Peertechz

Medical Group

Journal of Cardiovascular Medicine and Cardiology

DOI:http://dx.doi.org/10.17352/jcmc

ISSN: 2455-2976

CC By

Francisco Javier Calderón Montero*

Department of Health and Human Performance, School of Physical Activity and Sport Sciences (INEF), Universidad Politécnica de Madrid, Madrid, Spain

Received: 08 October, 2018
Accepted: 14 December, 2018
Published: 15 December, 2018

***Corresponding author:** Francisco Javier Calderón Montero, Department of Health and Human Performance, School of Physical Activity and Sport Sciences (INEF), Technical University of Madrid, Martín Fierro, 7, 28040 Madrid, Spain,
E-mail: franciscojavier.calderón@upm.es

https://www.peertechz.com

Check for updates

Review Article

The Ventricular Function of the "Athlete´S Heart". Part I: Systolic Function

Introduction

The knowledge of the so-called "athlete's heart"has been linked to the application of diagnostic techniques in cardiology. Perhaps, it is necessary to keep in mind that the "athlete's heart" is a physiologically "healthy" heart. The figure 1 shows the parameters that determine the mean arterial pressure. Physical exercise is the only physiological circumstance that precedes the regional metabolic demand to the control of the average blood pressure.

The analysis of figure 1 implies several questions that have been the subject of controversy in the studies on the mechanisms of adjustment and cardiac adaptation. The cardiac response to exercise is complex and includes the interaction of heart rate, preload, afterload and contractility. The discussion has been focused on knowing the relative percentage of each of the cardiac properties that are due to:

1. Procedure for measuring ventricular function in the intact heart [1-7].
2. Body position [8-17].
3. Intensity [12,18,19], type of effort [20] and duration [22,23].
4. Experimental subjects, animals [21,24,25] or human beings [22,23,26] and physical fitness.

All these methodological issues have led to the following questions:

1. Does the Frank-Frank-Starling mechanism intervene during the adjustment of the heart pump to the dynamic effort? If there is an increase in contractile performance due to an increase in preload, what is the relative importance of this mechanism in the ventricular response? A small increase in preload is a mechanism by which the heart can improve its performance during exercise. However, the verification of this fact presents the experimental difficulty of controlling for some factors (heart rate and sympathetic activity), which may the variations of the ventricular dimensions during physical exercise.
2. What role can the increase in contractile capacity (contractility) play in improving ventricular performance during exercise? Indirect data seem to indicate that the contractility improves during the dynamic efforts, being probably a fundamental mechanism of increase of the volume of ejection during the maximum efforts in which the duration of the diastole is reduced.
3. What determines and in what proportion do they intervene (Frank-Starling law and contractility) of the increase in cardiac output during dynamic exercise?

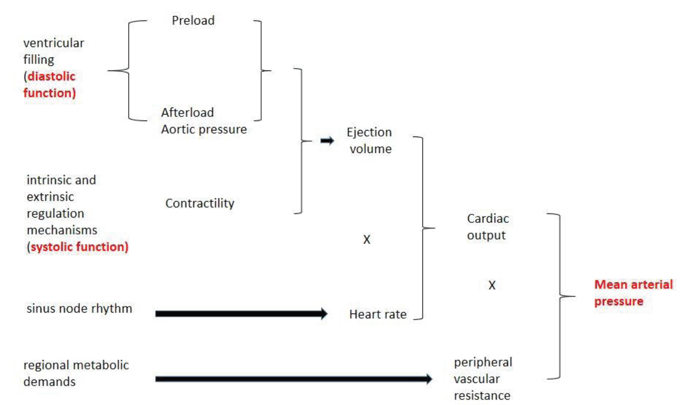

Figure 1

Citation: Calderón Montero FJ (2018) The Ventricular Function of the "Athlete´S Heart". Part I: Systolic Function. J Cardiovasc Med Cardiol 5(4): 088-093. DOI: http://doi.org/10.17352/2455-2976.000079

4. Does the serial disposition of the right heart imply an "accommodation" of its function to the left ventricular response?

5. Can the reduction of diastolic time at high heart rates be a modification of the diastolic function, also determining a greater atrial function?

6. Can ventricular function (left or right) be altered in the long term?

The answer to all these questions is of considerable importance given the degree to which training and competition have acquired, which, in many cases, have become more a potential source of pathology than a real health benefit. Although the progress made is considerable, the answers were not defined with absolute rotundity. In this first approach, I carry out a critical study of the systolic ventricular function of the astigmatism. In a second part, it will be analyzed the diastolic ventricular function will be analyzed. Although for some time, some researchers already intuited the possible "adaptation of diastole" in the trained heart, the reality is that it has been the application of diagnostic techniques in cardiology which has given certain corroborating guarantees to this the improvement of function diastolic.

Morfo-functional consequences of systematized exercise

The invasive cardiological examination techniques (hemodynamics) are difficult to carry out for the assessment of a healthy heart such as the athlete's heart. But the development of non-invasive techniques (echocardiography, ventriculography and nuclear magnetic resonance) has been a great impetus for the study of the athlete's heart. However, interest has been focused on establishing the differences between physiological adaptation, consecutive to training, and the pathologies that occur with changes in cardiac function similar to those that occur in the athlete's heart. This has motivated that many of the conclusions of the initial studies have been disparate.

To introduce this section, it is needed to delimit the characteristics of the athlete's heart that suggest a better systolic function. As was pointed out by Forteza et al. [27], the athlete's heart represents the upper limit of ventricular function, specifically in athletes who have a high dynamic component and a moderate static component, in their training and competition according to Mitchel's classification [28]. These athletes reach high values of cardiac output and as a consequence of venous return. Thus, in the present review only systolic ventricular function is analysed in these athletes.

First of all, it is worthy of consideration the enormous intuition of Henschen (cited by Rost [29], who, at the end of the 19th century, described a finding, which was later described with sophisticated techniques: dilation of the heart in a first stage and subsequently, a compensatory hypertrophy develops to the greater metabolic demand of the myocardium. In addition to this surprising and detailed description, this German author provided a fundamental finding as was able to make the differential diagnosis between the physiological adaptation of the heart and the cardiac diseases that present with dilatation and / or hypertrophy: the symmetry of cardiac dilation.

Cardiac dilatation : The development of echocardiography from the 70s was the confirmation of Henschen's intuition. Considering the cardiac adaptation of athletes who have a high dynamic and moderate static component, the table 1 shows the most relevant results to characterize cardiac adaptation as the maximum expression of systolic ventricular function. The increase in size of the left ventricle in these athletes compared to normal hearts reaches 35%, at most. Values of LVIDd above 64 mm, considered pathological, are exceptional. On the other

Table 1: Echocardiographic measurements suggesting enlargement.

Authors	Left ventricular internal dimension at end -diastole (LVIDd) (mm)	Relative diastolic diameter of the left ventricle (mm/m²)	relative end-diastolic volume (ml/m²)	diameter of the right ventricle
Serratosa [30] Men (n = 740) Females (n = 317)	57±3,9 51,8± 3,9	29,8±3,0 30,3±2,8	84±12,8 73,4±11,8	36±5,2 31,8±5,3
Serratosa [30] Canoe (n = 57) Rowing (n= 36) Cycling (n= 103) Long distance race (n = 74)	57,2± 3,0 60,3± 3,5 58,6± 3,3 56,7±3,7	29,2±1,9 29,8±2,4 31,9±2,1 32,8±2,4	82,5±10,1 90,3±12,4 92,9±11,1 91,9±12,8	36,1±5,2 37,9±4,5 36,2±5,5 36,3±4,8
Pellicia et al [31] Rowing (n = 95; 3 females) Cycling (n = 64; 15 females)	56±3,0 54,8±5			
Boraita et al [32] Men (n = 2039) Female (n = 1242)	55.3±4.4 49.3±3.9	28.5±2.8 29.5±2.6	77.2±13.2 68.9±10.9	
Urhausen et al [33] Rowing Males (n = 64) Females (n = 71)	56,7±3,7 52,2±3,6	26,4±1,8 26,9±1,3		

Citation: Calderón Montero FJ (2018) The Ventricular Function of the "Athlete´S Heart". Part I: Systolic Function. J Cardiovasc Med Cardiol 5(4): 088-093. DOI: http://doi.org/10.17352/2455-2976.000079

hand, it is from 2000 when detailed measurements of the right ventricle have been made. However, it can be stated that the right ventricle of athletes with a high dynamic component is larger, which suggests the "symmetry of hypertrophy." In addition, the criteria of proportionality between the various cardiac chambers, allows to affirm the harmony of the heart of athletes, not only in the ejection chambers but also in the inlet chambers, for example, the contribution of the left atrium to ventricular filling in normal conditions can be approximately 20 to 25%, but could reach 40% in endeavors where the heart rate limits the filling capacity.

Heart hypertrophy : Table 2 shows some echocardiographic measures that may represent the increase in the size of the myocytes and as a consequence of the myocardium. The measurement of thickness of the myocardium has been one of the priority objectives of the studies by echocardiography of the "athlete's heart" when defining the limits of the hypertrophy considered as "physiological".

Again, the maximum expression of hypertrophy is found in athletes who develop a high dynamic and moderate static component. But the differences regarding sedentary people do not exceed 20%. Similarly, the thickness of the interventricular septum is greater in dynamic athletes in a percentage similar to the LVPWd. Therefore, it is easy to deduce that the septum / wall ratio in the athlete's heart does not exceed unity, which in part helps to determine the symmetry of the hypertrophy.

The synthetic ventricular function of the trained heart

In the previous sections, we have summarized the morphological findings, assessed by echocardiography that could justify the improvement of the systolic function of the heart of alteta. In the phenomena of cardiac adaptation to exercise, the general principle is fulfilled: function makes the organ. As a result of the increase in energy demand (functional), the heart "has to adapt" in the way that nature has endowed, in order to "meet" the aforementioned increase. This adaptation should be translated into an improvement of systolic ventricular function. This principle would work as a feedback mechanism, since the greater demand morphologically modifies the heart, but such modification in turn allows the maintenance of cardiac output. Theoretically, greater degree of dilatation and hypertrophy better systolic ventricular function, assessed by echocardiography. Next, the different rates of assessment of systolic ventricular function are briefly discussed.

Table 3 shows the different parameters that assess ventricular function in rest situation. Parameters 1, 2, 3, 4 and 5 are of no interest from the point of view of "quantifying" the ventricular function in hearts adapted to the maximum level. Naturally, from the clinical point of view, it is important, they indicate that the athlete's heart is normal. As an example, EF is analyzed, the most widely studied parameter in the evaluation of the systolic function of the athlete's heart.

It is a global parameter that correlates with the prognosis in many heart diseases and that is considered an essential element, although sometimes not enough, to have a notion of systolic function as a whole.

Very influenced by the methods of measuring the volumes. Until relatively recent simple models were applied (Teizchol), but from the development of softwares more complex models are applied (Simpson).

In the heart adapted to increase the SV to the same extent as the VDF, the EF does not change with respect to a healthy heart. That is, it is a non-discriminative parameter.

It is a parameter that is very dependent on preload, afterload and heart rate.

Table 2: Echocardiographic measurements suggesting hypertrophy and symmetry of cardiac adaptation to training.

	Left ventricular posterior wall thickness at end-diastole (LVPWd) (mm)	Interventricular septum thickness at end-diastole (IVSd) (mm)	Relative left ventricle mass (gr/m²)	left ventricle mas	Index h/R
Serratosa [30] Men (n =740) Female (n=317)	9,2±1,0 7,9±0,9	9,3±1,1 (n = 740) 8,0±0,9 (n = 317)	107,1±19,9 (n = 740) 82,3±14,3 (n = 317)		0,33±0,04 (n = 740) 0,31±0,04 (n = 317)
Serratosa [30] Canoe Rowing Cycling long distance race (n = 74)	9,5±0,9 9,5±0,7 9,5±1,0 9,1±1,0	9,7±1,2 9,4±0,9 9,7±1,0 9,3±1,2	110,4±14,4 115±19,4 123,1±18,8 117,0±21,6		0,34±0,04 0,31±0.02 0,33±0,04 0,33±0,04
Pellicia et al [31] Rowing (n = 95; 3 females) Cycling (n = 64; 15 females)	11,3±1,3 10,4±1,1	±		121±22 115±23	
Boraita et al [32] (*) Men (n = 2039) Female (n = 1242)	8,9±1 7,5±0.9	9,2±1.2 7,7±0.9	97,3±19.6 74,6±14.5		
Urhausen et al [33] Rowing Males (n = 64) Females (n = 71)	11,1±1,2 9,9±1,1	11,2±1,2 9,7±1,1	120±17 98±15		0,40±0,04 0,38±0,04

(*) The data correspond to the entire population of athletes

Citation: Calderón Montero FJ (2018) The Ventricular Function of the "Athlete´S Heart". Part I: Systolic Function. J Cardiovasc Med Cardiol 5(4): 088-093. DOI: http://doi.org/10.17352/2455-2976.000079

Table 3:

Parameter	Description	Interest in the assessment of ventricular function in the athlete's heart
1. Beat volume (VL) or ejection or systolic volumen (SV)	Volume of blood that eject the left ventricle by the aorta (SV = EDV or TDV – ESV o TSV)	0
2. Cardiac output (Q)	Result of multiplying the stroke volume by heart rate (Q = SV · HR	0
3. Cardiac index	Result of dividing cardiac output by body surface (CI = Q/BS	0
4. Ejection fraction (EF)	Result of dividing the stroke volume by the end diastolic cvolume (EF = SV/EDV)	0 (dependent on contractility, loading, the disposition of myocardial fibers, the degree of hypertrophy and morphofunctional characteristics of the aorta)
5. Shortening fraction (SF)	Result of dividing the difference between the diastolic and systolic diameters and the final diastolic diameter (SF = (EDD – ESD)/EDD)	0
6. Circumferential shortening speed (CSS or CSV)	Result of dividing the shortening fraction. for the expulsive period or ejection time. (CSS = SF/ET)	↔ (independent preload parameter)
7. dp/dt	mathematical function that is the derivative of the ventricular pressure with respect to time	↔ (ethically questionable because catheterization is required
8. Strain (St)	deformation that the myocardium experiences when contracting measured in a given orientation	↑
9. Strain rate (SR)	it is defined as the speed at which the St occurs	↑
10. Telesystolic stress	is obtained by dividing the pressure by volume	↑ independent of the load conditions (preload and afterload), but dependent on myocardial mass and volume. Ethically questionable because catheterization is required
11. Elastance	telesystolic stress adjusted to the thickness of the myocardium	↑ (ethically questionable because catheterization is required)

EDV = end diastolic volumen; TDV = telediastolic volumen =; TSV = end systolic volumen; telesystolic volumen; HR = heart rate; BS = body Surface; EDD = end diastolic diameter; ESD = end systolic diameter; ET = ejection time)

It is sensitive to changes in contractility.

It is easy to measure and has a prognostic value in the clinic.

Therefore, the global systolic function indexes have little utility to quantify "physiologically" the maximum limit of cardiac adaptation to training. The cardiac output and the cardiac index do not show differences with respect to sedentary people, since the cardiac output depends on the size of the subject. Thus for two people of the same size, trained and untrained, the cardiac output is around 5 L / min [34]. On the other hand, in the review done by Maron [35], only two studies of the 29 reviewed articles found that the ejection fraction increased.

Parameters 6, 7, 8 and 9 (pre-ejective and deformation parameters) can be considered to be more objective when assessing ventricular function in athletes with high dynamic and moderate static components. They have the advantage over the ejection fraction or the shortening fraction in that they are not measures of systolic function based on changes in the ventricular cavity during blood ejection. However, they are not easy to obtain and interpret, since they must be measured in different planes (longitudinal, transversal or circumferential). For example, the St and Sr, when referring to the baseline myocardial state in end-diastole, is expressed as a percentage (%): 1) positive increase in dimension (for example transverse thickening) and 2) negative decrease (longitudinal systolic shortening). In addition, for each of these (St and Sr) for each orientation there will be a systolic Sr (speed at which it deforms) and a diastolic Sr (speed at which it returns to its basal state).

In summary, the quantification of the ventricular function of the athlete's heart at rest, as in diseased hearts, is difficult and complex. An ideal index of systolic function should be sensitive to inotropic changes, independent of loading conditions, ventricular size and mass. Unfortunately, all the proposed non-invasive echocardiography index do not meet all the indicated conditions, since the best indices of systolic function (invasive) are ethically questionable in the evaluation of the athlete's heart. On the other hand, although specific echocardiographic parameters of systolic function are better (for example St and SR) it is questionable whether deformation is synonymous with contraction, although SR is the parameter that is directly related to intrinsic contractility, that is, with dp / dt that is obtained by cardiac catheterization. The recent appearance of the speckle tracking technique may provide a different view of the echocardiographic indices for the quantification of ventricular function in the athlete's heart. Basically, this technique measures the change or displacement in space (tracking) of a series of "dots" (speckle) that are generated when the beam of ultrasound that emits the transducer is "returned" by the cardiac tissue. Using this technique some authors [36-40] have observed quantitative differences in the systolic function indexes measured by specke tracking.

Summary of the systolic ventricular function in the heart of athlete

As a result of continued training, athletes who perform a high dynamic and moderate static component show dilation and hypertrophy of marked physiological character. In general, the percentage increase of dilatation (diastolic diameter of

Citation: Calderón Montero FJ (2018) The Ventricular Function of the "Athlete´S Heart". Part I: Systolic Function. J Cardiovasc Med Cardiol 5(4): 088-093. DOI: http://doi.org/10.17352/2455-2976.000079

the LV) is greater than that corresponding to the increase in the thickness of the septum or free wall. The increase for the diastolic diameter of the left ventricle does not exceed 35%, while the increase in the thickness of the free wall does not exceed 20%. However, the h / R ratio (free wall thickness / left ventricle diameter) indicates the proportionality of the physiological adaptation phenomenon. When measuring the process of physiological adaptation as a determinant of systolic ventricular function, it is necessary to bear in mind the following considerations:

1) Training. It is necessary to keep in mind the training characteristics (intensity, volume and training time) to delimit the end of the ventricular function in systole.

2) Sex and race. Clearly, women show lower values than men for the same age and body surface, performing the same training load.

3) Anthropometric characteristics. The correction of echocardiographic parameters is usually done with the body surface. However, it is arguable, because it has not beendemonstrated that this is constant, so that some authors recommend making allometric corrections [41].

4) Experience of the echocardiography laboratory. The measurements echocardiographies are subject to considerable variability, in relation to the cut where the measurements are made. Despite the echocardiographic standardization, there is an inter-observer variability that can reach almost the amazing figure of 20%! Thus, it is necessary that the studies are carried out by different observers, including independent laboratories, without any of them knowing the examination carried out by the others. For example, the variations in the thickness of the septum or the free wall of the “athlete’s heart” with respect to the sedentary do not exceed 2 mm, which can be considered within the methodological errors inherent in the echocardiographic technique itself.

From the indicated morphological adaptation (dilation and hypertrophy) it is possible to think that the athlete’s heart will develop a better systolic ventricular function. The problem is that in resting state it is difficult to determine the best systolic function with a simple indices. The more precise indices require invasive methods, ethically questionable scans in healthy hearts. Another issue is that during exercise the higher cardiac output in trained athletes is partly a result of better systolic function. However, the demonstration of an improvement in inotropism is complex, since it “requires” that the preload, afterload and heart rate remain constant. If it is difficult to isolate these variables in very strict experimental conditions, the difficulty increases exponentially during the exercise. It is difficult to know if the contractility improves as a result of mechanisms of homeometric regulation or because of the great neurovegetative activity that occurs during physical exercise, although it is true that the mechanisms of homeometric regulation seem to be of little importance in the overall left ventricular performance, its role during isolated beats and above all in the ventricular interrelation can be fundamental, since the sympathetic stimulation cannot be maintained for long periods of time due to the phenomena of synaptic fatigue, in those efforts of high intensity and prolonged duration, the participation of intrinsic mechanisms for the “maintenance” of cardiac contractility can become important.

References

1. Collier CR (1956) Determination of mixed venous CO2 tensions by rebreathing. J Appl Physiol 9: 25-29. Link: https://goo.gl/dDL3rb

2. Defares JG (1958) Determination of PvCO2 from exponential CO2 rise during rebreathing. J Appl Physiol 13:159-164. Link: https://goo.gl/3YNmdg

3. Ferguson RJ, Faulkner JA, Julius S, Conway (1968) Compaison of cardiac output determined by CO2 rebreathing and dye-dilution methods. J Appl Physiol 25: 450-454. Link: https://goo.gl/Tz8FK4

4. Jernerus R, Lundin G, Thomson D (1963) Cardiac output in healthy subjects determined with a CO2 rebreathing method. Acta Physiologica Scandinavica 59: 390-399. Link: https://goo.gl/kjZSor

5. Klausen (1965) Comparison of rebreathing and acetylene methods for cardiac output. J Appl Physiol 20: 763-766. Link: https://goo.gl/BdN1YZ

6. Levett J, Replogle RL (1979) Thermodilution cardiac output: A critical analysis and review of the literature. J Surg Res 27: 392-404. Link: https://goo.gl/DZP3CL

7. Muiesan G, Sornini CA, Solinas E, Grassi V, Casucci G, et al. (1968) Comparison of rebreathing and direct Fick methods for determining cardiac output. J Appl Physiol 24: 424-429. Link: https://goo.gl/HoFVBG

8. Bevegard S. Freyschuss V, Strandell T (1966) Circulatory adaptations to arm and leg exercise in supine and sitting position. J Appl Physiol 21: 37-46. Link: https://goo.gl/E5MtMH

9. Bevegard,S, Holmgren,A, Jonsson B (1963) Circulatory studies in well trained athletes at rest and during heavy exercise with special reference to stroke volume and the influence of the body position. Acta Phisiol Scand 57: 26. Link: https://goo.gl/VuQJVv

10. Ekelund LG, Holmgren A (1964) Circulatory and Respiratory Adaptation, during Long-Term, Non-Steady State Exercise, in the Sitting Position 1. Acta physiologica Scandinavica 62: 240-255. Link: https://goo.gl/bWrs6w

11. Hermansen L, Ekblom B,,Saltin B (1970) Cardiac output during submaximal and maximal tread mill and bycicle exercise. J Appl Physiol 29: 82. Link: https://goo.gl/2Lrn1p

12. Holmgrem A, Jonsson B, SjšstrandT (1960) Circulatory data in normal subjects at rest and during exercise in the recumbent position,with special reference to the stroke volume at different work intensities. Acta Physiol Scand 49: 343-363. Link: https://goo.gl/bU7PZN

13. Poliner LR, Dehmer GJ, Lewis SE (1980) Left ventricular performance in normal subjects: A comparison of the responses to exercise in the upright and supine positions. Circulation 62: 528. Link: https://goo.gl/r7WUdu

14. Reves JT, Grover RF, Filley GF, Blount SG (1961) Circulatory changes in man during mild supine exercise. J Appl Physiol 16: 279-282. Link: https://goo.gl/Fetizo

15. Reves JT, Grover RF, Blount SG, Filley GF (1961) Cardiac output in response to standing and treadmill walking. J Appl Physiol 16: 283-288. Link: https://goo.gl/VVDqDK

16. Thadani U, Parker JO (1978) Hemodynamics at rest and during supine and sitting bicycle exercise in normal subjects. Am J Cardiol 41: 52-59. Link: https://goo.gl/s2oxje

17. Wang,Y, Marshall R, Shepherd JT (1963) Effet of changes in posture and of graded exercise on stroke volume in man. J Clin Invest 39: 1051.

Citation: Calderón Montero FJ (2018) The Ventricular Function of the “Athlete´S Heart”. Part I: Systolic Function. J Cardiovasc Med Cardiol 5(4): 088-093. DOI: http://doi.org/10.17352/2455-2976.000079

18. Chapman CB, Fisher JN, Sproule BJ (1960) Behavior of stroke volume at rest and during exercise in human beings. J.Clin.Invest 39: 1208. Link: https://goo.gl/MXYsDo

19. Astrand PO, Cuddy TE, Saltin B, Stenberg J (1964) Cardiac output during submaximal and maximal work. J Appl Physiol 19: 268-274. Link: https://goo.gl/Cr4j8B

20. Asmussen E (1981) Similarities and dissimila rities between static and dynamic exercise. Cir Res 46: I-3. Link: https://goo.gl/7RBCLa

21. Vatner SF, Franking D, Higgins CB, Patrick T, Braunwald E (1972) Left ventricular response to severe exertion in untethered dogs. J Clin Invest 51: 3052-3060. Link: https://goo.gl/kcoCDE

22. Saltin B, Stenberg J (1964) Circulatory response to prolonged severe exercise. J.Appl Physiol 19: 833. Link: https://goo.gl/MQBkGv

23. Ekelund LG (1966) Circulatory and respiratory adaptation during prolonged exercise in the supine position. Acta Physiol Scand 68 : 382-396. Link: https://goo.gl/7mZ9z6

24. Rushmer RF, Smith OA, Franklin D (1959) Mechanics of cardiac control in exercise. Circulation Res 7: 602-627. Link: https://goo.gl/RyAxgq

25. Rushmer RF (1959) Constancy of stroke volume in ventricular response to exertion. Amer J Physiol 196: 745-750. Link: https://goo.gl/p9ix6x

26. Shephard RJ (1991) Responses to acute exercise and training after cardiac transplantation : a review. Canadian Journal of sport sciences 16: 9-22. Link: https://goo.gl/wnUAQc

27. Forteza-Albertí JF, Sanchis-Gomar F, Lippi G, Cervellin G, Lucia A, et al. (2017) Limits of ventricular function: From athlete's heart to a failing heart. Clinical physiology and functional imaging 37: 549-557. Link: https://goo.gl/yQSxju

28. Mitchell JH, Haskell W, Snell P, Van Camp SP (2005) Task Force 8: classification of sports. Journal of the American College of Cardiology 45: 1364-1367. Link: https://goo.gl/YJrBbJ

29. Rost R (1982) The athlete's heart. European heart journal 3: 193-198. Link: https://goo.gl/ZGLx3m

30. Serratosa Fernández LJ (1998) Características morfológicas del corazón del deportista de élite: estudio ecocardiográfico. Link: https://goo.gl/Z5G9ns

31. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P (1991) The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. New England Journal of Medicine 324: 295-301. Link: https://goo.gl/v5JT3g

32. Boraita A, Heras ME, Morales F, Marina-Breysse M, Canda A, et al. (2016) Reference values of aortic root in male and female white elite athletes according to sport. Circ Cardiovasc Imaging 9: 005292. Link: https://goo.gl/JdJRxf

33. Urhausen A, Monz T, Kindermann W (1996) Sports-specific adaptation of left ventricular muscle mass in athlete's heart. International journal of sports medicine 17: S145-S151. Link: https://goo.gl/rw5LTJ

34. San Yang S (1988) From cardiac catheterization data to hemodynamic parameters. Davis 430. Link: https://goo.gl/swmQZY

35. Maron BJ (1986) Structural features of the athlete heart as defined by echocardiography. Journal of the American College of Cardiology 7: 190-203. Link: https://goo.gl/mVNBdD

36. Hedman K, Tamás É, Bjarnegård N, Brudin L, Nylander E (2015) Cardiac systolic regional function and synchrony in endurance trained and untrained females. BMJ open sport & exercise medicine 1: e000015. Link: https://goo.gl/zCYbYs

37. Kovács A, Oláh A, Lux Á, Mátyás C, Németh BT, et al. (2015) Strain and strain rate by speckle-tracking echocardiography correlate with pressure-volume loop-derived contractility indices in a rat model of athlete's heart. American Journal of Physiology-Heart and Circulatory Physiology 308: 743-748. Link: https://goo.gl/XgnWq5

38. Santoro A, Alvino F, Antonelli G, Caputo M, Padeletti M, et al. (2014) Endurance and strength athlete's heart: analysis of myocardial deformation by speckle tracking echocardiography. Journal of cardiovascular ultrasound 22: 196-204. Link: https://goo.gl/XiZQDi

39. Schattke S, Xing Y, Lock,J, rechtel L, Schroeckh S, et al. (2014) Increased longitudinal contractility and diastolic function at rest in well-trained amateur Marathon runners: a speckle tracking echocardiography study. Cardiovascular Ultrasound 12: 11. Link: https://goo.gl/4uA7Hg

40. Aksakal E, Kurt M, Öztürk ME, Tanboga IH, Kaya A, et al. (2013) The effect of incremental endurance exercise training on left ventricular mechanics: a prospective observational deformation imaging study/Artan dayaniklilik egzersiz egitiminin sol ventrikül mekanikleri üzerine etkisi: Ileriye-dönük gözlemsel bir deformasyon görüntüleme çalismasi. Anadulu Kardiyoloji Dergisi 13(5), 432. Link: https://goo.gl/gnYb51

41. Gunther B (1975) Dimensional analysis and theory of biological similarity. Physiological reviews 55: 659-699. Link: https://goo.gl/fPoBpU

Copyright: © 2018 Calderón Montero FJ. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Citation: Calderón Montero FJ (2018) The Ventricular Function of the "Athlete´S Heart". Part I: Systolic Function. J Cardiovasc Med Cardiol 5(4): 088-093. DOI: http://doi.org/10.17352/2455-2976.000079