LIMITATIONS OF UNIDIMENSIONAL INDEXES OF RIGHT VENTRICULAR CONTRACTILE FUNCTION IN CONSCIOUS DOGS

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Objective: Our goal was to examine the validity of unidimensional indexes of right ventricular contractile performance *in vivo*.

Methods: Unidimensional indexes and global measurements of right ventricular volume and contractile performance were compared in 6 conscious dogs. Vena caval occlusions were performed before (control) and during pulmonary arterial or aortic constriction.

Results: Moderately strong relationships were demonstrated between right ventricular septal-free wall indexes and global measurements of right ventricular end-diastolic and end-systolic volumes, stroke volume, stroke work, and the slope of the preload recruitable stroke work relationship, respectively, under control conditions (mean r^2 range 0.69-0.94). These relationships were shifted significantly, however, by increased right ventricular afterload. Increased left ventricular afterload significantly shifted the relationships between right ventricular septal-free wall dimensions and end-diastolic and end-systolic volumes. Relationships between the corresponding regional right ventricular free wall segmental indexes and global measurements under control conditions were weaker (mean r^2 range 0.12-0.65) and were significantly more sensitive to distortion by both increased right and left ventricular afterload, the effects of which were generally in opposite directions. These observations are consistent with significant ventricular interactive effects on the relationship between single right ventricular dimensions and right ventricular volume.

Conclusion: Unidimensional right ventricular measurements are not reliable surrogates for right ventricular volume when assessing right ventricular contractile performance in the intact heart. (*J Thorac Cardiovasc Surg* 2000;120:302-12)

The investigation of right ventricular (RV) function has been hampered by the difficulty of measuring the instantaneous volume of the RV due to its complex geometry. Several investigators have attempted to circumvent this difficulty by using single RV regional dimensions as surrogates for RV volume.¹⁻¹⁰ One poten-

tially important limitation of such unidimensional indexes of RV volume is the interactive effect of left ventricular (LV) volume on RV shape.¹¹⁻¹³ In isolated canine hearts, the relationship between RV free wall segment length and RV volume was linear at any given LV volume, but the position of this relationship shifted with changes in LV volume.¹³ When RV volume was held constant, RV free wall segment lengths increased or decreased in parallel with changes in LV volume, while the distance between the RV free wall and the interventricular septum was inversely related to LV volume.¹³ These observations led to the development of an ellipsoidal shell subtraction model of RV volume that was independent of LV volume, permitting assessment of RV function in the intact heart.^{4,13,14}

Recently, it has been argued that the complexity of this model and other methods of measuring RV volume¹⁵⁻¹⁷ may be unnecessary for many applications.⁹

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Unidimensional measurements of RV free wall segment shortening were linearly related to RV stroke volume when RV preload was altered.⁹ RV free wall regional stroke work, calculated as RV pressure-dimension loop area, was linearly related to the end-diastolic segment length,⁹ thus providing a potential unidimensional surrogate for the linear preload recruitable stroke work (PRSW) relationship between global RV stroke work (pressure-volume loop area) and end-diastolic volume as an index of RV contractile function.¹⁴

No attempt was made, however, to determine the susceptibility of these unidimensional measurements to the potentially confounding effects of ventricular interaction noted above. The observations were confined to the relatively symmetrical effects of preload reduction on both ventricles.⁹ Important distortions of both RV and LV geometry are well documented with RV volume or pressure overload,^{12,13,18} however, and interactive effects might also be expected with altered LV afterload.^{1,2,4,5,7,19,20} The impact of altered LV and RV afterload on unidimensional indexes of RV volume and contractile function remains untested in vivo. The purpose of this investigation was to document the relationship between unidimensional indexes and 3-dimensional measurements of RV volume and contractile function under experimental conditions of altered RV and LV afterload in vivo.

Materials and methods

The experimental procedures and protocols described below were approved by our institutional ethics committee and conformed to the guidelines of the National Health and Medical Research Council of Australia on the care and use of animals.

Experimental preparation. Six healthy adult dogs (22–44 kg) were anesthetized (halothane 1% to 2% and succinylcholine 0.3 mg/kg intravenously after thiamylal sodium 20 mg/kg intravenously). Through a left thoracotomy, ultrasonic dimension transducers were positioned across the base-apex (a) and anterior-posterior (b) axes of the LV and the septal-free wall minor axes of the LV (c) and RV (d).^{11–14} Particular care was taken to locate the septal transducer as close as possible to the RV endocardial surface. An additional pair of transducers was positioned to measure a regional segment length on the RV mid-free wall, perpendicular to the major axis.¹³ Pneumatic occluders were positioned around the inferior vena cava, the descending aorta, and the main pulmonary artery. Heparin-filled silicone catheters (2.6-mm inner diameter) were secured in the base of the left atrial appendage and apex of the RV. Another catheter with multiple side holes was positioned adjacent to the ventricular epicardium. The pericardium was left open and the thoracotomy repaired. After 7 to 10 days, the transducer leads, catheters, and occluder tubing were exteriorized from a subcutaneous

pouch. Each animal, lying quietly on its right side, was studied in the conscious state 1 hour after light sedation (morphine sulfate, 10 mg intramuscularly).

Data acquisition and experimental protocol. Micro-manometer-tipped catheters (model MPC-500; Millar Instruments, Inc, Houston, Tex) were passed via the implanted catheters to obtain LV, RV, and pleural pressures. Attenuation of autonomic reflexes was achieved with intravenous propranolol (1 mg/kg) and atropine (0.2 mg/kg). Data were recorded under steady-state conditions and during transient vena caval occlusion induced by inflation of the implanted occluder for approximately 10 seconds. After all variables had returned to their baseline levels, the pulmonary arterial occluder was partially inflated to achieve a stable increase in RV systolic pressure of at least 50%. Transient vena caval occlusion was repeated while the pulmonary arterial constriction was maintained. After release of both occluders and restabilization to baseline levels, transient vena caval occlusion was repeated. After restabilization, the descending aortic occluder was partially inflated to achieve a stable increment in LV systolic pressure of approximately 30%. Transient vena caval occlusion was repeated while the aortic constriction was maintained.

Data analysis. Data were digitized in real time at 200 Hz. RV chamber volume was calculated from the ultrasonic cardiac dimension measurements according to an ellipsoidal shell subtraction model ($\pi/6 a \cdot b \cdot d$ – RV free wall volume).¹³ RV free wall volume was determined post mortem by displacement in water. RV and LV transmural pressures were calculated as chamber pressure minus pleural pressure. End-diastole was defined initially 40 ms before a positive rate of pressure rise (dP/dt) of 200 mm Hg $\cdot$ s⁻¹, then visually adjusted to correspond with the maximum RV volume before ejection. End-systole was defined initially as the time of peak negative dP/dt , then visually adjusted to correspond with the minimum RV volume.¹⁴

Global stroke work was calculated as RV pressure-volume loop area for each beat. RV septal-free wall dimensional stroke work and RV free wall segment stroke work were calculated as the respective pressure-dimension loop areas for each beat. These stroke work values were plotted as a function of the corresponding end-diastolic measurements of volume or dimension obtained during vena caval occlusions. Global and regional RV contractile function were quantified by determining global and regional PRSW relationships by linear regression analyses (Fig 1).^{14,21}

Statistical analysis. Results are summarized as mean $\pm$ SD. The effects of increased RV or LV afterload on baseline hemodynamic variables were determined with analysis of variance. The relationships between unidimensional indexes and their global counterparts were determined by linear regression analyses. The global variables examined were RV end-diastolic volume, end-systolic volume, stroke volume, stroke work, and the slope and intercept of the global PRSW relationship. The corresponding unidimensional indexes of these global variables for both the septal-free wall dimension and RV free wall segment were the end-diastolic length, end-systolic length, systolic shortening, regional stroke work, and

Fig 1. Representative pressure-volume and pressure-dimension loops obtained from 1 dog during a single vena caval occlusion (*upper panels*) and the corresponding global and unidimensional PRSW relationships derived from these loops (*lower panels*). EDV, EDD, and EDL, End-diastolic volume, dimension, and length; RV, right ventricular; SW_G , SW_D , and SW_L , global, dimensional and segmental stroke work; M_{wG} , M_{wD} , and M_{wL} , global, septal-free wall dimensional and free wall segmental PRSW slopes; V_w , D_w , and L_w , global, dimensional and segmental PRSW x-axis intercepts.

the slope and intercept of the regional PRSW relationship, respectively. In determining these relationships, absolute dimensions and volumes were normalized to their respective baseline, steady-state values to account for differences in heart size and ultrasonic unidimensional transducer separation at the time of implantation.

The influence of increased RV or LV afterload on these relationships was determined by multiple linear regression analyses.²² The general multiple linear regression model was as follows:

$$y = b_0 + b_1 \cdot x + b_2 \cdot \text{IN} + \sum_{i=1}^5 d_i \cdot D_i + \sum_{i=1}^5 e_i \cdot D_i \cdot x + \sum_{i=1}^5 f_i \cdot D_i \cdot \text{IN} \quad (1)$$

where IN is a dummy variable coding for the loading condition (IN = 1 for control and -1 for aortic or pulmonary arterial constriction) and D_i represents dummy variables for each dog ($D_i = 1$ for dog = i [for $i = 1-5$], $D_i = 0$ for dog $\neq i$, and $D_i = -1$ for dog 6). Data obtained during increased RV or LV afterload were compared with control data obtained during the first vena caval occlusion.

Results

Representative RV pressure-volume and pressure-dimension loops obtained from 1 dog during a single vena caval occlusion are shown in Fig 1, together with the corresponding global and unidimensional PRSW relationships derived from these loops. Representative RV dimensions and RV and LV pressures recorded from 1 dog before and during aortic constriction and pulmonary arterial constriction are shown in Fig 2. The effects of pulmonary arterial constriction and aortic constriction on steady-state hemodynamic variables are summarized in Table I. Pulmonary arterial constriction increased RV mean ejection pressure by $83\% \pm 24\%$, with consequent increases in RV end-diastolic volume and reduced systolic shortening and stroke volume. Aortic constriction increased LV mean ejection pressure more modestly, by $18\% \pm 8\%$, with no significant changes in the other steady-state hemodynamic variables.

Fig 2. Representative recordings of cardiac dimensions and RV and LV pressure from 1 dog before and during aortic constriction and pulmonary arterial constriction, the onsets of which are indicated by the arrows. RV, Right ventricular; LV, left ventricular.

The effects of these interventions on the relationships between unidimensional indexes and the corresponding global measurements of RV end-diastolic volume, end-systolic volume, stroke volume, and stroke work are shown in Fig 3. The results of the linear regression analyses of the pooled data for these relationships are summarized in Table II. At end-diastole and end-systole, the coefficients of determination (r^2 values) in Table II indicate that changes in the septal-free wall dimension are moderately well accounted for by the changes in RV volume under control conditions and with increased RV or LV afterload. Increased RV afterload, however, significantly increased the slope of both relationships, whereas increased LV afterload caused a small but significant downward shift in both relationships.

The corresponding relationships between free wall segment lengths and end-diastolic and end-systolic volumes were generally weaker. There was significant distortion of the end-diastolic relationship by increased LV afterload. The slope of the end-systolic relationship increased significantly with both increased RV and LV afterload.

Strong relationships were evident between septal-free wall shortening and stroke volume under control conditions and with increased RV or LV afterload. The relationship shifted downward significantly, however, with increased RV afterload, but was not significantly influenced by increased LV afterload. A much weaker relationship was evident between free wall shortening and stroke volume under all conditions and was significantly distorted by both increased RV and LV afterload.

Table I. Effects of pulmonary arterial constriction and aortic constriction on steady-state hemodynamic variables

	HR (beats/min)	LVMEP (mm Hg)	RVMEP (mm Hg)	EDV (mL)	SV (mL)
Control	136 ± 11	126 ± 9	35 ± 3	54 ± 13	17 ± 9
PAC	143 ± 12*	119 ± 7	64 ± 6*	66 ± 14*	13 ± 8*
AC	132 ± 14	150 ± 9*	37 ± 2	54 ± 15	14 ± 10

Table II. Results of linear regression analyses of pooled unidimensional and global RV hemodynamic variables

Global variable	EDV*			ESV*		
	Slope	Y-int (mL)	r ²	Slope	Y-int (mL)	r ²
<i>Septal-free wall dimension</i>						
Control	0.23 (0.02)	0.75 (0.01)	0.69	0.20 (0.01)	0.79 (0.01)	0.76
PAC	0.39 (0.01)	0.66 (0.01)	0.90 ↑	0.34 (0.02)	0.72 (0.02)	0.84 ↑
AC	0.29 (0.02)	0.68 (0.02)	0.66 ↓	0.21 (0.01)	0.77 (0.01)	0.72 ↓
<i>Free wall segment length</i>						
Control	0.14 (0.02)	0.86 (0.01)	0.50	0.08 (0.01)	0.92 (0.01)	0.41
PAC	0.11 (0.01)	0.89 (0.00)	0.83	0.14 (0.01)	0.88 (0.01)	0.70 ↑
AC	0.22 (0.02)	0.81 (0.01)	0.54 ↑	0.16 (0.01)	0.88 (0.01)	0.70 ↑

The relationship between septal-free wall dimensional stroke work and global stroke work was strong under control conditions, but the relationship was weaker and shifted downward significantly with increased RV afterload. The relationship was not significantly influenced by increased LV afterload. The relationship between free wall segmental stroke work and global stroke work was much weaker and was even more sensitive to distortion by increased RV afterload, while also sensitive to increased LV afterload.

Representative examples of the global and unidimensional PRSW relationships obtained under control conditions and during pulmonary arterial constriction and aortic constriction are shown in Fig 4. The *upper panels* demonstrate the PRSW relationships from the dog with the best overall linear correlation coefficients. The *middle panels* show PRSW relationships from the dog with the worst overall linear correlation coefficients.

The *lower panels* show the PRSW relationships determined by multiple linear regression analysis of pooled data from all dogs. The mean data from all animals are summarized in Table III.

Linear correlation coefficients were high for the global PRSW relationship under all loading conditions. The global PRSW relationship was unchanged during pulmonary arterial constriction, whereas a statistically significant reduction in the slope was noted during aortic constriction. Linear correlation coefficients were generally lower for the unidimensional PRSW relationships, particularly for the free wall segment relationship during pulmonary arterial constriction. The slopes of the unidimensional PRSW relationships were subject to much greater variability (ratio of SD/mean values in Table III) than their global counterparts under conditions of increased RV or LV afterload. There was poor and variable concordance between the global and

SW_G ($\text{erg} \cdot 10^3$)	EDD (mm)	ΔD (mm)	SW_D ($\text{erg} \cdot 10^3 \text{ cm}^{-2}$)	EDL (mm)	ΔL (mm)	SW_L ($\text{erg} \cdot 10^3 \text{ cm}^{-2}$)
735 ± 403	36 ± 5	3.5 ± 2.7	14.6 ± 11.6	10.4 ± 3.5	0.7 ± 0.4	3.7 ± 1.7
$982 \pm 472^*$	$40 \pm 4^*$	$2.1 \pm 2.3^*$	15.6 ± 12.9	10.6 ± 3.7	$0.4 \pm 0.5^*$	3.5 ± 3.8
635 ± 444	35 ± 6	2.8 ± 2.6	12.4 ± 11.7	10.5 ± 3.5	0.6 ± 0.4	3.0 ± 1.9

Values are mean $\pm$ SD. PAC, Pulmonary arterial constriction; AC, aortic constriction; HR, heart rate; LVMEP, LV mean ejection pressure; RVMEP, RV mean ejection pressure; the remaining variables are right ventricular: EDV, end-diastolic volume; SV, stroke volume; SW_G , global stroke work; EDD, end-diastolic septal-free wall dimension; ΔD , septal-free wall dimensional shortening; SW_D , septal-free wall dimensional stroke work; EDL, end-diastolic regional segment length; ΔL , regional segment length shortening; SW_L , segment stroke work.

* $P < .05$ versus control.

SV			SW		
Slope (mm/mL)	Y-int (mm)	r^2	Slope (cm^{-2})	Y-int ($\text{erg} \cdot \text{cm}^{-2} \cdot 10^3$)	r^2
0.29 (0.01)	-1.09 (0.17)	0.92	0.025 (0.001)	-2.343 (0.502)	0.94
0.34 (0.01)	-1.83 (0.17)	0.90	0.026 (0.002)	-5.935 (1.244)	0.76
0.29 (0.00)	-1.11 (0.06)	0.98	0.027 (0.000)	-3.212 (0.239)	0.97
0.04 (0.00)	0.12 (0.06)	0.62	0.003 (0.000)	1.137 (0.193)	0.65
0.07 (0.01)	-0.42 (0.08)	0.62	0.006 (0.001)	-1.344 (0.461)	0.51
0.04 (0.00)	0.01 (0.02)	0.80	0.004 (0.000)	0.664 (0.122)	0.68

Y-int, Intercept on the ordinate. Other abbreviations as in Table I. $\uparrow$ and $\downarrow$ indicate that b_2 in the multiple linear regression model (equation 1) is significantly different from zero ($P < .05$) and the direction of difference.

*Normalized to baseline, steady-state value. Values in parentheses are SDs.

unidimensional PRSW relationships, even when the dog with the highest overall linear correlation coefficients is considered (Fig 4, upper panels).

This is demonstrated in Fig 5, which shows the relationships between the unidimensional and global PRSW slopes and x-axis intercepts in all of the dogs studied. There was no significant relationship between the unidimensional and global PRSW x-axis intercepts for either the septal-free wall or the free wall segment, with the exception of the septal-free wall during aortic constriction ($P = .01$). There was a strong relationship between the septal-free wall and global PRSW slopes under control conditions, but the relationship was significantly depressed ($P = .003$) and weaker with increased RV afterload. The trend toward an opposite effect of increased LV afterload did not achieve statistical significance ($P = .29$). The relationship between free wall segmental and global PRSW slopes was poor

and subject to distortion by both increased LV and RV afterload.

Discussion

A variety of methods have been developed to measure RV volume.^{3,13,15,23} Absolute RV volume measurement is not critical for RV contractile performance to be measured. It is important, however, that the relationship between the RV volume index and absolute RV volume be consistent over the range of hemodynamic conditions likely to be encountered in vivo. Because contractile performance is best determined over a wide range of volumes, frequently achieved by vena caval occlusion,^{3,4,7,9,11-14,21} it is desirable that the volume index consistently reflect instantaneous RV volume during such beat-to-beat changes.

Conductance volumetry provides an instantaneous volume index better validated for the LV.²⁴ The

Septal-Free Wall Dimension **Free Wall Segment Length**

Fig 3. Relationships between unidimensional and global RV hemodynamic variables derived from vena caval occlusions performed before (control) and during pulmonary arterial constriction (PAC) or aortic constriction (AC). End-diastolic and end-systolic data were normalized to the steady-state value under control conditions. *EDV*, *EDD*, and *EDL*, End-diastolic volume, dimension, and length; *ESV*, *ESD*, and *ESL*, end-systolic volume, dimension, and length; *SV*, stroke volume; *SW_G*, *SW_D*, and *SW_L*, global, dimensional, and segmental stroke work.

crescentic, thin-walled geometry of the RV limits conductance measurements of absolute RV volume due to inhomogeneity of the electric field.^{3,9} Several investigators have demonstrated moderate linear correlations between conductance estimates and RV stroke volume,^{3,9,25} but it has not been demonstrated that this relationship is unaltered by changes to the afterload on either ventricle. Further limitations include inconsistencies caused by movement of the conductance catheter and the necessity to position the catheter retrogradely across the pulmonic valve to avoid transduction of atrial volumes, which limits the technique to open-chest measurements.^{9,26}

Two-dimensional echocardiographic methods can provide accurate static measurements of RV volume,^{23,27} but the requirement for imaging in multiple planes precludes instantaneous volume measurements. Single-plane RV echocardiographic scanning of RV cross-sectional area has been advocated as a surrogate for volume measurement to overcome this limitation.¹⁷ Imaging of the short-axis RV cross-sectional area could account for much of the ventricular interactive effect on

RV shape because it accounts for changes in septal position and curvature.²⁸ Validation of this approach over the range of hemodynamic conditions examined in this study remains to be documented. Continuous echocardiographic recordings of short-axis RV cross-sectional area by automatic boundary detection techniques could facilitate pressure-area analyses of RV contractile function under altered loading conditions.

One of the advantages of the surgical implantation of ultrasonic dimension transducers is the measurement consistency provided by their fixed position. Similarly, the implantation of a multitude of radiographic markers that can be tracked by biplane fluoroscopy throughout the cardiac cycle to derive instantaneous volumes from the 3-dimensional marker coordinates^{7,15,29} provides consistent measurement within an individual and might be expected to account best for the complexity of RV geometry.

Nicolosi, Hettrick, and Warltier⁹ have suggested that RV unidimensional measurements are sufficient to characterize RV systolic performance, providing unidimensional surrogates of stroke volume and the

Fig 4. Examples of global and unidimensional PRSW relationships obtained under control conditions and during pulmonary arterial constriction (PAC) and aortic constriction (AC) for the dogs with the best (*upper panels*) and worst (*middle panels*) linear correlations. The *lower panels* represent the results of regression analyses of the pooled data from all dogs. EDV, EDD, and EDL, End-diastolic volume, dimension, and length; SW_G , SW_D , and SW_L , global, dimensional, and segmental stroke work.

PRSW relationship under the limited experimental conditions of preload reduction. Our findings indicate, however, that RV unidimensional measurements are subject to considerable distortion by altered RV and LV afterload: the relationships between RV unidimensional and global indexes shifted with altered RV or LV afterload, particularly the former, confirming the presence of significant ventricular interactive effects on RV unidimensional indexes of volume and contractility *in vivo*.

The RV free wall segment measurements in our study were orthogonal to those of Nicolosi, Hettrick, and

Wartier.⁹ It was demonstrated previously that both orientations provided good correlation with RV volume, but the orientation we used was more sensitive to RV volume changes.¹³ In contrast to our findings, Nicolosi, Hettrick, and Wartier⁹ found that the correlation between septal-free wall shortening and stroke volume was negative in half of their experiments yet positive in the other half. This inconsistency suggests variability of the location of the ultrasonic transducer within the interventricular septum. As described in the "Methods" section and previously,^{11,12} it is important to locate the septal transducer as close as possible to the RV endo-

Fig 5. Relationship between unidimensional and global PRSW slopes (*left panels*) and x-axis intercepts (*right panels*) under control conditions and during pulmonary arterial constriction (PAC) or aortic constriction (AC). M_{wG} , M_{wD} , and M_{wL} , Global, septal-free wall dimensional and free wall segmental PRSW slopes; V_w , D_w , and L_w , global, dimensional, and segmental PRSW x-axis intercepts. D_w and L_w were normalized to the steady-state end-diastolic dimensions under control conditions.

cardium to avoid distortion of the actual motion in the RV septal-free wall dimension by simultaneous leftward motion and thickening of the septum. This also may explain why Nicolosi, Hettrick, and Warltier⁹ observed little change in the end-diastolic RV septal-free wall dimension during preload reduction.

Our findings of interactive effects of RV or LV afterload on the relationship between single RV dimensions and RV volume in vivo are consistent with previous evidence from isolated heart experiments that RV free

wall and septal-free wall dimensions depend on the volume of *both* ventricles.¹³ For example, rightward septal shifting with increased LV volume decreased the RV septal-free wall dimension, necessitating a lengthening of RV free wall dimensions to accommodate the same RV chamber volume. Consequently, the position of the relationship between RV free wall dimensions and RV volume shifted upward with increased LV volume, while the relationship between the RV septal-free wall dimension and RV volume shifted downward.¹³

Table III. RV global and unidimensional PRSW relationship during control conditions, AC, and PAC

	Global			Septal-free wall			Free wall segment		
	M_{wG} ($\text{erg} \cdot \text{cm}^{-3} \cdot 10^3$)	V_w (mL)	r	M_{wD} ($\text{erg} \cdot \text{cm}^{-3} \cdot 10^3$)	D_w (mm)	r	M_{wL} ($\text{erg} \cdot \text{cm}^{-3} \cdot 10^3$)	L_w (mm)	r
Control	28.3 ± 10.9	28.7 ± 11.1	0.98 ± 0.01	26.3 ± 16.1	30.1 ± 4.3	0.94 ± 0.04	47.4 ± 31.8	9.4 ± 3.2	0.86 ± 0.18
PAC	29.7 ± 8.7	29.6 ± 12.6	0.97 ± 0.02	19.7 ± 10.5*	31.0 ± 6.3	0.89 ± 0.09	37.0 ± 27.8*	9.6 ± 3.6	0.56 ± 0.44
AC	23.1 ± 11.7*	26.2 ± 7.6	0.95 ± 0.04	21.9 ± 19.1	25.2 ± 7.7	0.72 ± 0.33	49.0 ± 31.5†	9.5 ± 3.2	0.84 ± 0.18

Values are mean ± SD. PRSW, Preload recruitable stroke work relationship; AC, aortic constriction; PAC, pulmonary arterial constriction; M_{wG} , slope of global PRSW; V_w , volume-axis intercept of global PRSW; M_{wD} , slope of septal-free wall PRSW; D_w , dimension-axis intercept of septal-free wall PRSW; M_{wL} , slope of free wall segment PRSW; L_w , length-axis intercept of free wall segment PRSW.

*P < .05 versus control, multiple linear regression analysis.

†P < .05 versus control, indicating a significant reduction in regional stroke work for a given segment length with AC by multiple linear regression analysis, but this was not reflected in the mean slope due to a large increase in slope in a single dog with $r = 0.54$.

In the same experiments, however, the highly linear relationship between ellipsoidal shell subtraction measurements of RV volume and actual RV volume ($r = 0.99 \pm 0.01$) was not significantly shifted by varying LV volume, and in vivo measurements demonstrated a strong linear correlation between RV stroke volumes measured with the model and flow-probe measurements (slope 1.07 ± 0.03 , $r = 0.994 \pm 0.001$).¹³

Our findings may have significant relevance to the conclusions that can be drawn from experiments that depend on unidimensional indexes of RV contractile function.^{1,5-7,30} For example, several investigators have used unidimensional indexes to determine the effects of LV assist device support on RV function.^{1,5,7} These investigators concluded that RV contractile function was impaired during LV assist device support, but they also demonstrated that this intervention causes significant ventricular interaction, with leftward shifting of the interventricular septum. In contrast to these unidimensional observations, no depression of the RV global PRSW relationship was found during LV assist device support.⁴

In conclusion, our findings indicate the need for considerable caution in using unidimensional measurements as surrogates for RV volume in the assessment of RV contractile function. Unidimensional measurements do not provide consistent indexes of RV contractile function due to their sensitivity to ventricular interactions, particularly when the afterload on either ventricle is altered.

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