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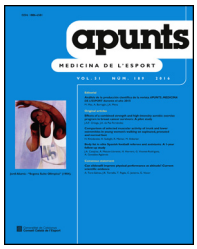

REVIEW

Cardiovascular effects and alterations from endurance sports. A systematic review

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KEYWORDS
Arrhythmia;
Arrhythmogenic risk;
Endurance training;
Cardiac pathology;
Athletic heart syndrome

Abstract The practice of endurance sports provides health benefits due to cardiovascular adaptation. Nevertheless, there has been a lot of research in which patients have shown some type of ventricular cardiac arrhythmia. This has worried some professionals who have been utilising this kind of training. As a result of this, professionals have begun revising and analyzing the scientific evidence from the last ten years. This investigation was focused on the review of possible development of arrhythmias and cardiovascular alterations that endurance sports can cause. In the following systematic review, we evaluate publications that show a direct relation between endurance sports and cardiac arrhythmias from 2006 to 2016. Twelve articles were identified and then grouped by different effects on the heart. Due to the high levels of cardiac stress that endurance-trained athletes are exposed to, there is a tendency for them to show different types of arrhythmias.

PALABRAS CLAVE
Arritmia;
Riesgo arritmogénico;
Entrenamiento de resistencia;

Efectos y alteraciones cardiovasculares provocados por la práctica de deportes de resistencia. Una revisión sistemática

Resumen La práctica de actividad física, especialmente el deporte de resistencia, produce adaptaciones cardiovasculares beneficiosas para la salud. Sin embargo, en varias investigaciones se ha reportado la presencia de algún tipo de arritmias cardíacas ventriculares

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Patología cardíaca;
Corazón de atleta

o supraventriculares, lo que ha desencadenado una alerta en los profesionales que recomiendan este tipo de estímulos. Consecuencia de lo anterior, fue imprescindible revisar y analizar la evidencia científica existente dentro de los últimos 10 años sobre las posibles arritmias y alteraciones cardiovasculares que generan los deportes de resistencia. En la siguiente revisión sistemática se evaluaron publicaciones entre los años 2006 y 2016 que relacionaron los deportes de resistencia con arritmias cardiacas. Se identificaron 12 artículos, que fueron agrupados según el efecto generado en el corazón (arritmias, fibrilación auricular, fibrosis cardíaca y cambios anatómicos). Debido a la alta exigencia cardíaca a que se exponen los deportistas de resistencia, existe una tendencia a la aparición de distintos tipos de arritmias.

Introduction

The regular practice of physical activity gives rise to major health benefits, reducing risk factors such as cardiovascular diseases and reducing the mortality associated with cardiac events.[1] Given this thinking, over recent years the practice of long-duration endurance sports has increased considerably,[2] with an increase in sudden deaths among marathon runners due to a predisposition to generate ventricular as well as supraventricular arrhythmia.[3] On the other hand, an association has been described between subject who practice endurance sports and the appearance of atrial fibrillation.[4,5] The latter is now one of the most common conditions among athletes, affecting from 0.4% to 1% of the general population.[6]

The lifelong practice of endurance sports, sports requiring strength or combinations of the same, gives rise to adaptations throughout the body.[7,8] At cardiovascular level, the most common adaptation is known as ''athlete's heart''.[9–12] This usually consists of cardiac remodelling with significant changes in diameters, large concentric hypertrophies and increased volumes ejected by both ventricles, larger than is the case in the general population. Some authors have reported lengthening of the left ventricle.[9,13] All of the above factors may lead to increased risk of developing negative heart conditions.[14–20]

Different physiopathological mechanisms have been suggested to explain the increased risk of developing atrial fibrillation associated with sport.[9,21,22] These explanations involve ectopic atrial beats as the first possible cause, referring to effects of ectopic pulmonary veins that may generate paroxystic atrial fibrillation.[3,9] Another hypothetic cause is modulation of the autonomous nervous system. This refers to the role played by the autonomous nervous system in affecting heart beat, depending on predominance of vagus or adrenergic stimulation.[3,9] Lastly, structural changes in the atria due to exercise have been postulated as another possible cause.[3,6,9] Other possible explanations include sex (female) and heart inflammation/dilation, although there is little scientific evidence in this respect.[3,6]

It has not been possible to scientifically establish whether the transformation of the myocardium of individuals who practice physical activity for long periods of time may be the cause of symptoms or physiopathological conditions such as ventricular arrhythmias or atrial fibrillation.[1,23,24] Nevertheless, there is evidence that there is a relationship between doing sports and the appearance of arrhythmias or atrial fibrillation,[14–16] as well as possible explanations for this relationship.[3,6,9] It is therefore clear that the existing information is ambiguous and hardly enables drawing any conclusions about this subject. The aim of this systematic revision is therefore to revise and analyse the existing scientific evidence which has emerged over the last ten years on arrhythmias and cardiovascular alterations that may be due to endurance sports.

Material and methods

Bibliographic search

A thorough search was carried out for this study in bibliographical references as well as electronic sources in different databases and search engines. The combinations of words shown in Table 1 were used for this search. The electronic search took place in Web of Science (WOS), Scopus, Sport Discuss, PubMed and Medline.

The search strategy was divided into five stages. Stage one consisted of electronically searching the different data bases, identifying 1654 papers, and after all duplicates had been erased 742 papers remained to be subjected to screening of their titles and abstracts. The second stage was the revision of all these remaining papers, after which 38 of them were left. In the third stage the 38 papers were read to identify the studies to be analysed. After revising the papers 30 were eliminated, all because they did not consist of experimental studies. Stage four consisted of searching articles guided by the bibliography. This stage included four new studies. Additionally, independent reviewers agreed on the inclusion of these twelve papers in the systematic review (Fig. 1).

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Table 1 Search strategy using the selection and combination of key words.

Steps	Strategy	WOS	Scopus	Sport Discuss	PubMed	Medline
1	Marathon	2603	3071	12,270	1370	1402
2	Endurance training	7768	9113	2326	6709	2592
3	Athlete heart	2702	4532	274	2559	226
4	#1 OR #2 OR #3	12,227	15,696	14,774	10,037	4161
5	Arrhythmia	34,636	67,920	1616	68,490	23,302
6	Myocardial fibrosis	7238	6624	62	7293	2704
7	Arrhythmogenic risk	756	1102	2	839	133
8	#5 OR #6 #OR #7	41,753	74,457	1674	75,256	25,979
9	#4 AND #8	354	804	33	431	32

Table 2 Inclusion criteria.

Study design	Experimental
Population	Adult men and women (trained and untrained)
Intervention	Performing endurance training
Comparator	Presenting arrhythmogenic risk factors
Results	Positive and negative
Languages	English and Spanish

Inclusion and exclusion criteria

The search limits were: papers published in the last 10 years (January 2006 to January 2016), written in English or Spanish, with no screening whatsoever as to the type of study sought.

The importance of each study was evaluated according to the inclusion criteria shown in Table 2. Studies which did not fulfil the inclusion criteria were excluded. The discrepancies which arose were resolved by consensus among the researchers.

Evaluation of methodological quality

The Physiotherapy Evidence Database scale (PEDro) was used to evaluate the quality of the studies. They were classified according to three selection criteria (maximum three stars), comparability (maximum five stars) and results (maximum two stars). The papers with a score from eight to eleven were considered to be of high methodological quality, while those with from four to seven were medium quality and those with fewer than four stars were considered low quality.

According to the PEDro scale no paper obtained a high score, while eleven were classified as moderate and one study was classified as low quality (Table 3).

Results

Cardiac adaptations to training

Cardiac adaptations as a result of long duration endurance sport training were sought, and electrical as well as anatomical changes were found.[3,6,9] The different effects that were

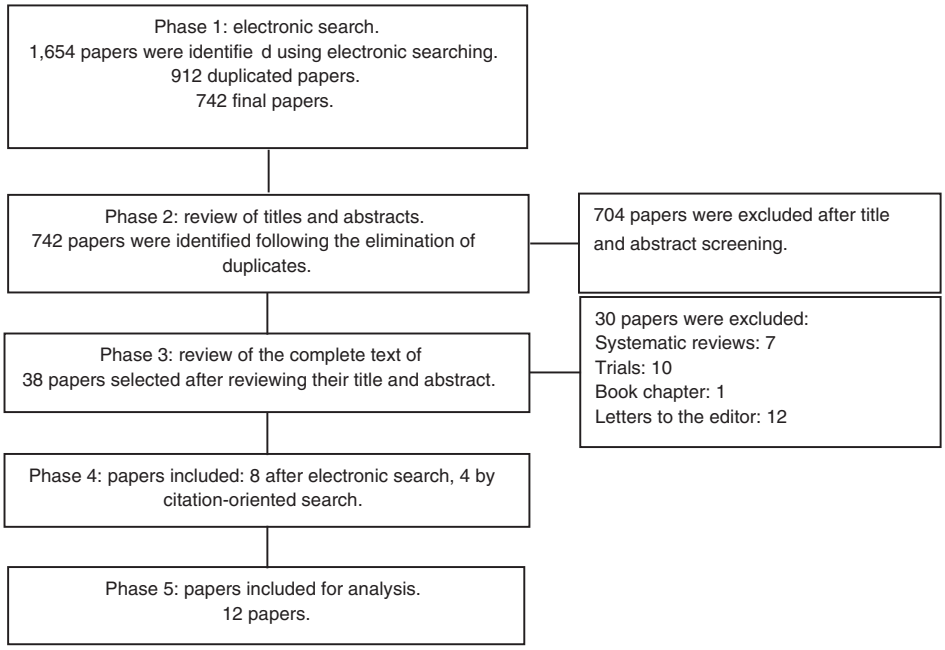

Figure 1 The identification of studies in the systematic review.

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Table 3 List of papers included with their score on the PEDro scale.

		Selection	Comparability	Results	Total
1	Biffi et al.[25]	**	**	**	6
2	Biffi et al.[26]	****	**	**	8
3	Fuchs et al.[27]	*	**	**	5
4	Myrstad et al.[28]	*	**	**	5
5	Verdile et al.[29]	*	**	**	5
6	Matelot et al.[30]	*	**	**	5
7	Brugger et al.[14]	*	**	**	5
8	Heidbuchel et al.[15]	*		*	2
9	Molina et al.[16]	**	**	**	6
10	Lindsay et al.[31]	**	**	**	6
11	Pellicia et al.[32]	*	**	**	5
12	Wilhelm et al.[33]	*	**	**	5

found made comparing and synthesising the results complicated. The papers found were divided into three categories based on different effects on the heart, to classify them and achieve the objective of this study: (1) Arrhythmia ($n = 7$), (2) atrial fibrillation ($n = 3$) and (3) cardiac fibrosis cardiac and anatomical changes ($n = 2$) (Table 4).

Below, and following a systematic, detailed and thorough review of the last ten years, only the papers about long-duration endurance sports and their effects on the heart are presented.

Arrhythmia (tachyarrhythmia, bradyarrythmia and arrhythmia)

This alteration refers to a loss of normal cardiac rhythm due to imbalances in the electrical conductivity of the heart, giving rise to irregular patterns of cardiac frequency (arrhythmia, tachyarrhythmia or bradyarrhythmia; ventricular and supraventricular).[25] On the other hand, in the Spanish literature the corresponding terms are ''arritmia'', ''taquiarritmia'' and ''bradiarritmia''. The search therefore has to be restricted to these exact terms.

Research by Biffi et al.[25] studied 37 physically active subjects and their levels of training. They were de-trained and then re-trained. At the end of the study an increase in ventricular tachyarrhythmia was found. Nevertheless, this increase reduced one year after they had returned to training. In the year 2008[26] this same authors carried out a study in which 175 elite athletes were selected who had been actively taking part in marathons and who had no signs or risk factors associated with cardiovascular diseases. The researchers compared their cardiac behaviour with that of other groups of subjects, each one with a different magnitude of training. They found non-significant differences between the mass of the left ventricle and the generation of ventricular tachyarrhythmia.

Similarly, Fuchs et al.[27] found elite and amateur athletes who presented arrhythmia during stress tests. At the end of the study all of the athletes, except for one with a long history of myocardiopathy, lived during the follow-up period of 70 ± 25 months. A study by Verdile et al.[29] of 5011 healthy athletes monitored their training and sports life during 7.4 ± 5 years. At the end of the study, benign arrhythmias that were not associated with cardiac events were reported. In the same way, Matelot et al.,[30] studied whether exercise associated with symptoms such as bradycardia predisposed to the development of arrhythmia or cardiac arrest. They compared athletes with bradycardia with sedentary subjects, and their results showed zero association of athletes with deep bradycardia and episodes of arrhythmia.

On the other hand, Brugger et al.[14] evaluated the impact of a life of aerobic training on anatomical remodelling and electrical adaptations in 95 athletes. The researchers divided the sample into groups according to the training time they had engaged in during their lives. When the study ended significant changes were reported in anatomical remodelling and electrical adaptations associated with the length of time they had been training. Additionally, Pellicia et al.[32] studied the association between high intensity training and the incidence of cardiac events or dysfunctions caused by training of this type. This study evaluated 114 Olympic athletes who had been training uninterruptedly and intensely for a long period of time. At the end of the study no link was found between training intensity and the appearance of cardiac events or ventricular dysfunction.

Atrial fibrillation

Atrial fibrillation is a common arrhythmia, and as such it should be included in the above category. However, given the large amount of data found during the review of this as a separate subject, the decision was taken to create its own sub-category to make it possible to examine in the best possible way the physical–pathological mechanisms developed by chronic exercise. Atrial fibrillation refers to a condition in which beating is faster than normal and which often causes deficient blood supply.[14–16] The term used in the Spanish literature for this condition is ''fibrilación auricular''.

Myrstad et al.[28] evaluated 3545 subjects and divided them into three groups according to how long they had been training for endurance sports. They searched for an association between length of lifetime training and the risk of developing atrial fibrillation. This study found a gradual increase in the risk of developing atrial fibrillation accompanied by heart murmur. Similarly, Heidbuchel et al.[15] studied 137 volunteer subjects who were evaluated according to

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Table 4 Characteristics of the publications that link long duration aerobic training and its effects on the heart.

Authors	Year	Consequence in the heart	Methods	Results	*n*
Biffi et al.[25]	2011	Arrhythmia	34 athletes without cardiovascular abnormalities and with frequent and complex ventricular tachyarrhythmia were monitored for 24 h using a Holter electrocardiograph, in three periods. Firstly, during their most intensive training, then in a period of de-training lasting for 3–6 months and lastly after 2, 6 and 123 months of re-training.	The subjects showed partial (101–500 premature ventricular complexes (PVC)/24 h) or marked (<100 PVC) reversibility in a arrhythmias after the de-training. Re-training at first gave rise to a significant increase in the frequency of arrhythmias compared to de-training (from 280 ± 475 to 1.54 ± 2.186 PVC; $P = .005$), ventricular pairs (0.14 ± 0.42 to 4.4 ± 8.2; $P = .005$) and unsustained ventricular tachycardia (of 0 to 0.8 ± 1.8; $P \leq .02$). Subsequently a gradual reduction was observed in the frequency of all the ventricular arrhythmias during one year of training (PVC 917 ± 1.630, bradycardial event 1.8 ± 4.2, and unsustained ventricular tachyarrhythmia 0.4 ± 1.2).	37
Biffi et al.[26]	2008	Arrhythmia	Cardiovascular examinations took place during the athletes' most intense period of training, and they were monitored for 24 h with a Holter electrocardiograph, including a session of conditioning (average 1 h); this intervention consisted of what the athlete usually did. Echocardiographic studies also took place during training sessions.	The athletes with and without left ventricular hypertrophy did not differ respecting the total numbers of PVC ($P = .58$), ventricular pairs ($P = .63$) and unsustained ventricular tachyarrhythmia (USVT) ($P = .61$). Additionally, athletes with only isolated uniform PVC ($n = 130$) and one with complex forms of ectopy (PVC multiforms, bradycardial events, or forced USVT; $n = 45$) showed not significant different in the mass of the left ventricle (97.9 ± 21 vs $102 \pm 22\ g/m^2$), $P = .2$).	175
Fuchs et al.[27]	2011	Arrhythmia	Initially: Electrocardiogram and stress test (Astrand's protocol). Then: Holter, echocardiogram, magnetic resonance and electrophysiological study.	A total of 56,462 athletes were subjected to an stress test for monitoring prior to taking part, identifying 192 under the age of 35 years old with ventricular arrhythmias during the stress test. Of the 192 athletes selected, 90 had 3 or more premature ventricular beats (PVB) (Group A) and 102 athletes had PVB and ventricular pairs or USVT (ranges from 3 to 30 consecutive beats) during the stress test (Group B).	284
Myrstad et al.[28]	2014	Atrial fibrillation.	Electrocardiogram and questionnaire	The accumulation of years of regular endurance exercise is associated with a gradual increase in the risk of atrial fibrillation (AF), with an OR of 1.16 (95% CI 1.06–1.28) per 10 years of exercise, 1.16 (95% CI 1.00–1.36) among the skiers and 1.20 (95% CI 1.06–1.35) among the men in the Oslo Health Study. Endurance exercise was also associated with a gradual increase in the risk of atrial flutter. The OR for atrial flutter per 10 years of exercise was 1.42 (95% CI 1.20–1.69) and there was no difference between the 2 cohorts).	3545

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Table 4 (*Continued*)

Authors	Year	Consequence in the heart	Methods	Results	*n*
Verdile et al.[29]	2015	Arrhythmia	Routine medical evaluation, physical examinations, electrocardiogram and two-dimensional echocardiogram. A stress test was performed before monitoring with an out-patient Holter for 24 h, magnetic resonance and average echocardiogram signal in selected athletes with ventricular arrhythmias.	Of the 5011 athletes, 367 (7.3%) showed ≥1 premature ventricular beat (PVB), including 331 with ≤10 PVBs and 36 (0.7%) with >10 PVBs and/or ≥ventricular pairs. The 331 athletes with 10 ≤ PVBs had no restriction in competitive sports, and repeating the stress test over 3–12 months showed a spontaneous reduction in the arrhythmias (from 5.2 ± 4 to 4 ± 6 PVBs; $P = .002$), including 83 of the 331 (23%) whose PVBs disappeared. In the other 36 athletes who were disqualified from doing sport this was due to the frequency and/or complexity of their arrhythmias; 23 showed a reduction in arrhythmias in 3–12 months (from 46 ± 42 to 28 ± 11 PVBs, from 8 ± 10 to 3 ± 3 ventricular pairs, and from 3.6 ± 6 to 1 ± 1 unsustained ventricular tachyarrhythmia; $P = .05$) and they were readmitted to competition. Of the other 13 athletes with persistent arrhythmias, six of them were successfully treated for their arrhythmias and they were allowed to return to their competitive sports during the follow-up time of 7.4 ± 5 years	5011
Matelot et al.[30]	2015	Arrhythmia	Echocardiogram in repose, maximal effort test, inclination test and 24 h-Holter.	Deep bradycardia in endurance athletes is not associated with increases in the number of incidents of cardiac arrhythmias or hypotensive susceptibility compared to endurance athletes without bradycardia or sedentary subjects.	46
Brugger et al.[14]	2013	Arrhythmia	Years of aerobic training.	No differences were found between groups, ages, blood pressures or diastolic function. The maximum left atrial volume and the conduction volume index increase significantly from the group of low level training to the high level training group, unlike the difference in the parameters of echography and stain following, in which the voltage of the pump and the voltage of the conduction, where there was no significant difference. The average signal of the P wave increased from the group of low level training to the high level training group. Four episodes of unsustained atrial fibrillation were recorded in a runner of the high level training group.	95
Heidbuchel et al.[15]	2006	Atrial fibrillation	Years of aerobic training.	31 (23%) of patients regularly practiced endurance sports before undergoing ablation, and 19 (14%) of them continued their regular sports activity. A history of endurance sport activity is a significant risk factor after ablation for atrial fibrosis (AF) (multivariate HR 1.96 (1.19–3.22, $P < .01$, and multivariate HR 1.81 (1.10–2.98), $P = .02$). Continuing endurance sports activity after ablation also showed a tendency to increase the risk of developing AF in spite of the fact that the sample was small. A 10–11% increased risk of developing AF is estimated per hour of sport per week before and after an ablation ($P < .01$ for both).	137

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Table 4 (*Continued*)

Authors	Year	Consequence in the heart	Methods	Results	*n*
Molina et al.[16]	2008	Atrial fibrillation	Years of aerobic training.	The group of marathon runners showed a higher risk of presenting isolated atrial fibrillation (HR: 7.38, CI: 95%: 1.10–49.63; $P = .040$).	557
Lindsay et al.[31]	2007	Cardiac fibrosis and anatomical changes.	Echocardiographic study of left ventricle mass, systolic and diastolic function. Collagen type 1 Carboxy-terminal propeptide (1CCTP), collagen type 1 Carboxy-terminal telopepetide (1CCTT) and matrix type 1 metal proteinase tissue inhibitor (MT1M) were measured as collagen synthesis, degradation and degradation inhibition markers, respectively.	The veteran athletes had significantly larger left ventricle dimensions and calculated mass index (LVCMI). Diastolic and systolic functions were normal. 1CCTP (259 vs 166 μg/l, $P > .001$), 1CCTT (5.4 vs 2.9 μg/l, $P < .001$) and MT1 M (350 vs 253 ng/ml, $P = .01$) were higher in the athletes. IMTM-1 was higher in athletes with left ventricle hypertrophy.	90
Pellicia et al.[32]	2010	Arrhythmia	Intense uninterrupted endurance training over a long period of time.	During the period of training and competition no cardiac event or new diagnosis of cardiomyopathy occurred in the 114 Olympic athletes. The overall systolic functioning of the left ventricle remained unchanged (ejection fraction: 62 ± 5 to $63 \pm 5\%$). Left ventricle volume (142 ± 26 ml to 144 ± 25 ml; $P = .52$) and left ventricle mass index (109 ± 21 g/m^2 to 110 ± 22 g/m^2; $P = .74$) remained unchanged, and left ventricle filling patterns remained within normal limits, although there was a slight increase in the size of the left ventricle (37.8 ± 3.7 mm to 38.9 ± 3.2; $P < .001$).	114
Wilhelm et al.[33]	2012	Cardiac fibrosis and anatomical changes.	Subjects who had run marathons.	Group 2 showed a significant left ventricle concentric increase in comparison with athletes in general. There were no significant differences between the groups in terms of the diastolic and systolic functions of the right ventricle.	122

PVC: premature ventricular complexes; USVT: unsustained ventricular tachyarrhythmia; PVB: premature ventricular beats; AF: atrial fibrosis; LVCMI: left ventricle calculated mass index; 1CCTP: type 1 collagen carboxy-terminal propeptide; 1CCTT: type 1 collagen carboxy-terminal telopeptide; MT1M: matrix type 1 metalloproteinase; HR: hazard ratio.

how long they had been training for endurance sports to observe the relationship of this kind of training with the risk of developing atrial fibrillation. This study showed that there is an association between length of training time and increased risk of developing atrial fibrillation. Moreover, Molina et al.[16] studied the sports behaviour of 252 men to look for an association between the duration of training and increased risk of developing atrial fibrillation compared to a group of healthy sedentary subjects. This study found an increased risk of developing atrial fibrillation in subjects who practice long endurance sports such as the Marathon.

Cardiac fibrosis and anatomical changes

Cardiac fibrosis refers to endomyocardial inflammation with fibrotic thickening of parts of the heart.[31] On the other hand, anatomical changes are intrinsic to sport and are known as ''athlete's heart''.[9,26] In the Spanish literature, cardiac fibrosis is referred to as ''fibrosis cardíaca''.

Lindsay et al.[31] examined 90 subjects non-invasively (see Table 4) for the presence of cardiac fibrosis, comparing groups of veteran athletes with healthy sedentary subjects. They found significant differences between these two groups regarding the presence of cardiac fibrosis. On the other

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hand, Wilhelm et al.[33] compared a group of marathon runners to a group of physically active subjects who did not run or compete in marathon races. The aim was to detect the impact of running on cardiac remodelling. At the end of the study the researchers found a concentric increase in the left ventricle in the group of marathon runners, although with no alteration in diastolic or systolic pressure.

Discussion

In connection with the aim of this review, the great majority of papers found showed the development of ventricular arrhythmia associated with the prolonged practice of endurance sports.[14–16,25,26,28–37] Secondly, different types of ventricular and supraventricular arrhythmia were found (tachyarrhythmia),[25,26,29,31] bradyarrhythmia,[30,38] atrial fibrillation[14–16,28,39] and arrhythmia.[25,28,30,33,40] Lastly, cardiac fibrosis was found[31] as well as anatomical changes in the heart.[14,25,26,30,33,41]

Although the studies which associate arrhythmia with endurance exercise may be divided into three categories, few studies have focussed solely on runners.[14,16,31,33] Many studies cover endurance sports in general, or a wide range of sports without differentiating between their types.[15,26,28,29,32] Additionally, the evaluation of morphological changes and damage requires a large number of tools to make them visible.[14,25,26,29–33]

9 of the 12 studies consulted determine the chronic effect of long duration exercise on arrhythmia, i.e., how long duration exercise leads to the appearance of different types of arrhythmia.[14–16,26,28,29,31,33] Of these nine studies, only four centred on marathon runners and runners.[14,16,31,33] Two studied a wide range of sports (basketball, football, skiing and running, etc.),[15,32] and three studied sports other than running.[26,28,29]

The other three studies centre on the acute effects of de-training and re-training,[25] monitoring athletes with ventricular arrhythmias[27] and the relationship between bradycardia and an increase in ventricular arrhythmia.[30]

After the systematic review it is also important to state that the majority of papers evaluate the effects and consequences of practicing this sport for years, while only one paper was a longitudinal study. This study observed how subjects with atrial or ventricular arrhythmia behaved and felt during a certain period of time.[27]

In connection with the secondary objective of this review, the majority of the papers studied were found not to mention problems or limitations when training or competing. Nevertheless, one paper which followed-up a large population of athletes reports that one subject died due to a long history of cardiomyopathy. However, all of the subjects lived during the follow-up period (70 ± 25 months).[27] One study which evaluated 367 athletes found that 36 of them were temporarily excluded from competing by the frequency and complexity of their arrhythmia. But in this group of excluded athletes, 23 of them were able to reduce this complexity and frequency over a period of from 3 to 12 months to an acceptable level that allowed them to return to competing. On the other hand, of the 13 remaining athletes who were still in a dangerous range, six were treated successfully and so were allowed to return to competition.[29]

Four of the papers studied state that the range of adaptations which arise due to the constant practice of endurance exercise do not cause limitations in athletes and that no such limitation was detected.[14,16,25,32] One of the papers investigated the effect of de-training and then re-training subjects with ventricular arrhythmia, and found that at the moment of ceasing to train a reduction in ventricular arrhythmia occurred, and that when training resumed it increased to an even higher level than it was at first. However, after one year of training the levels fell in comparison with the basic state.[25]

On the other hand, some studies mention damage and the possible consequences of systematically practicing endurance exercise.[16,28] However, in this case there is no prolonged follow-up of the athletes to observe their behaviour over years of training and competition.

Conclusion

According to the bibliographical review there is evidence showing that a range of structural and morphological adaptations of the cardiac muscle arise due to the systematic practice of endurance sports. More specifically, there is evidence showing harmful changes and adaptations in athletes. Nevertheless, it is still relatively unclear due to a lack of scientific evidence whether these changes tend to occur in endurance sports in general.

Due to the above consideration, more research is required after competitions. This would make it possible to learn the mechanisms which cause possible adaptations and/or negative alterations in athletes. As a result of this, carrying out long-term follow-ups to analyse the complexity, limitations and restrictions which may cause negative effects or alterations at a cardiovascular level due to participation in endurance sports is an alternative to prevent health and quality of life risks for these athletes.

Practical applications

In practical terms, systematic long duration exercise is a way of improving health and performance for competition. Notwithstanding this, certain factors must be taken into account:

Although the studies do not prove that greater complications arise in trained subjects with ventricular arrhythmia, if we wish to determine whether or not they continue in training or competitions we can use guides such as the ''Italian cardiological guidelines''[42] which offer ranges in which to consider whether athletes do so. Only subjects with 10 or fewer premature ventricular beats are accepted in this case).

A 24-hour Holter can be used to visualise the Electrocardiogram (ECG) of the subject, which will make it possible to monitor and quantify the degree of complexity and frequency of ventricular arrhythmia. Electrophysiological studies also allow us to study the electrical or electrophysiological pathology of individuals.

Lastly, and with the aim of observing how they adapt and the consequences of systematic practice, the researchers

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were invited to determine groups of similar activities, to then carry out a prolonged follow-up of the athletes.

Conflict of interests

Authors declare that they don't have any conflict of interests.

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