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Relationships of left ventricular strain and strain rate to wall stress and their afterload dependency

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ABSTRACT

Whether and how left ventricular (LV) strain and strain rate correlate with wall stress is not known. Furthermore, it is not determined whether strain or strain rate is less dependent on the afterload. In 41 healthy young adults, LV global peak strain and systolic peak strain rate in the longitudinal direction (LS and LSR, respectively) and circumferential direction (CS and CSR, respectively) were measured layer-specifically using speckle tracking echocardiography (STE) before and during a handgrip exercise. Among all the points before and during the exercise, all the STE parameters significantly correlated linearly with wall stress (LS: $r=-0.53$, $p<0.01$, LSR: $r=-0.28$, $p<0.05$, CS in the inner layer: $r=-0.72$, $p<0.01$, CSR in the inner layer: $r=-0.47$, $p<0.01$). Strain more strongly correlated with wall stress than strain rate ($r=-0.53$ for LS vs. $r=-0.28$ for LSR, $p<0.05$; $r=-0.72$ for CS vs. $r=-0.47$ for CSR in the inner layer, $p<0.05$), whereas the interobserver variability was similar between strain and strain rate (longitudinal: 6.2% vs. 5.2%, inner circumferential: 4.8% vs. 4.7%, mid-circumferential: 7.9% vs. 6.9%, outer circumferential: 10.4% vs. 9.7%), indicating that the differences in correlation coefficients reflect those in afterload dependency. It was thus concluded that LV strain and strain rate linearly and inversely correlated with wall stress in the longitudinal and circumferential directions, and strain more strongly depended on afterload than did strain rate. Myocardial shortening should be evaluated based on the relationships between these parameters and wall stress.

Key words: Strain, Strain rate, Speckle tracking echocardiography, Wall stress, Afterload

INTRODUCTION

Recent advances in 2-dimensional speckle tracking echocardiography (STE) have enabled us to accurately evaluate both regional and global, multidirectional, and layer-specific left ventricular (LV) myocardial deformation [1, 2]. The peak strain and systolic peak strain rate reflect the magnitude and maximal velocity of myocardial deformation during LV systole, respectively. These STE-derived parameters have been widely used in clinical settings to estimate myocardial shortening in various cardiac diseases. These parameters have been demonstrated to be useful for predicting the response to cardiac resynchronization therapy in patients with heart failure [3], detecting subclinical myocardial dysfunction in patients with hypertensive heart disease [4] or hypertrophic cardiomyopathy [5, 6], and predicting the prognosis of patients with various structural heart diseases and those with chronic heart failure [6-8].

The STE-derived strain and strain rate must depend on myocardial wall stress as the myocardial afterload. However, these STE parameters have not been interpreted with careful consideration of the wall stress in clinical studies and examinations. Moreover, the normal ranges of the relationships between LV wall stress and STE parameters have not been determined. It has also not been determined whether the myocardial strain or strain rate correlates with wall stress. Although it is generally considered that the strain rate is less dependent on the afterload than is strain, there have been few studies that compared the afterload dependency between these 2 parameters [9, 10]. We aimed to determine the relationships between LV wall stress and strain and those between wall stress and strain rate in the longitudinal and circumferential directions in healthy young subjects, and we compared the afterload dependency between strain and strain rate.

METHODS

Study Subjects

Forty-one healthy volunteers (30 men and 11 women) over 20 years old (mean age 29±5 years) participated in this study. The exclusion criteria included a history of cardiovascular disease, hypertension, diabetes mellitus, or dyslipidemia. None of the subjects had a smoking habit, and none were on medication. None had abnormal findings in the physical examination. All 41 of the subjects were in sinus rhythm and had normal electrocardiograms. This study was approved by the Research Ethics Committee of Hokkaido University Hospital, and all study subjects provided written informed consent.

Echocardiography

Each subject's height and body weight were measured, and the body surface area was calculated. A handgrip exercise was performed at 50% of the maximal grasping power, which had been measured prior to the exercise by using a digital hand-held dynamometer (Takei Scientific Instruments, Tokyo, Japan). The subjects held a handgrip in their left hand, squeezing it in 3 s and releasing it in 3 s repeated for a total period of 4 min. At each 1-min interval during the exercise, the subject's blood pressure and heart rate were measured.

Immediately before and 2 min after the initiation of handgrip exercise, transthoracic echocardiography was performed using an Artida ultrasound system (Toshiba Medical Systems, Tochigi, Japan) equipped with a PST-30BT transducer (3 MHz). The apical 4-chamber, 2-chamber, and long-axis views and the parasternal LV short-axis views at basal, mid-, and apical levels were recorded. The LV end-diastolic dimension (LVDd) and end-systolic dimension (LVDs) were measured in the parasternal LV long-axis view, and fractional shortening was calculated. Interventricular septal thickness and LV posterior wall thickness were measured at end diastole and end systole in the LV short-axis view at basal level. The LV ejection fraction

was measured using the biplane method of disks [11]. The LV meridional wall stress (MWS) was calculated using the following formula:

$$MWS = 0.334 \times SBP \times \frac{LVDs}{WTs \times (1+WTs/LVDs)},$$

where SBP and WTs were systolic blood pressure and end-systolic wall thickness averaged between interventricular septum and LV posterior wall, respectively [12]. The layer-specific circumferential wall stress (CWS) was also calculated, using the following formula:

$$CWS = ESP \times (LVDs/2)^3 \times \frac{1+(LVDs/2+WTs)^3/2\ [(LVDs/2)+(WTs \cdot In/It)]^3}{(LVDs/2+WTs)^3 - (LVDs/2)^3},$$

$$ESP = 0.98 \times [DBP+(SBP-DBP)/3] +11,$$

where DBP is diastolic blood pressure and ESP is estimated LV end-systolic pressure, and *In* is the number of incremental layers from the endocardial surface of the wall to the epicardium and *It* is the total number of the incremental layers [13]. Thus, 0, 0.5, and 1.0 were used as the *In*/*It* ratios to calculate the CWS at the innermost myocardial layer, midwall, and outermost myocardial layer, respectively.

Analysis of Speckle Tracking-Derived Strain

We analyzed the strain offline using the STE software program provided by Toshiba Medical Systems. The LV endocardial border was manually traced on the 3 apical views, and the longitudinal strain and strain rate curves were automatically generated. In the standard 16 segments in these 3 views, regional peak longitudinal strain (rLS) and regional systolic peak longitudinal strain rate (rLSR) were measured, and averaged to yield peak global longitudinal strain (LS) and systolic peak global longitudinal strain rate (LSR), respectively. The LV endocardial and epicardial borders were traced on the 3 short-axis views. The software algorithm discriminated the endocardial and epicardial borders as well as the midpoint on the end-diastolic

frame, and then those speckle patterns were tracked in a frame-by-frame manner. Consequently, the layer-specific circumferential strain and strain rate curves were generated. Similarly, in the 16 segments in these short-axis views, regional peak circumferential strain (rCS) in the inner layer, midwall, and outer layer were calculated, and averaged to yield peak global circumferential strain (CS). Also, the layer-specific regional systolic peak circumferential strain rate (rCSR) and systolic peak global circumferential strain rate (CSR) were measured. The strain and strain rate were taken as absolute values.

Statistical Analysis

We analyzed the data using SAS JMP software (version 11.0, SAS Institute, Cary, NC, USA). Continuous variables are expressed as mean±standard deviation. Paired *t*-test was used for the comparisons of parameter values obtained before and during the handgrip exercise. For the assessment of the correlations between wall stress and STE-derived parameters, we carried out linear regression analysis. An analysis of covariance was used to compare the strain before and during the exercise after adjusting for wall stress. The 95% confidence intervals for the values of STE parameters were computed and displayed on a regression plot. Two correlation coefficients drawn from the same sample were compared according to the method described by Cohen et al. [14]. The reproducibility of STE-derived parameters was assessed in 15 randomly selected subjects. Two independent blinded observers measured the peak strain and systolic peak strain rate from the same cine-loops, and one of them repeated the analysis on 2 separate days. Statistical significance was defined as $p<0.05$.

RESULTS

Relationships between Wall Stress and Global Strain/Strain Rate at Rest

The baseline characteristics of the study subjects are shown in **Table 1**. No abnormalities were detected by the echocardiographic examination in any subject. The subjects' hemodynamic and echocardiographic parameters before and during the handgrip exercise are shown in **Table 2**. None of the subjects had hypertension or tachycardia at rest. The CWS, CS, and CSR were highest in the inner myocardial layer and lowest in the outer layer.

The relationships between wall stress and global strain and those between wall stress and global strain rate at rest are shown in **Table 3**. The LS significantly correlated with MWS at rest. The CS significantly correlated with CWS in the inner layer and midwall, whereas that in the outer layer did not correlate with CWS. In contrast, there was no significant correlation between LSR and MWS or between CWS and CSR in any of the 3 layers at rest.

Their Relationships during Handgrip Exercise

Changes in hemodynamic and echocardiographic parameters by the handgrip exercise are shown in **Table 2**. SBP was significantly increased by the exercise. LVDs was also increased, whereas LVDd was not. The wall stress was increased to a greater degree than SBP, and the strain and strain rate were decreased to lesser degrees than both SBP and wall stress. The relationships between wall stress and global strain/strain rate during the exercise are shown in **Table 3**. The LS significantly correlated with MWS during the exercise. The CS also correlated with CWS in the inner layer and midwall. There was no significant correlation between CWS and CS in the outer layer, or between MWS and LSR or between CWS and CSR in any of the 3 layers.

Normal Range of the Relationships between Wall Stress and Global Strain/Strain Rate

There was no significant difference in the LS before and during the handgrip exercise after

adjusting MWS (p=0.47), indicating that the myocardial contractility did not change by the exercise. Regression analyses among all the points obtained before and during the exercise revealed that the LS significantly correlated linearly with MWS and that the CS also correlated linearly with CWS in all 3 layers (**Fig. 1**). Similarly, the LSR significantly correlated linearly with MWS, and the CSR correlated linearly with CWS in all 3 layers (**Fig. 2**).

Relationship between Wall Stress and Regional Strain/Strain Rate

We additionally analyzed the relationship between wall stress and rLS and that between wall stress and rCS in the inner layer (**Fig. 3, 4**). Similarly, the relationship between wall stress and regional strain rate was also analyzed (**Fig. 5, 6**). As a result, there was a significant linear correlation between wall stress and the regional strain/strain rate in almost all of 16 segments, and the regression coefficient in each segment was almost similar to that of global strain/strain rate.

Ratio of Individual Change in Global STE Parameters to Change in Wall Stress

Regarding global strain/strain rate, the individual slopes between the 2 points before and during the handgrip exercise were also measured and averaged among all of the subjects (**Fig. 1, 2**). The slopes were relatively constant among the subjects. The average value of the individual slope was similar to the regression coefficient of the wall stress-strain/strain rate relationship for LS and LSR, and for CS and CSR in the inner layer, midwall, and outer layer.

Comparison of Afterload Dependency between Strain and Strain Rate

The absolute value of percent change (%change) in LS by the handgrip exercise was significantly larger than that in LSR ($p<0.05$) (**Table 2**). In contrast, the differences in %change between CS and CSR did not reach significance in the inner layer or midwall (p=0.13, p=0.055,

respectively). In the outer layer, however, that in CS was significantly smaller than that in CSR ($p<0.05$).

The correlation of LS to MWS ($r=-0.53$) was significantly stronger than that of LSR ($r=-0.28$) ($p<0.05$). Similarly, that of CS ($r=-0.72$) was significantly stronger than that of CSR ($r=-0.47$) in the inner layer ($p<0.05$). The difference in correlation coefficients between CS and CSR in the midwall ($r=-0.57$ vs. $r=-0.46$, $p=0.36$) and outer layer ($r=-0.33$ vs. $r=-0.29$, $p=0.74$) did not reach significance, but the coefficient in CS tended to be higher than that in CSR.

Reproducibility

Intra- and interobserver variability values of strains were 4.1% and 6.2% for LS, 3.9% and 4.8% for CS in the inner layer, 5.8% and 7.9% for CS in the midwall, and 8.7% and 10.4% for CS in the outer layer, respectively. In contrast, those of strain rates were 4.5% and 5.2% for LSR, 3.3% and 4.7% for CSR in the inner layer, 5.3% and 6.9% for CSR in the midwall, and 7.9% and 9.7% for CSR in the outer layer, respectively.

DISCUSSION

The principal findings of the present study were as follows. (i) There was an inverse linear correlation between wall stress and both global and regional strain and between wall stress and both global and regional strain rate in the healthy young subjects. (ii) The ratio of individual change in global STE parameters to the change in wall stress induced by handgrip exercise did not differ notably among the subjects. (iii) Although strain and strain rate were decreased in response to the exercise to a lesser degree than the degree to which the wall stress was increased, the changes in these STE parameters did not appear negligibly small. (iv) The strain more closely correlated with wall stress compared to strain rate, whereas there was no significant difference in the variability of measurements between them, suggesting that the strain more strongly depends on afterload than does strain rate.

Wall Stress-Strain Relationship and Wall Stress-Strain Rate Relationship

Several studies have reported a significant inverse linear correlation between wall stress and LV fractional shortening [15, 16]. The ejection fraction was also reported to linearly correlate inversely with wall stress [16]. With respect to the relationship between wall stress and strain, there was an animal experiment in which MWS and LS were measured at various LV afterloads regulated by aortic banding [17]. As a result, the LS linearly correlated inversely with MWS. In contrast, 2 studies of human subjects revealed a significant inverse linear correlation between wall stress and strain [18, 19], whereas a study by Burns et al. [9] showed no significant correlation between them. Thus, it is not yet established whether strain correlates with wall stress. In the present study, there was a significant inverse correlation between wall stress and strain in the longitudinal and circumferential directions.

There have been few studies evaluating the relationship between wall stress and strain rate. Burns et al. [9] showed that both LSR and CSR correlated linearly with MWS in human subjects.

In their study, in which only 18 patients were enrolled, however, the regression analysis was performed using all of the data under different conditions, not only at rest but also during an infusion of glyceryl trinitrate and during volume overloading by the fluid infusion. As the strain and strain rate must depend on the preload [10], the relation to wall stress should be observed under a constant preload. In the present study, therefore, we paid close attention to keeping the LV preload and contractility. Actually, LVDd did not change by the exercise, thus we concluded that preload was maintained constant in our study. Consequently, both global and regional strain rate linearly correlated inversely with wall stress. Thus, this is the first study to demonstrate the inverse linear correlation between wall stress and myocardial strain rate in normal subjects.

Normal Range of Relationships between Wall Stress and Global Strain/Strain Rate

Our results suggested that there were no individual differences in the afterload dependency. In addition, the average of the ratio of individual change in STE parameters to the change in wall stress induced by the handgrip exercise among all subjects was almost identical to the regression coefficient derived from all of the data obtained before and during the exercise. The linear regression equations with 95% confidence intervals obtained in our healthy subjects can therefore be considered the normal range of relationships between wall stress and strain/strain rate under physiological conditions.

Strain and Strain Rate Changes in Response to Increase in Blood Pressure

Several studies have shown that strain and strain rate were decreased in response to an increase in wall stress [17]. Considering the states including resting and low-intensity exercise such as handgrip exercise as physiological, the range of variation as physiological responses in the wall stress and strain or strain rate in the present study were considerably wide. For example, the variation range in MWS was approximately $1000\times10^2\cdot mm^2\cdot dyn$, and that of LS was

approximately 4% as the strain value in response to the 18-mmHg rise in the subjects' SBP. Accordingly, clinicians should keep in mind that the variation range of strain due to the changes in blood pressure in the physiological response is not negligible when LV systolic function is evaluated using STE.

Comparison of Afterload Dependency between Myocardial Strain and Strain Rate

It is generally considered that the dependency of myocardial strain rate on the afterload is less than that of strain [20]. To our knowledge, however, there have been few studies that compared the afterload dependency between them. In one of the above-mentioned studies of human subjects by Burns et al. [9], the myocardial strain did not significantly correlate with wall stress, whereas strain rate did. In that study, however, the preload varied, and the afterload dependency should not be accurately compared. Thus, it has still not been established whether strain or strain rate is less dependent on the afterload.

Here we assessed the relationship between wall stress and strain and that between wall stress and strain rate under conditions in which wall stress was varied by a handgrip exercise but the LV preload and contractility were essentially maintained constant [21]. As a result, the strain more strongly correlated with wall stress compared to strain rate. Strain rate is generally considered to be noisier and less robust than strain when measured using STE. However, the reproducibility of strain and strain rate was similar in the present study, which suggested that the variability of measurements was not different between these parameters. We thus concluded that strain depends more strongly on the afterload than does strain rate. The reason for this might be referred from the difference in the time phase between the peak strain and systolic peak strain rate. The exercise-induced %change in the longitudinal strain during the latter half of systole was significantly greater than that during the early half of systole, suggesting that increased wall stress mainly affected LV wall deformation during the latter half of systole. Because the systolic

strain rate usually forms the peak during the early half of systole, it might be less dependent on the wall stress than is strain.

Clinical Implications

The present study demonstrated that the LV strain and strain rate considerably vary even in healthy subjects, and even in response to a slight elevation in blood pressure which could be considered as the physiological response. It further provided the normal range of relationships between LV wall stress and STE parameters in the longitudinal and circumferential directions. It is well known, in cardiac hypertrophy such as hypertensive heart disease and hypertrophic cardiomyopathy, that LV ejection fraction is often preserved due to a possible compensatory mechanism for impaired myocardial shortening through concentric LV hypertrophy [5, 22]. Heart failure with a preserved ejection fraction can develop under such conditions [23]. In these patients, myocardial function should be evaluated not by using STE parameters alone but by measuring both STE parameters and wall stress, and by reference to the normal relationships between them derived in the present study. The interpretation of STE parameters considering wall stress may better distinguish high-risk patients. The actual myocardial function cannot be determined only by STE parameters also in dilated LV, because the wall stress should be substantially increased. If STE parameters are used alone, therefore, myocardial function must be underestimated. Wall stress-corrected STE parameters are thus likely to be needed for a better understanding of myocardial function and prognostic prediction in such circumstances. Additionally, strain might more strongly depend on the afterload than did strain rate. Thus in some circumstances, the strain and strain rate could be used differently in the future.

Limitations

Several study limitations should be acknowledged. First, it is not clear whether the LV

contractility truly did not change in our study subjects. However, a previous study has demonstrated that the LV preload and contractility do not change by the handgrip exercise [21]. In addition, we confirmed that there was no significant difference in LS before and during the exercise after adjusting for MWS.

Second, our subjects' heart rates were slightly but significantly increased by the handgrip exercise. It was reported that an increase in heart rate led to a decrease in strain, and thus we should take into account the change in heart rate [24]. However, in our present study there was no significant correlation between heart rate and LS ($p=0.12$) or heart rate and LSR ($p=0.07$) among all of the points obtained before and during the exercise. We therefore believe that the increase in heart rate by the exercise did not have a significant impact on the present results.

CONCLUSIONS

In healthy young subjects, the LV strain and strain rate linearly correlated inversely with wall stress in the longitudinal and circumferential directions, and strain more strongly depended on the afterload than did strain rate. Because the variation range of these parameters was not negligibly small even in response to a slight elevation in blood pressure induced by a handgrip exercise, myocardial shortening should be evaluated based on the relationships between these STE parameters and wall stress.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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FIGURE LEGENDS

Figure 1. Relationships between wall stress and global myocardial strain among all points obtained before and during handgrip exercise.

The regression lines (**solid line**) and 95% confidence intervals (**dashed line**) are provided. The individual slopes between the two points before and during the handgrip exercise were calculated and averaged. Abbreviations are the same as in Table 2.

Figure 2. Relationships between wall stress and global myocardial strain rate among all points obtained before and during handgrip exercise.

The regression lines (**solid line**) and 95% confidence intervals (**dashed line**) are provided. The individual slopes between the two points before and during the handgrip exercise were calculated and averaged. Abbreviations are the same as in Table 2.

Figure 3. Relationships between wall stress and regional longitudinal strain among all points obtained before and during handgrip exercise

rLS, regional peak longitudinal strain. MWS, meridional wall stress.

Figure 4. Relationships between wall stress and regional circumferential strain among all points obtained before and during handgrip exercise

rCS, regional peak circumferential strain. CWS, circumferential wall stress.

Figure 5. Relationships between wall stress and regional longitudinal strain rate among all points obtained before and during handgrip exercise

rLSR, regional systolic peak longitudinal strain rate. MWS, meridional wall stress.

Figure 6. Relationships between wall stress and regional circumferential strain rate among all points obtained before and during handgrip exercise

rCSR, regional systolic peak circumferential strain rate. CWS, circumferential wall stress.

Table 1. Baseline characteristics of study subjects (n = 41)

Age (yrs)	29.0±5.0
Male/female	30/11
Body height (cm)	168.6±7.7
Body weight (kg)	62.7±10.5
Body surface area (m^2)	1.71±0.17
LVDd (mm)	46.1±2.9
LVDs (mm)	30.4±3.1
IVST (mm)	7.6±0.9
PWT (mm)	7.6±1.0
FS (%)	34.0±5.5
LVEF (%)	63.1±4.1

LV, left ventricular. LVDd, LV end-diastolic dimension. LVDs, LV end-systolic dimension. IVST, interventricular septal thickness. PWT, posterior wall thickness. FS, fractional shortening. LVEF, LV ejection fraction.

Table 2. Changes in hemodynamic and echocardiographic parameters by handgrip exercise

	Before handgrip	During handgrip	%change	p-value
Systolic blood pressure (mmHg)	97.6±11.1	116.0±14.9	+18.9±7.4%	<0.01
Diastolic blood pressure (mmHg)	52.2±9.6	62.0±12.2	+19.8±18.3%	<0.01
Heart rate (bpm)	63.8±9.4	70.0±10.5	+10.4±11.9%	<0.01
LVDd (mm)	46.1±2.9	46.3±3.0	+0.3±1.4%	NS
LVDs (mm)	30.4±3.1	33.2±3.2	+9.5±7.9%	<0.01
WTs (mm)	11.1±1.3	10.2±1.2	–8.4±4.4%	<0.01
MWS (dyn・mm^{-2})	660±130	975±174	+49.2±18.2%	<0.01
CWS (dyn・mm^{-2})				
Inner layer	885±123	1159±172	+31.5±14.6%	<0.01
Midwall	499±104	728±138	+47.9±21.9%	<0.01
Outer layer	373±89	564±119	+54.2±25.2%	<0.01
LS (%)	17.2±1.7	15.7±1.4	–8.4±4.8%	<0.01
CS (%)				
Inner layer	25.0±1.4	22.3±1.9	–10.7±5.9%	<0.01
Midwall	16.9±1.2	15.2±1.7	–9.8±6.9%	<0.01
Outer layer	11.5±1.4	10.6±1.8	–7.2±13.2%	<0.01
LSR (s^{-1})	0.80±0.09	0.75±0.08	–6.4±5.5%	<0.01
CSR (s^{-1})				
Inner layer	1.25±0.12	1.10±0.11	–12.1±7.1%	<0.01
Midwall	0.88±0.09	0.77±0.09	–11.8±8.7%	<0.01
Outer layer	0.62±0.09	0.56±0.09	–9.9±13.7%	<0.01

WTs, end-systolic wall thickness averaged between interventricular septum and left ventricular posterior wall. MWS, meridional wall stress. CWS, circumferential wall stress. LS, peak global longitudinal strain. CS, peak global circumferential strain. LSR, systolic peak global longitudinal strain rate. CSR, systolic peak global circumferential strain rate. Other abbreviations are the same as in Table 1.

Table 3. Linear regression analyses for relationships between wall stress and global myocardial strain/strain rate before and during handgrip exercise

	Before handgrip exercise			During handgrip exercise		
	Regression equation	r	p-value	Regression equation	r	p-value
MWS-LS	y=−0.0041x+19.9	−0.32	<0.05	y=−0.0033x+18.9	−0.40	<0.05
CWS-CS						
Inner layer	y=−0.0062x+30.4	−0.53	<0.01	y=−0.0055x+28.7	−0.51	<0.01
Midwall	y=−0.0038x+18.8	−0.34	<0.05	y=−0.0048x+18.8	−0.39	<0.05
Outer layer	y=−0.0024x+12.4	−0.15	0.34	y=−0.0038x+12.7	−0.25	0.11
MWS-LSR	y=−0.00010x+0.87	−0.14	0.38	y=−0.000041x+0.79	−0.09	0.59
CWS-CSR						
Inner layer	y=−0.00018x+1.41	−0.19	0.25	y=−0.000095x+1.21	−0.14	0.38
Midwall	y=−0.00015x+0.95	−0.17	0.28	y=−0.00014x+0.87	−0.21	0.19
Outer layer	y=−0.000040x+0.64	−0.04	0.81	y=−0.00010x+0.62	−0.13	0.42

Abbreviations are the same as in Table 2.

Fig. 1

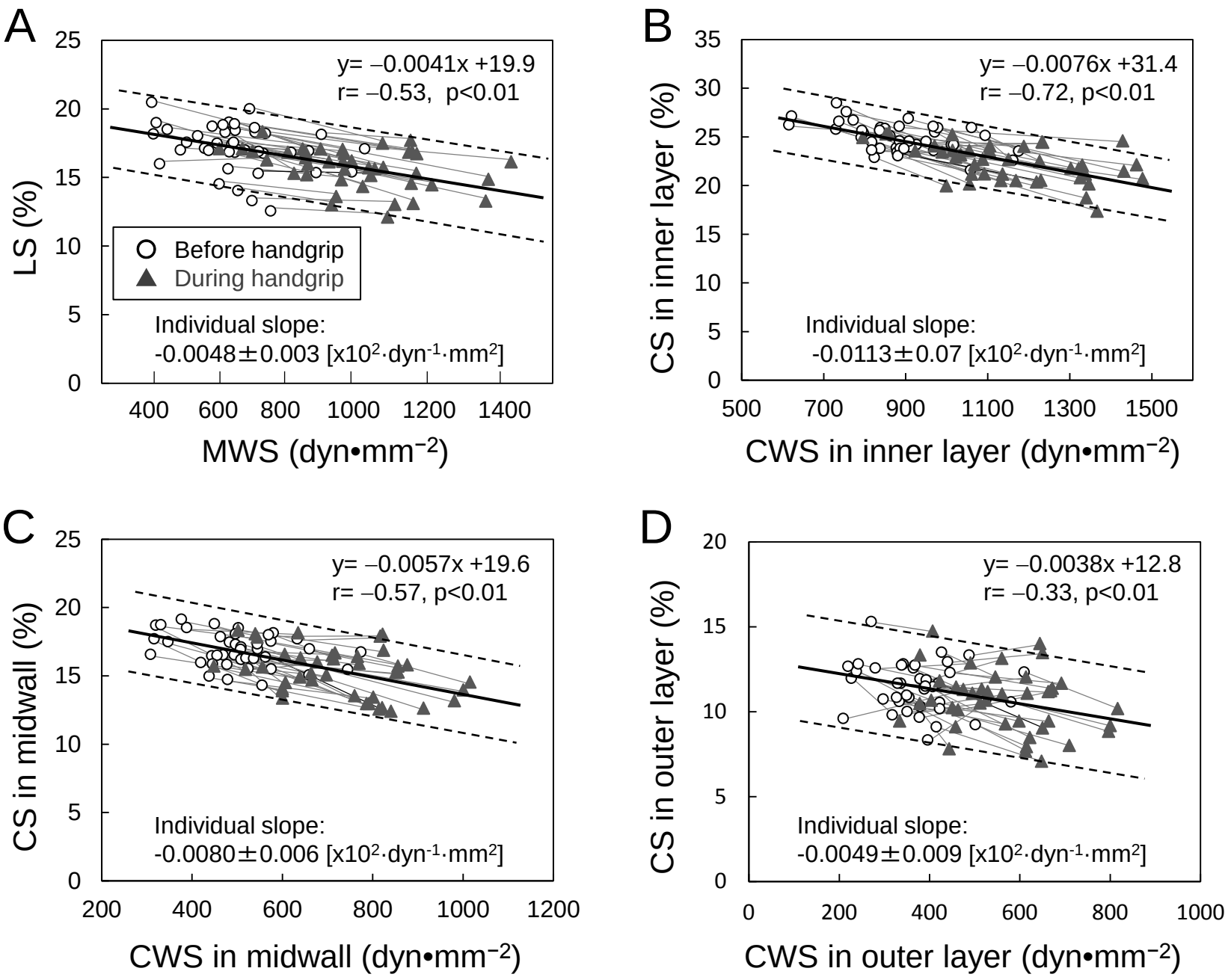

A
y= –0.0041x +19.9
r= –0.53, p<0.01
Before handgrip
During handgrip
Individual slope:
-0.0048±0.003 [x10²·dyn⁻¹·mm²]
LS (%)
MWS (dyn•mm⁻²)
B
y= –0.0076x +31.4
r= –0.72, p<0.01
Individual slope:
-0.0113±0.07 [x10²·dyn⁻¹·mm²]
CS in inner layer (%)
CWS in inner layer (dyn•mm⁻²)
C
y= –0.0057x +19.6
r= –0.57, p<0.01
Individual slope:
-0.0080±0.006 [x10²·dyn⁻¹·mm²]
CS in midwall (%)
CWS in midwall (dyn•mm⁻²)
D
y= –0.0038x +12.8
r= –0.33, p<0.01
Individual slope:
-0.0049±0.009 [x10²·dyn⁻¹·mm²]
CS in outer layer (%)
CWS in outer layer (dyn•mm⁻²)

Fig. 2

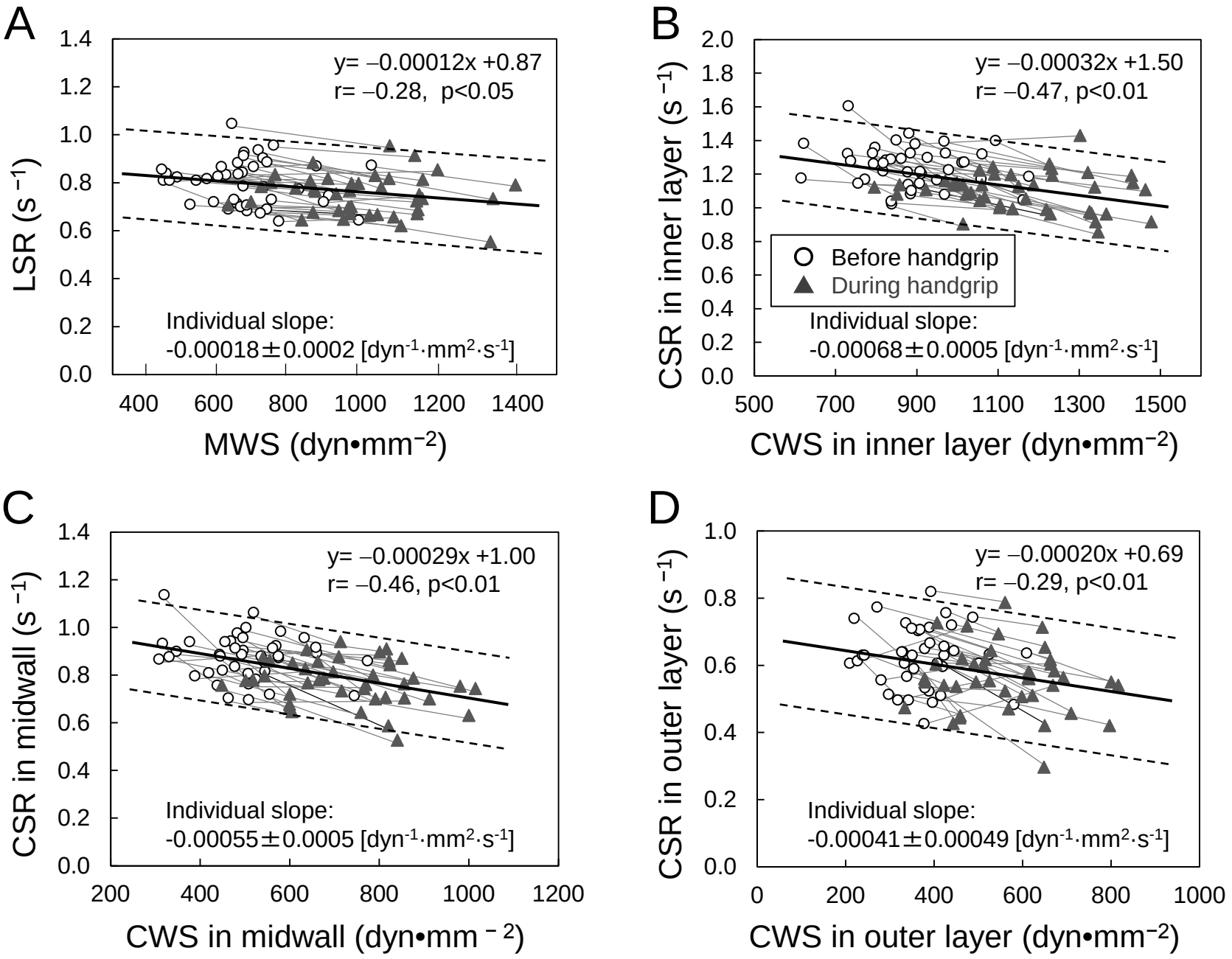

A
y= –0.00012x +0.87
r= –0.28, p<0.05
Individual slope:
-0.00018±0.0002 [dyn-1·mm2·s-1]
LSR (s –1)
MWS (dyn•mm–2)
B
y= –0.00032x +1.50
r= –0.47, p<0.01
Before handgrip
During handgrip
Individual slope:
-0.00068±0.0005 [dyn-1·mm2·s-1]
CSR in inner layer (s –1)
CWS in inner layer (dyn•mm–2)
C
y= –0.00029x +1.00
r= –0.46, p<0.01
Individual slope:
-0.00055±0.0005 [dyn-1·mm2·s-1]
CSR in midwall (s –1)
CWS in midwall (dyn•mm – 2)
D
y= –0.00020x +0.69
r= –0.29, p<0.01
Individual slope:
-0.00041±0.00049 [dyn-1·mm2·s-1]
CSR in outer layer (s –1)
CWS in outer layer (dyn•mm–2)

Fig. 3

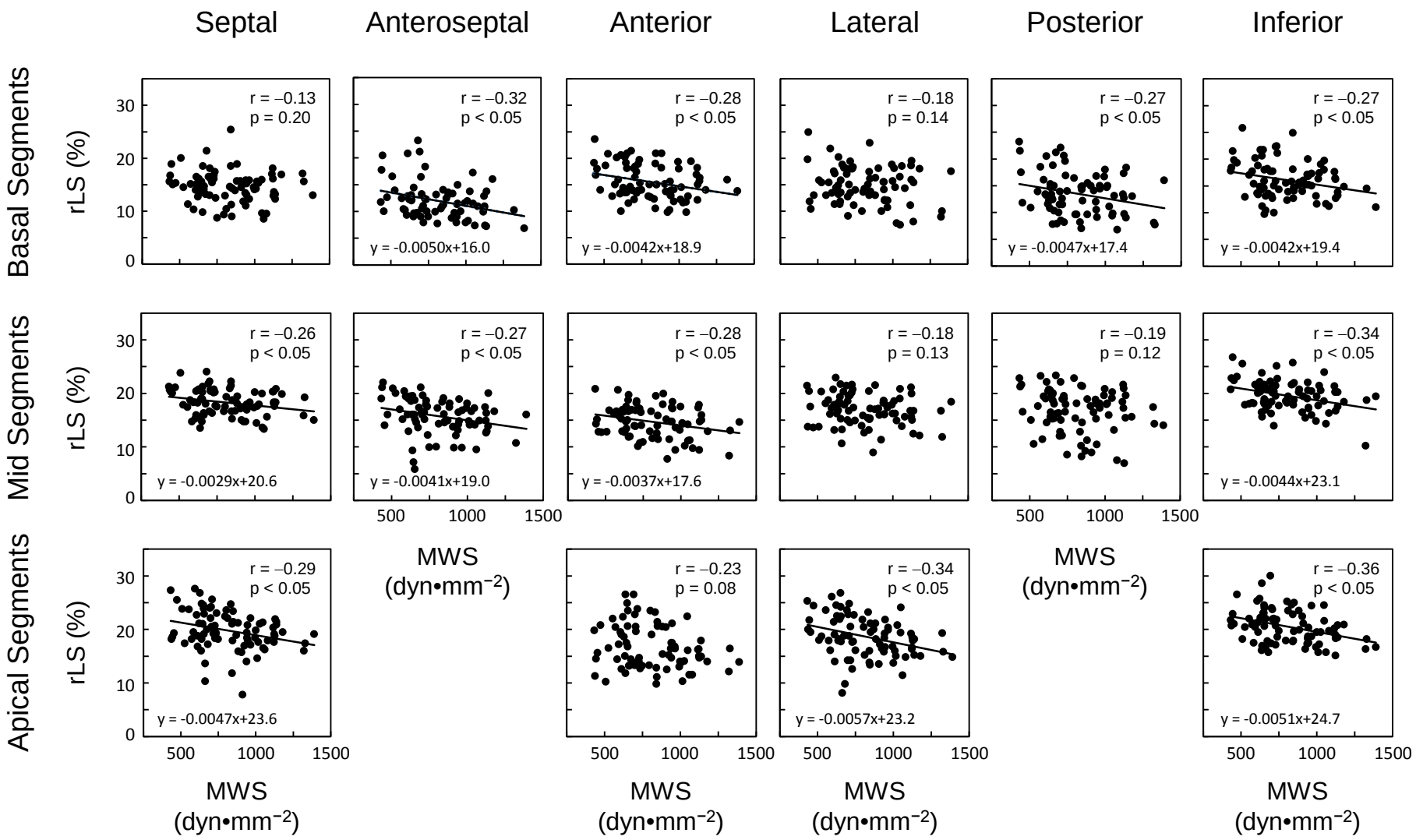

Septal
Anteroseptal
Anterior
Lateral
Posterior
Inferior
Basal Segments
Mid Segments
Apical Segments
rLS (%)
MWS (dyn•mm⁻²)
r = –0.13
p = 0.20
y = -0.0050x+16.0
r = –0.32
p < 0.05
y = -0.0042x+18.9
r = –0.28
p < 0.05
r = –0.18
p = 0.14
y = -0.0047x+17.4
r = –0.27
p < 0.05
y = -0.0042x+19.4
r = –0.27
p < 0.05
r = –0.26
p < 0.05
y = -0.0029x+20.6
r = –0.27
p < 0.05
y = -0.0041x+19.0
r = –0.28
p < 0.05
y = -0.0037x+17.6
r = –0.18
p = 0.13
r = –0.19
p = 0.12
r = –0.34
p < 0.05
y = -0.0044x+23.1
r = –0.29
p < 0.05
y = -0.0047x+23.6
r = –0.23
p = 0.08
r = –0.34
p < 0.05
y = -0.0057x+23.2
r = –0.36
p < 0.05
y = -0.0051x+24.7
0
10
20
30
500
1000
1500

Fig. 4

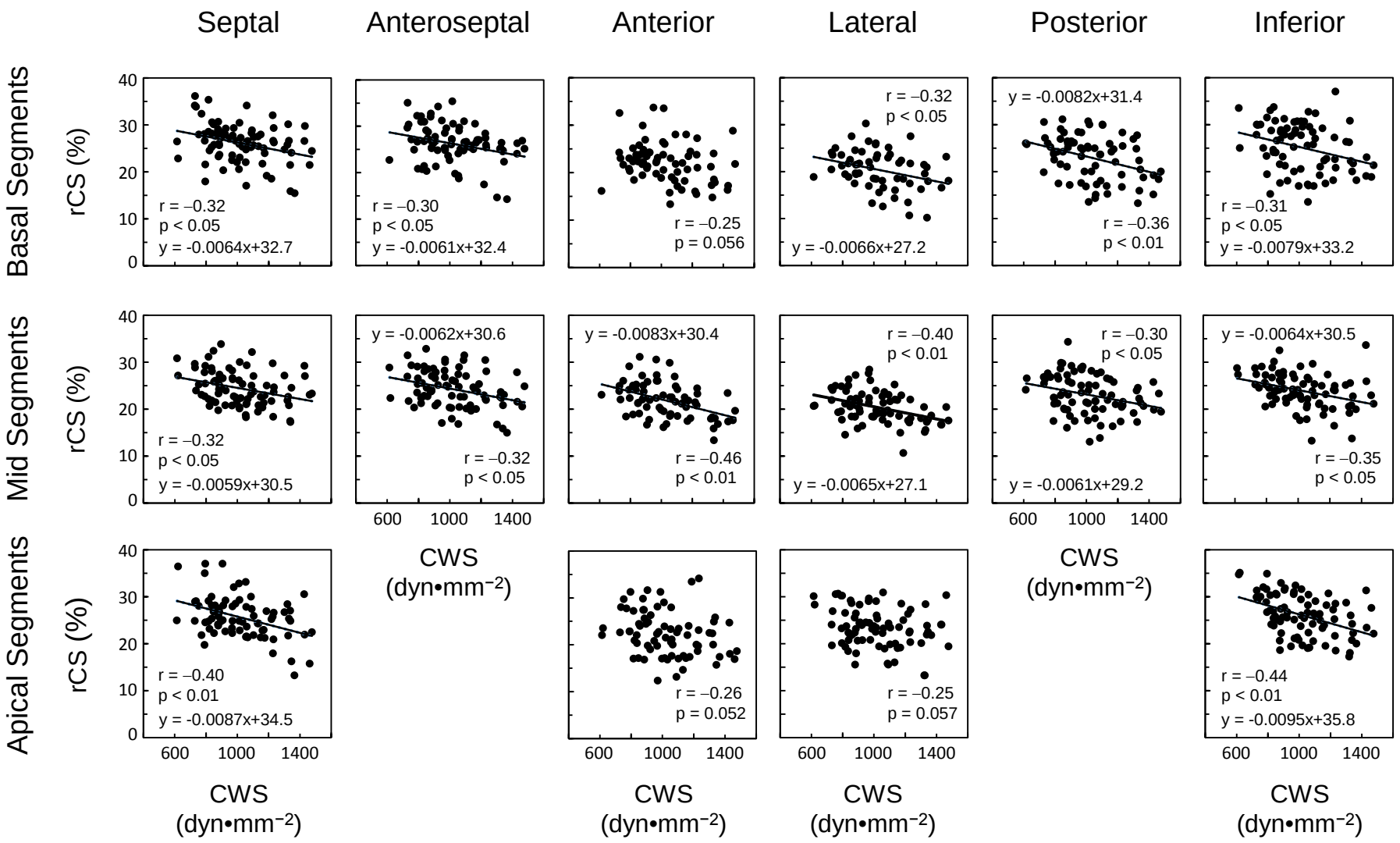

Septal
Anteroseptal
Anterior
Lateral
Posterior
Inferior
Basal Segments
Mid Segments
Apical Segments
rCS (%)
CWS (dyn•mm⁻²)
r = –0.32
p < 0.05
y = -0.0064x+32.7
r = –0.30
p < 0.05
y = -0.0061x+32.4
r = –0.25
p = 0.056
r = –0.32
p < 0.05
y = -0.0066x+27.2
y = -0.0082x+31.4
r = –0.36
p < 0.01
r = –0.31
p < 0.05
y = -0.0079x+33.2
r = –0.32
p < 0.05
y = -0.0059x+30.5
y = -0.0062x+30.6
r = –0.32
p < 0.05
y = -0.0083x+30.4
r = –0.46
p < 0.01
r = –0.40
p < 0.01
y = -0.0065x+27.1
r = –0.30
p < 0.05
y = -0.0061x+29.2
y = -0.0064x+30.5
r = –0.35
p < 0.05
r = –0.40
p < 0.01
y = -0.0087x+34.5
r = –0.26
p = 0.052
r = –0.25
p = 0.057
r = –0.44
p < 0.01
y = -0.0095x+35.8
0
10
20
30
40
600
1000
1400

Fig. 5

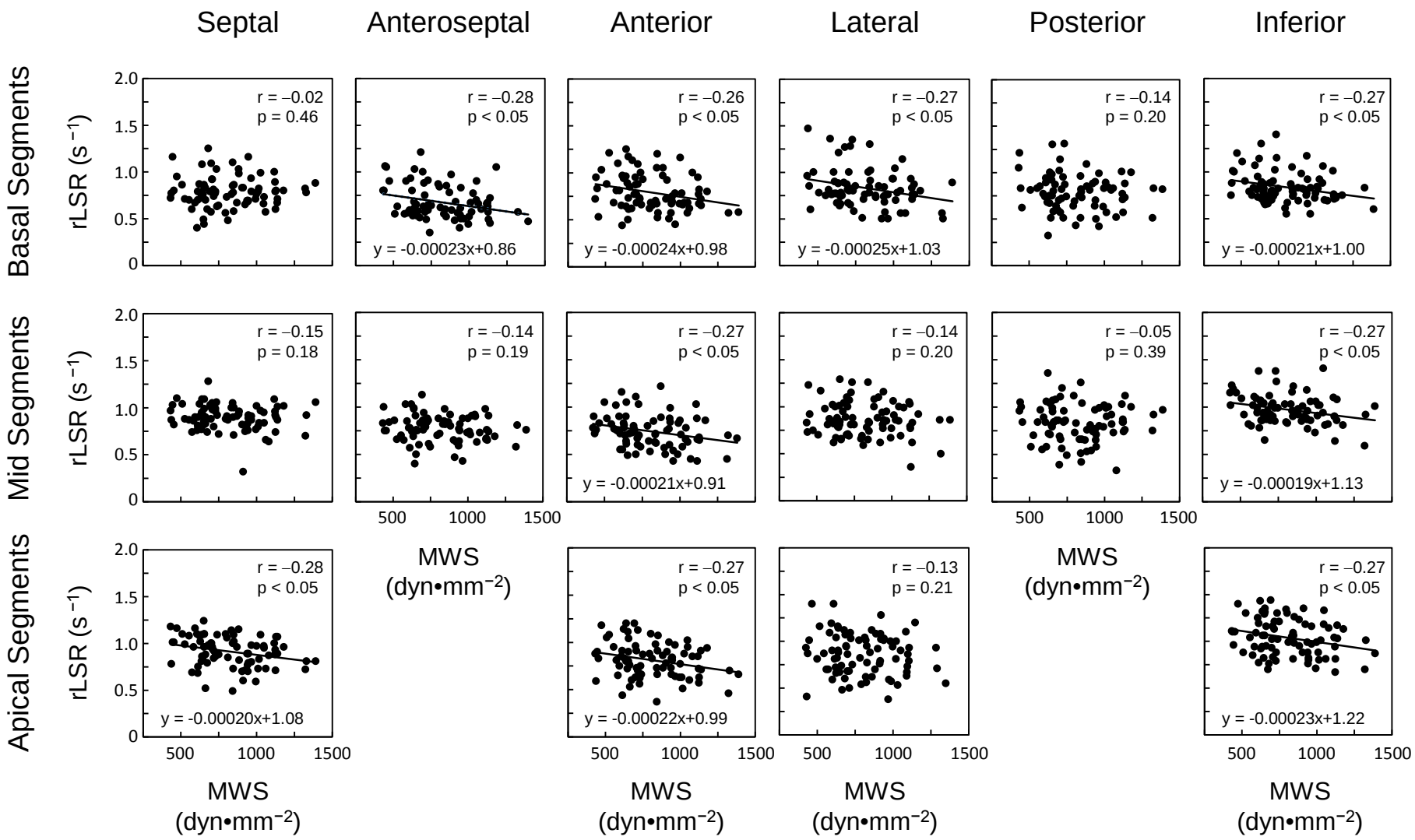
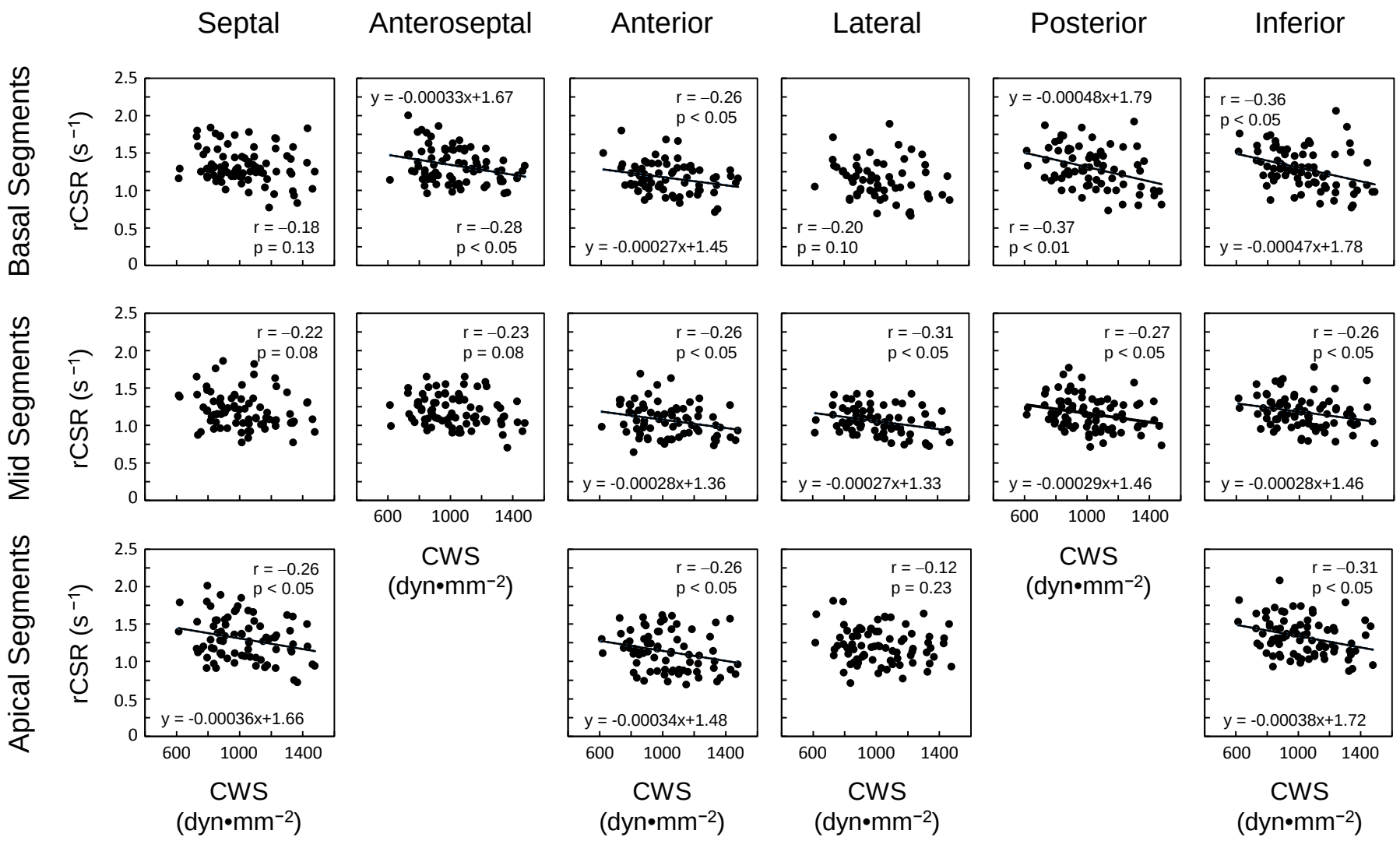

Septal
Anteroseptal
Anterior
Lateral
Posterior
Inferior
Basal Segments
Mid Segments
Apical Segments
rLSR (s⁻¹)
MWS (dyn•mm⁻²)
r = –0.02
p = 0.46
r = –0.28
p < 0.05
y = -0.00023x+0.86
r = –0.26
p < 0.05
y = -0.00024x+0.98
r = –0.27
p < 0.05
y = -0.00025x+1.03
r = –0.14
p = 0.20
r = –0.27
p < 0.05
y = -0.00021x+1.00
r = –0.15
p = 0.18
r = –0.14
p = 0.19
r = –0.27
p < 0.05
y = -0.00021x+0.91
r = –0.14
p = 0.20
r = –0.05
p = 0.39
r = –0.27
p < 0.05
y = -0.00019x+1.13
r = –0.28
p < 0.05
y = -0.00020x+1.08
r = –0.27
p < 0.05
y = -0.00022x+0.99
r = –0.13
p = 0.21
r = –0.27
p < 0.05
y = -0.00023x+1.22

Fig. 6


Septal
Anteroseptal
Anterior
Lateral
Posterior
Inferior
Basal Segments
Mid Segments
Apical Segments
rCSR (s⁻¹)
CWS (dyn•mm⁻²)
y = -0.00033x+1.67
r = –0.18
p = 0.13
r = –0.28
p < 0.05
r = –0.26
p < 0.05
y = -0.00027x+1.45
r = –0.20
p = 0.10
y = -0.00048x+1.79
r = –0.37
p < 0.01
r = –0.36
p < 0.05
y = -0.00047x+1.78
r = –0.22
p = 0.08
r = –0.23
p = 0.08
r = –0.26
p < 0.05
y = -0.00028x+1.36
r = –0.31
p < 0.05
y = -0.00027x+1.33
r = –0.27
p < 0.05
y = -0.00029x+1.46
r = –0.26
p < 0.05
y = -0.00028x+1.46
r = –0.26
p < 0.05
y = -0.00036x+1.66
r = –0.26
p < 0.05
y = -0.00034x+1.48
r = –0.12
p = 0.23
r = –0.31
p < 0.05
y = -0.00038x+1.72