CARDIAC STRUCTURE AMONG DIFFERENT SPORTS PLAYERS

Luca Silvestrini[1], Cecilia Pera[2], Luca Marin[3,4,5], Massimiliano Febbi[3,4,6], Dario Silvestri[6], Vittoria Carnevale Pellino[5], Matteo Vandoni[5], Nicola Lovecchio[7], Alessandro Gatti[5,*]

[1]Department of Industrial Engineering, University of Tor Vergata, Rome, Italy - [2]Corilab Ambulatory, Frascati (Rome), Italy - [3]Department of Physiotherapy, Faculty of Medicine, University of Ostrava, Czech Republic - [4]Laboratory for Rehabilitation Medicine and Sport (LARMS), Rome, Italy - [5]Laboratory of Adapted Motor Activity (LAMA), Department of Public Health, Experimental and Forensic Medicine, University of Pavia, Pavia, Italy - [6]Asomi College of Sciences, Research Department, Malta - [7]Department of Human and Social Science, University of Bergamo, Bergamo, Italy

ABSTRACT

Introduction: *Competitive sports activities involve morphological and functional cardiac adaptations allowing to optimize cardiac function. The adaptations derived from sports practice are related to genetic factors, age and sport activity, type and intensity of training protocols, which can define different functional and morphological adaptation models (endurance vs resistance vs power/strength). The purpose of our study was, therefore, to verify the presence and extent of specific cardiovascular adaptations to different sports.*

Materials and methods: *We conducted a cross-sectional study on 80 healthy males divided into five groups based on the sport practiced: bodybuilding (BB), volleyball (VB), cycling (CG), running (RN), and no practitioners (SY). During a routine medical visit at 8.00 AM with the same environmental condition (room temperature at 24°), all the participants were asked to undergo a mono and bidimensional echocardiography and tissue Doppler imaging to investigate cardiac dimensions.*

Results: *Between BB and SY, our results showed only a higher LVED and LVES for BB compared to SY ($p<0.05$). BB had also a lower BSA, LVM, and PP ($p<0.05$) than the VB group. No differences were found between CG and RN ($p>0.05$). Furthermore, our findings revealed that all outcomes except for BSA and LVED index were higher for CG than for SY ($p<0.05$).*

Conclusions: *The continuous practice of bodybuilding is not uniquely associated with an increase in left ventricular mass. From the data obtained in our experimental study, there are no cardiological adaptations worthy of note, having found differences with the sedentary group only in left ventricular systolic and diastolic diameters. Moreover, our results confirm that specific types of training and sports activities influence cardiac output differently in healthy males.*

Keywords: *cardiac output, sports, strength training, endurance training.*

DOI: 10.19193/0393-6384_2023_6_171

Received *February 15, 2023;* ***Accepted*** *June 20, 2020*

Introduction

Competitive sport activities involve morphological and functional cardiac adaptations allowing to optimize cardiac function[1-3]. The primary functional adaptation is represented by bradycardia at rest, resulting from a new sympathetic-vagal balance[4-6], while the main morphological adaptation is represented by myocardial hypertrophy, based on the harmonic increase in myocardial mass that, in turn, depends on the intensity of practiced sport[2, 7-10]. This type of hypertrophy involves both myocytes and vessels to maintain proper blood supply to increase myocardial mass[11-12]. Interstitial fibrosis detectable in hypertrophic heart diseases, such as hypertrophic cardiomyopathy or cardiac amyloidosis, is not detectable in physiological cardiac adaptations, avoiding detrimental effects on diastolic function (loss of distensibility) or wall stress increase, which represent negatives consequences of pathological irreversible illnesses[11-12]. These adaptations (derived from sport practice) are related to genetic factors (genotype), age and sport activity, type and intensity of training protocols, which can define different functional and morphological adaptation models (endurance vs resistance vs power/strength)[13-21].

Intense exercise, able to stimulate skeletal muscle tone, and bone density and exert metabolic effects on insulin sensitivity, is often not considered a therapeutic approach tool by physicians, mainly due to the strong cardiovascular impact related to peak pressure and sudden increase in heart rate[22, 23]. We studied a selected population of high-ranked competitive body-builders, and we compared their cardiovascular adaptations with similar high-ranked sportsmen from different disciplines. With respect to the literature, this group of athletes did not show a homogeneous pattern of adaptation at all (most of them had no one), contrary to what we found in other sports population[24-30]. These considerations led us to conclude that resistance training (RT), even when conducted in an intense and prolonged way, was not able to really impact on the cardiovascular system in strenuous work overload, as it seems to happen in sports like cycling or endurance[31-33].

The purpose of our study was, therefore, to verify the presence and extent of specific cardiovascular adaptations to the sport of bodybuilding. In addition, within the study, we wanted to verify whether we can find differences between those who practice this discipline. Finally, a further comparison was made with a group of apparently healthy sedentary people.

Methods

Study design and participants

We conducted a cross-sectional study on 80 healthy males (age 24.47±4.92 years; BMI 23.96±3.87 kg/m² mean±SD). They were enrolled and divided into five groups based on their sport participation: bodybuilding (BB), volleyball (VB), cycling (CG), running (RN), and no practitioners (SY). In particular (Table 1), the 20 subjects' no-sport-practitioners (24.3±3.66 years; BMI 23.58±2.83 mean±SD) did not take regular exercise in the last 12 months.

The inclusion criteria were to be an athlete (high-ranked competitive) of bodybuilding, volleyball, and cycling, and run for at least three years with at least five training sessions per week; age>18 years; to be in possession of a valid sport medical examination and the capacity to understand the Italian language. The exclusion criteria were orthopedics injuries in the last six months and cardiovascular or respiratory diseases. Written informed consent was obtained by all participants prior to engaging in the study. All the participants were free to withdraw from the study at any time. All the procedures were in accordance with the Declaration of Helsinki, as revised in 2013. The study protocol and procedure were approved by the Ethical Committee of Diagnostica e Medicina Specialistica-RXCA (011110841002).

Procedure

	Age (years)	Height (m)	Weight (kg)
BB	27±5.23	1.73±0.07	86.6±11.4
CG	23.8±3.47	1.76±0.04	69.2±4.83
RN	27.9±5.31	1.75±0.06	62.0±4.39
SY	25.3±3.96	1.77±0.06	74.0±10.7
VB	21.7±3.96	1.93±0.06	86.3±4.51

Table 1: Descriptive characteristics of the sample stratified by the sport practiced.

Anthropometrics characteristics

Before the measurements, all the subjects refrained from alcohol consumption and intensive physical activity for 24 hours and from caffeine consumption for 5 hours in order to avoid acute effects on endothelial function and oxidative stress.

Height and weight are collected before the echocardiography assessment. Standing height was measured using a Harpenden stadiometer (Holtain Ltd., Cross-well, UK) with a fixed vertical backboard and an adjustable headpiece. The measurement was taken on each subject in an upright position, without shoes, with their heels together and toes apart, hands at sides, aligning the head in the Frankfort horizontal plane. Weight was assessed with participants not wearing shoes and in light clothing, standing upright in the center of the scale platform (Seca, Hamburg, Germany) facing the recorder, hands at sides, and looking straight ahead.

Two measurements were taken for each parameter, and a third was obtained if a discrepancy was noted between the initial measurements for weight (>500 g) and height (>0.5 cm). BMI was calculated as body weight (Kilograms) divided by height (meters squared).

Echocardiography and tissue Doppler imaging measurements

During a routine medical visit at 8.00 AM with the same environmental condition (room temperature at 24°), all the participants were asked to undergo a mono and bidimensional echocardiography and tissue Doppler imaging to investigate cardiac dimensions (Left and Right end-diastolic ventricular diameter, wall thickness, diastolic and systolic function as well as myocardial stiffness related parameters as isovolumetric relaxation time).

Studies were performed with a commercially available ultrasound machine equipped with tissue Doppler (SONY-ACUSON CV 70), using a 1.5-5 MHz transducer. Heart rate and blood pressure were measured just before the cardiac measurements in a landing position.

All the measurements were averaged from at least three consecutive cardiac beats. Conventional echocardiography consisted of M-mode, 2D, and Doppler (color and tissue) blood flow measurements that were performed according to the current guidelines of the American Society of Echocardiography[(3)]. In particular, with the M-mode tracings from the parasternal long and short-axis view, a series of cardiac indexes were measured as described in Table 2.

Measure	Unit of measures	Description	Acronimous
Aortic root diameter	mm		AR
Left atrium diameter	mm		LA
Left ventricular telesystolic diameter	mm		LVES
Left ventricular telediastolic diameter	mm		LVED
Diastolic septal	mm		SIV
Posterior wall thickness	mm		PP
Fractional shortening	%		FS
Left Ventricular mass index	gr/m^2	Hypertrophy was considered present when the mass index exceeded 116 g/m^2	LV
End-diastolic area	cm^2		EDA
End-systolic area	cm^2		ESA
Ejection fraction	%	Assessed using left ventricular lengths and following modified biplane Simpson's method	EF%
Flow propagation velocity	cm/sec		FPV
Diastolic function	</=/>1	Assessed by pulsed-wave Doppler of the transmitral flow and ejection on area ratio?	EAR
Left ventricular inflow	cm/sec	recorded by color M-mode echocardiography	LV
Peak myocardial velocities in systole	cm/sec		STDI
Peak myocardial velocities in diastolic early	cm/sec		ETDI
Peak myocardial velocities in diastolic late	cm/sec		ATDI
Left ventricular end-diastolic pressure	27	Measured as the ratio of Ejection on ETDI ratio	

Table 2: Cardiac measures and index collected.

Statistical analysis

All the data were summarized as mean and standard deviation (SD), and the mean difference was calculated to describe differences between groups. We tested for normality by Shapiro-Wilk tests and graphically checked for linearity. We used a parametric ANOVA with Bonferroni correction or its correspondent non-parametric ANOVA of Krustal-Wallis with dwass-steel-critchlow-fligner correction as appropriate to evaluate the differences between groups. All the significance was set at a p-value less than 0.05. Statistical analyses were performed using The Jamovi Project (2021).

Jamovi Version 1.6 for Mac (Computer Software), Sydney, Australia; retrieved from https://www.jamovi.org (accessed on 5 May 2022).

Results

Tables 1 and 3 show the descriptive characteristics of the sample, while Table 4 reports the differences in cardiac output between all the groups.

	BB	CG	RN	SY	VB
BSA	2.01± 0.15	1.85± 0.09	1.75± 0.09	1.85± 0.17	2.17± 0.08
LVED (mm)	55.00± 5.64	57.13± 2.92	57.00± 2.39	50.35± 3.03	57.60± 2.35
LVED index	27.43± 1.95	30.98± 1.64	32.55± 2.05	27.37± 3.43	16.75± 1.09
LVES (mm)	36.00± 5.10	36.53± 3.98	35.40± 3.58	30.85± 2.45	36.33± 2.38
LVES index	17.93± 1.86	19.82± 2.26	20.21± 2.23	16.70± 2.19	16.75± 1.09
LVM (gr)	375.77± 41.23	441.67± 16.12	438.27± 18.11	362.80± 20.66	435.23± 21.33
LVM index	187.92± 21.49	239.48± 10.71	250.41± 18.51	97.38± 22.16	200.74± 12.02
SIV (mm)	10.43± 1.39	12.13± 0.83	11.73± 0.80	9.60± 0.75	5.47± 0.40
SIV index	5.22± 0.69	6.58± 0.51	6.71± 0.62	5.21± 0.59	5.47± 0.40
PP (mm)	9.91± 1.38	12.07± 0.46	12.27± 0.46	10.15± 0.67	12.00± 0.80
PP index	4.96± 0.75	6.54± 0.27	7.01± 0.52	5.54± 0.70	5.50± 0.42

Table 3: Descriptive characteristics of the sample stratified by sports.
Note: The data are presented as mean + SD.

Table 4 shows that BB had a higher BSA ($p<0.05$) compared to CG and RN. BB had also a higher SIV ($p<0.05$) and a lower BSA, LVM, and PP ($p<0.05$) than the VB group. Between BB and SY, our results showed only a higher LVED and LVES for BB compared to SY ($p<0.05$). Furthermore, our findings revealed that all outcomes except for BSA and LVED index were higher for CG than for SY (p<0.05). No differences were found between CG and RN (p>0.05). For RN, our results showed that all outcomes, except for BSA, were higher in RN than in SY (p<0.05).

RN also had a higher PP index, SIV index, LVM index, and lower BSA than VB (p<0.05). Between SY and VB, SY had lower direct cardiac measures than VB (p<0.05), while the calculated indexes were not different between the two groups (p>0.05).

Discussion

The aim of our study was to evaluate the different cardiac adaptations among different sports. The results from our study showed no relevant cardiac adaptations in the BB group. They only differed from the SG group for increased left telesystolic and telediastolic diameters, but when adjusting for BSA, this difference disappeared. Although this difference with sedentary adults, our results are in accordance

		CG	RN	SY	VB
BSA	BB	0.16 (p=0.01)**	0.26 (p<0.001)***	0.16 (p>0.05)	-0.16 (p=0.034)*
	CG		0.10 (p>0.05)	0.00 (p>0.05)	-0.32 (p<0.001)***
	RN			-0.10 (p>0.05)	-0.42 (p<0.001)***
	SY				-0.32 (p<0.001)***
LVED (mm)	BB	-2.13 (p<0.05)	-2.00 (p<0.05)	4.65 (p<0.001)***	-2.60 (p>0.05)
	CG		0.13 (p>0.05	6.78 (p<0.001)***	-0.47 (p>0.05)
	RN			6.65 (p<0.001)***	-0.60 (p>0.05)
	SY				-7.25 (p<0.001)***
LVED index	BB	-3.55 (p<0.001)***	-5.12 (p<0.001)***	0.06 (p>0.05)	0.88 (p>0.05)
	CG		-1.57 (p>0.05)	3.61 (p>0.05)	4.43 (p<0.001)***
	RN			5.18 (p<0.001)***	6.00 (p<0.001)***
	SY				0.82 (p>0.05)
LVES (mm)	BB	-0.53 (p>0.05)	0.60 (p>0.05)	5.15 (p<0.001)***	-0.33 (p>0.05)
	CG		1.13 (p>0.05)	5.68 (p<0.001)***	0.20 (p>0.05)
	RN			4.55 (p=0.008)**	-0.93 (p>0.05)
	SY				-5.48 (p<0.001)***
LVES (index)	BB	-1.89 (p>0.05)	-2.28 (p=0.034)*	1.23 (p>0.05)	1.18 (p>0.05)
	CG		-0.39 (p>0.05)	3.12 (p=0.006)***	3.07 (p=0.006)**
	RN			3.51 (p=0.006)**	3.46 (p<0.001)***
	SY				0.05 (p>0.05)
LVM (gr)	BB	-65.9 (p<0.001)***	-62.5 (p<0.001)***	12.31 (p>0.05)	-60.12 (p<0.001)***
	CG		3.4 (p>0.05)	78.87 (p<0.001)***	6.44 (p>0.05)
	RN			75.47 (p<0.001)***	3.04 (p>0.05)
	SY				-72.43 (p<0.001)***
LVM (index)	BB	-51.56 (p<0.001)***	-62.49 (p<0.001)***	-9.46 (p>0.05)	-12.82 (p>0.05)
	CG		-10.93 (p>0.05)	42.10 (p<0.001)***	38.74 (p<0.001)***
	RN			53.03 (p<0.001)***	49.67 (p<0.001)***
	SY				-3.36 (p>0.05)
SIV (mm)	BB	-1.70 (p=0.005)**	-1.30 (p=0.033)*	0.83 (p>0.05)	1.44 (p=0.016)*
	CG		0.40 (p>0.05)	2.53 (p<0.001)***	0.26 (p>0.05)
	RN			2,13 (p<0.001)***	-0.14 (p>0.05)
	SY				-2.27 (p<0.001)***
SIV (index)	BB	-1.36 (p<0.001)***	-1.49 (p<0.001)***	0.01 (p>0.05)	-0.25 (p>0.05)
	CG		-0.13 (p>0.05)	1.37 (p<0.001)***	1.11 (p<0.001)***
	RN			1.50 (p<0.001)***	1.24 (p<0.001)***
	SY				-0.26 (p>0.05)
PP (mm)	BB	-2.16 (p<0.001)***	-2.36 (p<0.001)***	-0.24 (p>0.05)	-2.09 (p<0.001)***
	CG		-0.20 (p>0.05)	1.92 (p<0.001)***	0.07 (p>0.05)
	RN			2.12 (p<0.001)***	0.27 (p>0.05)
	SY				-1.85 (p<0.001)***
PP (index)	BB	-1.58 (p<0.001)***	-2.05 (p<0.001)***	-0.58 (p=0.024)*	-0.54 (p>0.05)
	CG		-0.47 (p>0.05)	1.00 (p<0.001)***	1.04 (p<0.001)***
	RN			1.47 (p<0.001)**	1.51 (p<0.001)***
	SY				0.04 (p>0.05)

Table 4: Cardiac output stratified by the sport practiced.
*Note: The data are presented as mean difference (p-value) for the comparison between groups (e.g., for the BSA; BB (mean 2.01) – CG (mean 1.85) = 0.16). *p<0.05; **p<0.01; ***p<0.001.*

with a study conducted by Silva et al. (34), which showed no cardiac adaptations in strength compared to endurance athletes. Also, in contrast to our results, other works indicated that bodybuilding would lead to an increase in the internal dimensions of the left ventricle, the thickness of the interventricular septum or the posterior wall, and therefore globally, the ventricular mass.[35-38].

Despite this, ventricular mass (dependent on organ thickness) in bodybuilders is lower compared to populations of other athletes, all of whom (excluding volleyball players, whose body size justifies the difference) are characterized by disciplines that subject the heart to prolonged and intense efforts (where intensity refers to values of heart rate relative to the maximum theoretical heart rate). Moreover, another interesting result is the similarity between CG and RG in all cardiac outcomes; this could be explained by the similarity of the sports practiced and the type of training performed by these athletes. Indeed, the persistent demand for increased blood flow (in terms of stroke volume per heart rate) to actively contracting muscles, combined with greater venous return due to rhythmic contraction of the exercising musculature, leads to central adaptations characterized by a harmonious increase in chamber dimensions and cardiac wall thicknesses.

Despite this, our results are in contrast with precedent studies that showed that cardiac adaptations in cyclers and runners were different[39-41]. For example, Hoogsteen et al.[39] showed an increased left ventricular mass in cyclists and triathletes compared to marathon runners. One possible explanation for these contrasting results could be the evolution in the type of training for both these disciplines in recent years, so future studies should focus on evaluating the specific training program of these disciplines and analyze the adaptations in cardiac output to confirm our results. Supporting the hypothesis that various activities result in distinct cardiac adaptations, the VB group serves as confirmation. In our study, the VB group exhibited the most pronounced divergence when compared to the other groups.

This divergence could be attributed to the specific nature of sports that involve prolonged engagement in explosive activities, such as jumps and dives. In accordance with our results, several studies explained that from different training stimuli occur different cardiac adaptations. For example, Mihl et al.[42] showed that resistance training induces modification in left ventricular wall thickness, while endurance training showed an increase in cardiac output and volume load in both left and right ventricles, leading to a mild to moderate dilation of the left ventricle in the trained heart. Moreover, Spirito et al.[43] confirmed that strength and power disciplines have a large impact on left ventricular wall thickness.

In conclusion, the continuous practice of bodybuilding is not uniquely associated with an increase in left ventricular mass. From the data obtained in our experimental study, there are no cardiological adaptations worthy of note, having found statistically significant differences only with the sedentary group in left ventricular telesystolic and telediastolic diameters. This difference is also canceled by indexing the outcomes obtained for the body surface area. The literature also shows that the type of adaptations found among subjects dedicated to this type of discipline is not absolutely univocal: most cases would be characterized by concentric hypertrophy, while the minority by an eccentric one. The presence or absence of adaptations would depend on multiple factors: the execution of the Valsalva maneuver, which, as widely discussed, would minimize the increase in transmural pressure and, therefore, wall stress, the type of training adopted, the age of start of sports practice, the age of the subjects taken into consideration, the seniority of training.

We must not forget the role of genetics, for which some subjects may be "predisposed" to have a globally large heart and, therefore, a larger left ventricle: further exposure to workloads typical of bodybuilding could exert an additive effect, realizing more marked central adaptations.

References

1) Mitchell JH, Wildenthal K. Static (isometric) exercise and the heart. Physiological and clinical considerations. Annu Rev Med 1974; 25: 369.
2) Grossman W, Jones D, McLaurin MP et al. Wall stress and patterns of hypertrophy in the human left ventricle. J Clin Invest 1975; 56: 56.
3) Keul J, Dickhuth H, Lehmann et al. The athlete's heart - Haemodynamics and structure. Int J Sports Med 1982; 3: 33.
4) Fagard R, Aubert A, Staessen J et al. Cardiac structure and function in cyclists and runners. Comparative echocardiographic study. Brit Heart J 1984; 52: 124.

5) Weicker H. Sympathoadrenergic regulation. Int J Sports Med 1986; 7 (suppl. 1): 16.

6) Zeppilli P. The Athlete's Heart. Practical Cardiology 1988; 14, 8: 61.

7) Devereux, R. B., & Reichek, N. (1977). Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. Circulation, 55(4), 613–618. https://doi.org/10.1161/01.cir.55.4.613

8) Ceccarelli G, Benedetti L, Arcari ML, Carubbi C, Galli D. Muscle Stem Cell and Physical Activity: What Point is the Debate at? Open Med (Wars). 2017 Jul 24; 12: 144-156.

9) Eshani AA, Hagberg JM, Hickson RC et al. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. Am J Cardiol 1978; 42: 52.

10) Rost R, Hollmann W. Athlete's Heart-A review of its historical assessment and new aspects. Int J Sports Med 1983; 4: 147.

11) Blomqvist CG, Saltin B. Cardiovascular adaptations to physical training. Ann Rev Physiol 1983; 45: 169.

12) Agati L, Fedele F, Gagliardi MG. A supernormal behavior of diastolic echocardiographic data in athletes despite left ventricular hypertrophy. Int J Sports Cardiol 1985; 2: 11.

13) Spataro A, Pelliccia, A, Caselli et al. Ventricular repolarization disturbances and hypertrophy in athletes. Int J Sports Cardiol 1986; 3: 17.

14) Hollmann W, Frenkl R, Berteau P, Rost R. The Cardiovascular System. In: Dirix A, Knuttgen HG and Tittel K (eds). The Olympic Book of Sports Medicine. Blackwell Scientific Publications 1988.

15) Pelliccia A, Maron BJ, Spataro A et al. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991; 324: 295.

16) Thompson PD, Sadaniantz A, Cullinane EM, Bodziony KS, Catlin DH, Torek-Both G, Douglas P. Left ventricular function is not impaired in weight-lifters who use anabolic steroids.JACC Vol 19, n° 2, February 1992; 278-82.

17) Sullivan J, Hanson P, Rahko PS et al. Continuous measurement of left ventricular performance during and after maximal isometric deadlift exercise. Circulation 1992; 85: 1406.

18) Fagard RH. Impact of different sports and training on cardiac structure and functions. Cardiol Clin 1992; 10(2): 241.

19) Missault L, Duprez D, Jordaens L et al. Cardiac anatomy and diastolic filling in professional road cyclists. Eur J Appl Physiol 1993; 66(5): 405.

20) Pelliccia A, Spataro A, Caselli G and Maron BJ. Absence of left ventricular wall thickening in athletes engaged in intense power training. The American Journal of Cardiology, 72, November 1,1993.

21) Lentini AC, McKelvie RS, McCartney N, Tomlinson CW and MacDougall JD. Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise. Journal Applied Physiology 75 (6): 2703-2710, 1993.

22) Spirito P, Pelliccia A, Proshan MA et al. Morphology of the "athlete's heart" assessed by echocardiography in 947 elite athletes representing 27 sports. Am J Cardiol 1994; 74(8): 802.

23) Pelliccia A. Determinants of morphologic cardiac adaptation in elite athletes: the role of athletic training and constitutional factors. Int J Sports Med 1996; 17: 157.

24) Fagard RH. Impact of different sports and training on cardiac structure and function. Cardiol Clin 1997; 15 (3): 397.

25) Sagiv M, Sagiv A, Ben-Sira D, Ben-Gal S, Soundry M. Effects of chronic overload training and aging on left ventricular systolic function. Gerontology 1997; 43(6): 307-15.

26) Colan SD. Mechanics of left ventricular systolic and diastolic function in physiologic hypertrophy of the athlete's heart. Cardiol Clin 1997; 15(3): 355.

27) Dickerman RD, Schaller F, McConathy WJ.Left ventricular wall thickening does occur in elite power athletes with or without anabolic steroid use. Cardiology 1998; 90: 145-148.

28) Urhausen A, Kindermann W. Sport-specific adaptations and differentiation of the athlete's heart. Sports Med 1999 ; 28(4) : 237.

29) Qualls E.J. Stevens, Rob D. Dickerman. Surpassing the upper limits of physiologic concentric left ventricular hypertrophy: atrial fibrillation in an elite power athlete. Int J of Card. 2001; 81: 275-6.

30) Haykowsly MJ, Taylor D, Teo K, Quinney A and Humen D. Left ventricular wall stress during Leg-Press Exercise performed with a brief Valsalva Maneuver. Chest 2001; 119:150-154.

31) Scharhag J, Schneider G, Urhausen A, Rochette V, Kramann B, Kindermann W. Right and left ventricular mass and function in male endurance athletes and untrained individuals determined by magnetic resonance imaging. JACC Vol 40, n° 10; November 20, 2002: 1856-63.

32) Haykowsky MJ, Dressendorfer R, Taylor D, Mandic S, Humen D. Resistance training and cardiac hypertrophy: unraveling the training effect. Sports Med. 2002; 32(13): 837-49.

33) American College of Sports Medicine position stand progression models in resistance training for healthy adults. Med Sci Sports Exerc 2002; 34: 364-80.

34) Silva, D. V., Waclawovsky, G., Kramer, A. B., Stein, C., Eibel, B., Grezzana, G. B., Schaun, M. I., & Lehnen, A. M. (2018). Comparison of Cardiac and Vascular Parameters in Powerlifters and Long-Distance Runners: Comparative Cross-Sectional Study. Arquivos brasileiros de cardiologia, 111(6), 772–781.

35) Abinader EG, Sharif D, Sagiv M, et al. The effects of isometric stress on left ventricular filling in athletes with isometric or isotonic training compared to hypertensive and normal controls. Eur Heart J 1996; 17: 457-61.

36) Deligiannis A, Zahapoulou E, Mandroukas K. Echocardiographic study of cardiac dimensions and function in weight lifters and bodybuilders. Int J Sports Cardiol 1988; 5: 24-32.

37) Fleck SJ, Bennet III JB, Kreamer WJ, et al. Left ventricular hypertrophy in highly strength trained males. In: Lubich T, Venerando A, Zeppilli P, editors. Sports Cardiology 2nd International Congress; 1987; Sorrento. Bologna: Aulo Gaggi Publisher, 1987: 303-12.

38) George KP, Batterham AM, Jones B. The impact of scalar variable and process on athlete-control comparisons of cardiac dimensions. Med Sci Sports Exerc 1998; 30: 824-30.

39 Hoogsteen, J., Hoogeveen, A., Schaffers, H., Wijn,

P. F., van Hemel, N. M., & van der Wall, E. E. (2004). Myocardial adaptation in different endurance sports: an echocardiographic study. The international journal of cardiovascular imaging, 20(1), 19-26.

40 Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE. The athlete's heart. A meta-analysis of cardiac structure and function. Circulation. 2000 Jan 25;101(3):336-44. doi: 10.1161/01.cir.101.3.336.

41 Hoogsteen, J., Hoogeveen, A., Schaffers, H., Wijn, P. F., & van der Wall, E. E. (2003). Left atrial and ventricular dimensions in highly trained cyclists. The international journal of cardiovascular imaging, 19(3), 211-217.

42 Mihl, C., Dassen, W. R., & Kuipers, H. (2008). Cardiac remodeling: concentric versus eccentric hypertrophy in strength and endurance athletes. Netherlands heart journal: monthly journal of the Netherlands Society of Cardiology and the Netherlands Heart Foundation, 16(4), 129-133.

43 Spirito, P., Pelliccia, A., Proschan, M. A., Granata, M., Spataro, A., Bellone, P., Caselli, G., Biffi, A., Vecchio, C., & Maron, B. J. (1994). Morphology of the "athlete's heart" assessed by echocardiography in 947 elite athletes representing 27 sports. The American Journal of Cardiology, 74(8), 802-806.

Corresponding Author:
ALESSANDRO GATTI
Email: alessandro.gatti08@universitadipavia.it
(Italy)