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Resistance Training and Cardiac Hypertrophy

Unravelling the Training Effect

Mark J. Haykowsky,[1,2] *Rudolph Dressendorfer*,[1] *Dylan Taylor*,[2] *Sandra Mandic*[1] and *Dennis Humen*[3]

1 Faculty of Rehabilitation Medicine, University of Alberta, Edmonton, Alberta, Canada
2 Division of Cardiology, Faculty of Medicine, University of Alberta, Edmonton, Alberta, Canada
3 Division of Cardiology, Faculty of Medicine, University of Western Ontario, London, Ontario, Canada

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Abstract

Resistance training (RT) is a popular method of conditioning to enhance sport performance as well as an effective form of exercise to attenuate the age-mediated decline in muscle strength and mass. Although the benefits of RT on skeletal muscle morphology and function are well established, its effect on left ventricular (LV) morphology remains equivocal. Some investigations have found that RT is associated with an obligatory increase in LV wall thickness and mass with minimal alteration in LV internal cavity dimension, an effect called concentric hypertrophy. However, others report that short- (<5 years) to long-term (>18 years) RT does not alter LV morphology, arguing that concentric hypertrophy is not an obligatory adaptation secondary to this form of exertion. This disparity between studies on whether RT consistently results in cardiac hypertrophy could be caused by: (i) acute cardiopulmonary mechanisms that minimise the increase in transmural pressure (i.e. ventricular pressure minus intrathoracic pressure) and LV wall stress during exercise; (ii) the underlying use of anabolic steroids by the athletes; or (iii) the specific type of RT performed. We propose that when LV geometry is altered after RT, the pattern is usually concentric hypertrophy in Olympic weightlifters. However, the pattern of eccentric hypertrophy (increased LV mass secondary to an increase in diastolic internal cavity dimension and wall

thickness) is not uncommon in bodybuilders. Of particular interest, nearly 40% of all RT athletes have normal LV geometry, and these athletes are typically powerlifters. RT athletes who use anabolic steroids have been shown to have significantly higher LV mass compared with drug-free sport-matched athletes. This brief review will sort out some of the factors that may affect the acute and chronic outcome of RT on LV morphology. In addition, a conceptual framework is offered to help explain why cardiac hypertrophy is not always found in RT athletes.

Resistance training (RT) programmes are well known to improve muscle strength and endurance for sport. RT has also gained popularity as an effective form of exercise to improve general health-fitness.[1] In addition, RT is accepted as a safe and effective therapeutic exercise intervention to attenuate the age-related decline in muscle mass and functional capacity.[2] However, despite these established benefits, disagreement exists concerning the effect of RT on left ventricular (LV) morphology. Previous reviews indicate that RT increases LV internal cavity dimension,[3,4] ventricular septal wall thickness,[3,4] posterior wall thickness,[3,4] relative wall thickness,[3,4] and LV mass.[3,4] A widely held belief in sport cardiology and exercise physiology is that serious RT for sport produces cardiac hypertrophy, which is usually defined as concentric hypertrophy (i.e. increased LV mass secondary to an increase in LV wall thickness with minimal alteration in internal cavity dimension). In contrast, some investigations have shown that short- (<5 years) to long-term (>18 years) RT was not associated with an alteration in LV internal cavity dimension,[5-7] ventricular septal or posterior wall thickness,[5-7] relative wall thickness,[6,7] or LV mass[5-7] in either male or female resistance-trained athletes. Moreover, no resistance-trained athlete was found to have an absolute LV mean wall thickness above normal clinical limits (i.e. >12mm).[6,7] Taken together, these studies suggest that RT does not necessarily produce concentric hypertrophy.[8] Disparate findings may be caused by the type of resistance-trained athletes that have been studied (i.e. bodybuilders, powerlifters, or Olympic weightlifters) or the underlying use of anabolic steroids, a practice sometimes used by these athletes.[9]

The purpose of this brief review is to sort out some of the factors that may affect the acute and chronic effects of RT on LV morphology. A conceptual framework is used to describe the development of three types of cardiac hypertrophy. In addition, a hypothesis is offered to help explain why cardiac hypertrophy is not always found in resistance-trained athletes. For the purpose of this review, resistance-trained athletes are considered those who specialise only in the types of RT typical of bodybuilders, powerlifters and Olympic weightlifters.

1. Acute Effects of Resistance Exercise on Left Ventricular (LV) Systolic Function and Wall Stress: Laplace Law Revisited

Numerous investigations have shown that the immediate response to resistance exercise is a transient and marked increase in systolic pressure;[10-14] however, few studies have assessed the acute effects of RT on LV systolic function and wall stress. Using two-dimensional echocardiography combined with invasive arterial pressure monitoring, Lentini et al.,[15] examined the effects of repetitive leg-press exercise at 95% of 1 repetition maximum (1RM) performed with a brief Valsalva manoeuvre (VM) on LV volumes and systolic function in younger healthy males. The major finding was that LV end-diastolic and end-systolic volumes decreased during exercise compared with resting values. Consequently, preload reserve and stroke volume declined (figure 1). However, since leg-press exercise mediated greater LV contractil-

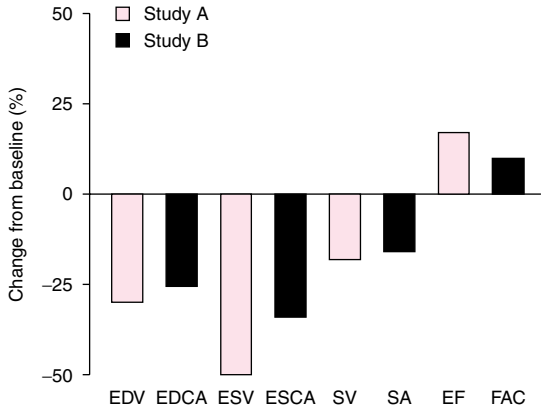

Fig. 1. Percentage change in left ventricular volumes (areas) and systolic function during repetitive submaximal (95% 1 repetition maximum) leg-press exercise in healthy young men. Study A = Lentini et al.[15]; Study B = Haykowsky et al.[17] **EDCA** = end-diastolic cavity area; **EDV** = end-diastolic volume; **EF** = ejection fraction; **ESCA** = end-systolic cavity area; **ESV** = end-systolic volume; **FAC** = fractional area change; **SA** = stroke area; **SV** = stroke volume.

ity and heart rate, cardiac output and ejection fraction increased (figure 1). These investigators also found that the acute increase in systolic blood pressure during RT was due in large part to elevated intrathoracic pressure associated with performing a brief VM. More importantly, LV transmural pressure (i.e. the pressure stressing the ventricular walls and calculated as LV pressure minus intrathoracic pressure) was lower than the measured systolic blood pressure during exertion. Although positive swings in intrathoracic pressure transmitted to the heart and arterial vasculature increases systolic pressure, the heightened intrathoracic pressure paradoxically prevents a rise in LV transmural pressure (see Hamilton et al.[16]). This finding is of utmost importance in understanding the potential benefit of performing a brief VM. MacDougall et al.[13] found that a brief VM was a compensatory response during repetitive RT performed at ≥85% maximal voluntary contraction or during submaximal exercise to volitional fatigue. A limitation of their investigation, however, was that LV wall stress was not measured during exertion.

Recently, Haykowsky et al.[17] examined the acute effects of repetitive submaximal (80 and 95% 1RM) and maximal leg-press RT performed with a brief (phase I) VM on LV cavity areas, fractional area change and wall stress in younger healthy males. The main finding was that leg-press exercise with a brief VM decreased preload reserve (i.e. decreased end-diastolic cavity area), which was offset by increased LV contractile reserve, resulting in increased fractional area change during lifting (figure 1). More importantly, this form of exercise was not associated with an acute alteration in LV end-systolic wall stress. The findings of Lentini et al.[15] and Haykowsky et al.[17] suggest that LV systolic function does not decline in healthy young males who perform submaximal and maximal leg-press exercise with a brief VM. In addition, LV end-systolic wall stress was unchanged compared with resting values. This finding is in direct conflict with the widely held belief in sport cardiology that systolic pressure loading is the mechanism of LV hypertrophy in resistance-trained athletes.

The law of Laplace states that LV wall stress is directly related to systolic pressure and radius of

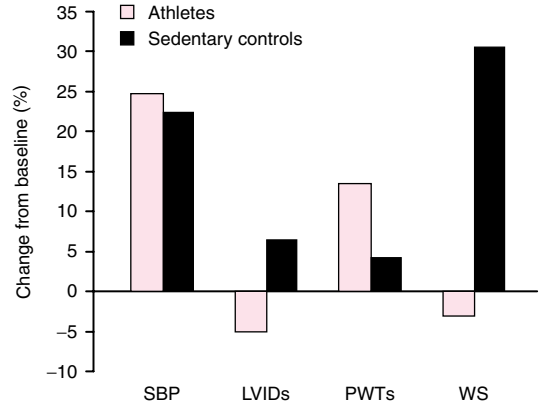

Fig. 2. Percentage change in left ventricular dimensions, wall stress and blood pressure during isometric handgrip exercise (without a Valsalva manoeuvre) in athletes and sedentary controls (data from published tables from Galanti et al.[18]). **LVIDs** = left ventricular internal dimension in systole; **PWTs** = posterior wall thickness in systole; **SBP** = systolic blood pressure; **WS** = end-systolic meridional wall stress.

curvature, and indirectly related to LV wall thickness. However, the important factor contributing to cardiac hypertrophy is LV transmural pressure rather than systolic pressure. Furthermore, as shown in figure 2, acute changes in LV geometry such as decreased cavity size with a concomitant increase in wall thickness may also occur during RT.[18] Such changes attenuate the increase in LV wall stress during exertion. We propose that compensatory changes in LV transmural pressure and LV geometry can occur during RT to blunt the increase in LV wall stress. This hypothesis may help to explain the lack of agreement between studies on whether RT leads to cardiac hypertrophy.

2. Cross-Sectional Investigations of Resting LV Morphology in Resistance-Trained Athletes

Over 20 cross-sectional investigations have examined the effects of RT on resting LV morphology and systolic function in athletes (table I). In each study, LV cavity dimensions, wall thickness and mass were compared between age-matched athletes or healthy nontrained controls, or comparisons were made with predicted normal values. Based on these investigations, it appears that RT can produce a wide range of LV morphologic adaptations. Some investigations showed that resistance-trained athletes have significantly larger absolute ventricular septal wall thickness,[19-29] posterior wall thickness,[19-21,23-34] or absolute LV mass[19-22,25,27-32,35] compared with healthy controls or normal predicted values. However, other studies reported that RT did not alter ventricular septal wall thickness,[6,7,31,32,34,36-38] posterior wall thickness,[6,7,22,31,36-38] or absolute LV mass.[6,7,26,31,34,36] Notably, only a few studies found that resistance-trained athletes had a greater LV wall thickness or mass after absolute values were indexed to body surface area (table I). These heterogeneous results also apply to resistance-trained female athletes, as increased LV wall thickness and mass have been reported in some studies,[39] while others[5,40] found no alterations in LV morphology (table II). Irrespective of gender, nearly all cross-sectional studies indicated that RT is not associated with an alteration in resting LV systolic or diastolic cavity dimensions or systolic function. Additional training variables that may determine the effect of RT on LV morphology are the type of RT performed and the use of anabolic steroids.

2.1 Type of Resistance Training (RT) and Subsequent Alterations in LV Morphology and Geometry

A limitation of comparisons in LV morphologic adaptations between different resistance-trained athletes is that the acute cardiovascular response depends on the mode of RT. For example, Falkel et al.[42] compared powerlifters and bodybuilders performing submaximal and maximal unilateral knee extension and squatting movements. The bodybuilders showed cardiac volume overload with significantly higher stroke volume and cardiac-output responses. Consequently, RT performed by bodybuilders could induce LV cavity enlargement, in contrast to the programmes preferred by powerlifters. This suggestion is consistent with the findings of Pelliccia et al.[41] who found that bodybuilders had a significantly larger LV diastolic cavity dimension and LV mass compared with powerlifters or Olympic weightlifters. It may be possible, therefore, to predict the pattern of LV geometric adaptation based on the type of RT performed.

Figure 3 shows the four LV geometrical patterns that we have interpreted from studies using echocardiographic measurements of LV mass index (i.e. LV mass/body surface area; normal values for men:[43] 116 g/m^2 and women:[43] 104 g/m^2) and relative wall thickness, which is calculated as two times end-diastolic posterior wall thickness divided by LV internal dimension (normal value[43] is less than 0.43). The geometric pattern is considered normal when LV mass index and relative wall thickness are both within the norm. Concentric remodelling is indicated when the LV mass index is normal but relative wall thickness is >0.43. Increased LV mass index with normal relative wall

Table I. Summary of cross-sectional studies assessing the effects of resistance training on left ventricular (LV) dimensions, mass, geometry and systolic function in male athletes and controls

Participants	Age (y)	Calibre	Training (y)	n	LVIDd	LVIDs	FS (%)	VST	PWT	LVM	h/R	LVG (EPD)	Reference
CT	31			9	45mm	33mm	32	10mm	10mm	165g	0.44 (EPD)		19
										84 g/m^2			
WL	30	NL	Min 4	8	43mm	28mm*	34	15mm*	13mm*	280g*	0.6 (EPD)	CH	
										158 g/m^{2*}			
CT	26			19	51mm		36			168 g/m^2			35
										87.8 g/m^2			
RT	26		Min 1	19	53mm		37			190g*			
										95.1 g/m^2			
CT	23			7	54mm	34mm	37	9mm	9mm	225g	0.33 (EPD)		36
					27 mm/m^2	17 mm/m^2		4.0 mm/m^2	4.0 mm/m^2	112.7 g/m^2			
WL	26	9C (4 years)	4	12	54mm	35mm	36	9mm	9mm	242g	0.33 (EPD)	EH	
					29 mm/m^{2*}	19 mm/m^2		5.0 mm/m^2	5.0 mm/m^{2*}	129.4 g/m^2			
CT	22			33	48mm	32mm	32		9mm	168g	0.38 (EPD)		30
										98 g/m^2			
PL	24	NL	>4	11	54mm*	34mm	37*		13mm*	373g*	0.49 (EPD)	CH	
										165 g/m^{2*}			
CT	23			15	49mm	27.8mm		7.6mm	7.6mm	155g	0.31 (EPD)		20
					25 mm/m^2			3.9 mm/m^2		79 g/m^2			
WL	23	C	6.4	15	50mm	30.8mm*		11.2mm*	10.9mm*	265g*	0.44 (EPD)	CH	
					25.8 mm/m^2			5.7 mm/m^{2*}		136 g/m^{2*}			
BB	24	C	5.5	15	53mm*	31.6mm*		10.8mm*	10.2mm*	270g*	0.38 (EPD)	EH	
					26.5 mm/m^2			5.4 mm/m^{2*}		134 g/m^{2*}			
CT	28			17	53mm	34mm		11mm	12mm	269g			21
					25 mm/m^2	16 mm/m^2		5.2 mm/m^2	5.6 mm/m^2	124 g/m^2			
BB,PL,WL	28	C-EL		17	55mm	34mm		13mm*	14mm*	352g*			
					27 mm/m^2	16 mm/m^2		6.2 mm/m^{2*}	6.6 mm/m^{2*}	164 g/m^{2*}			
CT	17.8			14	41.1mm	36.7mm		10.5mm	11.3mm				37
					21.4 mm/m^2	19.3 mm/m^2		5.5 mm/m^2	5.9 mm/m^2				
JWL	18.4	EL		14	44.9mm	23.4mm*		9.8mm	11.4mm				
					25.8 mm/m^{2*}	14.1 mm/m^{2*}		5.6 mm/m^2	6.5 mm/m^2				

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Table I. Contd

Participants	Age (y)	Calibre	Training (y)	n	LVIDd	LVIDs	FS (%)	VST	PWT	LVM	h/R	LVG (EPD)	Reference
CT	22			45	52.4mm			9.1mm	9.3mm	206g	0.35 (EPD)		22
										109 g/m^2 (EPD)			
WL	23	NL	10	11	54.2mm			10.8mm*	10.0mm	262g*	0.37 (EPD)	EH	
										132 g/m2 (EPD)			
YC	22			8	52.1mm	32.9mm	37	9.4mm	9.4mm	179g	0.4		6
										94.1 g/m^2			
JPL	21	EL	4.4	8	53.2mm	33.5mm	37	9.4mm	9.2mm	185g	0.3		
										95.0 g/m^2		NG	
MAC	47			11	51.8mm	31.4mm	40	9.7mm	9.5mm	184g	0.4		
										93.3 g/m^2			
MPL	46	EL	18.3	12	53.0mm	33.0mm	38	9.4mm	9.0mm	183g	0.3		
										91.3 g/m^2		NG	
CT	31			10	51.8mm			10.1mm	9.3mm	187g	0.4		7
										92.5 g/m^2			
PL	33	EL	10	21	54.4mm			9.7mm	9.6mm	200g	0.4		
										100.2 g/m^2		NG	
CT	25.5			23	51.6mm			9.8mm	9.9mm				23
BB	25.1			20	54.8mm			12.2mm*	12.1mm*				
WL	26.5			14	53.7mm			11.2mm*	11.2mm*				
CT	24			10	51.8mm			9.4mm	9.1mm	109.6 g/m^2	0.35 (EPD)		24
WL	25			10	53mm			14.5mm*	11.7mm*	176.9 g/m^2*	0.44 (EPD)	CH	
HC	26.9			14	50mm			8.3mm	8.0mm	150g	0.32		31
										71 g/m^2			
CWL	26.9			17	50mm			9.3mm	9.4mm*	190g*	0.38		
										90 g/m^2*			
AWL	28.1			7	49mm			9.8mm	8.3mm	172g	0.34		
										86 g/m^2			
CT	20-36			9	51.9mm			9.3mm	8.5mm		0.33		38
BB	20-39	C	15	9	52.3mm			11.0mm	9.7mm		0.37		
CT	23			44	49.5mm			9.0mm	8.7mm	192g			25
										100 g/m^2 (EPD)			
PA	22	NL		38	50.4mm			10.2mm*	9.8mm*	238g*			
										120 g/m^2 (EPD)			

CT	25			10	51mm		37	9mm	8mm	165g	0.31		32
					27 mm/m^2					87 g/m			
WL	28	C	>2	16	56mm*		34	10mm	9mm*	241g*	0.32	NG	
					27 mm/m^2					114 g/m^{2*}			
BB	30	NL	>3		57mm*			9.6mm	8.8mm	204g*			41
										102 g/m^{2*}			
WL	23	NL	>3		52mm			9.6mm	8.8mm	177g			
										92 g/m^2			
PL	22	NL	>3		51mm			9.8mm	8.7mm	170g			
										90 g/m^2			
ST	27		Min 3	46	55mm*				9.6mm*	265g*			33
										131 g/m^2			
PRED					52mm				9.2mm	197.4g			
CT	29			14	44mm	31mm		11mm	10mm	158g	0.45		26
										91.3 g/m^2			
WL	28	NL	5	14	43mm	28mm		14mm*	14mm*	232g	0.65	CH	
										129 g/m^2			
CT	24			17	50.4mm			8.5mm	8.2mm	170g			34
WL	25		7	14	51mm			9.3mm	9.6mm*	210g			
CT	23			50	46.7mm	32.7mm		9.7mm	8.4mm	163g	0.36 (EPD)		27
					27.2 mm/m^2	19.0 mm/m^2		5.6 mm/m^2	4.8 mm/m^2	94 g/m^2			
BB	22	Int		14	53.5mm*	36.5mm		12.4mm*	11.3mm*	305g*	0.42 (EPD)	EH	
					27.1 mm/m^2	18.4 mm/m^2		6.2 mm/m^2	5.7 mm/m^2	154 g/m^{2*}			
WL	26	Int		10	52.3mm*	35.0mm		12.7mm*	11.5mm*	302g*	0.44 (EPD)	CH	
					24.3mmm/m^{2*}	16.3 mm/m^2		5.4 mm/m^2	4.9 mm/m^2	139 g/m^{2*}			
CT	31			10	51.3mm	34.7mm	32.9	7.6mm	8.6mm	157g	0.35		28
BB	33		>5	11	49.7mm	32.3mm	34.9	11.0mm*	11.8mm*	210g*	0.48*		
CT	29			9	52mm	34mm		8.6mm	8.3mm	93g			29
WL	29	NL		8	55mm	35mm		10.7mm*	9.8mm*	128g*			

AWL = amateur weightlifters; **BB** = bodybuilders; **C** = competitive; **CH** = concentric hypertrophy; **CT** = control; **CWL** = competitive weightlifters; **EH** = eccentric hypertrophy; **EL** = elite; **EPD** = estimated from the published data; **FS** = fractional shortening; **HC** = heavy controls; **h/R** = relative wall thickness; **Int** = international; **JPL** = junior powerlifters; **JWL** = junior weightlifters; **LVG** = LV geometry; **LVIDd** = LV internal dimension in diastole; **LVIDs** = LV internal dimension in systole; **LVM** = LV mass; **MAC** = middle-aged controls; **Min** = minimum; **MPL** = master powerlifters; **n** = number of participants; **NG** = normal geometry; **NL** = national level; **PA** = power athletes; **PL** = powerlifters; **PRED** = predicted; **PWT** = posterior wall thickness; **RT** = resistance trained; **ST** = strength trained; **VST** = ventricular septal wall thickness; **WL** = weightlifters; **YC** = young controls; * $p < 0.05$ *vs* CT or appropriate comparison.

Table II. Summary of cross-sectional studies assessing the effects of resistance training on left ventricular (LV) dimensions, mass, geometry and systolic function in female athletes and controls

Participants	Age (y)	Calibre	Training (y)	n	LVIDd	LVIDs	FS (%)	VST	PWT	LVM	h/R	LVG (EPD)	Reference
CT	22			10	46.8mm 28.8 mm/m^2	30.2mm	35	6.6mm 4.08 mm/m^2	7.5mm 4.6 mm/m^2	116g 71 g/m^2			40
PA	22	C	>2	10	48.6mm 27.5 mm/m^2	32.2mm	34	6.9mm 3.9 mm/m^2	8.0mm 4.52 mm/m^2	134g 75 g/m^2			
CT	23			46	48.4mm			7.7mm	7.5mm	137g 82.5 g/m^2 (EPD)	0.31		39
WL	25	EL	6	15	46.2mm*			9.0mm*	8.7mm*	158g 96 g/m^2 (EPD)	0.38*	NG	
CT	35			6	48mm			9mm	8mm	134g 81 g/m^2	0.33		5
PL	31	EL	5.8	8	49mm			7mm	7mm	120g 69 g/m^2	0.29	NG	

C = competitive; **CT** = control; **EL** = elite; **EPD** = estimated from the published data; **FS** = fractional shortening; **h/R** = relative wall thickness; **LVG** = LV geometry; **LVIDd** = LV internal dimension in diastole; **LVIDs** = LV internal dimension in systole; **LVM** = LV mass; **n** = number of participants; **NG** = normal geometry; **PA** = power athletes; **PL** = powerlifters; **PWT** = posterior wall thickness; **VST** = ventricular septal wall thickness; **WL** = weightlifters; * $p < 0.05$ *vs* CT.

thickness suggests eccentric hypertrophy. The pattern of concentric hypertrophy is identified by increased LV mass index and relative wall thickness.

Based on these criteria, 13 investigations (with 16 different comparisons) provide sufficient information to estimate the pattern of LV geometry and relate it to the type of RT performed (table I and table II). The most common LV patterns were normal geometry (37.5%) and concentric hypertrophy (37.5%), with only 25% of athletes demonstrating eccentric hypertrophy. Interestingly, no resistance-trained athlete was found to have a concentric remodelling pattern of LV geometry. Associating these patterns of cardiac hypertrophy with the type of RT reveals that two-thirds of athletes with normal geometry were powerlifters while the remaining one-third were Olympic weightlifters. The opposite finding was observed for the concentric hypertrophy pattern, as a majority of resistance-trained athletes exhibiting this pattern were weightlifters and a smaller number (<20%) were powerlifters. Finally, in the four investigations that observed an eccentric pattern of LV hypertrophy, the athletes were either weightlifters or bodybuilders. Thus, when LV geometric changes do occur with RT the most common type is a concentric pattern that is almost exclusive to weightlifters. In contrast, eccentric hypertrophy is less common and is seen more often in weightifters or bodybuilders. The concentric remodelling pattern

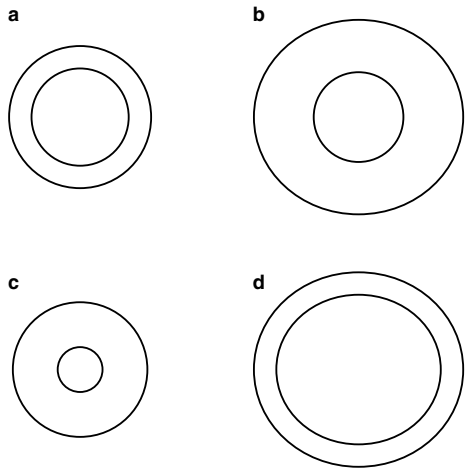

Fig. 3. Patterns of left ventricular geometry associated with resistance training: (**a**) normal geometry: common in power lifters; (**b**) concentric hypertrophy: common in Olympic weightlifters; (**c**) concentric remodelling: not found in resistance-trained athletes; (**d**) eccentric hypertrophy: common in bodybuilders.

Table III. Effects of anabolic steroids on left ventricular (LV) dimensions, mass, geometry and systolic function

Participants	Age (y)	Calibre	Training (y)	n	LVIDd	LVIDs	FS/EF	VST	PWT	LVM	LVG (EPD)	Reference
CT	32		Min 5	10	47.2mm 25.2 mm/m^2			9.8mm	9.9mm	104 g/m^2		51
WLNU	30		Min 5	10	50.1mm* 24.7 mm/m^2			12.2mm*	11.7mm*	123 g/m^2*	CH	
WLU	33		Min 5	10	53.1mm* 26.1 mm/m^2			12.3mm*	11.6mm*	146 g/m^2* **	CH	
CT	25	?		14	50.5mm 26.6 mm/m^2	30.9mm 16.0 mm/m^2	68.6	9.8mm 5.1 mm/m^2	9.8mm 5.1 mm/m^2	226g 117 g/m^2		49
DFBB	26	?	3.5	14	53.1mm 26.8 mm/m^2	34.9mm 17.6 mm/m^2	62.7	10.5mm 5.3 mm/m^2	9.8mm 4.9 mm/m^2	247g 125 g/m^2	EH	
DUBB	26	?	4.2	14	55.2mm* 28.1 mm/m^2	35.8mm* 18.1 mm/m^2*	63.4	11.0mm* 5.6 mm/m^2*	10.3mm 5.3 mm/m^2	281g* ** 142 g/m^2* **	EH	
DFBB	27	C	10.9	8	57mm	36mm	37	8.7mm	10.3mm			45
DUBB	27	C	10.9	8	56mm	35mm	36	11.2mm**	12.1mm**			
DFWL	23	C	6.4	15	50.8mm		70	11.0mm	11.1mm	267g 132 g/m^2 (EPD)	CH	50
DUWL	23	C	6.2	15	53.4mm		69	11.8mm	11.6mm	313g** 150 g/m^2 (EPD)	CH	
CT	26	?		9	46.8mm			10.9mm	10.9mm	184.5g 97 g/m^2 (EPD)		47
DFBB	27	?	Min 2	9	46.1mm			16.3mm*	16.4mm*	318.9g* 164 g/m^2 (EPD)	CH	
DUBB	25	?	Min 2	9	45.9mm			16.6mm*	18.3mm* **	374.5g* ** 184 g/m^2 (EPD)	CH	
DFBB	27		5	13	55.7mm		37	9.3mm	9.5mm	169g 83 g/m^2 (EPD)	NG	44
DUBB	27		5	11	59.1mm**		35	11.1mm**	11.2mm**	210g** 99 g/m^2 (EPD)	NG	
DFBB	34	?	?	10			41	8.7mm	8.7mm	199g 101 g/m^2		46
DUBB	37	?	?	10			41	10mm**	9.8mm**	245g** 117 g/m^2		
DFBB	26	C	8	7	57.4mm 27.4 mm/m^2		32	11.6mm 5.52 mm/m^2	10.3mm 4.92 mm/m^2			48
DUBB	28	C	6.3	14	54.7mm 25.8 mm/m^2**		35	12.6mm 5.89 mm/m^2**	12.5mm** 5.90 mm/m^2**			

C = competitive; **CH** = concentric hypertrophy; **CT** = control; **DFBB** = drug free bodybuilders; **DFWL** = drug free weightlifters; **DUBB** = drug using bodybuilders; **DUWL** = drug using weightlifters; **EF** = ejection fraction; **EH** = eccentric hypertrophy; **EPD** = estimated from the published data; **FS** = fractional shortening; **LVG** = LV geometry; **LVIDd** = LV internal dimension in diastole; **LVIDs** = LV internal dimension in systole; **LVM** = LV mass; **Min** = minimum; **n** = number of participants; **NG** = normal geometry; **PWT** = posterior wall thickness; **VST** = ventricular septal wall thickness; **WLNU** = weightlifters not using anabolic steroids; **WLU** = weightlifters using anabolic steroids; **?** indicates unknown; * $p < 0.05$ vs CT, ** $p < 0.05$ *vs* sport-matched athletes not using anabolic steroids.

does not appear to occur in resistance-trained athletes. A finding that requires further reinforcement is that nearly 40% of all resistance-trained athletes had a normal LV geometry and powerlifters most often met this classification. Hence, it is tenable that the type of RT performed may result in divergent acute cardiovascular responses. Long-term RT may result in diverse morphologic and geometric patterns. A discussion of the effect of RT on cardiac morphology should, therefore, consider the type of training programme.

2.2 LV Morphology and Geometry in Athletes Using Anabolic Steroids

The disparity between investigations reporting the effect of RT on LV mass may also be due to the use of anabolic steroids. For example, a series of investigations indicated that athletes using anabolic steroids have a significantly greater LV diastolic cavity dimension,[44] ventricular septal wall thickness,[44-46] posterior wall thickness,[44-48] LV mass,[44,46,47,49,50] or LV mass index[51] compared with sport-matched nonsteroid using athletes (table III). A more alarming finding from one investigation[47] was that posterior wall thickness exceeded 18mm in 66% of resistance-trained athletes using anabolic steroids (3 athletes had a wall thickness ≥20mm), while cavity dimension was normal. This pattern of LV hypertrophy is suggestive of marked concentric hypertrophy. Consistent with the observation that the cardiac effect of RT is training-specific, is the finding that a majority (60%) of resistance-trained steroid users appear to have concentric hypertrophy. Of these, 67% were weightlifters and 33% were bodybuilders. Eccentric hypertrophy was found in 20% (all bodybuilders) and the remaining 20% had normal geometry. No resistance-trained athlete who used anabolic steroids was found to have concentric remodelling. A survey by Wagman et al.[9] suggests that two-thirds of elite resistance-trained athletes use anabolic steroids to enhance sport performance. Consequently, the disparity of the findings in the studies from table I and table II may be caused, in part, by the underlying use (or unreported use) of anabolic steroids by the study participants.

2.3 Effect of Short-Term RT on LV Morphology

Another possible explanation for the disparity between studies is that the LV morphologic adaptations may occur soon after initiating a RT programme with no further increases in LV size as training continues. Eight investigations[52-59] have assessed the short-term (4 to 16 weeks) effects of RT on resting LV morphology. The average exercise intensity in these investigations was between 30 to 90% of 1RM and a wide variety of upper and lower extremity exercises were performed for 2 to 5 days/week (table IV). In five of these investigations,[52-56] the participant mean age was under 23 years while in the remaining three studies[57-59] the participants were greater than 63 years of age. Finally, with the exception of one investigation,[52] all of the study participants were men. These investigations indicated that short-term RT is not associated with a significant alteration in LV internal cavity dimension,[53-55,58,59] wall thickness[53,56-59] or LV systolic function.[54,56-58] Despite no change in LV cavity dimensions or wall thicknesses, four out of six investigations found significant increases in LV mass or LV mass index after RT.[53-56]

Although alterations in LV mass may occur in the early period after beginning an exercise programme, the absolute changes in reported LV wall thickness are within the methodological error of M-mode echocardiography. Of particular interest, the mean age of the participants in studies that found an increase in LV mass with RT was less than 23 years, whereas in the investigations that found no change in LV mass, the participants were in their seventh decade of life. This observation suggests that the senescent heart may have a diminished capacity to change its shape with RT. In contrast, an earlier cross-sectional study found that master athletes had significantly larger LV wall thickness and mass compared with sport-matched younger athletes.[60] However, these morphologic changes in the master athletes were associated with long-term training (i.e. 3 decades). Thus, pre-

Table IV. Effects of short-term resistance training on left ventricular (LV) dimensions, mass and systolic function in male and female athletes and controls

Time	Age (y)	Type/freq/int	Duration (wk)	n	LVIDd	LVIDs	FS/EF	VST	PWT	LVM	Reference
Pre	16	IE, 3 d/wk, 30% MVC × 2 min	12	15			68.2	7.7mm	7.3mm		52
Post							69.6*	8.5mm*	8.3mm*		
Pre	19	WL, 3 d/wk, 7 sets × 5RM	20	8	47.6mm			10.7mm	10.7mm	234g	53
Post					48.4mm			11.4mm	10.9mm	244g*	
Pre	22	IE, 3 d/wk, int ?	10-12	6	44.5mm 23 mm/m^2		43	8.3mm	9.5mm	65 g/m^2	54
Post					44.5mm 23 mm/m^2		45	8.7mm*	10.9mm*	77 g/m^2*	
Pre	23	RE, 5 d/wk, 3-5 sets, 5 reps	10	9	51mm	35mm	32	9.3mm	7.6mm	82g	55
Post					53mm	34mm	36*	9.4mm	8.5mm*	92.3g*	
Pre	23		4	10	48.2mm	31.6mm	78	8.7mm	9.0mm	166.8g	56
Post					53.7mm*	36.3mm*	69	9.3mm	8.3mm	202.8g*	
Pre	68	IE, 3 d/wk, 30% MVC	12	20			58	10mm	9.8mm		57
Post							59	10mm	10.2mm		
Pre	68	IT, 3 d/wk, 68-80% 1RM	16	10	54mm	34mm	40	11mm	10mm	212g	58
Post					54mm	33mm	40	10mm	10mm	202g	
Pre	60-75	RE, 2 d/wk, 85-90% 1RM	16	9	50.1mm			12.8mm	10mm	268g	59
Post					52.2mm			12mm	9.4mm	258g	

EF = ejection fraction; **freq** = frequency; **FS** = fractional shortening; **IE** = isometric exercise; **int** = intensity; **IT** = isotonic exercise; **LVIDd** = LV internal dimension in diastole; **LVIDs** = LV internal dimension in systole; **LVM** = LV mass; **MVC** = maximal voluntary contraction; **n** = number of participants; **Post** = post-training; **Pre** = pre-training; **PWT** = posterior wall thickness; **RE** = resistance exercise; **reps** = repetitions; **RM** = repetition maximum; **VST** = ventricular septal wall thickness; **WL** = weightlifting; **?** indicates unknown; * $p < 0.05$ *vs* Pre.

viously sedentary older individuals may require a greater training duration than younger individuals to induce LV morphologic changes similar to that of younger individuals. Alternatively, it is possible that RT may not produce an acute rise in LV wall stress in older individuals, thereby blunting the stimulus for LV hypertrophy. Consistent with this hypothesis was the finding that LV wall stress was not altered after 16 weeks of RT in previously sedentary older males.[58] It seems possible that in younger resistance-trained athletes, increased LV mass may occur with short-term training whereas individuals who initiate a RT programme later in life may require a longer training period to induce LV hypertrophy.

3. Conclusion

In conclusion, we propose that increased LV mass is not an obligatory adaptation associated with heavy RT. Furthermore, RT is not associated with a homogeneous alteration in LV geometry. It appears that when LV geometry is altered after RT, the concentric pattern of hypertrophy occurs more often than the eccentric pattern. Concentric hypertrophy is found more often in Olympic weightlifters whereas eccentric hypertrophy is more common to bodybuilders. Of particular interest, nearly 40% of all resistance-trained athletes have normal LV geometry, which is typical of powerlifters. Whether serious RT promotes LV hypertrophy probably depends on multiple variables. The disparity between studies on whether RT increases LV mass could be partly caused by the performance of a brief (phase I) VM during exercise. A brief VM may minimise the acute increase in LV transmural pressure and wall stress. The underlying use of anabolic steroids, the specific type of RT performed and the age of the indi-

vidual are some important factors affecting the outcome of RT. Furthermore, with the exception of a few investigations, the absolute change in LV dimension is small and close to the methodological error of M-mode echocardiography. In addition, RT is rarely associated with an acute or chronic alteration in LV systolic function. It is also possible that some resistance-trained athletes may be genetically predisposed to having a larger LV, which combined with intense RT exposure, may result in an increase in LV mass.

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Correspondence and offprints: *Mark J. Haykowsky*, Faculty of Rehabilitation Medicine, University of Alberta, 2-50 Corbett Hall, Edmonton, AB T6G 2G4, Canada.
E-mail: mark.haykowsky@ualberta.ca