



Left ventricular vortex formation time in elite athletes

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Abstract

Vortex formation time (VFT) is a continuous measure of the left ventricular (LV) filling that integrates all phases of diastole. This has been previously studied in patients with heart failure. This study examined the differences in VFT between healthy controls and elite athletes. We compared echocardiographic indices between elite male athletes ($n=41$) and age-, weight- and sex-matched sedentary volunteers ($n=22$). VFT was obtained using the validated formula: $4 \times (1 - \beta) / \pi \times \alpha^3 \times \text{LVEF}$, where β is the fraction of total transmitral diastolic stroke volume contributed by atrial contraction (assessed by time velocity integral of the mitral E- and A-waves) and α is the biplane end-diastolic volume (EDV)^{1/3} divided by mitral annular diameter during early diastole. Diastolic function was measured by the ratio of mitral peak velocity of early filling (E) to early diastolic mitral annular velocity (e') (E/ e' ratio) and the ratio of E to mitral peak velocity of late filling (A) (E/A ratio). The heart rate was lower (63 ± 10 vs. 74 ± 6 beats per minute, $p < 0.001$) and the LV end diastolic diameter was larger in athletes as compared to controls (56 ± 3 vs. 50 ± 4 mm, $p < 0.001$). The VFT was lower in the sedentary group compared to athletes (3.1 ± 0.4 vs. 4.0 ± 0.8 , $p < 0.001$). Similarly, E/ e' was higher in sedentary controls compared to athletes (7.5 ± 1.8 vs 4.2 ± 1.0 , $p < 0.001$). Furthermore, there was a modest correlation between VFT and E/A ($r=0.47$, $p < 0.001$) as well as E/ e' ($r = -0.33$, $p=0.012$). In conclusion, the VFT was elevated among elite athletes compared to healthy sedentary controls.

Keywords Vortex formation time · Athletes · Left ventricular fluid dynamics · Diastolic function · Echocardiography

Background

Vortex formation time as assessed by echocardiography has been found to have important clinical applications [1]. Indeed, conventional echocardiography including Doppler tissue imaging is the modality of choice in the assessment of athletic cardiac physiology [2–4]. Quantification of diastolic

function often provides insights on the athletic heart. However, current methods for measurement of diastolic function have limitations. Specifically, the transmitral inflow Doppler profile may pseudonormalise with progression from normal to prolonged LV relaxation and before restrictive pattern [5]. In addition, tissue Doppler assessments are angle-dependent and often focus only on the mitral annular motion and neglect blood flow from the left atrium to the ventricle [6]. It has been documented that during early LV diastolic filling a rotating fluid mass called vortex ring was formed [7, 8]. The use of a vortex ring for fluid transport is more energy efficient than that by a steady straight jet of fluid [9]. Gharib et al. have demonstrated that vortex formation time (VFT) was a dimensionless measure of the time needed for optimal vortex ring formation and has been shown to be a measure of left ventricular filling efficiency. An abnormal VFT by echocardiography has been shown previously to reliably differentiate between healthy control subjects and those with heart failure patients [1, 10, 11].

Pathological left ventricular hypertrophy as a response to hypertension, or valvular disease, has been associated with impaired diastolic relaxation, diastolic dysfunction and

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symptomatic heart failure [12, 13]. Conversely, prior work has demonstrated that despite thicker left ventricular walls, elite athletes had enhanced myocardial relaxation [14]. Vortex formation time has not been previously assessed in elite athletes. We hypothesized that VFT would be significantly increased among elite athletes over healthy controls and may be related to cardiac adaptation as a response to exercise and training.

Methods

Study population and data collection

Transthoracic echocardiography was performed in 41 elite male athletes. Athletes were competitive inter-county Hurlers from County Kilkenny, Ireland. The Hurlers were actively involved in training regimes during the time course of the study and no hurler was accepted into the study if training had ceased for more than 24 h prior to the study.

At the time of data acquisition, all hurlers were involved in preparation phase training programmes which involved 3–8 hr of three low intensity base aerobic conditioning sessions per week, training in hurling techniques, alternating with 2–3 hr per week of higher intensity aerobic work in or around the lactate anaerobic threshold. In addition, most hurlers participated in up to three resistance weight training and core training sessions. Running consisted of 18–25 runs at 80–85% of maximal predicted heart rate. Runs could be 100 m and 20 sec intervals of rest between them and also core and recovery sessions followed by weights sessions. Group sessions consist of weight and core training to include Hurling drills, and running. This weekly activity is followed by end of week matches. The hurlers also performed some aerobic cross training such as running, cycling or swimming on active recovery days.

The control population consisted of 22 normal sedentary subjects. One age-, weight- and sex-matched control was recruited approximately for every two athletes recruited. These controls comprised of healthy hospital staff and their relatives or acquaintances, who were all non-athletes and all gave informed consent for participation in this study. Conduct of this study was in a hospital in County Kilkenny, Ireland.

At the time of echocardiographic examination, the subject's height, weight, heart rate and blood pressures were recorded. All the subjects studied had undergone comprehensive echocardiographic study which included two-dimensional, pulsed, continuous Doppler as well as myocardial tissue Doppler studies. The mitral annulus diameter (D, mm) was measured in early diastolic from the four-chamber view. Left ventricular ejection fraction and end-diastolic volume were quantified by the biplane method of discs. VFT was

obtained using the formula: $4 \times (1 - \beta) / \pi \times \alpha^3 \times \text{LVEF}$, where β is the fraction of total transmitral diastolic stroke volume contributed by atrial contraction (assessed by time velocity integral of the mitral E- and A-waves) and α is the biplane end-diastolic volume (EDV)^{1/3} divided by mitral annular diameter during early diastole [1]. Measurements were made by qualified sonographers and reported by cardiologists who were blinded to the identity and category of the subjects studied. VFT was correlated with demographic, cardiac parameters, and compared with normal controls. This study was approved by the local hospital ethics committee and informed consent was obtained from all participants.

Statistical analyses

Descriptive statistics were presented as means (standard deviations, SD) for continuous data and number and percentage for categorical data. Comparisons between athletes and controls were made using t tests for normally distributed variables, Mann–Whitney U test for non-normal data and Chi square tests for categorical data. Multivariable regression analysis was used to adjust for the effect on confounding variables (if any) on the difference in VFT observed between the two groups. In examining the relationship between VFT and other echocardiographic parameters, correlations between continuous variables across the entire study population (athletes and controls) were made using Pearson's correlation. Analyses performed using SAS v9.3 and significance at $p < 0.05$ assumed.

Results

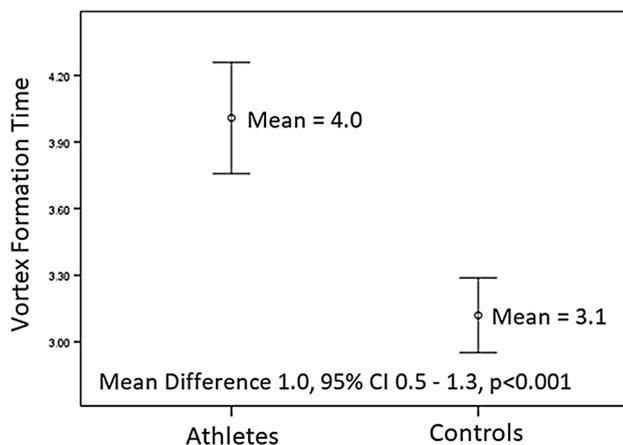
Forty-one athletes were compared against 22 age-, sex- and weight-matched controls. There were no significant differences in age, gender, body surface area, systolic or diastolic blood pressure between the controls and athletes (Table 1). The heart rate was significantly lower

Table 1 Baseline demographic and clinical parameters in athletes compared to controls

Parameter	Athletes (mean \pm SD) n=41	Control (mean \pm SD) n=22	P value
Age (years)	24 \pm 4.3	27 \pm 4	0.03
BMI (kg/m ²)	25.0 \pm 1.9	24.0 \pm 4.0	0.60
Body surface area (m ²)	1.97 \pm 0.19	1.89 \pm 0.19	0.27
LV mass (g)	214 \pm 39	187 \pm 37	0.05
LV mass index (g/m ²)	111 \pm 28	100 \pm 21	0.23
Systolic BP (mmHg)	134 \pm 10	129 \pm 6	0.07
Diastolic BP (mmHg)	81 \pm 6	81 \pm 6	0.86
Heart rate (min ⁻¹)	63 \pm 10	74 \pm 6	<0.001

Table 2 Differences in echocardiographic profile between athletes and controls

Parameter	Athletes, mean (\pm SD), n=41	Control, mean, (\pm SD), n=22	P value
LV ejection fraction (%)	67 \pm 6	70 \pm 6	0.04
Left atrial size (mm)	35 \pm 3	35 \pm 2	0.82
Left atrial volume (ml)	38 \pm 14	40 \pm 5	0.37
LV end diastolic diameter (mm)	56 \pm 3	50 \pm 4	<0.001
Septal size (mm)	10.1 \pm 1.0	8.0 \pm 0.9	0.001
Posterior wall size (mm)	9.0 \pm 0.9	8.0 \pm 0.8	0.001
Mitral annular diameter (mm)	34.3 \pm 2.2	35.0 \pm 2.0	0.109
Stroke volume (ml)	101 \pm 16.0	79 \pm 19	<0.001
End-systolic volume (ml)	52 \pm 11	41 \pm 12	0.001
Time velocity integral (cm/s)	5.2 \pm 1.8	5.3 \pm 1.0	0.79
Isovolumic relaxation time (ms)	75 \pm 5	80 \pm 3	0.014
E/A ratio	2.2 \pm 0.6	2.3 \pm 0.6	0.61
e' (cm/s)	20.1 \pm 2.4	14.7 \pm 1.6	<0.001
E/e'	4.2 \pm 1.0	7.5 \pm 1.8	<0.001
Vortex formation time	4.0 \pm 0.8	3.1 \pm 0.4	<0.001

**Fig. 1** Difference in vortex formation time between athletes and controls

(63 \pm 10 vs. 74 \pm 6 beats per minute, $p < 0.001$, Table 1) and the LV end diastolic diameter was significantly larger in athletes as compared to controls (56 \pm 3 vs. 50 \pm 4 mm, $p < 0.001$, Table 2). The VFT was significantly elevated in the athletes as compared to sedentary controls (4.0 \pm 0.8 vs. 3.1 \pm 0.4, $p < 0.001$, Table 2). The mean difference for VFT between athletes and controls was 1.0 (95% confidence interval 0.5–1.3, Fig. 1). On multivariable regression analysis adjusting for heart rate, the adjusted mean difference remained significantly elevated in athletes

compared to the sedentary controls (0.9, 95% confidence interval 0.5–1.1).

There was no significant correlation between VFT and age, gender, body surface area or tissue Doppler systolic myocardial velocities but it correlated positively with tissue Doppler early diastolic myocardial velocities ($r = 0.40$, $p < 0.001$). There was a significant difference in E/e' ratio between the sedentary (7.5 \pm 1.8) and athlete (4.2 \pm 1.0) groups ($p < 0.001$). There was a significant correlation between VFT and e' ($r = 0.46$, $p < 0.001$), VFT and E/e' ($r = -0.33$, $p = 0.012$), VFT and E/A ($r = 0.47$, $p < 0.001$), and VFT and LV mass ($r = 0.38$, $p = 0.002$).

Discussion

Tissue Doppler echocardiography and speckle tracking imaging have been used to distinguish maladaptive from physiological remodelling of the right and left ventricles of the elite athlete [15]. The physiological changes that occur with training are predicted to alter the passive pressure–volume characteristics of the left ventricle by reducing myocardial stiffness and increasing compliance [14, 15].

The preferred non-invasive method to assess the athletic heart and measurement of diastolic function has been transthoracic echocardiography [16]. Most of these indexes are empiric and are not based on mechanisms and are irrespective of causal relations. VFT is a composite index, and may be better than Doppler component parts along with deformation imaging as these do not provide insights on fluid mechanics within the LV. VFT is a novel index that could become a useful indicator of biological fluid transport efficiency [1, 10, 11].

There has been some controversy surrounding the use of VFT. Previous authors have suggested that VFT may not be a reliable index of ventricular function because of dynamic conditions within the LV. The VFT concept was based on hydrodynamic experiments with unconfined (large tank) flow, whereas the LV is a confined space of filling with dynamic conditions of expansion and relaxation within [17]. However, the work by Poh et al. demonstrated important clinical applications of VFT, with significant differences in VFT in patients with heart failure and preserved ejection fraction compared to controls, while Gharib et al. also tested VFT in patients with dilated cardiomyopathy [1, 9].

VFT showed a modest negative correlation with E/e' in our study. Indeed, we demonstrated that VFT was lower and E/e' was correspondingly higher in the sedentary group. E/e' is an important estimate of LV filling pressures which may correspond with diastolic dysfunction. From previous studies, recoil (Ea) occurs during isovolumic relaxation before filling, and thus the Doppler mitral annular tissue velocities and Doppler mitral inflow velocities are influenced by

preload conditions in a normal population. The dissociation between filling and recoil has been described, and the influence of preload on the rate of filling means that in normal or supra-normal conditions, E/e' and VFT would be unlikely to correlate [18]. Since this study examined healthy subjects that were unlikely to have diastolic dysfunction or elevated LV filling pressures, we observed only a modest negative correlation between VFT and E/e' .

Vortex formation may be affected by cardiac dysfunction and has been previously quantified using echocardiographic parameters to derive VFT. VFT ring formation in the LV cavity manifests nature's solution to the atrium to ventricular mass transfer problem [10, 11]. The blood flow inside the normal left ventricle is dynamically arranged with regions of straight and turning flow and this causes vortices to develop and disappear with the phases of the cardiac cycle. Flow within the left ventricular cavity can be described and divided into components. The total inflow into the left ventricle consists of direct and retained non-ejected flow. The inflow from the left atrium arrives with kinetic energy funnelling into the left ventricle, which then collides with the residual flow creating the vortex flow structures [6]. The process of vortex ring formation has been studied in *in vitro* experiments, where it has been demonstrated that fluid transport by vortex ring is more efficient than by a steady, straight jet of fluid [19].

While VFT has been shown to be reduced in heart failure, this study demonstrated that VFT was increased in athletes [1]. This study demonstrated using a physiological based expression of VFT that optimization of vortex filling times were present in elite athletes compared to normal controls [20]. Therefore the VFT may be a useful performance indicator. The index is dimensionless therefore it could be compared across different patient groups and may be interpreted without considering patients specific effects.

However, it was also important to note the significant difference in resting heart rate between the elite athletes and the control group. Although all echocardiographic studies were done at rest, it was not surprising that the elite athletes had a significantly slower resting heart rate compared to the sedentary controls. To our knowledge, no previous study has quantified the effect of heart rate on VFT. Nevertheless, the lower resting heart rate and consequently slightly longer duration of diastole may have been contributory to the higher VFT in athletes compared to controls. We adjusted for the effect of heart rate on multivariable analysis, and found that VFT still remained significantly higher in athletes compared to the control group.

Of note, LVEF was not dissimilar between athletes and controls. For performance athletes, there are “efficiencies” such as, for example, mechanical efficiency, metabolic efficiency, or blood transport (hemodynamic) efficiency, which may characterize the cardiac working regimen

[21]. These “efficiencies” may or may not be aligned with the results of conventional measurements of LV function such as LV ejection fraction (EF) and LV pressure. Cardiac adaptation and remodeling in athletes may have led to more optimal hemodynamic or fluid dynamic conditions suitable for formation of the vortex, leading to the observed differences in VFTs between the athletes studied and the controls. This study may form the basis for further research testing the association between VFT and performance measures, and thus whether VFT can differentiate between physiological and pathological cardiac adaptation.

Limitations

This study recruited a small-sized cohort comparing athletes of a specific sport to healthy controls. The applicability of the findings to cohorts of athletes in sports with different physical demands remained to be elucidated. Our study remains exploratory and evaluates the potential for VFT to distinguish athlete heart from sedentary controls.

Larger, prospective studies would be required to validate these findings and evaluate its clinical use. We also did not explore the correlation of VFT with sports performance measures, as it was not the focus on our study. Further study may also evaluate the effect of LV sphericity on difference in VFT, which was not evaluated in this study. A possible source of error may have arisen from the use of the approximated vortex formation definition of ‘T’ which incorporated the maximum mitral valve exit diameter rather than the time-dependent diameter. However the mitral exit diameter was easily measured images obtained from healthy athletes and normal controls and we consistently measured the diameter at the same phase of diastole.

Conclusion

VFT was demonstrated to be a novel measure of left ventricular filling efficiency. Athletes had more optimal vortex formation in the LV compared to healthy controls. Further research on the association between VFT and performance measures and whether VFT can differentiate between physiological and pathological cardiac adaptation are warranted.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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