MEETING REPORT

Report from the Annual Conference of the British Society of Echocardiography, November 2016, Queen Elizabeth II Conference Centre, London

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Foreword

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It was a pleasure to welcome delegates to the annual conference of the British Society of Echocardiography (BSE) in November 2016. This was held at the prestigious Queen Elizabeth II Conference Centre in the City of London, with fabulous views directly out over Westminster Cathedral and the Houses of Parliament. There was an extremely full and varied program over 2 days meaning that delegates had little time to go sightseeing! The numbers of delegates attending the conference has increased every year and 2016 was no exception, with almost a thousand delegates and 26 exhibitors, including representatives from all the major echocardiography companies. Lectures were spread over three auditoria during the conference, with parallel sessions meaning that there were plenty of options from which to choose. In addition, there were practical training sessions using simulators for hands-on experience, and live discussions during the breaks on hot topics in echocardiography, both technical and societal.

In this supplement for Echo Research and Practice, we have summarized some of the highlights of the conference. These include the invited lecture from our national speaker, Prof. Martin Cowie who proved incredibly popular with the delegates. We have also summarized the lectures that were given in the session sponsored by Echo Research and Practice. These lectures were by leaders in their field and are a reflection of the high-quality, evidence-based research and practice that is presented at our conference and that makes up the contents of the journal. Finally, we have included some of the high-quality abstracts that were presented during the conference. For the main abstract session, this includes the winning abstract by Janaki Srinivasan, investigating deformation changes that occur in Takutsubo cardiomyopathy. In support of our future researchers, we have included the abstracts from the Scientific Training Programme (STP) day, including the winning abstract from Dean Thomas.

The 2017 conference will take place on the 10th and 11th November in Edinburgh, at the Edinburgh International Conference Centre. We are delighted to welcome Allan Klein, current President of the American Society of Echocardiography, as our invited international speaker on Saturday 11th November 2017. We look forward to seeing you there!

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National Invited Lecture 2016

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The heart failure epidemic: a UK perspective

There are more than half a million people living with heart failure (HF) in the United Kingdom, and the NHS spends 1–2% of its budget on this (60–70% related to the costs of hospitalization). Echocardiography is key to the diagnosis of HF, being the most readily available cardiac imaging modality.

In the most recently available data from the national (hospital) audit, echocardiography was performed in around 90% of hospitalized patients, with the use higher in those patients admitted to a cardiac ward (around 50% of patients) than those admitted to a more general ward. HF specialist nurses reach an additional 20% of patients.

The echocardiographic data collected in the national audit are not detailed, but it shows that 70% have obvious LV systolic dysfunction. Valve disease is very common (over 30% of cases), with isolated diastolic dysfunction recorded for around 10% of cases.

The National Institute for Health and Care Excellence (NICE) has issued clear guidance to the NHS in England as to what good care looks like for both chronic and acute (hospitalized) HF. It has also issued Quality Standards so that health care services, commissioners, patients and the public can judge the quality of care using a small number of carefully chosen metrics.

For chronic HF, people with suspected HF should have an echocardiogram and specialist assessment. If the patient has had a previous myocardial infarction or has a very high level of serum natriuretic peptides, then the echocardiogram and assessment need to be delivered within 2 weeks. NICE does not stipulate the location of the imaging, leaving this up to local services to determine whether community or hospital-based facilities are the more appropriate.

For acute (hospitalized) HF, new cases should have a measurement of natriuretic peptide in the emergency room or acute admissions unit, and if this elevated and HF is suspected then an echocardiogram should be performed within 48 h of admission. Also, such patients should have input from the specialist HF team within 24 h of admission.

Major issues remain regarding access to diagnostic tests in primary and secondary care, with huge variability from one part of the country to another. The All Party Parliamentary Group on Heart Disease has issued a report entitled ‘Focus on heart failure’. In its submission to that group of MPs and peers, the Alliance for Heart Failure highlighted the shortage of echocardiographers in the UK. The current manpower issues for echocardiography must be overcome if the NHS can deliver the quality of care that NICE, health care professionals and patients demand.

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Prof. Cowie is a Non-Executive Director of the National Institute for Health and Care Excellence, but the opinions expressed in this article are his own and should not be taken to represent the official position of NICE.

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Right ventricular function in tricuspid regurgitation
Bushra S Rana

The natural history and treatment of tricuspid regurgitation (TR) remains poorly understood. Right ventricular function is a key factor in determining prognosis, timing for intervention and longer-term outcome. Right ventricular (RV) morphology underpins RV function. The RV is a thin-walled chamber with a predominance of longitudinal fibers and a shared ventricular septum. In health, the low pressure system of the pulmonary circulation generates a highly compliant RV that is well equipped to respond to changes in preload but sensitive to even small alterations in afterload. Key principles of ventricular function assessment are emphasized and highlight the importance of RV size, volumes and ejection fraction, particularly in risk stratification in TR.

The paucity of published data reflects some uncertainty in the management of TR and timing of intervention. Historic data have suggested surgical treatment of TR carries high mortality. However, these data reflect the cohort studied, namely residual TR seen some years post mitral valve surgery. These data emphasize the importance of understanding the etiology of TR and defining more precisely RV dysfunction. Early surgery should be considered in primary severe TR with RV dilatation even prior to significant symptoms and emphasizes the importance of functional assessment and exercise contractile reserve. Functional severe TR represents a different cohort of patients who have pulmonary hypertension and/or primary RV dysfunction responsible for annular dilatation and severe secondary TR. Those presenting with RV failure reflect a potentially high-risk group with often complex disease, involving RV dysfunction, pulmonary hypertension and hepatic and renal complications, with additional pulmonary and left heart disease who may have undergone previous cardiac surgery. Careful assessment and work up are needed to ensure intervention is performed in a timely and appropriate manner.

The left ventricle in aortic stenosis
John B Chambers

The left ventricle (LV) responds to pressure load by myocyte hypertrophy, which is a means of minimizing increased wall stress. Unfortunately, this process is also accompanied by fibrosis and this is maladaptive. Left ventricular hypertrophy (LVH) is associated with an increased risk of adverse events before and after surgery. LVH occurs once a threshold LV mass index is exceeded, often taken as 109 g/m² in women and 134 g/m² in men. However, some patients develop asymmetric hypertrophy mainly affecting the septal and anteroseptal segments. Others develop LV cavity dilatation and hypokinesis at end-stage or relatively early in their natural history.

What determines these different patterns of LV response? The grade of valve obstruction only affects LV geometry and function in severe aortic stenosis. Systemic hypertension is also a major determinant particularly of asymmetric hypertrophy. Gender exerts an important effect. Women tend to develop thick-walled, but small LV cavities while men are more likely to develop thinner more dilated LV cavities. This may be because the pattern of collagen deposition is different between the two sexes. Coronary disease is associated with LV cavity dilatation and hypokinesis even with subsurgical disease. A genetic component is likely to explain about 30% of variability.
in wall thickness in white and 40% in black patients. The nature of the genetic influence is still not known although the angiotensin-converting enzyme is a likely candidate. It is also possible that there is a relationship with hypertrophic cardiomyopathy, which is more common in relatives of patients with asymmetric hypertrophy than those without.

What is the clinical significance of this? The LV response is variable with severe LV systolic or diastolic dysfunction developing as a result of severe aortic stenosis in some patients but in the presence of more moderate aortic stenosis in others. At its most extreme is the situation of low gradient normal EF severe aortic stenosis where there are effectively two separate disease processes, HFPEF (heart failure with preserved ejection fraction) and aortic stenosis. This usually develops without the intermediate stage of high-gradient normal aortic stenosis and responds less well than usual to invasive therapy. To prevent this increasingly common entity, we probably need to treat systemic hypertension as a potential cause of LV dysfunction much earlier than we do now and using angiotensin-converting enzyme inhibitors.

Monitoring the heart during mitral regurgitation

Simon Ray

Severe primary mitral regurgitation (MR) imposes a state of volume overload on the left ventricle (LV). The ventricle is able to adapt to this state with an increase in end diastolic volume to accommodate the increased stroke volume of severe MR while maintaining a normal end-systolic wall stress. This phase of ‘compensated’ MR may be apparently stable for months or years but if the regurgitation continues unchecked then at some point left ventricular dilation becomes progressive, end-systolic volumes and wall stress begin to increase and ejection fraction to fall. It has been recognized for years that once this ‘decompensated’ phase is reached, or patients become significantly symptomatic, survival is reduced even if the valve is then repaired or replaced. Therefore, patients need to be referred for surgery before this stage is reached and echocardiography is used to monitor serial changes in the LV – the so-called ‘watchful waiting’ strategy.

Conventionally, end-systolic diameter and LV ejection fraction have been the principal measures used to identify incipient decompensation with end-systolic dimension (ESD) of ≥40mm or an LV ejection fraction (EF) of ≤60% considered Class 1 indicators for surgery in reparable valves. The most recent guidelines from the American Heart Association/American College of Cardiology categorize asymptomatic patients in this phase as being in stage C2 of MR as distinct from stage C1 where individuals are asymptomatic with ESD of <40mm and EF >60%. This is a useful conceptual model.

There is however some evidence that reliance on these simple parameters to refer patients for surgery may miss important degrees of LV dysfunction that only become apparent postoperatively when the MR has been abolished. Various methods have been used in an attempt to refine the identification incipient transition to stage C2. These include exercise echo, longitudinal function using speckle tracking to derive global longitudinal strain (GLS), left atrial function, cardiac magnetic resonance (CMR) imaging and biomarkers. Of these, the most promising is probably GLS with a value of around >−20% indicative of LV dysfunction postoperatively in patients referred for surgery.

In practice, a holistic approach is required. Conventional parameters of ESD and LVEF should be monitored scrupulously on a regular basis, at least annually. The addition of GLS can be useful in echo labs that are experienced in its use and in patients where image quality is good. Left atrial (LA) volumes are useful as if the LA is not enlarged then the MR is not chronic and severe. BNP measurements may be useful as if normal they provide reassurance that the LV is compensated. There should be a low threshold for symptomatic exercise testing to identify patients who are not truly asymptomatic and for confirmatory testing with CMR. There is emerging evidence that a regurgitant volume of >55 mL or a regurgitant fraction of >40% on CMR are makers of a worse prognosis.

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Abstract 1: Left ventricular mechano-temporal alterations during the apparent recovery of acute stress-induced (Tako-tsubo) cardiomyopathy

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Aims

Tako-tsubo syndrome is an increasingly recognized cause of chest pain and occasionally of cardiogenic shock. However, the recovery course of this condition remains unresolved. Here, we describe the medium-term recovery in Tako-tsubo patients presenting predominantly with ST-elevation type ECG or malignant arrhythmias and with apical ballooning.

Methods and results

We prospectively recruited 47 patients with a clear-cut diagnosis of Tako-tsubo and 28 healthy controls of the same age, gender and cardiovascular co-morbidity distribution. Patients underwent comprehensive assessment of left ventricular (LV) myocardial deformation acutely and at 4-month follow-up. The patients’ mean age was 66 years (range 41–87) and 92% were women. Most of the abnormal echocardiographic indices observed acutely in Tako-tsubo patients improved significantly at follow-up. However, significant mechano-temporal alterations characterizing both systole (global longitudinal strain, apical circumferential strain, both \(P<0.01\); left ventricular twist/twist rate and torsion, all \(P<0.001\)) and diastole (untwist rate and time to peak untwisting, all \(P<0.001\)) persisted at 4-month follow-up when compared with controls, despite normalization of LV ejection fraction and end-diastolic volume.

Conclusion

In patients with the most clinically severe spectrum of Tako-tsubo cardiomyopathy, regional LV systolic and diastolic deformation abnormalities persist beyond the acute event, despite normalization of global LV function and size.

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Abstract 2: Right ventricular structure and function in veteran ultrarunners: is there evidence for chronic maladaptation?

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Purpose

It has been proposed that long-term participation in prolonged strenuous exercise may result in maladaptation of the right ventricle (RV). The aim of the study is to quantify size and function of the RV in a veteran population of ultrarunners and compare these findings to a sedentary control group. We hypothesize that ultrarunners will have larger RVs and impaired regional myocardial function.

Methods

40 ultrarunners and 24 age- and gender-matched sedentary controls (Table 1) volunteered to take part in the study. Conventional 2D and tissue Doppler (TDI) echocardiography were used to establish RV size and function. Structural data were scaled allometrically based on the rules of geometric similarity. Myocardial speckle tracking echocardiography was used to determine peak global and regional strain ($\varepsilon$) of the RV lateral free wall. Regional assessment of RV insertion points were made at the basal and apical anteroseptum and infersoseptum using longitudinal and circumferential left ventricular $\varepsilon$.

Results

All conventional RV parameters are presented in Table 2. Scaled structural parameters of RV size were significantly larger ($P<0.05$) in ultrarunners compared to controls. Ultrarunners also had significantly higher RV fractional area change (FAC) and tissue Doppler (TDI) $S'$, $E'$ and $A'$. There were no significant differences in RV regional or global $\varepsilon$ between the two groups. There was significantly higher basal circumferential $\varepsilon$ at the RV insertion points in ultrarunners compared to controls, as well as higher longitudinal $\varepsilon$ at the apical anteroseptum (Table 3).

| Parameter                  | Ultrarunners ($n = 40$) (mean ± s.d.) | Controls ($n = 24$) (mean ± s.d.) | $P$ value |
|---------------------------|-------------------------------------|----------------------------------|-----------|
| Age (years)               | 46 ± 8                              | 46 ± 7                           | 0.986     |
| Gender (%)                | M = 82.5%, F = 17.5%                | M = 83.3%, F = 16.7%             |           |
| Height (cm)               | 177 ± 8                             | 174 ± 7                          | 0.112     |
| Weight (kg)               | 71.3 ± 10.1                         | 85 ± 14                          | <0.001    |
| Body surface area         | 1.87 ± 0.16                         | 2.02 ± 0.19                      | 0.002     |
| Training (years)          | 18 ± 12                             | 4 ± 11                           |           |
| Training (h/week)         | 11 ± 4                              | 1 ± 1                            |           |
| Number of marathons       | 61 ± 80                             | 0                                |           |
Conclusion

This study demonstrates RV enlargement with higher conventional measures of function in veteran ultrarunners compared to age- and gender-matched controls. There is no evidence of maladaptation as demonstrated with normal and in some cases superior measures of global, regional and insertion point deformation. Our data suggest that regular exposure to endurance exercise has no negative consequences for the RV in the veteran ultrarunner population.

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Table 2  Conventional echocardiographic data of RV structural and functional parameters from ultrarunners and a sedentary age-matched control group.

| Parameter                        | Ultrarunners (n=40) (mean±s.o.) | Controls (n=24) (mean±s.o.) | P value |
|----------------------------------|----------------------------------|-----------------------------|---------|
| RVOTPLAX index (mm/(m²)⁰.⁵)      | 23 ± 3                           | 21 ± 2.7                    | 0.043   |
| RVOT1 index (mm/(m²)⁰.⁵)         | 24 ± 3.4                         | 21 ± 2.6                    | <0.001  |
| RVOT2 index (mm/(m²)⁰.⁵)         | 18 ± 1.8                         | 16 ± 1.9                    | 0.003   |
| RVD1 index (mm/(m²)⁰.⁵)          | 31 ± 3.8                         | 26 ± 3.6                    | <0.001  |
| RVD2 index (mm/(m²)⁰.⁵)          | 21 ± 3.4                         | 16 ± 2.6                    | <0.001  |
| RVD3 index (mm/(m²)⁰.⁵)          | 64 ± 5.4                         | 55 ± 5.6                    | <0.001  |
| RVD area index (cm²/m²)          | 13 ± 2.4                         | 9 ± 2.3                     | <0.001  |
| RVS area index (cm²/m²)          | 7 ± 1.9                          | 5 ± 1.5                     | <0.001  |
| RA volume index (mL/(m²)¹.⁵)     | 25 ± 10.8                        | 12 ± 4.9                    | <0.001  |
| RVFAC (%)                        | 50 ± 0.1                         | 44 ± 0.1                    | 0.013   |
| TAPSE (mm)                       | 24 ± 4                           | 23 ± 3.6                    | 0.321   |
| TDI S’ (cm/s)                    | 17 ± 2.9                         | 13 ± 1.7                    | <0.001  |
| TDI E’ (cm/s)                    | 15 ± 3.2                         | 12 ± 2.8                    | <0.001  |
| TDI A’ (cm/s)                    | 1.83 ± 0.46                      | 1.54 ± 0.40                 | 0.019   |
| RA, right atrial; RV, right ventricular; RVD, RV dimensions 1 (base) 2 (mid) 3 (longitudinal); RVD and RVS area, RV diastolic and systolic area; RVFAC, RV fractional area change; RVOT1 and 2, RV outflow tract 1 (proximal) and 2 (distal); RVOTPLAX, RV outflow tract from parasternal long axis view; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue Doppler imaging during ventricular systole (S’) and during early (E’) and late (A’) ventricular diastole.

Table 3  Regional and global strain from the RV free wall and RV insertion points of ultrarunners and sedentary age-matched controls.

| Parameter                        | Ultrarunners (n=40) (mean±s.o.) | Controls (n=24) (mean±s.o.) | P value |
|----------------------------------|----------------------------------|-----------------------------|---------|
| RV basal ε (%)                   | −27 ± 4.5                        | −29 ± 4.3                   | 0.215   |
| RV mid ε (%)                     | −28 ± 3.8                        | −30 ± 4.5                   | 0.174   |
| RV apical ε (%)                  | −32 ± 4.4                        | −32 ± 4.7                   | 0.819   |
| RV base to apex gradient (%)     | 4.3 ± 5.4                        | 3.2 ± 5                     | 0.396   |
| LV circumferential               |                                  |                             |         |
| Basal anterior septum ε (%)      | −26 ± 7.9                        | −21 ± 5.7                   | <0.001  |
| Basal septum ε (%)               | −25 ± 6                          | −16 ± 9.3                   |         |
| LV radial                        |                                  |                             |         |
| Basal anterior septum ε (%)      | 39 ± 18.8                        | 34 ± 17.3                   | 0.259   |
| Basal septum ε (%)               | 43 ± 19                          | 38 ± 17.9                   | 0.117   |
| LV longitudinal                  |                                  |                             |         |
| Basal anterior septum ε (%)      | −19 ± 3.2                        | −18 ± 2.2                   | <0.001  |
| Apical anterior septum ε (%)     | −28 ± 6.8                        | −22 ± 4.3                   |         |

ε, indicates strain; LV, left ventricular; RV, right ventricular.
MEETING REPORT

Abstract 3: Feasibility, efficacy and safety of physiologist-led stress echocardiography in a rapid access chest pain setting

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Introduction

A rapid access chest pain clinic (RACPC) ideally should entail same day clinical assessment and investigation for the detection of coronary artery disease (CAD) to allow rapid management plan implementation. Ease of accessibility, rapid performance time and real-time interpretation make stress echocardiography (SE) the ideal test compared to other non-invasive imaging in the RACPC setting. SE has been shown to be more efficacious (both for diagnosis and prognosis), safe and more cost-efficient than exercise ECG: it is the most widely used initial test for the assessment of CAD in the United Kingdom. We sought to assess whether physiologist-led SE, which is inherently cost-saving, in the setting of RACPC is feasible, efficacious and safe.

Methods and results

Between May 2014 and May 2015, 768 consecutive patients (mean pre-test probability of CAD 36 ± 1.3%) referred from RACPC underwent SE, performed by a team of trained physiologists with physician availability and with all tests being interpreted by the lead consultant. In total, 675 (88%) patients had same day SE. Of the 768 patients, 704 (92%) underwent treadmill exercise SE with dobutamine testing in the remainder. Diagnostic tests were achieved in 750 (98%) patients. Contrast was used in 725 (94%) patients. Significant adverse events were seen in only 2 (0.26%) patients. Of the 60 (8%) patients with inducible myocardial ischemia, 59 patients underwent coronary angiography. Flow-limiting CAD was demonstrated in 52 (88%) patients of whom 36 (69%) underwent revascularization. Of the 690 (92%) patients with normal SE, 2 (0.29%) suffered acute coronary syndrome over a follow-up period of at least 1 year.

Conclusion

Physiologist-led SE in the RACPC setting was highly feasible and safe, allowing rapid communication of SE results in almost 90% of patients and translated into a same day implementation of a management plan. SE also resulted in appropriate management, appropriate revascularization in those with positive SE and excellent outcome in those discharged with a negative SE. Given the high volume of referral to RACPC, physiologist-led SE is feasible, safe and efficacious, may be a cost-efficient alternative to other non-invasive diagnostic modalities.

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Abstract 4: Prognostic value of simultaneous stress echocardiography and carotid ultrasound in patients with suspected coronary artery disease

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Aims

The clinical significance of carotid disease in patients undergoing stress echocardiography (SE) for new-onset chest pain without known coronary artery disease (CAD) is unknown. We hypothesized that information of ischemia and atherosclerosis, which can be achieved simultaneously by ultrasound, may provide incremental prognostic information in these patients.

Methods

Consecutive patients with no previous history of CAD investigated with SE for suspected angina underwent carotid ultrasound (US) prospectively. Carotid plaque presence and burden were assessed. Patients were followed up for combined major adverse events (MAE) of all-cause mortality, non-fatal myocardial infarction and unplanned coronary revascularization.

Results

Out of 591 recruited patients, 580 (98%) (269 male (46%), mean age 59 ± 11 years) were available for follow-up. SE demonstrated myocardial ischemia in 12%, but prevalence of carotid plaques was 59%. During a mean
follow-up of 1117±361 days, 40 first MAE occurred. In the multivariable regression model, only abnormal SE ($P<0.0001$) and carotid plaque burden ($P<0.0001$) predicted MAE. MAE rate/year increased from 0.9% vs 1.97% vs 4.3% vs 9.7% in patients with no carotid plaque and normal SE vs presence of plaque and normal SE vs no plaque and abnormal SE vs presence of plaque and abnormal SE, respectively ($P<0.0001$). In hierarchical analysis, plaque burden provided incremental prognostic value over pre-test probability of CAD and SE (Fig. 1), likewise SE was incremental to pre-test probability of CAD and plaque burden ($P<0.0001$ for both) (Fig. 2).

**Conclusion**

In patients with suspected stable angina without known CAD, simultaneous SE (for ischemia) and US (for atherosclerosis) provided synergistic prognostic value.

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Abstract 5: Long-term echocardiographic follow-up in transcatheter aortic valve implantation

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Purpose

Transcatheter aortic valve implantation (TAVI) is a well-established procedure for treating severe aortic stenosis in elderly, non-surgical cases; however, long-term echocardiographic data is lacking. This study aims to collate several key echocardiographic parameters over long-term follow-up and highlight incidence rates of valvular degeneration.

Methods

49 patients were selected who had undergone TAVI (Sapien; Sapien XT; Sapien 3) and were followed up within the Papworth physiologist-led valve service (Table 4). Retrospective analysis was performed on standard 2D/Doppler-derived echo data and compared during three follow-up stages (FU1 1 year post TAVI, FU2 2 years post, FU3 3 years post) and between different sizes of TAVI valve implanted (Table 5).

Table 4  Baseline characteristics (n=49).

| Parameter                              | Value          |
|----------------------------------------|----------------|
| Age (years)                            | 79 ± 7         |
| Male                                   | 42 (86)        |
| Diabetes                               | 15 (31)        |
| Hypertension                           | 18 (37)        |
| Coronary artery disease                | 39 (80)        |
| Previous myocardial infarction         | 5 (10)         |
| Previous open heart surgery            | 37 (76)        |
| Previous cerebrovascular accident      | 7 (14)         |
| COPD                                   | 3 (6)          |
| Atrial fibrillation                    | 13 (27)        |
| Anticoagulation                        | 13 (27)        |
| Antiplatelet                           | 42 (86)        |
| Trans-femoral TAVI                     | 41 (84)        |
| TAVI size 23mm                         | 9 (18)         |
| TAVI size 26mm                         | 20 (41)        |
| TAVI size 29mm                         | 20 (41)        |

Values are mean ± s.d. or n (%). Statistical analysis by Students paired t-test. Bold indicates the mean value.

Table 5  Mean echo parameters (baseline to FU2).

|                  | FU1 (n=49) | FU2 (n=47) | FU3 (n=26) | Change FU1–FU3 (%) | P value (FU1–FU2; FU1–FU3; FU2–FU3) |
|------------------|------------|------------|------------|--------------------|---------------------------------------|
| PPG (mmHg)       | 21.8 ± 13  | 22.2 ± 12.6| 23.4 ± 15.4| 7.5                | 0.91; 0.72; 0.83                       |
| MPG (mmHg)       | 11.5 ± 7.2 | 12.1 ± 7.3 | 13.3 ± 9.3 | 15.5               | 0.45; 0.80; 0.65                       |
| AVA (cm²)        | 1.70 ± 0.59| 1.61 ± 0.50| 1.69 ± 0.51| −0.4               | 0.19; 0.70; 0.56                       |
| AVAi (cm²/m²)    | 0.87 ± 0.34| 0.84 ± 0.24| 0.87 ± 0.27| 0                  | 0.48; 0.71; 0.53                       |
| VR               | 0.5 ± 0.15 | 0.45 ± 0.12| 0.5 ± 0.15 | 0                  | 0.11; 0.55; 0.17                       |
| EF (%)           | 50.8 ± 9.1 | 50.6 ± 9.6 | 54.3 ± 7.8 | 7.0                | 0.35; 0.12; 0.04                       |
| SVI (mL/m²)      | 41.4 ± 14  | 38.7 ± 10.3| 39.7 ± 8.5 | −4.1               | 0.21; 0.52; 0.56                       |
| sPAP (mmHg)      | 36.1 ± 9.6 | 33.5 ± 9.27| 34.3 ± 7.1 | −5.0               | 0.32; 0.76; 0.77                       |

Values are mean ± s.d. or n (%). Statistical analysis by Students paired t-test. Bold indicates the mean value.
Results

26 patients reached FU3 (53%), with a total mortality rate of 8%. There were no notable changes seen across traditional mean prosthetic valve Doppler measurements from FU1 to FU3 (P=0.05), while LVEF improved slightly between FU2 and FU3 (51–54%; P=0.04). Secondary analysis comparing different valve sizes indicated that the smaller valve size (23 mm) was slightly more obstructive, reflected by higher peak/mean pressure gradients and lower aortic valve area (AVA), AVA index and velocity ratio (Figs. 3, 4, 5, 6 and 7). At FU1, the incidence of paravalvular leak (PVL) was 53%, of which 16% of cases were considered more significant ( ≥ moderate) (Fig. 8). A 3% increase in incidence of significant PVL was noted from FU1 to FU3, while the 29 mm valve size showed the greatest increase in incidence of PVL from FU1 to FU3 (10–21%) (Fig. 9). 3 patients were identified to have developed thrombus formation on their valve (6%), with significantly obstructive valvular parameters, which then resolved somewhat upon commencing anticoagulation therapy.

Conclusion

Our data show a range of traditional prosthetic valve Doppler values, which may be used to guide sonographers when assessing different TAVI valves and highlight rates of significant valvular degeneration. Further studies with larger sample sizes than the present study are required to further assess the validity of these findings.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this article.

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Figure 7
VR by valve size (FU1–FU3).

Figure 8
Paravalvular leak incidence from FU1 to FU3 ($\eta$).

Figure 9
Paravalvular leak incidence from FU1 to FU3 accounting for valve size.

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MEETING REPORT

Abstract 6: The CHA₂DS₂VASc risk score appropriately risk stratifies patients prior to atrial fibrillation ablation and reduces the requirement for trans-esophageal echocardiography

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Background

Prior to atrial fibrillation (AF) ablation, current recommendations suggest patients undergo trans-esophageal echocardiography (TOE) to exclude left atrial appendage (LAA) thrombus. Patients undergoing AF ablation are usually anticoagulated thus making the presence of thrombus unlikely. This study aimed to determine whether the CHA₂DS₂VASc scoring system can be used for risk stratification to identify patients that do not require TOE prior to AF ablation.

Methods

In this single-center retrospective cohort study, local institutional and primary care databases and electronic patient records were searched to identify patients that had undergone TOE prior to AF ablation and also correlated with cath lab records of these patients. Patient demographics, CHA₂DS₂VASc score, TOE findings and anti-coagulation status were collected retrospectively.

Results

Over a 7-year period (2008–2014), 346 patients underwent TOE prior to proposed AF ablation of which 14 patients were excluded due to incomplete data, leaving a total sample size of 332 patients (age 57 ± 10 years; 74% male). CHA₂DS₂VASc scores of 0, 1, 2 and >2 were found in 39, 34, 15 and 12% of patients respectively. The prevalence of LAA thrombus was 0.6% (2 patients) and these 2 patients had risk scores of 2 and 4. No patients with a score of 0 or 1 had LAA thrombus.

Conclusions

Patients that are classed as low risk by the CHA₂DS₂VASc score (score of 0 or 1) do not require a pre-ablation TOE to screen for LAA thrombus provided they have been on anti-coagulation with a stable therapeutic INR. This would lead to a significant reduction in health care expenditures by reducing unnecessary TOE requests and also thereby improve patient experience.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this article.

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The role of strain imaging in assessing CRT response

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Background

Despite advancements in research, cardiac resynchronization therapy (CRT) response is still defined clinically by symptomatic improvement. Speckle tracking strain imaging with its ability to evaluate left ventricular (LV) dysfunction and dyssynchrony would provide an ideal parameter to assess CRT response.

Aim

To validate speckle tracking strain imaging for assessing the difference in LV dysfunction and dyssynchrony between responders and non-responders following 3 months of CRT. To examine the relationship between symptomatic response and physiological response to CRT.

Methods

In 28 patients, echocardiography and Minnesota Living with Heart Failure Questionnaire (MLHFQ) was performed at baseline and following 3 months of CRT. Response to CRT was defined as a \( \geq 15\% \) reduction in MLHFQ score between baseline and 3 months. Speckle tracking strain imaging was used to assess LV dysfunction and dyssynchrony.

Results

At follow-up, 79\% of patients were classified as responders with 21\% as non-responders to CRT. A significant difference in baseline global longitudinal strain (GLS) was found between responders and non-responders (mean 8.2 ± 2.72\% vs 6.22 ± 1.73\%; \( P < 0.05 \)). There was an insignificant difference in GLS, time to peak radial and longitudinal strain between responders and non-responders following 3 months of CRT (\( P > 0.05 \)). The percentage of biventricular pacing was greater in responders vs non-responders (mean 96.2 ± 4.1\% vs 88.8 ± 13.2\%; \( P = 0.03 \)). 3.8\% of the change in symptomatic response is accounted for by change in GLS.

Conclusion

Both responders and non-responders have a mechanical response to CRT. GLS can predict response to CRT. Assessment of biventricular pacing at follow-up is important factor influencing response.

Declaration of interest

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