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REVIEW

Adaptation of the aorta to training. Physiological perspective

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Abstract
The damping of the blood pressure oscillations makes the aorta an ''extension'' of the ventricular systolic function. This function is even more evident during endurance exercise, with an increase in the ejection index of 4 times the resting values. Similarly, as a result of training, an adaptation occurs in cardiac morphology, in which the aorta undergoes a change in its structure that allows for better damping function. Athletes that adapt more may experience both of the heart chambers and the aorta, are those who demand a high cardiovascular stress, a high dynamic component and moderate static component. By using non-invasive measurements in humans (mainly echocardiography and MRI), the size of the aorta has been shown to increase in athletes with greater cardiovascular demands.

PALABRAS CLAVE
Ejercicio de resistencia;
Aorta;
Adaptación

Adaptación de la aorta al entrenamiento. Perspectiva fisiológica

Resumen
La función de amortiguación de las oscilaciones de la presión de la sangre hace de la aorta una «prolongación» de la función ventricular sistólica. Esta función se pone aún más de manifiesto durante el ejercicio de resistencia, durante el cual se produce un incremento del índice de eyección de unas 4 veces los valores de reposo. De la misma manera que a consecuencia del entrenamiento se produce una adaptación de la morfología cardiaca, la aorta experimenta una modificación de su estructura que permite una mejor función amortiguadora. Los deportistas que mayor grado de adaptación pueden experimentar, tanto de las cavidades cardiacas como de la aorta, son aquellos que demandan una elevada exigencia cardiovascular, alto componente dinámico y moderado estático. En seres humanos, mediante medidas incruentas (ecocardiografía, resonancia magnética, fundamentalmente), se ha demostrado un incremento del tamaño de la aorta en los deportistas con mayor exigencia cardiovascular.

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Introduction

The aorta has 3 clear functions. The most intuitive is "peripheral conduction" of blood *(conduction function)*, "centrally" propelled by the left ventricle. The second function is as a "temporary blood store" *(reservoir function)* while the left ventricle relaxes and distends to accept blood from the venous territory. The third function is to "convert" "pulsatile flow" to an almost stable flow in the peripheral vessels, "buffering blood pressure oscillations" *(protection function)*.[1] The latter function is critical, because it is estimated that the volume change in the aorta during the cardiac cycle might be around 12.5 ml, assuming a pulse pressure of 25 mmHg and distensibility of 0.5 ml/mmHg.[1]

The reservoir capacity enables considerable volume change throughout the arterial tree with very small pressure changes. If the aorta were not "elastic" or lose its elasticity, as occurs with age, which was demonstrated some time ago[2-4] and has been currently demonstrated,[5-10] the very high pressure exercised by the left ventricle could restrict emptying and pressure in the aorta would fall so rapidly that the ventricle would not be able to fill during diastole with normal pressure values in the left atrium.

The objectives of this review will be realised from a physiological perspective and are as follows:

1. To describe the mechanical properties of the aorta based on the characteristics of the vessel's layers. The mechanical compression behaviour of the aorta might serve to explain the process of adaptation or otherwise of the aorta in athletes with high cardiovascular demand.
2. To review and study whether or not a process of adaptation in the aorta is induced as a consequence of exercise that is demanding of the cardiovascular system. Considerable pressure oscillations occur during intense exercise. This is consistent with the aorta possibly changing in structure and consequently changing its mechanical behaviour to become more efficient.

Mechanical properties of the aorta

The increase in pressure during the entire isovolumetric phase and rapid emptying means that the aorta must maximise its radius and thus reduce resistance to flow. Therefore flow must be as laminar as possible so as not to reach a Reynolds number approaching 1000, which would cause turbulence. Therefore, the radius of the aorta must be proportional to cardiac output and the proportionality constant would be given by the equation ($K = \sigma/\eta \cdot Re \cdot \pi$, η being blood viscosity and σ density). With a blood viscosity level of about 0.03 cm^2/s, the radius of the aorta should be the result of multiplying this level by cardiac output (*radio* = $\frac{Q}{1000 \cdot \pi \cdot 0{,}03}$ = 1 *cm*). However, although this theoretical value corresponds with the measurements taken[11,12] for the majority of the animal species studied, the predictive value is low; it is lower for small animals and higher for larger animals. Nonetheless, correction for body surface area (aortic area = $BS^{0.72}$) greatly adjusts the proportionality relationship between cardiac output and the area of the aorta. However, since the duration of systole varies inversely with the square root of pulse frequency, the linear velocity of blood during systole is greater in small animals. This would mean that small animals use relatively more energy than large animals[11,12] for the same cross section of the aorta and cardiac output relative to size.

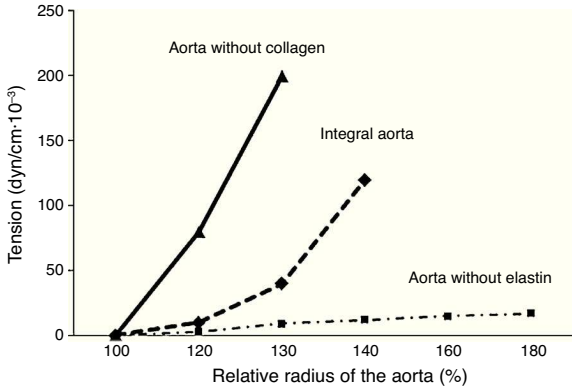

Figure 1 Relationship between tension of the aortic wall and its relative radius. Prepared by self, based on reference data: Shadwick RE. Mechanical design in arteries. J Experimen Biol. 1999;202(23):3305-3313.

The structure of the aorta wall as justification for mechanical properties

It is currently considered that many of the arterial wall structures are interconnected and do not form an exclusive part of each of the layers, in such a way that they mutually influence one another. It has been demonstrated that aortas from which the collagen has been removed by chemical treatment but that have elastic fibres (Fig. 1) show an increased capacity to distend that is not very different from that observed in the lower range of the length-tension relation, corresponding to the intact vessel.

This would mean that the smooth muscle fibres, of elastin and collagen, would be interrelated forming a "single network" which would function in series and in parallel, so that the distensibility of the wall as a whole would be the result not only of the individual properties of each of the fibres, but of the interrelation between them.[13]

Due to their special relevance in the compression of the mechanical properties of the aorta, I provide a brief description of the layers of the wall of the elastic arteries such as the aorta.[13] In particular, the characteristics of the middle layer offer some explanation of the mechanical properties of the arterial walls, and how they can change in certain diseases. Three fibrillin types have been described in the human genome and fibrillin-1 mutations result in Marfan syndrome, which is characterised by cardiac (aortic root dilatation), skeletal and ocular disorders.[14] Fibrillin-2 mutations are associated with autosomal dominant disorders that present, amongst other anomalies, with vascular disorders.[15] In animals, fibulin deficiency triggers evident changes in the aortic arch.[16] In humans, type 1 collagen mutations can result in a form of autosomal recessive disorder (Ehlers-Danlos syndrome).[17] Finally, it should be noted that the function of other proteins (emilin-1, lysine-oxidases) in the walls of the arteries is not known.[13]

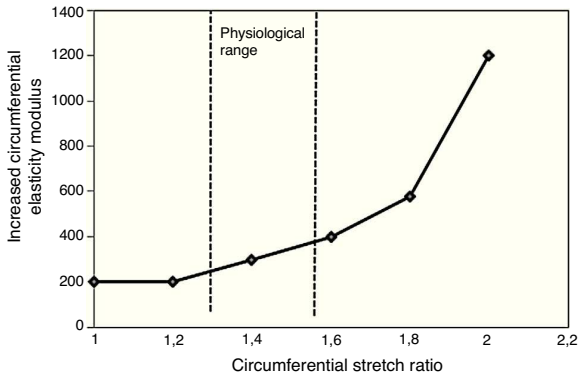

Figure 2 Relationship between circumferential stretch and increased circumferential elasticity modulus. Prepared by self, based on reference data: Wagenseil JE, Mecham, RP. Vascular extracellular matrix and arterial mechanics. Physiol Rev. 2009; 89(3):957.

Mechanical characteristics of the aorta

It has been demonstrated with aortic ring preparations that the relationship between tension ($T = dF/dA$; F being force and A surface area or area) of the aortic wall and its length have no linear relationship and do not obey Hook's law, especially based on a range.[18] This author made an analogy of the behaviour of the aorta with that of a rubber tube covered with a relatively stiff coating.[18] Under the current nomenclature, stress (force per unit of surface area) and strain (change in length given an original length) are given as follows:

$$\frac{F}{A} = Y \cdot \frac{\Delta L = L - L_0}{L_0} \quad (1)$$

where Y is a constant (Young's model), which depends on the composition of the material. A is the area of the cross section and T is the tension exercised for the material of an initial length (L_0) and a change in length.

General characteristics of the tension-length relation

Applying equation 1 to the aorta results in the behaviour explained by Remington and subsequently corroborated (Fig. 2). However, when an attempt was made to explain the general behaviour according to the 2 principal components of the artery walls (elastic and collagen) (Fig. 1), a variation in Young's model was observed. At low tension values, the elastic module of elastin dominates the mechanical behaviour of the entire set of wall structures and this is relatively extensible.[13] From a certain length value, the tension experienced increases exponentially; a slight increase in length results in a considerable increase in tension.

The explanation offered by Wagenseil and Mecham[13] is that at high tension values, the aorta is also distensible, the collagen rather than the elastin however, so that for a determinate value the characteristics of collagen predominate, because the wall is relatively inextensible. Since the elastic vessels are subject to cyclical pressure variations they "expand and contract" with changes in pressure. This prevents the vessels being subjected to very high pressures. The behaviour of the intact vessel, without collagen or elastin, is shown in Figure 1. Note how the radius-tension relation moves towards collagen when tension is high and towards elastin when tension is low. The intact aorta shows a mean behaviour, which from analysis of the figure is clearly not the result of an algebraic sum of behaviours when the wall is without collagen or without elastin.

Mathematical models relating to the artery's mechanical behaviour

The non-linear relation makes a mathematical description of the tension-length relation difficult. Thus, stress, a determinate length value, can be calculated by a simple linear equation when the relation is in the physiological range, but constants need to be established for exponential or polynomial behaviour when the physiological range is exceeded. Despite the difficulties of establishing mathematical models to explain the tension-length relation, these are necessary to compare between different animal species and especially in extreme conditions such as during exercise. It is true that with current imaging techniques, if the length-tension relation is known, tension can be calculated and predicted stability of the vessel wall determined.[13]

Mathematical models are difficult to make, because it is necessary to bear in mind that the tension-length relation presents, like many other biological structures, the hysteresis phenomenon (viscoelasticity or pseudoelasticity). This means that a tension-length relation should be considered for when the aorta distends and another during the process of elastic recovery. It is not know whether or not the hysteresis phenomenon "accumulates" when repeated deformation actions occur. When the hysteresis phenomenon occurs it is possible that the different components of the arterial wall undergo a reorganisation on the circular and longitudinal planes. If, in effect, this were the case, it might be very important in establishing what happens in the aortic root of athletes.

Presumably, mathematical descriptions of the length-tension relation are equations whose constants refer to the properties of the materials. The problem is that it is necessary to make certain specifications when approaching the artery's geometry:[1] 1) establish the length and diameters of the aorta in all its segments, and 2) the transformation of laminar fluid to turbulent fluid has to be taken into account, and therefore a series of dimensional parameters such as the Reynolds and Womerseley number need to be taken into account. Nonetheless, sophisticated imaging methods (magnetic resonance, computerised tomography, positron emission tomography, ultrasound, computerised microtomography, optical tomography at tissue level, interference microscopy, multiphoton microscopy and electronic tomography at cellular level, and X-ray crystallography at molecular level) have now enabled the different mathematical methods to be assessed with relative precision. Some of these methods have been used for imaging of the aorta.[1,19,20]

Simply put, mathematical models with continuity (continuous methods) suggest that all the components of the wall intervene as a whole so that the changes produced in the vessel wall are due to the whole. The disadvantage is that they do not take into account the changes that occur

in the different components of the wall. They consider the wall to behave viscoelastically or pseudoelastically, which means that the behaviours on loading and unloading are treated separately. The vessels are considered non-linear, anisotropic (different properties in different directions) and incompressible. All the continuous models describe the mechanical behaviour of the length-tension relation reasonably well.[1]

Models without continuity (microstructural methods) assign a different mechanical behaviour to each of the wall's components with limits on how one deforms compared to the other and the complete artery. The changes in composition modify the properties of the material of the complete artery and result in improved outcomes in patients with peripheral arterial disease treated with ramipril.[13] In general, these models attribute different mechanical behaviours to the following arterial wall components: elastin (linear behaviour) and collagen (non-linear isotropic and anisotropic behaviour).

Irrespective of the model considered, Kassab[1] concludes that, in general, the vessel walls can model or remodel in response to pressure changes occurring in the arterial system, so that the force exercised on the wall can be kept constant by the regulation mechanisms on the diameter of the vessels (constriction or dilation). Other researchers suggest that blood pressure is in "opposition" to the properties of elastin, collagen and smooth muscle fibres which are arranged in layers, so that the mean tension of each lamella is quite constant (ΔL).

Adaptation of the aorta to training

In athletes, it would seem clear that the maximum expression of aortic adaptation is in hearts undergoing greater degrees of dilation and hypertrophy, assessed by ultrasound, given that athletes' cardiovascular systems are subjected to high levels of stress. In this regard, athletes develop a high dynamic component and a moderate to high static component in their training and during competitions, according to the classification of Mitchell et al.[21] Therefore this section focuses on these athletes.

Animal experimentation

Various studies have approached analysis of the mechanical characteristics of the arteries and have not found differences in Young's module (arterial distensibility) in trained rats[22] or this index was less in trained rats compared to un trained rats.[23] However, Young's module does not demonstrate that aortic distensibility (dV/dP) changes, according to the argument of Koutsis et al.[24] According to Laplace's Law, a change in Young's module might be counteracted by modification of the vessel's radius, as occurs with advancing age, where decreased distensibility is compensated by a greater aortic diameter.

Koutsis et al.[24] demonstrated that, as a consequence of training, the passive distensibility of the aortic wall improved in trained rats. The improvement was certainly significant only in the upper end of the relationship between the variation in aortic length in relation to the variation in tension (ΔL/ΔT), L/T, i.e., a difficult situation to achieve under *in vivo* conditions. The differences in passive distensibility of the aorta between untrained and trained rats were due to the increased collagen in the media in sedentary rats which would explain, in particular, the differences in extension velocities observed when collagen was the main factor determining stiffness. Neither the amount of collagen nor the amount of elastin was lower in the media of the aortas of the trained rats compared to the untrained rats.

Finally, to better understand the response of the aorta to exercise, it is important to determine the differences in the response of maximal tension to the increase of catecholamines, since during exercise there is an increase in sympathetic-adrenal activity. The possible changes of the maximum tension in trained rats respect to the sedentary ones could be the consequence of: 1) an hypertrophy of the smooth muscle fibers of the wall of the arteries, and 2) a smaller number of alpha-adrenergic receptors or to a lower sensitivity of these to the concentration of catecholamines in general and noradrenaline in particular. These possible explanations are yet to be proved.

Humans

Haemodynamic characteristics which impact on possible adaptation of the aorta

The ejection index (ejection volume/body surface area) increases from about 6-15 ml/m^2 (at rest) to 8-25 ml/m^2 or in absolute values, from 5-6 to 25-40 l/min.[25,26] These data suggest an increase in the pressure exercised by the left ventricle. Measurements in animals or indirect measurements in humans (brachial artery pressure) suggest what might occur in the ascending aorta.[26] It has been demonstrated that systolic pressure measured in the arm is overestimated and that the mean pressure is similar in the ascending aorta during a treadmill exercise.[27] In any case, although there is a difference between the levels found between a peripheral artery and the aorta, it is obvious that great "sustained" oscillations are caused during dynamic efforts, although they might even be higher in isometric efforts.[28]

Furthermore, the work of the heart during exercise must reach levels that could impact on the function of the aorta. Total cardiac work is the sum of : 1) work to develop blood pressure and to pump a certain volume ($W_{heartbeat}$ = pressure · volume); 2) kinetic energy $Ec = \frac{1}{2}m \cdot v^2$, and 3) the energy developed to generate tension during the period of isovolumetric contraction ($E_{tension} = K \cdot T \cdot \Delta t$; T being ventricular wall tension and Δt that the ventricle maintains tension). It is considered that at rest kinetic energy is negligible, but it might be important in high intensity physical exercise, reaching up to 15%, when blood velocity can reach levels that exceed Reynolds critical number. During exercise, the work on each beat might be even more important and especially the energy developed on isovolumetric contraction, where it is presumed that the highest pressure levels are generated.

Thus, probably the "highest ventricular pressure levels and the total work developed during intense exercise" are achieved in athletes who have a very high dynamic compo-

nent and moderate static component according to the classification of Mitchell et al.[21]

Aortic size as an adaptation phenomenon

The upper limits of cardiac dilation and hypertrophy have been demonstrated in rowers and cyclists in 2 of the largest published series, 947[29] and 4,739.[30] Table 1 shows data on the size of the left ventricle, wall thickness and size of the aorta only for athletes with high cardiovascular demand. The accepted mode for aortic diameter values was one dimensional (M mode), although they are currently made in two-dimensional mode. Taking into account the values measured in M mode alone, it is accepted by the scientific community as a whole that aortic diameter is increased in athletes with high cardiovascular demand compared to the normality values set by Roman et al.[31] and accepted by the European and American ultrasound societies.

In a meta-analysis,[32] aortic root size is greater (2.2 mm) in endurance-trained athletes than non-athletes. Although these authors consider that the increased aortic root size is slight, I consider that from an anatomo-functional perspective it should be taken into account. Firstly, because the difference in aortic size between athletes and non-athletes are similar to the increase in ventricle size and myocardial thickness, at around 20%. Secondly, it should be borne in mind that the aortic root is inside the fibrous pericardium along with the pulmonary artery, therefore there is a much reduced capacity for stretching. Furthermore, dilation of the aortic root in athletes is exceptional,[33] since 17 out of 2,317 Italian athletes had aortic root sizes ≥ 99th percentile of the population studied (> 40 mm in the males and > 34 mm in the females).

Changes at ultra-structural level as a consequence of training

Although measurements of the aorta in endurance-trained athletes are clearly greater than in non-athletes, the question arises as to whether this increase is the consequence of morpho-functional modification of the aortic wall. As mentioned earlier, animal experimentation seems to confirm improved passive distensibility of the ascending aorta. The different studies in humans do not unanimously indirectly demonstrate an improvement of the aorta's mechanical properties.

Burr et al.[34] observed that ultra-marathon exercise might cause an alteration in arterial elasticity but that it was reversible. Other authors have provided evidence that endurance training causes a reduction in arterial stiffness[35-37] or does not trigger changes.[38-40] Finally, several authors argue that training improves the mechanical characteristics of the arteries[41-47] or induces changes depending on the type of training.[48,49]

In general, the authors that indicate better adaptation of the aorta have demonstrated differences in arterial distensibility in endurance-trained athletes. To be specific, D'Andrea et al.[46] found differences between endurance-trained athletes and strength-trained athletes and controls (4.7, 2.8 and 3.1 in $dyn^{-1}\cdot cm^{-2}\cdot 10^{-6}$, for the endurance-trained athletes, the strength-trained athletes and the controls respectively). The stiffness index calculated by D'Andrea et al.[46] was greater in the strength-trained ath-

Table 1 Aortic volume, thickness and diameter in endurance athletes

	VI telediastolic volume (mL)	Posterior wall thickness (mm)	Septal wall thickness (mm)	Aortic ring (mm)	Sinuses of Valsalva (mm)	Supra-aortic crest (cm)	Ascending aorta (mm)	Maximum ascending aorta diameter (mm)	Minimum diameter (mm)
D'Andrea	150.4 ± 13.9	9.2 ± 2.1	9.7 ± 3.1	21.0 ± 6	31 ± 0.6	2.9 ± 6	31 ± 6		
Boraita	151.4 ± 28.6	9.0 ± 1.1	9.3 ± 1.3	30.3 ± 3.4	263 ± 3.5	26.7 ± 3.8			
Pellicia	118-179			32.22 ± 22.7					
Calderón	162.2 ± 23.9	9.2 ± 1.0	9.3 ± 1.0	31.2 ± 1.5					
Carlson	163.1 ± 23.9	10.3 ± 1.2	10.9 ± 1.0	31 ± 1.5					
Dzudie	70-118	9.2 ± 1.2	10.6 ± 1.2	29.6 ± 3.6					
Galanti	70-118	9.74 ± 0.92	10.12 ± 0.93	29.55 ± 7.85	30.40 ± 6.15	27.34 ± 5.83	28.54 ± 6.46		
Lamont							28 ± 2		
Krol		1.2 ± 0.1	12.0 ± 1.0		33 ± 4		33 ± 4		
Magalski	70-118	10.0 ± 1.0	9.0 ± 1.0		29.0 ± 3.0	25 ± 3.0			
Pellicia							30.3 ± 2		
Aquaroaa								29.2 ± 7.02	27.11 ± 7.1

Measurements taken by ultrasound.
[a] Measurements taken by nuclear magnetic resonance.

letes compared to the endurance-trained athletes and the control subjects (9.2, 6.9 and 6.6 is adimensional, therefore the equation is aortic stiffness = ln (SBP/DBP)/[(AoS – AoD)/AoD], for the strength-trained athletes, endurance-trained athletes and controls respectively). These authors[46] attribute the increased aortic stiffness in the strength-trained athletes to the pressure overload that must be produced in this type of effort.

Due to the considerable methodological limitations of the different studies that either do or do not support an improvement in mechanical characteristics, the results found are certainly very coherent. As indicated earlier, it appears logical that in athletes that have to pump a high blood volume, the aorta becomes more distensible and at the same time less stiff, in other words, more elastic. The variations in the aorta's mechanical properties proposed are maintained by the increased concentrations of the different components (collagen and elastin) of the arterial walls.

In sum, there is scientific evidence as to how the wall adapts to sustained training. This adaptation can be considered physiological and would be more notable in athletes with high cardiovascular demand, as occurs in those with a high dynamic component and moderate static component according to the classification of Mitchell et al.[21] The highest aortic diameter values of endurance athletes measured ultrasonically are indirect evidence of the aorta's adaptation to high cardiovascular demand. Studies on animals show that adaptations of the wall are consecutive to structural phenomena which occur in the wall (quantitative changes of the components), but are difficult to explain or justify in human beings.

Conflict of interests

The author has no conflicts of interest to declare.

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