The Relationship Between Oxygen Uptake and the Rate of Myocardial Deformation During Exercise

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ABSTRACT

Background: The relationship between resting echocardiographic measures of cardiac function and exercise capacity is weak. The details of ventricular augmentation may provide insight into determinants of cardiac efficiency for optimal exercise performance. The aims of this study were to establish how much of the variability in exercise performance could be explained by myocardial recruitment and which parameters describing cardiac function were most closely related to exercise performance.
Methods: Untrained volunteers were recruited before training for the London Marathon. All performed a cardiopulmonary exercise test combined with stress echocardiography. Systolic and diastolic longitudinal velocities (S′ and E′), left ventricular ejection fraction (LVEF), stroke volume (SV), and strain (GLS) were obtained throughout exercise.
Results: A variety of parameters including S′, E′, GLS, and SV showed a correlation with $\dot{V}o_2$ throughout exercise ($\rho = 0.83$, $P < 0.0001$). At the prespecified sample point (respiratory exchange ratio > 1.0) only SV and S′ were predictive of $\dot{V}o_2$peak. LVEF and E′ as well as both global longitudinal and circumferential strain showed no correlation with $\dot{V}o_2$peak. The systolic efficiency slope (SES) that we developed by determining the individual regression lines for $\dot{V}o_2$ and S′ showed a relationship between with $\dot{V}o_2$ peak for both septal S′, $r = 0.57$, $P < 0.001$, and lateral S′, $r = 0.53$, $P < 0.001$.
Conclusion: A detailed description of myocardial function is described, linear for S′ and E′ and a plateau for EF and GLS. S′ during exercise is a better predictor of exercise performance than LVEF, SV, or GLS. The SES slope predicted $\dot{V}o_2$peak, suggesting the process driving systolic velocity and its augmentation is a key determinant of exercise ability. *Journal of Clinical Exercise Physiology*. 2021;10(3):85–93.

Keywords: cardiopulmonary exercise testing, stress echocardiography, myocardial recruitment

INTRODUCTION

In those with cardiac failure, the relationship between resting echocardiographic measures of cardiac function and exercise capacity is weak (1–4). At peak exercise, left ventricular (LV) function, especially longitudinal motion, is better correlated with maximum exercise capacity (5). The shape of the augmentation curve, as well as the relative contribution of various measurable facets of heart function is

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poorly described. Among athletes with phenotypically abnormal hearts, augmentation of LV ejection fraction (LVEF) by more than 10% differentiates those with adaptive rather than cardiomyopathic hearts (6,7). Understanding the shape of the relationship between contractile reserve and exercise attainment is vital to understand the physiology and to establish target parameters that might be useful when investigating disease states. We therefore hypothesized that detailed measures of exercise myocardial recruitment would explain individual exercise performance and that longitudinal function would be the primary adaptive strategy. This was achieved by recording oxygen uptake ($\dot{V}o_2$) and echocardiography simultaneously across an incremental exercise protocol in a cohort of healthy volunteers.

METHODS

Full details of the protocol have been previously published (8), and this was a preplanned ancillary analysis. Healthy participants were recruited in 2016 before undertaking training their first marathon (Virgin Money London). Inclusion criteria age was less than 35 y at recruitment, no past significant medical history, and no previous marathon running experience. All procedures were in accordance with the principles of the Helsinki declaration. All participants gave written informed consent, and the study was approved by the London-Queen Square National Research Ethics Service Committee (15/LO/0086).

All measurements were conducted before training started over a 3-week period (3 consecutive weekends after ballot place announcement) 6 months before the marathon.

Data Acquisition and Analysis

Cardiopulmonary exercise testing (CPET) was performed according to exercise testing guidelines (9). Protocols were individually determined with work rate (15–30 W) increased every minute until voluntary exhaustion, aiming for 10 minutes of exercise. To permit concurrent transthoracic echocardiography, studies were performed using a semirecumbent cycle ergometer (ERG 911 S/L, Schiller, Baar, Switzerland). A 1-minute rest period was included followed by a 3-minute warmup. Heart rate (HR), blood pressure, and oxygen saturation were monitored throughout. Here, $\dot{V}o_2$ was continuously measured using a calibrated breath-by-breath analyzer (Cosmed Quark CPET, Rome, Italy). Participants were verbally encouraged to exercise until maximal exertion. Here, $\dot{V}o_2$peak was expressed as the highest value from an average of 30 seconds during the final stage of the exercise test. To fully assess submaximal efforts, the oxygen uptake efficiency slope (OUES) was calculated automatically from $\dot{V}o_2$ against the logarithm of V_E ($\log V_E$) (10).

Echocardiography was performed using a GE Vivid E95 platform (Vingmed-General Electric, Horten, Norway) equipped with a phased-array transducer (1.4–4.6 MHz). A detailed protocol was collected at rest, at 5 minutes into exercise (excluding the 3-minute warmup period), and when the respiratory exchange ratio (RER) was above 1.0. This included the apical 4-chamber view (with and without tissue velocity imaging [TVI]), the apical 2-chamber, apical long-axis view, parasternal short-axis view at the base and apical level, and a Pulse Wave Doppler at the level of the LV outflow tract (LVOT; 1 cm below the aortic valve). An abbreviated protocol was obtained every 1 minute and 15 seconds and included the apical 4-chamber view (with and without TVI). As part of the protocol 12-lead electrocardiogram and cardiac magnetic resonance was undertaken but not included in this analysis.

Echocardiographic Analysis

All analyses were carried out offline using the GE Echopac software (version 113). S′ was derived from color-derived TVI images, where the sample volume was placed at the annulus of the septal and lateral walls. S′ was defined as the highest velocity during systole after the end of isovolumetric contraction. Three cardiac cycles were obtained, and S′ was averaged for each time point. During the full protocol, Simpson's biplane was measured (in the apical 4- and 2-chamber view), global longitudinal strain (GLS) in the 3 apical views, and the LVOT velocity time integral. Speckle-tracking strain analysis was accepted when the software and visual inspection indicated adequate tracking. If tracking was inadequate, manual adjustments were made, or the analysis was excluded from overall analyses. GLS was obtained from the apical 4-chamber view (basal septum, midseptum, apical septum, basal lateral, midlateral, and apical lateral), the apical 2-chamber view (basal inferior, midinferior, apical inferior, basal anterior, midanterior, and apical anterior), and the apical long-axis view (basal posterior, midposterior, basal anteroseptal, midanteroseptal). If data were of insufficient quality, they were excluded from analyses. This was defined as poor TVI tracing, exclusion of the mitral valve annulus (for color TVI measurements), or poor endocardial border definition (for biplane). Eleven participants were excluded from analysis.

Statistical Analyses

All data are expressed as mean ± standard deviation (SD) for continuous variables. Categorical data are expressed as absolute values and percentages. All data were tested for normality using the Kolmogorov-Smirnov statistical test. Pearson's correlation coefficients (r) or Spearman's coefficients (ρ) were used to determine relationships between hemodynamic and echocardiographic responses. Here, $\dot{V}o_2$ was averaged every 10 seconds and linked with corresponding S′ values every minute. Also, E′, LVEF, GLS, and circumferential strain values were linked with corresponding S′ values at rest, 5 minutes, and when the RER was over 1. Values at rest, at 5 minutes, and when RER was over 1.0 were compared using 1-way analysis of variance and Tukey post hoc analyses to determine differences between groups. A scatter plot with 95% confidence interval was plotted. Correlations were performed for $\dot{V}o_2$ measures of myocardial function throughout exercise and between myocardial function and $\dot{V}o_2$peak. Interobserver and intra-observer measurement variability was performed for 5 randomly selected

TABLE 1. Baseline characteristics.

Parameter	Value, Mean ± SD, All Participants (N = 57)
Height (cm)	175 ± 10.3
Weight (kg)	71.6 ± 12.9
Age (y)	29.2 ± 3.3
Male (%)	32 (56)
$\dot{V}o_2$peak (L·min^{-1})	2.8 ± 0.7
$\dot{V}o_2$peak (mL·kg^{-1}·min^{-1})	38.7 ± 6.5
Predicted $\dot{V}o_2$ (%)	107 ± 17
WR peak (W)	226 ± 55
HR peak (b·min^{-1})	168 ± 17.5
Peak RER	1.23 ± 0.09
OUES (mL·min^{-1})·(L·min^{-1})$^{-1}$	2936 ± 749

HR = heart rate; OUES = oxygen uptake efficiency slope; RER = respiratory exchange ratio; $\dot{V}o_2$peak = peak oxygen uptake; WR = work rate.

participants for echocardiographic parameters and for 15 randomly selected participants for CPET parameters. Two British Society of Echocardiography-accredited operators (JVZ, SB) with extensive experience in performing CPET simultaneously with echocardiography performed the interobserver and intra-observer measurements. The intra-class correlation coefficient (ICC) and 95% confidence intervals were used to quantify reproducibility. All statistical analyses were performed using SPSS (version 20.0; SPSS, Inc). A P value of <0.05 was considered statistically significant.

RESULTS

Population

Of the 68 patients recruited, 11 were excluded (9 studies were of insufficient image quality for meaningful analysis, and 2 studies did not have TDI switched on during exercise). Fifty-seven participants were included in the final analysis. Thirty-two (56%) were male, aged 29.2 ± 3.3 y, mass of 71.6 ± 12.9 kg (157 ± 28 lb), and height of 175 ± 10.3 cm (69 ± 4 in). Baseline LVEF, end-diastolic (EDV), and end-systolic volumes (ESV) were within normal ranges (LVEF: 61.0 ± 2.8%; EDV: 120 ± 30.2 mL; ESV: 46.9 ± 13.3 mL; Tables 1 and 2).

Exercise Performance

Peak RER was 1.23 ± 0.09 with 56 (98%) of participants achieving an RER of >1.10 (1 participant reached a peak RER of 1.08). Absolute $\dot{V}o_2$peak was 2.8 ± 0.7 L·min^{-1}, and relative $\dot{V}o_2$peak was 38.7 ± 6.5 mL·kg^{-1}·min^{-1}. OUES was 2936 ± 749 (mL·min^{-1})·(L·min^{-1})$^{-1}$ (Table 1).

TABLE 2. Hemodynamic and echocardiographic responses at baseline and exercise (mean ± SD).

Hemodynamic and Echo Variable	Rest	5 min	RER > 1.0	% Change From Rest to 5 min	% Change from rest to RER > 1
HR (b·min^{-1})	69 ± 10.6	130 ± 14.8[a]	151 ± 14.0[b]	88	119
SV (mL)	67.0 ± 14.9	81.1 ± 17.0[a]	84.6 ± 19.1	21	26
CO (L)	4.6 ± 1.4	9.4 ± 1.7[a]	12.7 ± 2.6	104	176
WR (W)	NA	113 ± 22[a]	169 ± 38[b]	NA	-
Proportion of maximal load	NA	51 ± 0.1	75 ± 0.1	NA	-
Septal S′ (cm·s^{-1})	7.2 ± 1.4	11.3 ± 1.8[a]	13.7 ± 1.8[b]	57	90
Lateral S′ (cm·s^{-1})	8.4 ± 1.6	12.5 ± 1.6[a]	14.9 ± 1.7[b]	57	77
Septal E′ (cm·s^{-1})	10.1 ± 1.4	15.3 ± 2.8[a]	18.1 ± 2.7[b]	51	79
Lateral E′ (cm·s^{-1})	12.8 ± 2.0	17.0 ± 2.7[a] 18.8 ± 2.5[b]	18.8 ± 2.5[b]	33	47
LVEF (%)	61.0 ± 2.8	68.8 ± 3.6[a]	69.9 ± 3.9	13	15
EDV (mL)	120 ± 30.2	113 ± 38.0	112 ± 25.8	−5.8	-6.7
ESV (mL)	46.9 ± 13.3	36.7 ± 11.5[a]	34.3 ± 9.2	−22	-27
GLS (%)	−18.0 ± 2.4	−21.4 ± 3.1[a]	−21.8 ± 2.7	−19	-21
Circumferential strain (%)	−20.1 ± 4.7	−23.2 ± 4.3	−23.3 ± 4.3	−15	- 16

CO = cardiac output; E′ = diastolic velocity; EDV = end diastolic volume; ESV = end systolic volume; GLS = global longitudinal strain; HR = heart rate; LVEF = left ventricular ejection fraction; NA = not available; RER = respiratory exchange ratio; S′ = systolic velocity; SV = stroke volume; WR = work rate.
[a]Baseline versus 5 min; $P < 0.05$
[b]5 min versus RER > 1.0; $P < 0.05$

Mechanical Augmentation

All parameters except for EDV significantly augmented after 5 minutes of exercise (Table 2). Ejection fraction increased by 13 ± 7.2%, stroke volume by 22 ± 17%, GLS by 22 ± 15%, and septal S′ and lateral S′ by 52 ± 24% and 61 ± 27%, respectively. At RER > 1.0, only longitudinal velocities (systolic and diastolic, septal and lateral) continued to show statistically significant augmentation. All other parameters demonstrated an augmentation plateau (Table 2; Figure 1).

Longitudinal Contractile Reserve—$\dot{V}o_2$ Relationship

The augmentation of longitudinal velocity showed a strong correlation with $\dot{V}o_2$ throughout exercise for the septal S′ ($\rho = 0.86$, $P < 0.0001$), the lateral S′ ($\rho = 0.78$, $P < 0.0001$), and for the average S′ ($\rho = 0.83$, $P < 0.0001$) throughout exercise (Figure 2). Diastolic velocities also demonstrated a good relationship with $\dot{V}o_2$ (septal E′: $\rho = 0.8$, $P < 0.0001$; lateral E′: $\rho = 0.78$; Table 2). The relationship was stronger with the septum than the lateral wall. A modest relationship was found for $\dot{V}o_2$ and SV, for $\dot{V}o_2$ and GLS, and a weak correlation was found for CO and $\dot{V}o_2$ ($r = 0.23$) throughout exercise (Table 3).

When looking at the correlation with $\dot{V}o_2$peak only S′ ($r = 0.60$, $P < 0.05$) and SV ($r = 0.64$, $P < 0.05$) at RER > 1.0 showed a significant correlation with $\dot{V}o_2$peak. Diastolic parameters, LVEF, as well as both global longitudinal and circumferential strain showed no correlation with $\dot{V}o_2$peak.

Systolic Efficiency Slope (SES)—The S′/$\dot{V}o_2$ Relationship

We took advantage of the linear augmentation pattern of systolic velocity and its close relationship with $\dot{V}o_2$ to derive individual regression lines for $\dot{V}o_2$ and S′. This new parameter represents the amount of S′ associated with a 1 unit increase in $\dot{V}o_2$ and therefore a measure of myocardial

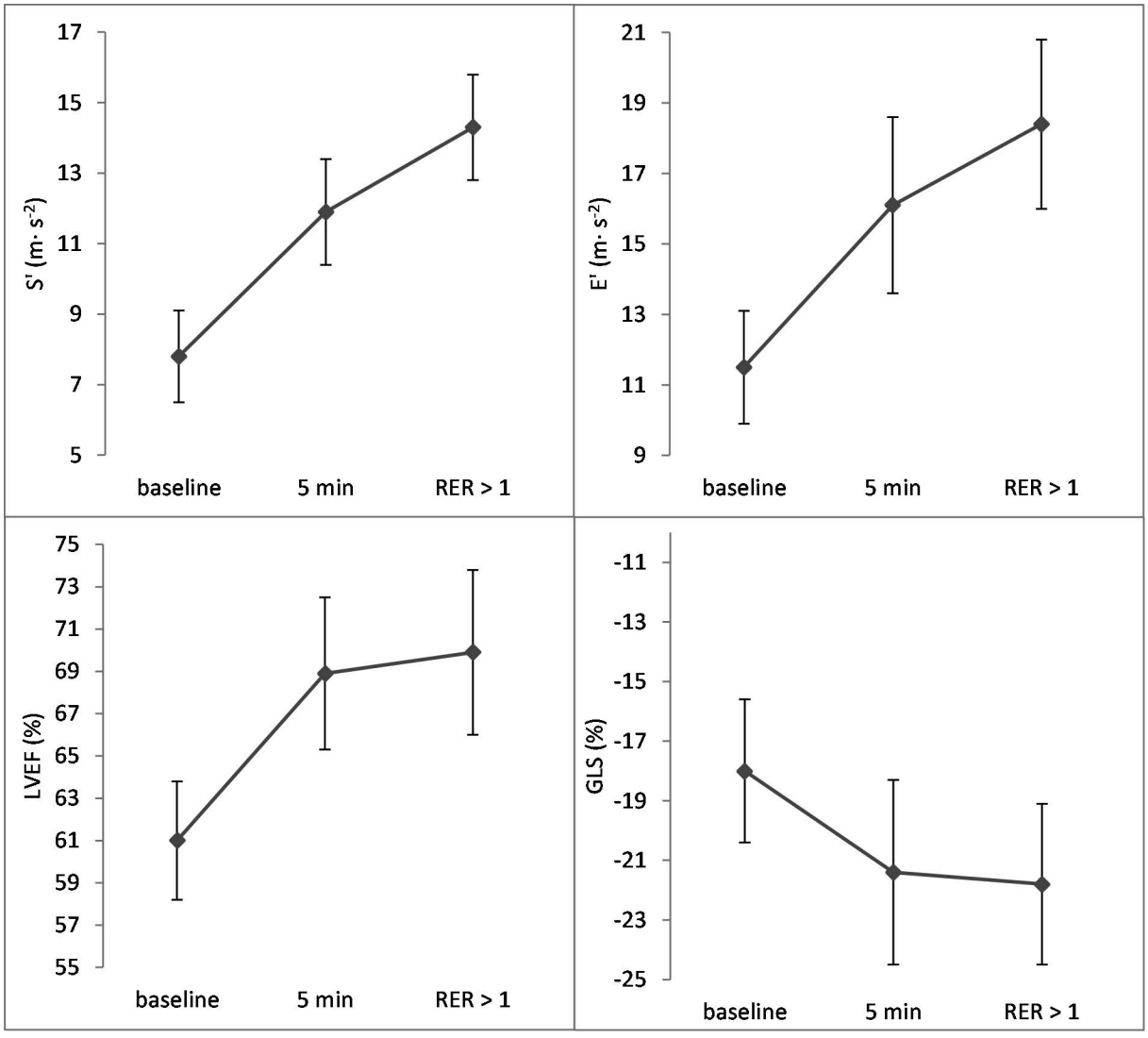

FIGURE 1. Mechanical augmentation for S′, E′, LVEF, GLS throughout exercise. S′ = systolic velocity; E′ = diastolic velocity; LVEF = left ventricular ejection fraction; GLS = global longitudinal strain; $\dot{V}o_2$ = oxygen consumption.

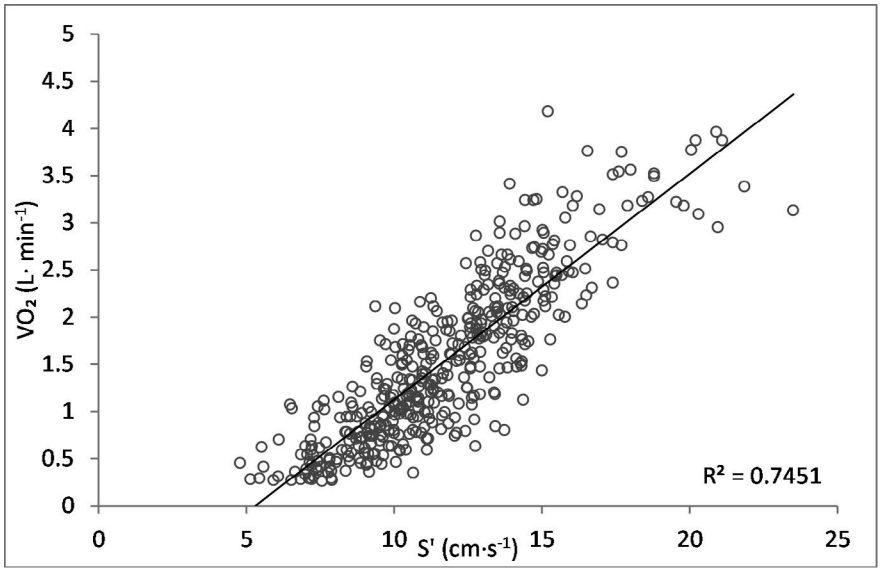

FIGURE 2. S′-$\dot{V}o_2$ relationship throughout exercise. S′ = systolic velocity; $\dot{V}o_2$ = oxygen consumption.

efficiency. A relationship between the SES slope was observed for the $\dot{V}o_2$peak and septal S′, $r = 0.57$, $P < 0.001$, and lateral S′, $r = 0.53$, $P < 0.001$; Figure 3. We also confirmed the validity of these findings by creating models for OUES, a submaximal measure shown to predict total cardiopulmonary capacity (10,11). A significant relationship was found between SES slope and the OUES (septal $r = 0.54$, $P < 0.001$, lateral $r = 0.52$, $P = 0.001$). Contractility also increased with increase in heart rate (force-frequency relationship). Here, S′/HR correlation was $R^2 = 0.6$, whereas the S′/$\dot{V}o_2$ relationship was $R^2 = 0.75$.

TABLE 3. Correlations between $\dot{V}o_2$ measures of myocardial function throughout exercise.

Myocardial Function Measure	R Value
Septal S′ (cm·s^{-1})	0.86[a]
Lateral S′ (cm·s^{-1})	0.80[a]
Septal E′ (cm·s^{-1})	0.78[a]
Lateral E′ (cm·s^{-1})	0.70[a]
LVEF (%)	0.66[a]
ESV (mL)	−0.17
SV (mL)	0.56[a]
CO (L)	0.23[a]
GLS (%)	−0.46[a]

CO = cardiac output; E′ = diastolic velocity; ESV = end systolic volume; GLS = global longitudinal strain; LVEF = left ventricular ejection fraction; S′ = systolic longitudinal velocity; SV = stroke volume; $\dot{V}o_2$ = oxygen uptake.
[a]$P < 0.05$

Interobserver and Intra-observer Reproducibility

There was excellent interobserver and intra-observer reproducibility for all measurements by CPET: $\dot{V}o_2$peak (ICC: 0.99–0.99) and OUES (ICC: 0.98–1.00). For echocardiography parameters, there was good to excellent interobserver and intra-observer reproducibility for S′ and E′ at rest and exercise: Sep S′ (ICC: 0.87–0.99), Lat S′ (ICC: 0.9–0.99), Sep E′ (ICC: 0.81–0.96), and Lat E′ (ICC: 0.81–0.96). There was lower interobserver and intra-observer reproducibility seen for LVEF and GLS both at rest and exercise: GLS (ICC: 0.61–0.93) and LVEF (ICC: 0.63–0.88).

DISCUSSION

There are significant gaps in the understanding of myocardial augmentation and its relationship with exercise performance. This study addresses these by demonstrating that systolic and (to a lesser extent) diastolic myocardial velocities increase incrementally as exercise work rate increases in a linear fashion, in contrast to parameters that are measured throughout systole (LVEF, SV, GLS), which show a plateau after low-intensity exercise. We provide ranges for the expected augmentation in healthy young adults both at low intensity and at peak exercise. We demonstrate that systolic velocities are very closely linked to $\dot{V}o_2$ throughout exercise, and in our study, systolic velocities during exercise are a better predictor of exercise performance than LVEF, SV, or GLS, and we define a new parameter (SES) equivalent to the slope of this relationship that can predict exercise capacity.

Although the augmentation of normal and pathological heart function has been measured, this is usually at the start, at some fixed point, during, or after the cessation of exercise. This makes comparison complex. We found a 14% increase in LVEF during exercise with most of the increase happening during the initial bout of exercise, which is very comparable with the existing literature which suggests that LVEF

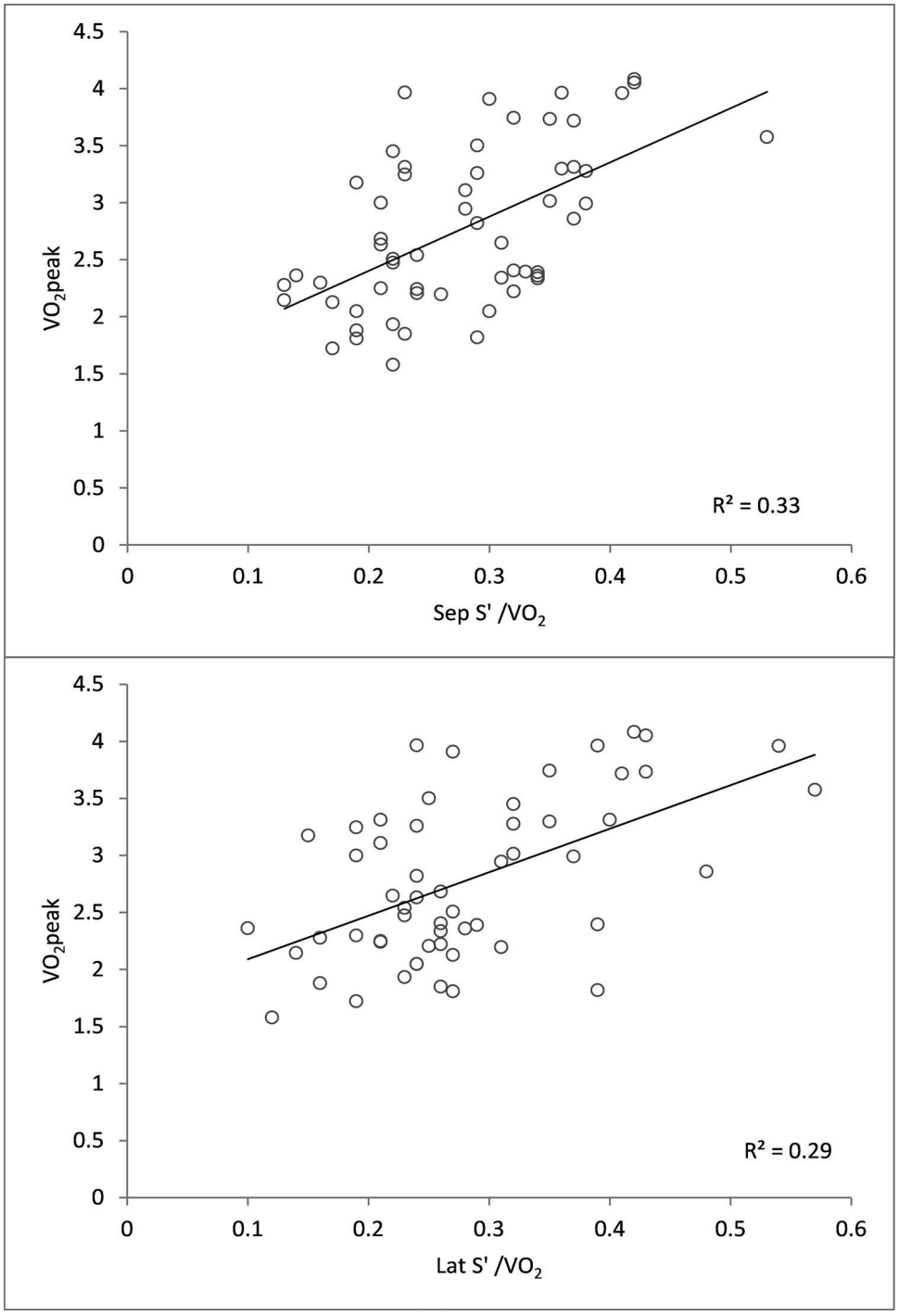

FIGURE 3. Systolic efficiency slope (SES)-S′-$\dot{V}o_2$ relationship. S′ = systolic velocity; $\dot{V}o_2$ = oxygen consumption; sep = septal; lat = lateral.

beyond the ventilatory threshold shows a slight plateau response (12). Body position effects the change in EDV and ESV during exercise (13,14). In a recumbent position, EDV remains unchanged during exercise, and an increase in seen in ESV (15). We used a semirecumbent cycle ergometer and found a similar response to volumes.

GLS is a more advanced method of measuring LV function. Wang et al. (2014) (16) showed a significantly increase in GLS and S′ during exercise in healthy control patients. Our data showed a 25% increase in GLS and 89% increase in S′, which is similar. However, GLS is more problematic to measure at peak exercise because high heart rates may lead to speckle misregistration, although, anecdotally, higher-than-recommended heart rates are tolerated by the software. Cifra et al. (2016) (17) described a linear relationship between HR and GLS during exercise and showed that S′ obtained using color Doppler tissue imaging showed excellent interobserver and intra-observer variability. GLS measurements were more challenging to obtain during exercise due to lung interference and more excessive cardiac motion.

However, although peak systolic strain values showed higher interobserver variability, it was still considered acceptable for clinical use (17).

There is limited data on the relationship between myocardial augmentation and exercise performance. We have previously published that peak systolic velocity and $\dot{V}o_2$ are closely correlated across a very wide range of diagnoses (5,18). These data, while an interesting proof of concept, do not resolve the question of whether the heart is behaving differently or whether those with less severe disease are simply achieving more exercise and hence more myocardial augmentation. This central weakness, the confounding effect of exercise ability, runs through much of the contractile reserve literature. Attempts to resolve this by imaging at submaximal exercise are vulnerable to an incomplete understanding of the pattern of augmentation for each measured parameter.

The use of a semirecumbent cycle ergometer allows the combination of CPET and echocardiography, providing additional insight into any observed changes in heart function. There have been limited attempts to relate changes on myocardial function on echocardiography with changes in $\dot{V}o_2$. In a study of 31 patients (12 with HF, 15 with preserved LVEF, 16 with reduced LVEF, and 15 controls), they demonstrated an increase in S′ and E′ from baseline to the ventilatory threshold up to maximal effort, a result like the data in a healthy cohort. GLS and LVEF also augmented, but the relationship was weaker, as did the additional measures of the RV and circumferential strain (19). Resting LVEF is not able to predict $\dot{V}o_2$peak (4,20,21). However, obtaining LVEF during exercise does have incremental prognostic value. HF patients who can increase LVEF by more than 5% have a better prognosis than those who do not (22). Evaluating GLS at rest is an independent and incremental prognostic tool regarding long-term risk in cardiovascular morbidity and mortality (23). However, GLS at exercise requires more investigation.

In our study, we selected S′ as our principle long-axis evaluation parameter, as it is a reproducible measurement during exercise (2). Mechanistically, longitudinal S′ has previously been shown to be an early indicator for ventricular dysfunction, as it is one of the principle engines of systole, and a fall often precedes a subsequent fall in LVEF (24) during disease; unlike other parameters of both volumetric and longitudinal function, S′ augmentation remained linear during exercise. We propose that this is because the S′ augmentation is both displacement and frequency dependent, and thus, heart rate will still increase velocity even when augmentation has reached a plateau.

In the analysis, S′ was more predictive of $\dot{V}o_2$peak than ejection fraction or GLS, both at 5 minutes of exercise, when all parameters showed augmentation, and at peak exercise, when LVEF and GLS had plateaued. It is easily forgotten that strain and velocity measure different aspects of systolic performance and cannot be considered interchangeable. Work by Gu et al. (2017) (25) looking at the first phase ejection fraction, during which, with normal activation, peak velocity is reached, has demonstrated that, within systole, the initial phase of contraction is the most important. This provides a rationale for the superiority of systolic velocity over the whole systolic measures of ejection fraction and GLS. Our study confirms the very tight relationship between $\dot{V}o_2$ and S′ throughout the whole of exercise, making it a very useful surrogate for $\dot{V}o_2$ and suggesting that the processes that increase myocardial velocity are central to determine exercise function.

By inference, therefore, variation in or changes to cardiac function would result in different relationships between $\dot{V}o_2$ and S′. This is important because of the potential that augmentation of S′ is simply reflecting a longer exercise time or greater workload, making the relationship tautological. To understand the relationship between $\dot{V}o_2$ and S′ in greater detail, we hypothesized that the individual ratios of S′ to $\dot{V}o_2$ (the amount of myocardial augmentation required to increase $\dot{V}o_2$ by a single unit) could predict $\dot{V}o_2$peak values. We devised the term the systolic efficiency slope (SES) to describe this. There was a relationship between the SES and $\dot{V}o_2$peak, suggesting that this relationship may be important. However, given that peak exertion can be heavily effort dependent, we also looked at OUES (a well-validated submaximal measure known to be highly related to $\dot{V}o_2$peak). Again, there was a significant relationship. This contraction-metabolic coupling relationship has not been previously described in this way, to our knowledge. The SES slope is a slope that does not require a prespecified heart rate or maximal exercise to be achieved. While the data are not strong enough to suggest this as a clinical tool, we propose these may be suitable methods for future research. Integrating CPET with exercise echocardiography provides unique data that can provide new and valuable insights into disease processes. This study demonstrates greater insights into contracting myocardium and exercise tolerance. The slope between S′ and $\dot{V}o_2$ showed a strong positive relationship with $\dot{V}o_2$peak, suggesting that submaximal slope values can predict $\dot{V}o_2$peak values, which is valuable in patients who cannot exercise to maximal exertion. This is a new way to describe heart function and critically appraised in more detail than ever before. Integrating CPET with exercise echocardiography provides unique and robust data that can provide new and valuable insights into disease processes. These results would need to be applied to clinical caseloads to establish the effect of aging and comorbidity as well as disease states.

Our analysis has several limitations. The population was young and verified to be without cardiac disease, and despite this, 11 patients were excluded mainly due to insufficient image quality. Only participants with analyzable image quality were included. The imaging protocol was arduous; however, the findings (threshold values for ejection fraction and GLS-continuous augmentation of S′) would allow simplification in future studies that would also be more clinically achievable. Noninvasive blood pressure was measured as part of the protocol; however, the sampling from the automatic detection devices was consistently poor, resulting in less than 50% evaluable data. Analysis requiring

blood pressure was therefore not performed. These findings may not apply to a disease population or those from other age groups. TVI measurements were optimized for perpendicular septal measurements. Lateral wall TVI may have been off axis, which can introduce error. Transmitral Doppler was not recorded because the technical challenge of obtaining these values at high rate show complete early/late active filling fusion, making detection of the true Doppler E wave unreliable and hence an unreliable E/E′. The GLS measured by speckle tracking may not be accurate at the higher heart rate. Our data would have permitted the evaluation of myocardial work, but this did not form part of the original protocol and is vendor specific; therefore, this analysis has not been undertaken.

CONCLUSIONS

We have described in detail the augmentation of a variety of systolic parameters during exercise in young and fit adults, demonstrating 2 patterns of augmentation: linear for S′ and E′ and a plateau for ejection fraction and GLS. The magnitude of the augmentation (14% for ejection fraction and 89% for S′) is like previous research. The study describes the relationship between longitudinal function (as expressed by S′) and $\dot{V}o_2$ throughout exercise. Systolic velocity, which represents early and midsystolic function, is more important than whole systolic parameters such as LVEF, including GLS, in predicting exercise performance. The SES described for the first time shows that the regressive relationship between S′ and $\dot{V}o_2$ predicts peak exercise tolerance, which proves that the relationship is more than simply collinearity. It also offers a novel research opportunity to evaluate disease states where this relationship may be different. Our data suggest a move from velocity to strain may not be justified or helpful in evaluating myocardial performance during exercise.

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