Left ventricular vortex formation time in elite athletes: novel predictor of myocardial performance

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ABSTRACT

Background Efficient transportation of blood through the left ventricle (LV) during diastole depends on vortex formation. Vortex formation time (VFT) can be measured by echocardiography as a dimensionless index. As elite athletes have supranormal diastolic LV function, we aim to assess resting and post-exercise VFT in these athletes and hypothesised that VFT may predict myocardial performance immediately post-exercise.

Method Subjects were world class speedskaters training for the Winter Olympic Games. Echocardiographic measurements were obtained before and immediately after 3000 m of racing. VFT was computed as $4 \times (1 - \beta) \pi \alpha \times 1/3$ divided by mitral annular diameter during early diastole.

Results Baseline VFT was 2.6 ± 0.7 (n=24, age 22 ± 3 years, 67% males). Post-exercise, heart rates increased (64 ± 10 vs 89 ± 12 beats/min, p<0.01); however, VFT was unchanged (2.9 ± 1.0, p>0.05). VFT at rest correlated modestly with post-exertion early diastolic mitral inflow velocity (E; r=0.59, p=0.01), tissue Doppler-derived early mitral annular velocity (E'; septal and lateral, both r=0.59, p=0.01) and systolic annular velocity (S'; septal; r=0.46, p=0.01 and lateral; r=0.48, p=0.02) but not late diastolic mitral inflow velocity (A; r=0.06, p>0.05) or annular velocity (A'; septal; r=0.34, p=N5 and lateral: r=0.35, p>0.05).

Conclusion There was no significant difference between VFT at rest and immediately post-exercise. However, VFT at rest correlated with immediate post-exercise augmented systolic and early diastolic tissue Doppler indicators of myocardial performance in elite athletes.

INTRODUCTION

It has been demonstrated that in a normal heart, flow of blood from the left atrium into the left ventricle (LV) during early diastole results in the formation of a vortex ring. Using cardiac magnetic resonance velocity mapping and echocardiographic particle image velocimetry, this transmitial flow has been demonstrated to transform through vortex ring into a laminar and unidirectional LV outflow. This form of fluid transportation by vortex ring formation is more efficient than by a steady straight jet. Gharib et al have quantified the process of vortex ring formation using a dimensionless index. Subsequently, we used non-invasive transcranial echocardiography to quantify vortex formation time (VFT) index using the formula $4 \times (1 - \beta) \pi \alpha \times 1/3$ left ventricle ejection fraction (LVEF) (where $\alpha$ represented LV geometry ratio and $\beta$ was fraction of total transmitial diastolic filling contributed by atrial contraction) and had previously found that it was reduced in clinical conditions.

What is already known about this subject?

► Vortex formation time is a dimensionless echocardiographic index that has a role in prognostication in heart failure, dilated cardiomyopathy, hypertrophic cardiomyopathy and is increasingly studied in other pathological cardiac conditions.

What does this study add?

► This study examined vortex formation time in elite athletes. The study demonstrated that the efficiency of vortex formation in the LV at rest correlated with and appeared to predict post-exercise augmented systolic and early diastolic tissue Doppler myocardial performance in elite athletes.

How might this impact clinical practice?

► This study demonstrates the expanding interest and use of vortex formation time in various clinical settings. It may be used to monitor the progress and training of elite athletes. It may also serve as a springboard for the application of vortex formation time in other cardiac conditions.

METHODS

We analysed 24 world class speedskaters training for the Winter Olympics Games. All subjects had undergone a comprehensive echocardiographic examination (two-dimensional, pulsed and continuous Doppler) as well as myocardial tissue Doppler studies, before and immediately after 3000 m of racing. Echocardiographic images were obtained within 5 min of termination of exercise. We measured the mitral annulus diameter (D, mm) in early diastole from the four-chamber view. LVEF and end-diastolic volume (EDV) were quantified using the biplane method of disks. $\alpha$ was determined by $4 \times (1 - \beta) \pi \alpha \times 1/3$ divided by D. Transmitial flows were assessed using pulsed Doppler in the four-chamber view. Velocity time integrals of (early) and late
Figure 1 (A) Relationship between left ventricular (LV) geometry (α) and vortex formation time (VFT). (B) Relationship between tissue Doppler-derived early mitral annular velocity and VFT.

Table 1 Baseline clinical and echocardiographic profile at rest of the athletes studied

| Parameter                  | Athletes (n=24), mean (±SD) |
|----------------------------|----------------------------|
| Age (years)                | 22±3                       |
| Male gender (n)            | 16 (67%)                   |
| Body surface area (m²)     | 1.6±0.2                    |
| Resting heart rate (beats per min) | 64±10                |
| Systolic blood pressure (mm Hg) | 110±7                 |
| Diastolic blood pressure (mm Hg) | 66±8                 |
| Left atrial volume index (mL/m²) | 33±7                |
| LV end-diastolic dimension (cm) | 5.0±0.4                |
| LV end-systolic dimension (cm) | 3.5±0.4                |
| LV end-diastolic volume (mL) | 125±24                   |
| LV end-systolic volume (mL) | 51±12                     |
| LV interventricular septum (mm) | 8.4±1.3                |
| LV posterior wall dimension (mm) | 9.4±1.5                |
| LV mass index (g/m²)       | 105±21                     |
| LV ejection fraction (%)   | 60±5                      |
| Peak E velocity (cm/s)     | 8.5±1.7                   |
| Peak A velocity (cm/s)     | 4.9±2.1                   |
| E/A ratio                  | 1.9±0.6                   |

A, late diastolic in-flow velocity; E, early diastolic in-flow velocity; LV, left ventricular.

Table 2 Pearson correlations between VFT index and other continuous variables

| Postexercise echo parameters (cm/s) | VFT   | r     | P value |
|-------------------------------------|-------|-------|---------|
| Mitral inflow Doppler               |       |       |         |
| Peak E velocity                     | 0.59  | 0.01  |         |
| Peak A velocity                     | 0.06  | NS    |         |
| Septal annular TDE                  |       |       |         |
| Peak S′ velocity                    | 0.46  | 0.02  |         |
| Peak E′ velocity                    | 0.53  | 0.01  |         |
| Peak A′ velocity                    | 0.34  | NS    |         |
| Lateral annular TDE                 |       |       |         |
| Peak S′ velocity                    | 0.48  | 0.02  |         |
| Peak E′ velocity                    | 0.53  | 0.01  |         |
| Peak A′ velocity                    | 0.35  | NS    |         |

NS = Not statistically significant, p>0.05
A′, late diastolic annular velocity; E′, early diastolic annular velocity; S′, systolic annular velocity; TDE, tissue Doppler echocardiography; VFT, vortex formation time.

**RESULTS**

Compared with non-athletes, these 24 elite athletes, aged 22±3 years, 67% males, had larger atrial and LV volumes, lower LV filling pressures and evidence of right ventricular remodeling.

(A) diastole were measured to calculate β which was the fraction of total transmitral diastolic filling contributed by atrial contraction. VFT was computed using the formula 4×(1−β)/π×α×LVEF.5 Peak systolic (S′), early (E′) and late (A′) diastolic tissue Doppler echocardiographic (TDE) velocities were measured at the septal and lateral mitral annulus in the apical four-chamber view. An average of three measurements was made for each parameter.

Continuous variables were expressed as mean value±SD. Paired Student’s t-tests were used to examine differences between pre-exercise and post-exercise variables. Correlation coefficients (r) were obtained between relevant parameters and resting VFT. Statistical significance was attributed when p<0.05. SPSS for Windows (V.15.0) was used for all analyses.

**DISCUSSION**

VFT had been dubbed as a ‘universal parameter’ of LV filling. It was conceived initially on fluid dynamic studies and has since been growing in clinical interest and applications.8 9 Nevertheless, there have also been conflicting studies that suggested that VFT cannot represent an index of diastolic ventricular function.10

Existing methods to non-invasively evaluate diastolic cardiac function have had limited success. VFT had been proposed as a viable alternative to existing echocardiographic parameters. However, it had many limitations. It was based on in vitro experiments with measurement of fluid dynamics in large-volume tanks.8 9 The LV with which this VFT has been applied is instead a viable alternative to existing echocardiographic parameters. It was conceived initially on fluid dynamic studies and has since become growing in clinical interest and applications.8 9 Neverthe-

With the growing clinical interest, however, there have also been studies with more attenuated findings. It had been assumed that VFT represented an index of diastolic ventricular function, where patients with impaired relaxation or restricted ventricular filling would exhibit lower than normal values of VFT. More recent work, however, has shown that VFT did not correlate with diastolic ventricular dysfunction, but it was instead related to the intraventricular pressure difference during early filling.11 12 Despite these inconsistencies, many studies had found applications for VFT in various clinical scenarios, ranging from heart failure to dilated cardiomyopathy.13 14

To the best of our knowledge, this was the first investigation assessing LV VFT and post-exertional changes in athletes. VFT, as immediate post-exercise early diastolic mitral in-flow velocity (E, r=0.59, p=0.01), tissue Doppler (TDE)-derived early mitral annular velocity (E′; septal (figure 1B) and lateral, both r=0.59, p=0.01) and systolic annular velocity (S′; septal: r=0.46, p=0.02 and lateral: r=0.48, p=0.02) but not late diastolic mitral in-flow velocity (A; r=0.06, p>0.05) or annular velocity (A′; septal: r=0.34, p>0.05 and lateral: r=0.35, p=NS) (table 2). VFT correlated better with change (difference between postexertion to baseline) in E′ (r=0.52, p=0.009) than with change in E velocity (r=0.42, p=0.08).
mentioned, would reflect early ventricular filling and efficiency of fluid propagation through the LV. This study did not find any significant differences between the VFT at rest and the VFT and repeated measurement taken within 5 min immediate post-exercise. The athletes’ VFT appeared slightly lower than healthy controls, but higher than those with heart failure. We found that the LV geometry ($\alpha$) correlated well with VFT suggesting the importance of LV remodelling in fluid propagation through the LV. The relationship of $\alpha$ to VFT appeared similar to that in healthy subjects, which was altered in systolic heart failure.

Importantly, the small sample size in this study may have meant that it may have been underpowered to demonstrate a significant difference in the VFT at rest compared with immediately post-exercise. Similarly, the increased heart rate immediately post-exercise and correspondingly shorter time of diastole may also have affected the measurement of VFT. Furthermore, in the immediate post-exercise state, athletes may be more volume depleted compared with measurements taken at rest, which would affect the LV load as well. To our knowledge, the effect of heart rate and volume status on the measurement of VFT has not been evaluated in previous studies. Nevertheless, these may be important factors that affect early ventricular filling and thus would consequently also affect measurement of VFT. These differences may also therefore have abolished any significant difference in the VFT measurements made post-exercise when compared against the VFT at rest.

A previous study on subjects with hypertrophic cardiomyopathy also failed to demonstrate a significant difference in VFT in patients with hypertrophic cardiomyopathy compared with controls. VFT was, however, shown to correlate well with exercise tolerance in patients with hypertrophic cardiomyopathy. It remained unclear from this study if VFT could be used to differentiates hypertrophy from an athlete’s heart compared with pathological conditions or healthy controls, but this may be an important aspect for future study.

However, we demonstrated reasonable correlations of baseline VFT with post-exertional early diastolic and systolic Doppler parameters. The process of vortex ring formation occurs mainly during early LV diastole. In fact, although it was assumed that VFT coincided with the completion of the early filling E-wave, it had been found that VFT correlated with the onset of the early filling wave deceleration instead. True enough, in our athletes, VFT at rest correlated modestly with $E'$ and TD-derived $E'$ but not $A'$. Therefore, in athletes, the late diastolic annular $A'$ may contribute less to diastolic fluid propagation through the LV compared with $E'$. In addition, we found VFT at rest correlated with the difference in $E'$ from rest to immediate post-exercise. It would appear that athletes with higher baseline VFT can improve their early diastolic myocardial function better on exertion. Previous studies have shown that the exercise capacity of endurance athletes was best predicted from diastolic dimensions and function during exercise. Dynamic exercise training in elite athletes may improve cardiac performance by an effect on diastolic filling and fluid propagation through the LV.

**Limitations**

We did not systemically image fluid propagation and vortices in the LV in the elite athletes at rest and post-exercise. This may be performed with aid of contrast echocardiography or digital particle imaging velocimetry (PIV). However, VFT has been extensively verified in mechanical and controlled models and shown to be robust. Although the echocardiographic images obtained were clear and of high quality, we did not report interobserver or intraobserver variability for the measurement of VFT in this study, and hence the reproducibility of the measurements made may not be clear. In a prior study using an identical method to measure VFT, the interobserver and intraobserver variabilities as assessed by the intraclass correlation coefficients (r) were: 0.96 (95% CI 0.84 to 0.99, $p<0.0001$) and 0.87 (95% CI 0.58 to 0.97, $p=0.0002$), respectively.

The values of VFT in our study were slightly lower compared with healthy subjects in prior studies. This may be because we examined elite athletes. Cardiac adaptation in these athletes as well as changes in LV morphology may influence VFT. Although not the focus of our study, the difference in VFT between elite athletes and sedentary controls may be of interest in future study. We did not demonstrate any significant differences in VFT at rest compared with immediately post-exercise in the athletes studied. This may have been due to the relatively small sample size that meant the study was underpowered to demonstrate a statistically significant difference in the VFT. Nevertheless, the findings remain exploratory and hypothesis-generating. VFT at rest correlated with post-exercise systolic and early diastolic tissue Doppler velocities. This may be the subject of future larger prospective studies that may establish the role of VFT in athletic cardiac physiology.

**CONCLUSIONS**

VFT, a dimensionless index incorporating LV geometry, systolic and diastolic contributions to fluid dynamics can be derived using transthoracic echocardiographic measurements in elite athletes. Although VFT did not differ significant immediately post-exercise compared with rest, the VFT at rest appeared to correlate with immediate post-exercise augmented systolic and early diastolic tissue Doppler indicators of myocardial performance.

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