxysmal AF were prospectively recruited for the study from a dedicated AF clinic run at St Georges Hospital, in London, UK between 2008 and 2010. Patients with atrial flutter or persistent AF were excluded. All patients had stroke prophylaxis based on CHA2DS2-VASc score.

Inclusion criteria into the study were AF < 18 months duration, age between 25 and 75 years, LV ejection fraction (EF) >50% without the presence of hypertrophy and adequate mobility for exercise. Exclusion criteria included structural and coronary artery disease, and pulmonary hypertension of any aetiology.

Nineteen out of thirty patients were on amiodarone, one patient was on amiodarone plus diltiazem, one on diltiazem plus digoxin and another one was on flecainide. The remaining patients were not on antiarrhythmic drugs.

Ethics approval was obtained from the local ethics committee and informed consent was obtained from all the study subjects

**Echocardiography**

All patients underwent transthoracic echocardiography at the baseline visit, and at 3 and 12 months follow-up. Two-dimensional, pulsed, colour and tissue Doppler images were recorded through optimal parasternal, apical and sub-xiphoid views using Vivid 9 Vingmed General Electric ultrasound scanner (GE Vingmed, Horten, Norway). Left ventricular EF was estimated by Simpson’s biplane method. LV filling pressure was assessed using the E/E´ ratio.

Pulmonary artery systolic pressure was estimated using the peak tricuspid regurgitation and in the inferior vena cava collapsibility with respiration, according to the recommendations of the American society of echocardiography. Right atrial pressure, in other words, the central venous pressure, is reflected proximally into the inferior vena cava. During normal RA pressure, about 3 mmHg (range, 0-5 mmHg), diameter of the inferior vena cava remains < 2.1 mm with more than 50% inspiratory collapse.

For RA measurements, the RV end systolic frame just before tricuspid valve opening was identified in the apical 4C view. Supero-inferior and medio-lateral RA diameters were measured. RA area was calculated in the same frame by tracking the endocardial border, paying attention to exclude the area between the tricuspid leaflets and annulus. Cardiac chamber quantification was performed in accordance with the American Society of Echocardiography guidelines [8]. All 2D RA and RV measurements were averaged over three consecutive cardiac cycles when in SR with an ECG sweep speed of 100mm/sec. When in AF, an index beat was used, namely the RR interval following 2 preceding cardiac cycles of equal duration, A time difference of <60 ms was allowed between preceding RR and pre-preceding RR intervals of the index beat [21]. Offline analysis was performed using GE Echopac (Version 11.0).

RA myocardial function was assessed by 2D STE. The RA endocardium was manually traced just before the QRS complex, at its minimum volume. The software then automatically generated a 15mm wide region of interest with a review feature allowing visual confirmation of tracking points or permitting manual adjustment to ensure optimal tracking of the RA during the entire cardiac cycle. Six segments in the atria were assessed and the average of these values was taken. After approval of tracking points, a longitudinal strain curve was generated that included RA reservoir (providing a positive strain curve) and RA booster strain, as shown in Figure 1, Panels (A) and (B). Right atrial reservoir strain corresponds to RV end-systole and is considered to be the maximal RA strain. Right atrial booster strain curves appear after the P wave or in case of AF right before the QRS complex. Right atrial booster strain represents atrial contraction and hence appears below the zero reference line, (Figure 1). For the atrial deformation assessment, we set the zero reference point as that time from the beginning of the P wave in patients (so called PP gating) with SR and the start of QRS wave in those with AF (RR gating).

Using a non-foreshortened RV focused apical 4-chamber view, the RV mid cavity and basal diameters were measured at end diastole [9]. The RV outflow tract in the sub-pulmonary region was measured from the parasternal short-axis view. For quantitative measurement of RV systolic function, percent fractional area change (FAC %) and tricuspid annular plane systolic excursion (TAPSE, cm) were used.

RV strain was assessed by tracking the endocardial border using the same GE EchoPAC software. Peak longitudinal systolic strain at the end of RV systole was computed from the RV free wall.

**Follow up**

The study subjects were contacted monthly to document the presence of symptoms for AF recurrence. At one month, patients had a full clinical review with a resting 12 lead ECG, 24-hour Holter monitor and a repeat conventional and 2D strain echocardiogram at 3 and 12 months. All patients were followed up to 12 months.

**Statistical methods**

Data are expressed as mean ± SD unless otherwise stated. For variables with normal distribution, comparisons between the AF and SR groups were performed using Fisher’s exact test for categorical data and the Student t test for unpaired continuous variables. As the primary outcome measure was freedom from AF at 12 months and the predictive value of RA and RV strain on AF recurrence, univariate and multivariate regression analyses were performed to identify independent predictors of AF recurrence (AFR). A covariate-adjusted sequential forward step-wise regression was used. The covariates measured at 1 year included: age, baseline NT-proBNP, RA booster strain, RA maximum volume, RV basal and mid wall strain (Table 3).

Correlation between NTproBNP levels and right heart parameters were evaluated using Pearson’s correlation.

**Inter- and intra-observer variability**

All thirty patients were studied in duplicate 1 week apart, by 2 observers (AK and SKS). Bland-Altman plots and linear regression analysis was used to assess reproducibility.

All analyses were performed using SPSS 20.0 (SPSS Inc., Chicago, IL, USA) and MedCalc version 12.5 (MedCalc Software, Mariakerke, Belgium). The level of significance for all tests was 5%.

**Results**

Fifty eight percent of study subjects (n= 30) were men (n=18). All thirty patients were in SR at the time of recruitment into the study and by 1 month, 14 had documented evidence of AF on ECG or Holter. By 3 months, 15 patients had recurrent AF. Baseline characteristics of patients are presented in Table 1, based on rhythm status at 1 month. RA diameter/area and indexed LA volumes did not differ between those with recurrent AF and SR at 1 month. All patients were either on anti-arrhythmic therapy (n=24), and/or rate controlling drugs (n=13). Baseline characteristics were essentially similar in both SR and recurrent AF groups. All thirty patients were followed up to 12 months, at which time, 11 patients remained in SR and 19 had AF recurrence. Of these 19 patients, 15 were on anti-arrhythmic drugs whilst 7 out of the 11 patients in sinus rhythm were on anti-arrhythmics, (p=0.06).

At 3 months, 3 out of the 15 patients with recurrent AF had their echocardiograms recorded in the presence of AF and an index beat was used to take measurements. At 12 months, 5 of the 19 patients had their echocardiograms performed whilst in AF. Comparisons of right heart parameters at the 3- and 12- month follow up visits between those maintaining SR and those who had recurrent AF are presented in Table 2.

RA booster strain was significantly greater in the group maintaining SR than in those that went on to have AF recurrence. The atrial deformation curves of the sinus rhythm subjects were markedly contrasting to the patients in AF, Figure 2, Panels (A) and (B).

At baseline, there were no significant differences in RV function between the 2 groups based on AF recurrence at 1 month. However, the 3-month follow up echocardiogram demonstrated differences in RA reservoir strain (SR 27% vs AFR 17%, p<0.01). RA booster strain% was significantly different between the groups (SR 11% vs AFR 6%, p<0.01) as well. Peak longitudinal RV basal strain values in SR were 32% vs 28% in AFR group at 3 months.

Figure 3 illustrates a notable difference in the curves between the sinus rhythm patients compared with a patient that had studies performed whilst in AF, with not only lower strain values but less synchronised strain curves.

By 12 months, all right heart parameters were significantly greater in the SR compared to the recurrent AF patients, Table 2. Interestingly, baseline resting ANP and BNP levels showed no differences between those going on to maintain SR up to 12 months and those with early relapse to AF.

In the univariable analysis, NT-proBNP, RV basal and mid systolic strain, RA booster strain and RA maximum volume were significantly associated with the primary outcome, while in the multivariable analysis, RA booster strain remained the only predictor of AF recurrence at 1 year post-cardioversion (Table 3).

**Cut-off value of RA booster strain at 1 year to predict AF recurrence**

A cut-off value of 11.5 % RA booster strain provides the best specificity of AF recurrence of AF (94%), while a cut-off value of 0.6 % of RA booster strain provides the best sensitivity of AF recurrence at 1 year (93 %). In simpler terms, a strain of 11.5 % is predictor of sinus rhythm while a strain of 0.6 % or less is expected to predict AF recurrence with 94% sensitivity.

Figure 5 shows the echocardiographic parameters measured at 1 year post-cardioversion.

NT-proBNP correlated marginally significantly with RV basal strain% (r=-0.37, p=0.05) but significantly with age (r=0.58, p=0.001). Right atrial area correlated negatively with RV basal strain (r= -0.38, p=0.05) and trended towards a significant negative correlation with RV mid wall strain (r= -0.32, p=0.08), suggesting a possible RA-RV mechanical coupling.

**Reproducibility of right heart measurements**

Intra- and inter-observer variability is presented in Tables 4 and 5. In the linear regression model, the adjusted R2 values ranged from 0.92-0.99. In the Bland-Altman plots, the mean difference (bias) was not significant, Fig 4, a-c.

**Discussion**

The main findings of this study are: (i) RA & RV deformation properties are significantly impaired in recurrent AF compared to those who maintain SR for up to 12 months, (ii) higher RA booster strain% independently predicted SR maintenance for up to 12 months, (iii) impaired RV mechanics in AF reverses with SR by 3 months and (iv) the load- and angle independent 2D-STE method is robust to study the right heart mechanics in AF with excellent reproducibility.

Atrial fibrillation is a progressive bi-atrial disease and yet, the RA, similarly to RV, is often the forgotten chamber as in many cardiac disease states. The RA has been shown to have prognostic effects in several clinical conditions such as primary pulmonary hypertension, congestive heart failure and cardiomyopathy [22-25]. The results of the present study also support a plausible association between AFR and RA strain, particularly RA booster strain %.

LA remodelling has been the main focus and studies have reported it as a risk factor for AF recurrence after successful catheter ablation [26]. Left atrial functional impairment, uncovered by use of strain imaging, has been shown to be related to LA wall fibrosis in a study using delayed enhancement magnetic resonance to detect fibrosis of the atrial myocardium [18]. LA strain was mildly impaired in patients with PAF and more significantly impaired in persistent AF as the degree of fibrosis and remodelling in the latter being more extensive. Such data are not available for the RA but similar findings would be expected. To the best of our knowledge this study is the first one to investigate the role of RA mechanics in AFR.

RA remodelling has been reported in AF, but few studies have focused upon it as a risk factor for AF recurrence [27,28]. This is possibly due to the elusive nature of accurate RA assessment and also the known electrophysiological circuits connecting the LA to the pulmonary veins resulting in high success rates with catheter ablation [29,30]. Despite these success rates, AFR remains a major problem in the short and longer terms.

Studies in AF and non AF patients undergoing cardiac surgery have shown that the degree of interstitial fibrosis in the RA appendage is greater than that in the LA appendage in patients with paroxysmal and persistent AF compared to those without AF [31]. Previously, pacing and ablation in the high RA, Bachmann’s bundle and inter-atrial septum was used to prevent AF, as multiple re-entry circuits were identified in the RA [32,33].

Akutsu et al have shown an association with RA remodelling and AFR, RA and LA volumes (>87ml and >97ml respectively) being predictive of AF recurrence post catheter ablation[26,27]. They also demonstrated a similar degree of myocardial remodelling in both atria by voltage mapping. In the heart failure model, both RA and LA pressures were increased similarly in early and severe heart failure and this bi-atrial remodelling contributed to the development of atrial arrhythmias [27]. Interestingly, whilst Akutsu et al [26] only studied atrial volumes in the paroxysmal AF group, our study has shown that whilst RA booster strain was significantly different between AFR and SR groups, other indices of baseline RA function such as RA size and area were not. This suggests that diminished baseline RA booster strain, which may be predictive of recurrent AF, may represent more subtle RA contractile stunning that could otherwise not be detected with standard measures of RA function. Hence, 2D-STE may be a more sensitive tool with predictive capacity in detecting early subclinical RA dysfunction and patchy myocardial changes.

Right ventricle has also been increasingly recognised as one of the most important outcome determinants in various cardiovascular diseases such as pulmonary arterial hypertension, cardiomyopathy [34-36] and ischaemic heart disease [34]. Yet, its role in AF is undefined.

Assessment of RV is more complex due to its distinct micro-anatomical and geometrical challenges [37]. Despite the differences compared with LV, studies of RV mechanics are feasible and reproducible using 2D-STE, even though current software is not customized for RV. A recent study by Su et al showed that global LV systolic strain, not EF, was a superior predictor of cardiovascular events in AF [38]. However, since AF patients are at high risk of developing heart failure with preserved EF, LVEF may not be as accurate in predicting adverse cardiovascular events. Similarly, RV systolic function may be equally impaired in AF even in the absence of failure and standard measures of RV function such as TAPSE and FAC. TAPSE alone may not detect subclinical dysfunction or early pressure and volume loading abnormalities in the presence of normal ejection values [39]. These early changes, however, may reflect a good prognosis as our study showed improvement in the strain percentage after 3 months of SR maintenance, whilst those with AFR had a progressive decline in RV strain. However, baseline NT-proBNP levels were not different amongst the groups or predictive of AF recurrence, as they truly were not in heart failure, neither clinically nor otherwise.

**Limitations**

The relatively small sample size due to the strict entry criteria is a limitation as multiple covariates were considered in the regression model. Hence, caution must be taken to draw definitive conclusions without further examination with larger sample size to validate our findings. Finally, currently available STE software was developed for assessment of LV function and its use in RA/LA and RV function assessment has not yet been fully validated. In this project, we have not compared the relative importance of right versus left sided heart in AF. Although the left atrium has been extensively studied and normative data published [40], to the best of our knowledge, the right atrial normative data have never been studied as only a handful of right atrial mechanics has been studies such as one by one of us [41].

**Conclusions**

Two-dimensional STE is useful and highly reproducible in assessing right heart function in AF patients. RA booster strain function in our patient group was predictive of sinus rhythm maintenance for up to 1 year. However, larger study groups are required to confirm these results.

**Compliance with Ethical Standards - Statements**

This study on patients with PAF was performed at St George’s University of London and St George’s Hospital, NHS Trust, London, UK

**Disclosures:**

The British Heart Foundation (BHF) grant reference number was PG/08/038/ 24217.

The title was “Utility of natriuretic peptides in patients with atrial fibrillation undergoing direct-current cardioversion or those requiring rate-control”

The Principal Investigator and grant holder was Professor A John Camm.

The BHF grant only covered the peptides in atrial fibrillation. However, we have reported in our manuscript a correlation between peptides and echocardiographic parameters.

Dr Samir Kanti Saha performed the statistical analysis and created the 1st draft of the manuscript in conjunction with MG and AK.

Professor A John Camm was a BHF Professor until June 2012. The grant started on the 01/07/2008. Dr Malini Govindan was funded by this BHF grant as a Research Fellow in order to complete her PhD thesis.

The study was approved by the Local Ethics Committee at St George's University of London, London, UK and informed consent was obtained from eligible patients.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Conflict of Interest: Malini Govindan, Anatoli Kiotsekoglou, Samir Kanti Saha, John Camm declare that they have no conflict of interest.**

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**Tables**

**Table 1:** Baseline characteristics of patients based on rhythm status at 1 month

|  |  |  |  |
| --- | --- | --- | --- |
| **Clinical and other variables** | **SR (n = 16)** | **AF (n = 14)** | **P-value** |
| **Age (years)** | 63 ± 10.1 | 68 ± 4.9 | 0.21 |
| **Body mass index** | 31 ± 4.1 | 32 ± 5.7 | 0.35 |
| **Heart rate, beats/minute** | 81 ± 15 | 97 ± 18 | 0.25 |
| **Systolic blood pressure (mmHg)** | 128 ± 14 | 131 ± 12 | 0.17 |
| **Diastolic blood pressure (mmHg)** | 75 ± 7 | 79 ± 8 | 0.54 |
| **Anti-arrhythmic therapy** | 13 | 12 | 0.68 |
| **CHA2DS2-VASc risk score** | 2.0 | 3.0 | 0.84 |
| **NT-proBNP (pg/ml)** | 838 ± 491 | 1056 ± 491 | 0.13 |
| **ANP (nmol/L)** | 6.8 ± 2.1 | 7.4 ± 2.9 | 0.8 |
| **Left ventricular ejection fraction (%)** | 59 ± 11 | 56 ± 9 | 0.68 |
| **E/E’ ratio** | 7 ± 4 | 9 ± 2 | 0.14 |
| **LAVi (ml/m2)** | 37 ± 6 | 37 ± 7 | 0.92 |
| **Right atrial diameter (cm)** | 4.5 ± 0.5 | 4.3 ± 0.4 | 0.23 |
| **Right atrial area (cm2)** | 17 ± 8 | 21 ± 3 | 0.07 |
| **Right ventricular basal dimension (cm)** | 3.9 ± 0.5 | 3.9 ± 0.6 | 0.68 |
| **Right ventricular outflow tract diameter (cm)** | 3.3 ± 0.3 | 3.1 ± 0.3 | 0.06 |
| **Pulmonary artery systolic pressure (mmHg)** | 28±5 | 33±6 | 0.03 |

Results are represented as Mean ± SD. P-values are obtained from t-tests, adjusted for unequal variances if necessary. SR: sinus rhythm; AF: atrial fibrillation; ANP: atrial natriuretic peptide; E: early left ventricular filling; E’: peak early diastolic annular myocardial velocity; LAVi: indexed left atrial volume.

**Table 2:** Echocardiographic analysis of Right Heart function at 1, 3 and 12 months

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Variables** | **RA reservoir strain%** | **RA booster strain%** | **RV basal strain%** | **RV mid strain%** | **RV apical strain%** | **FAC**  **(%)** | **TAPSE cm** |
| ***Baseline values at 1 month*** | | | | | | | |
| **SR=16** | 21±9 | 10±3\*\* | 25±6 | 23±4 | 21±5 | 36±4 | 1.8±0.4 |
| **AF=14** | 18±5 | 8±2 | 22±5 | 22±4 | 21±5 | 34±17 | 1.3±0.8 |
| ***At 3 Month*** | | | | | | | |
| **SR=15** | 27±5\* | 11±5\* | 32±6\* | 27±3\* | 24±7 | 43±3 | 2.2±0.3 |
| **AF=15** | 17±5 | 6±0.8 | 25±3 | 26±3 | 26±3 | 34±7 | 1.8±0.5 |
| ***At 12 Month*** | | | | | | | |
| **SR=11** | 27±9 \*\* | 13±4 \*\* | 28±6 \*\* | 26±6 \*\* | 24±4 \*\* | 44±5 \*\* | 2.1±0.5\*\* |
| **AF=19** | 14±8 | 5±0.8 | 16±10 | 14±9 | 12±8 | 30±10 | 1.3±0.5 |

RA: right atrial; SR: sinus rhythm; AF: atrial fibrillation; FAC: fractional area change; TAPSE: tricuspid annular plane systolic excursion. \*P-value <0.01, \*\*P-value <0.001

**Table 3:** Univariable and multivariable predictors of rhythm status 1 year post-cardioversion

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Univariable analysis Multivariable analysis | | | | | | |  | |
| Variables | R2 | Co-efficient | SE | 95% CI (Co-efficient) | P | R2 | Co-efficient | P |
| Age, years | 0.1 | -0.4 | 0.8 | -1.3 - 2.2 | 0.59 |  |  |  |
| NT-proBNP, pg/ml | 0.09 | 0.4 | 0.16 | 0.07 - 0.7 | <0.05 |  |  |  |
| RA booster strain, % | 0.8 | 1.1 | 0.1 | 1 - 1.2 | <0.001 | 0.8 | 1.1 | <0.001 |
| RV basal strain, % | 0.2 | 1.5 | 0.4 | 0.8 - 2.3 | <0.001 |  |  |  |
| RV mid wall strain, % | 0.2 | 1.5 | 0.01 | 0.8 - 2.2 | <0.001 |  |  |  |
| RA maximum volume, ml | 0.3 | -0.14 | 0.3 | -0.7 - 0.43 | <0.05 |  |  |  |

RA: right atrium; RV: right ventricle. R2: co-efficient of determination; SE: standard error; CI: confidence interval; P: P-value.

Baseline at 1 month, RA booster strain % also predicted rhythm status, both as a univariate as well as a multivariate predictor (p<0.001).

**Table 4:** Inter-observer variability

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **2D strain measures** | **Bias** | **95% CI** | **P-value** | **R2** | **95% CI** | **P-value** | **Kappa** |
| **RA reservoir strain%** | 0.08 | -0.65 to 0.83 | 0.8 | 0.98 | 0.96 to 0.99 | <0.0001 | 0.78 |
| **RA booster strain %** | -0.11 | -0.5 to 0.30 | 0.5 | 0.97 | 0.91 to 0.99 | <0.0001 | 0.79 |
| **RV basal strain%** | 0.33 | -0.3 to 0.95 | 0.2 | 0.99 | 0.96 to 0.99 | <0.0001 | 0.78 |
| **RV mid wall strain%** | -0.25 | -0.7 to 0.20 | 0.2 | 0.99 | 0.96 to 0.99 | <0.0001 | 0.83 |
| **RV apical strain%** | -0.01 | -0.99 to 0.97 | 0.9 | 0.92 | 0.78 to 0.97 | <0.0001 | 0.68 |

**Table 5:** Intra-observer Variability

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **2D strain measures** | **Bias** | **95% CI** | **P-value** | **R2** | **95% CI** | **P-value** | **Kappa** |
| **RA reservoir strain%** | -0.14 | -0.78 to 0.5 | 0.6 | 0.99 | 0.97 to 0.99 | <0.0001 | 0.85 |
| **RA booster strain %** | -0.40 | -0.9 to 0.12 | 0.1 | 0.98 | 0.95 to 0.99 | <0.0001 | 0.79 |
| **RV basal strain%** | 0.40 | -0.3 to 1.0 | 0.2 | 0.97 | 0.94 to 0.99 | <0.0001 | 0.80 |
| **RV mid wall strain%** | -0.30 | -0.96 to 0.3 | 0.3 | 0.97 | 0.93 to 0.99 | <0.0001 | 0.84 |
| **RV apical strain%** | -0.20 | -0.9 to 0.6 | 0.6 | 0.97 | 0.93 to 0.99 | <0.0001 | 0.81 |

2D: two-dimensional; CI: confidence intervals; R2: coefficient of determination; RA: right atrium; RV: right ventricle.

**Figure Legends**

**Figure 1:** Two-dimensional speckle tracking (STE) obtained right atrial strain curves.

Left panel: right atrial tracking by STE.

Right panel: right atrial strain curve showing reservoir, conduit and booster strain.

**Figure 2:** right atrial strain curves obtained from a patient in sinus rhythm vs atrial fibrillation

Left panel: good booster function represented by the curve below the zero line in a patient with restoration of sinus rhythm.

Right panel: markedly lower booster function in atrial fibrillation recurrence.

The shifting zero reference line was set in sinus rhythm at the beginning of the P wave whilst in patients with atrial fibrillation at the start of QRS wave. This may have caused some distortion of the strain profile.(1)

**Figure 3:** right ventricular strain curves obtained from a patient in sinus rhythm vs atrial fibrillation

Panel A: longitudinal strain from the apical four chamber view in a patient in sinus rhythm vs AF, Panel B.

**Figure 4:** Bland Altman and scatter plots for right atrial reservoir, booster and basal right ventricular wall strain: left panel inter-observer; right panel intra –observer plots.

a) Right atrial booster strain; b) Right atrial reservoir strain; c) Right ventricular basal strain

**Figure 5:** Bar diagram (mean ± SEM) showing the difference of echocardiographic variables in AF (atrial fibrillation) and in SR (sinus rhythm) measured at one year. SEM: standard error of the mean; LVEF: left ventricular ejection fraction; RV: right ventricle; RA: right atrium; RA MIN D: minimum diameter of the right atrium measured in the 4-chamber apical view at end-systole; 4CH: 4-chamber apical view; RVOT: right ventricular outflow tract; LAX: long-axis parasternal view; E/E´ ratio: surrogate of left ventricular filling pressure; PASP: pulmonary artery systolic pressure.

\* <0.05 (AF vs SR).