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Aortic compliance variation in long male distance triathletes: A new insight into the athlete's artery?

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ABSTRACT

Objectives: To assess cardiac and vascular adaptations in long-distance male triathletes and the influence of an increased training volume on these parameters.
Design: Case-control study using long-distance male triathletes (Tri) (n = 12) and an age-matched cohort of sedentary volunteers (Ctrl).
Methods: All participants gave an informed consent and underwent a Cardiovascular Magnetic Resonance imaging (CMR) exam to measure left and right ventricle functional parameters, and aortic parameters (surface, strain, compliance, pulse wave velocity). This exam was repeated in the triathletes' group after an increased training volume of at least 2 h/week for six weeks.
Results: Compared to control volunteers, triathletes presented at baseline a typical pattern of athlete's heart (higher end-diastolic, end-systolic and stroke volumes index, $p \leq 0.009$, and lower cardiac rate, $p = 0.015$) but similar vascular characteristics except a trend towards an enlarged ascending aorta (surface 942 ± 106 vs $812 \pm 127\ mm^2$, $p = 0.058$). Between the two visits, the triathletes increased their weekly training time from 9.67 ± 2.43 (Tri1) to 12.15 ± 3.01 h (Tri2): no modifications were found regarding cardiac parameters, but compliance and distensibility of the ascending aorta increased, from 2.60 to $3.34\ mm^2/mmHg$ ($p = 0.028$) and from 3.36 to $4.40 \times 10^{-3}\ mmHg^{-1}$ ($p = 0.048$) respectively.
Conclusions: Using CMR, we showed that vascular characteristics of the ascending aorta may vary along the sport season in endurance athletes. This remodelling could be considered as a physiological adaptation, but could eventually lead to an adverse vascular remodelling.

1. Introduction

The health benefits of regular moderate intensity exercise are well demonstrated,[1] but the upper limit of this relation is regularly questioned, especially regarding possible sports-related cardiac injuries in endurance athletes.[2] Dealing with this specific population, most studies focused on the cardiac remodelling consecutive to prolonged intense endurance exercise, and the athlete's heart characteristics are now well established.[3,4,5] Intense endurance exercise bouts have also been shown to involve more acute functional and biochemical cardiac dysfunctions, with an unclear significance.[6] Overall, it appears that exercise in excess may sometimes specifically harm the heart.[2] Some evidence also suggest the concept of an "athlete's artery", even if these vascular adaptations in athletes have not yet been fully characterized. Interestingly, studies dealing with the effect of long term and regular endurance exercise training on central blood pressure reveal contrasting findings. For instance, some authors show that, compared to healthy controls, endurance athletes have lower aortic pressure[7] whereas some other show comparable aortic function.[8] In these contrasting studies, the lack of adjustment for confounding factors (among them, no doubt that the amount of training volume plays an important role) is generally discussed as a possible explanation. Cardiac Magnetic Resonance imaging (CMR) has now been accepted as the reference for cardiac assessment in the athletic population,[9] but most studies dealing with vascular adaptation in athletes are

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based on non-invasive tonometry therefore excluding the direct evaluation of central large arteries.[10,11]

Therefore, using CMR, our study was designed to assess cardiovascular characteristics in a long distance male triathlete population to test the hypothesis that not only cardiac but also aortic adaptations may further be modified according to the amount of training volume.

2. Methods

In this prospective single-centre pilot study, approved by the local ethics committee, conducted in a tertiary centre, twelve male non-professional triathletes, practising an endurance activity for several years, were enrolled between January and April 2014. Informed consent was obtained from all participants, who were screened for contra-indications to CMR, including non-sinus rhythm. Inclusion criteria were: age between 18 to 45 years old at the time of inclusion, a regular workout of at least eight hours per week in preparation either for a long distance race (1900-m swim/90-km bike/21.1-km run) or an Ironman (3800-m swim/180-km bike/42.2-km run). All triathletes underwent two CMR, the first one (Tri1) within one month the beginning of their training, considered as their baseline, and the second (Tri2) six to eight weeks after having increased their weekly volume of training by at least two hours. Twelve sedentary people (control group = Ctrl), matched on sex and age, were recruited by means of advertising; sedentary lifestyle, as defined by the World Health Organisation, was a lack of physical activity.[12] Exclusion criteria were: chronic diseases including cardiovascular, medical therapy susceptible to influence cardiovascular remodelling such as antihypertensive or lipid-lowering drugs, current tobacco smoking of at least 5 cigarettes per day.

A medical examination was conducted prior to CMR: height and weight were measured and body surface area (BSA) calculated using the Mosteller standard equation.[13]

CMR was performed on a 3-T unit (Signa HDx, General Electric Healthcare, Milwaukee, Wisconsin, USA) using a dedicated 8-channel cardiac coil. Participants were in supine position, arms along the body. A cuff was placed on their left arm, at the same level as the heart, to allow automated blood pressure recording (Maglife C; Schiller Medical, Wissembourg, France); three measurements were obtained throughout the CMR examination and median values were stored for further analysis. For left ventricle (LV) and right ventricle (RV) functional assessment, a vertical, a horizontal long axis and a stack of contiguous short-axis slices were obtained during 10–15 s breath-hold using a balanced Steady State Free Precession (bSSFP) sequence. Typical parameters were as follows: 30 reconstruction phases per cardiac cycle; 35 cm-field of view; 224 × 224-mm matrix; 8 mm thickness; 1.5 ms-echo time; 45°-flip angle; 3.7–4.1 ms-repetition time; parallel imaging with an acceleration factor of 2; 12 views per segment, yielding a temporal resolution of 45–50 ms. Image analysis was performed using a dedicated software (Mass (MR Analytical Software System) v2013-EXP, Leiden University Medical Center, Department of Radiology, Division of Image Processing, Leiden, NL); LV and RV end-diastolic volumes (EDV), end-systolic volumes (ESV) and LV mass were obtained after manual contouring, following the Society for Cardiovascular Magnetic Resonance (SCMR) recommendations.[14] Papillary muscles were included into LV cavity. All values were indexed to body surface area and the concentric remodelling index calculated as LV mass/LVEDV (Left Ventricle End-Diastolic Volume).[15]

To measure ascending and descending aortic surfaces, a slice perpendicular to the ascending aorta in its middle part, at the level of the right pulmonary artery, was recorded during a breath-hold, with a SSFP sequence similar to that described above, but with a higher temporal (20–25 ms) and spatial resolution (26 cm-field of view). Ascending and descending aorta contours were automatically detected and propagated throughout the cardiac cycle; minimal and maximal surfaces were stored for further analysis, being Aomin and Aomax respectively. Aortic strain (AS) was defined as (Aomax − Aomin)/Aomin; distensibility calculated as AS/brachial pulse pressure, in $mmHg^{-1}$ or $cm^2\,dyn^{-1} \times 10^{-6}$, and compliance as (Aomax − Aomin)/brachial pulse pressure, in $mm^2\,mmHg^{-1}$.

Flow measurements were performed with a phase contrast cine sequence, with the following parameters: electrocardiogram (ECG) gating; 40 cm-field of view; 256 × 128 mm-matrix; 10 mm thickness; 3.3 ms-echo time; 20°-flip angle; 200 cm/s velocity encoding; 6 ms-repetition time; 4 views per segment; 30 cardiac phases reconstruction. Aortic stroke volume was obtained at the middle part of the ascending aorta, using CV Flow software (v2011-EXP, Leiden University Medical Centre, Department of Radiology, Division of Image Processing, Leiden, NL) for analysis; velocities were corrected by a region of interest-based method in case of evident offset error.[16] Aortic pulse wave velocity was measured as the ratio of the distance between two measurements sites, at the mid ascending aorta and the distal descending aorta, to the transit time of the velocity curves, obtained with a 8 ms-temporal resolution phase contrast cine sequence, as previously described.[3,17]

Quantitative results were expressed as mean ± standard deviation. Nonparametric tests were used as a normal distribution could not be assumed owing to an insufficiently high number of cases. Differences between means of triathletes and the sedentary group (i.e. Tri1 vs Ctrl) were assessed by using the non-parametric test of Wilcoxon for independent samples. Mean differences between before and after training session for triathletes (i.e. Tri1 vs Tri2) were assessed by using paired Wilcoxon test.

All tests were also adjusted for multiple comparisons according to Bonferroni–Holm correction. Throughout the analysis, a two-sided p value of less than 0.05 was considered statistically significant. All computations were performed with software R (version 3.2.0, the R Foundation for Statistical Computing, Vienna, Austria).

Table 1
Physical characteristics of triathletes and control volunteers.

Parameter	Tri1 (n = 12)	Ctrl (n = 12)	P value
Age (year)	32.3 ± 7.1	33.1 ± 8.8	>0.99
Weight (kg)	74.2 ± 6.1	80.5 ± 13.3	>0.99
Height (m)	1.81 ± 0.07	1.80 ± 0.08	>0.99
BMI (kg/m^2)	22.60 ± 1.06	24.93 ± 3.98	0.302
BSA (m^2)	1.93 ± 0.11	2.00 ± 0.18	>0.99

BMI, body mass index; BSA, body surface area; Ctrl, control volunteers; Tri1, triathletes at inclusion.
Data are given as means ± standard deviations. P values are adjusted for multiple comparisons according to Bonferroni–Holm correction.

3. Results

The anthropometric characteristics of all participants at the inclusion are presented in (Table 1). The two groups were well matched, without significant difference between Tri1 and Ctrl. Cardiac and vascular characteristics of control volunteers and triathletes, both at inclusion and follow-up, are detailed in (Table 2).

As compared to Ctrl, Tri1 had greater left and right ventricles volumes, with adapted LV mass, increased LV stroke volume, and lower resting heart rate, typical of athlete's heart, but no difference regarding vascular characteristics of the aorta were observed, except a trend towards an enlarged ascending aorta (surface 942 ± 106 vs 812 ± 127 mm^2, p = 0.058).

The delay between Tri1 and Tri2 was 66.25 ± 11.13 days and the amount of weekly training volume increased from 9.67 ± 2.43 h to

Table 2
Comparison of the cardiac and vascular characteristics between the triathletes at baseline and the control volunteers, and the triathletes before and after an increased workout.

Parameter	Ctrl ($n=12$)	Tri1 ($n=12$)	Tri2 ($n=12$)	P value (Tri1/Ctrl)	P value (Tri1/Tri2)
Resting heart rate (bpm)	73 ± 11	60 ± 11	55 ± 11	0.015*	0.098
Blood pressure (mmHg)					
Systolic	123 ± 11	126 ± 8	112 ± 4	>0.99	0.001*
Mean	89 ± 9	86 ± 11	79 ± 7	>0.99	0.122
Diastolic	70 ± 10	66 ± 12	61 ± 5	>0.99	0.355
Median pulse pressure (mmHg)	53 ± 8	61 ± 7	52 ± 4	0.090	0.007*
Left ventricle					
ED volume index (mL/m^2)	84.3 ± 11.5	113.7 ± 14.3	116.7 ± 12.2	<0.001*	0.904
ES volume index (mL/m^2)	37.6 ± 6.3	49.8 ± 10.5	52.1 ± 9.1	0.009*	0.611
Stroke volume index (mL/m^2)	46.7 ± 7.5	63.9 ± 8.9	64.6 ± 7.0	<0.001*	>0.99
Mass index at end diastole (g/m^2)	55.7 ± 5.8	73.2 ± 8.0	74.1 ± 7.3	<0.001*	>0.99
Ejection fraction (%)	55.3 ± 4.5	56.4 ± 6.0	55.5 ± 4.6	>0.99	>0.99
Right ventricle					
ED volume index (mL/m^2)	81.2 ± 12.6	112.5 ± 12.0	114.3 ± 10.8	<0.001*	0.798
ES volume index (mL/m^2)	35.6 ± 7.4	48.5 ± 7.8	49.7 ± 8.5	0.001*	>0.99
Stroke volume index (mL/m^2)	45.6 ± 7.7	64.0 ± 8.8	64.6 ± 6.5	<0.001*	>0.99
Ejection fraction (%)	56.2 ± 5.1	56.9 ± 5.2	56.7 ± 4.9	>0.99	>0.99
Cardiac index (L/min/m^2)	3.3 ± 0.4	3.9 ± 0.8	3.7 ± 0.7	0.062	>0.99
Ascending aorta					
Aomax (mm^2)	812 ± 127	942 ± 106	957 ± 98	0.058	>0.99
Aortic strain (%)	18.5 ± 5.4	20.2 ± 7.3	22.9 ± 9.2	>0.99	0.388
Distensibility (10^{-3} mmHg^{-1})	3.44 ± 0.67	3.36 ± 1.17	4.40 ± 1.58	>0.99	0.048*
Compliance (mm^2/mmHg)	2.36 ± 0.59	2.60 ± 0.71	3.34 ± 0.90	>0.99	0.028*
Descending aorta					
Aomax (mm^2)	497 ± 67	576 ± 86	591 ± 90	0.113	0.816
Aortic strain (%)	25.6 ± 6.2	25.0 ± 6.0	23.4 ± 7.3	>0.99	0.611
Distensibility (10^{-3} mmHg^{-1})	3.86 ± 0.88	3.30 ± 0.65	3.62 ± 0.73	0.340	0.388
Compliance (mm^2/mmHg)	1.92 ± 0.54	1.89 ± 0.38	2.1 ± 0.48	>0.99	0.388
Aorta PWV (m/s)	5.05 ± 0.65	4.74 ± 0.51	5.1 ± 0.79	>0.99	>0.99

Aomax, maximal cross-sectional area at systole; Ctrl, control volunteers; ED, end-diastolic; ES, end-systolic; PWV, pulse wave velocity; Tri1 and Tri2, triathletes at baseline and follow-up respectively.
Data are given as means ± standard deviations; P values are adjusted for multiple comparisons according to Bonferroni–Holm correction.
* Statistically significant.

12.15 ± 3.01 h ($p=0.011$). Systolic blood pressure and median pulse pressure decreased, but no further difference was found regarding left and right ventricle parameters. Compliance and distensibility of the ascending aorta (respectively 2.60 vs 3.34 mm^2/mmHg, $p=0.028$; 3.36 vs 4.40 × 10^{-3} mmHg^{-1}, $p=0.048$) were increased but not those of the descending aorta (respectively 1.89 vs 2.1 mm^2/mmHg, $p=0.388$; 3.30 vs 3.62 × 10^{-3} mmHg^{-1}, $p=0.388$).

4. Discussion

The health benefits of regular moderate intensity exercise are well established, both on cardiovascular and noncardiovascular morbi-mortality.[1] However, repeated prolonged intense endurance exercise can also induce significant cardiac remodelling (termed the "athlete's heart") sometimes leading to rare but remarkable sudden cardiac events.[18] The triathletes involved in our study undoubtedly presented the characteristic cardiac adaptations of the athlete's heart (Tables 1 and 2).[4] The athlete's heart is now a generally accepted term with numerous studies focusing on the structural, functional and electrical remodelling that accompanies regular exercise.[4] Some authors indeed hypothesized that excessive intense exercise may harm the heart[2]; this has been particularly suggested for excessive bouts of exercise (i.e. too intense or with too short recovery period) possibly leading to cardiac injury with proarrhythmic remodelling (predominantly affecting the right ventricle).[19]

In contrast, the vascular adaptation in athletes has not been fully characterized, even if some evidences suggest the concept of an "athlete's artery".[10] The CMR imaging technique used in our study allowed us to assess the vascular characteristics of the aorta whereas most studies dealing with athlete's artery[20] only considered more peripheral arteries based on pulse wave analysis method.[11] Facing the athlete's heart and the hemodynamic stress imposed during exercise, the ascending aorta acts as the first (i.e. just beyond the heart) elastic buffering chamber known as the Windkessel function.[21] The Windkessel function is directly related to the elastic properties (i.e. compliance) of the aorta, and has a critical influence on the peripheral circulation but also on the heart function (reduction of left ventricular afterload, improvement in coronary blood flow and left ventricular relaxation). The original finding of this study was the increased compliance and distensibility of the ascending aorta between Tri1 and Tri2, i.e. after increasing the training volume, but not of the descending aorta. Considering the close relationship of the heart and the aorta, this could be considered as an adaptive remodelling of the vascular structure of our triathlete population. Even if far less attention has been devoted to athlete's arteries than athlete's heart, some evidences support that endurance athletes have enlarged arteries with decreased wall thickness as a result of prolonged training interventions.[20] This specific athlete's wall thickness changes should be considered a physiological adaptation on wall remodelling in healthy arteries under repetitive exercise stress, but the long-term vascular consequences are currently unknown. Furthermore, this increased compliance of the ascending aorta between Tri1 and Tri2 associated with the absence of any significant differences in arterial compliance between Ctrl and Tri1 (Table 2) may also highlights a kind of dose-response relationship for the exercise-related benefits in vascular adaptation, involving a minimum amount of time training to be effective (i.e. the minimal threshold to trigger this vascular adaptation). Indeed, the increased compliance of the ascending aorta was observed after our triathlete population had increased their weekly training hours from 9.67 ± 2.43 to 12.15 ± 3.01 h for at least two months. These results support that, once the heart is already adapted in athletes (i.e. athlete's heart), a further increased

in training hours (i.e. between Tri1 and Tri2) may not significantly impact the heart but may predominantly affect the vascular structure beyond the heart. Thus, by analogy with the suspected adverse cardiac remodelling of excessive exercise,[2] we could speculate that the variation of aortic compliance according to the amount of training hours during a training season may also lead to some adverse vascular remodelling. Indeed, available data now show that aortic root enlargement can be found in elite athletes in the absence of apparent aetiology.[22] Initially, it was found that aortic root diameter was greater in strength-trained athletes compared with endurance-trained ones and that the duration of high-intensity strength training appeared to be the strongest predictor of aortic dimensions.[23,24] More recently Pellicia et al. found that aortic dimensions were larger in endurance athletes than in strength athletes but the cause of this aortic enlargement in a few athletes remains to be clarified.[25] Despite the limited size of the population, we indeed found a trend toward a larger ascending aorta of Tri1 than Ctrl (Aomax; $p = 0.058$). In the light of our major result, we could speculate that the repeated variations of compliance of the ascending aorta over the years in endurance athletes may sometime lead to pathological vascular remodelling and possibly "exercise-induced aortic root dilatation". As these results were obtained in a limited population of male triathletes, further studies are needed to confirm them in both male and female athletes, and to investigate the impact of long-term endurance exercise on arterial structure and remodelling.

Apart from the limited size of the population, we acknowledge that we didn't obtain central pressures but relied on brachial arterial pressure to calculate aortic compliance and distensibility, as usual for MRI assessment of these parameters.[17,26,27] The other limitation of this study is that no follow-up study was obtained in the control subjects, as this study was designed to assess aortic adaptations according to the athletes' training volume.

5. Conclusion

Vascular remodelling in the athletic population results in the emergence of the concept of "athlete's artery", but the long-term consequences of such a remodelling are currently unknown. Using CMR in a specific population of trained long distance triathletes, our study demonstrated an increased compliance and distensibility of the ascending aorta related to the amount of training volume. Thus, vascular characteristics may vary during the sport season depending on the training and recovery periods in endurance athletes. If this remodelling can be considered as a physiological adaptation, it also questions the potential for some possible adverse vascular remodelling, especially in the light of the increasing reports of aortic root enlargement in elite athletes. Then, whether repeated prolonged intense exercise may not only harm the heart but also the arteries needs to be clarified.

Practical implications

- This study shows that long distance triathletes present with LV and RV remodelling typical of an athlete's heart.
- It also demonstrated that these ventricular parameters were not further modified during an 8-week period of increased training, whereas aortic compliance and distensibility increased in the meantime.
- These modifications of vascular characteristics during a sport season, along with a trend towards dilation of the ascending aorta brings new evidence of an athlete's artery that could lead to cardiovascular events.

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Ethical approval

This study was approved by the local ethical committee and informed consent was obtained from each participant.

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