BRADYCARDIA IN ATHLETES: DOES THE TYPE OF SPORT MAKE ANY DIFFERENCE? – A SYSTEMATIC REVIEW

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Descritores: Esportes; Atletas; Bradicardia; Fisiologia cardiovascular; Sistema nervoso autônomo.

RESUMEN

La bradicardia en atletas puede variar de moderada a grave, y los factores que contribuyen para la reducción de la frecuencia cardíaca son complejos. Los estudios que investigan los mecanismos asociados a la bradicardia son controvertidos y, posiblemente, vinculados al tipo de ejercicio. Se realizó una investigación bibliográfica sistemática para discutir los mecanismos de bradicardia en atletas de diferentes modalidades deportivas. Las bases de datos consultadas fueron Pubmed (MEDLINE), Clinical Trials, Cochrane, Scopus, Web of Science, SciELO, Sport Discus y PEDro. Se incluyeron artículos en inglés, que avaliaram atletas de diferentes modalidades deportivas, publicados hasta enero de 2019. Se encontraron ciento noventa y tres artículos y diez preencheram os critérios de inclusão, perfazendo 1.549 homens e mulheres atletas de diversas modalidades esportivas. A frequência cardíaca em repouso e a estrutura cardíaca foram estudadas em associação com a modalidade esportiva, por variabilidade da frequência cardíaca, eletrocardiograma, ecaecardiograma e bloqueio farmacológico. Os estudos sugerem que a redução da frequência cardíaca em repouso não é explicada apenas pelo aumento da modulação vagal, mas também por alterações da estrutura cardíaca. De acordo com os estudos, diferentes esportes parecem produzir diferentes respostas cardíacas e a bradicardia encontrada em atletas pode ser explicada por mecanismos não autonômicos e autonômicos. Este achado parece depender do tipo de esforço ou modalidade esportiva praticada. No entanto, o mecanismo envolvido na redução da frequência cardíaca em cada modalidade esportiva ainda não está claro.

Keywords: Sports; Athletes; Bradycardia; Cardiovascular physiology; Autonomic nervous system.
INTRODUCTION

In the field of sports, athletes commonly have a resting heart rate below 60 bpm, a chronic adaptation known as athlete's bradycardia. Functionally, bradycardia induced by physical training increases ventricular filling time and, consequently, contributes to increase systolic volume in resting and physical exercise conditions. However, despite an assumed consensus in the literature as to the athlete's brady cardiac phenomenon induced by physical training, as well as its functionalities, the mechanisms responsible for such adaptation are still current targets of debate. In this respect, it has been elegantly demonstrated through pharmacological blockade that athlete bradycardia can be the result of a change in cardiac autonomic balance. Specifically, these studies suggest that increased parasympathetic activity and reduced sympathetic activity of the heart are responsible for the reduction in the frequency of sinus node shots. On the other hand, the autonomic hypothesis has been refuted in some other studies, suggesting that the main adaptation responsible for athlete's bradycardia is the intrinsic reduction of the sinus node firing frequency.

Despite that, since different training models and professions may result in different forms of cardiovascular overload, and considering that physiological chronic adaptations induced by physical training are specific to the type of stimuli offered during successive training sessions, it is plausible to hypothesize that the mechanisms responsible for bradycardia are dependent on the athletic modality. In fact, functional and structural differences have been reported in hearts of athletes involved in different athletic modalities.

Therefore, given the controversy about the mechanisms primarily responsible for athlete's bradycardia and the possibility of such divergences being driven by the athletic modality, the objective of the present study is to conduct a systematic review of studies that have investigated the mechanisms responsible for bradycardia in athletes involved in different athletic modalities. To the best of our knowledge, this is the first study to carry out a review and critical analysis of the literature on the subject.

METHODS

The PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) were adopted in the systematic review process. Two reviewers (MM, LG) conducted the search independently and a senior reviewer (MG) was to be invited to resolve any divergence, which was not necessary. The agreement analysis between the two independent reviewers was evaluated using the Kappa index with a 95% confidence interval.

We searched in the following databases: Pubmed (MEDLINE), Clinical Trials, Scopus, Web of Science, SciELO (Scientific Electronic Library), Sport Discus and PEDro, up to January 2019, in view of the time needed for data extraction, analysis of results and writing of the manuscript. The Cochrane database was consulted to verify the existence of revisions about this topic.

The following keywords were used for the searches: “sports AND nervous system; parasympathetic AND sympathetic”; “sports AND exercise” AND “bradycardia”; “athletes AND nervous system AND parasympathetic AND sympathetic”; “athletes AND bradycardia”; “exercise AND nervous system AND parasympathetic AND sympathetic”; “exercise AND bradycardia”. The descriptors used were extracted from the PUBMED (Medical Subjects Headings- MeSH) and DeCS (Health Sciences Descriptors) databases.

References were added to a reference manager for collection and subsequent analysis of titles and abstracts. After careful reading of the articles, studies published in English that directly or indirectly evaluated the possible mechanisms associated with sinus bradycardia of athletes involved in different sports modalities were included in the review. Consequently, we did not include studies in a language other than English, nor studies with children, adolescents or master athletes; animals; or evaluating only non-athletes or sedentary individuals without comparison to athletes; with unhealthy subjects or that did not specify the sport modality practiced by the volunteers. Information about age, sport modality, form of bradycardia evaluation and proposed mechanisms for bradycardia were extracted from the articles and organized as a table.

RESULTS

The concordance analysis among the reviewers showed a Kappa value of 0.85, which indicates almost perfect agreement. We found 193 articles in the databases referred above. Of these, 155 were excluded based on their title and abstract and 10 papers were duplicate articles. Of the 28 remaining manuscripts, two were in a language other than English, nine were crosstalk, viewpoints or conference papers, two did not specify the sport evaluated and five evaluated adolescents or children. However, these, although excluded from the main analysis, provided subsidies for a better discussion of the articles. Thus, 10 articles were selected for this review, based on the inclusion criteria. Figure 1 describes the article selection process.

The total sample consisted of 1549 athletes aged 17-36 years, who practiced different athletic modalities such as a) cycling (71 athletes or 4.6%), b) running (13 athletes or 0.8%), c) ball sports and martial arts (1036 athletes or 66.9%), d) gymnastics (111 athletes or 7.2%), and e) resistance sports (318 athletes or 20.5%).

Three studies compared athletes of different athletic modalities. Most of the studies evaluated men (8 studies) and two did not specify the sex of the individuals evaluated. Two studies evaluated women; however, none of them reported menstrual cycle phase control, which may influence heart rate variability and resting heart rate itself.

The studies used pharmacological blockade, electrocardiogram, echocardiogram, and heart rate variability to evaluate mechanisms that could explain heart rate reduction after an exercise training period. The descriptive analysis of the articles is summarized in Tables 1 and 2 and they are organized in order of the year of publication.

DISCUSSION

The main findings of this study are that the effect of physical training on athletes' heart rhythm, in resting condition, does not strongly suggest...
the possible influence of the type of athletic modality as an intervening variable in training-related bradycardia, autonomic or non-autonomic. On the other hand, only two recently published studies\(^1^7,1^9\) showed that athletic modality (running, cycling and wrestlers) may possibly influence the type of adaptation that occurs in the physiological mechanisms of cardiac control in athletes in resting condition.

This review shows that six (60%) articles selected evaluated cyclists (71 cyclists) and observed that bradycardia may be explained by non-autonomic mechanisms. Four of these studies\(^2,1^0,2^0,2^3\) evaluated bradycardia and its possible mechanisms through the heart rate variability method at rest. These studies suggest that heart rate reduction is not entirely dependent on autonomic control or specifically on increased vagal activity at rest. Molina et al.\(^1^0\) compared a group of professional cyclists with a group of non-athlete physically trained individuals. Although the group of cyclists presented bradycardia in resting condition, compared to the group of physically trained individuals, the authors did not see a difference in cardiac autonomic control from heart rate variability in the supine and orthostatic positions, as well as after active postural change (orthostatic test). The authors suggest that the bradycardia observed in cyclists seems to be related to intrinsic adaptation of the sinus node.
In addition, Martinelli et al.\(^2\) argues that bradycardia in cyclists may be related to intrinsic adaptations of the sinus node. This finding was also observed by Bonaduce et al.,\(^{23}\) who concludes that intrinsic heart rate reduction seems to determine bradycardia in cyclist athletes. In this regard, two papers raise the hypothesis of a possible relation between sport modality and bradycardia mechanism, evaluating athletes at rest and post-exertion.\(^{15,24}\) Interestingly, these studies used heart rate variability for evaluation.

To try to explain the possible physiological mechanisms involved in the bradycardia of athletes practicing different athletic modalities, Azevedo et al.\(^{17}\) compared cyclists and runners, using double pharmacological blockade and echocardiography. They concluded that the magnitude of bradycardia observed in cyclists could be explained by intrinsic heart rate reduction, possibly associated with concentric and eccentric hypertrophy observed in the walls of the heart. This hypothesis is supported by studies such as the one by Lewis et al.,\(^{16}\) who observe intrinsic heart rate reduction in cyclists and speculate that dilation of the cardiac chamber would cause a stretch of the sinoatrial node, leading to its inhibition as an acute response. Still in this regard, the study by Azevedo et al.\(^{17}\) showed that the athletic modality (running or cycling) could determine the magnitude of the vagal effect and the intrinsic heart rate. The article suggests that vagal modulation could coexist with changes in the sinus node in highly trained runners and the opposite could be applied to professional cyclists.

In studies evaluating cardiac structure, there is a concern with the type of the training and dimensions of the heart. Research using the echocardiogram method\(^{25-28}\) to investigate the effects of physical training on the heart, ventricular wall and mass dimensions in athletes who practiced diverse sports demonstrated that athletes who engage in sports with greater overload to heart resistance such as, for example, exercise that consists of predominately isometric work (e.g. weight lifting) develop concentric hypertrophy in the heart. In contrast, athletes who perform sports with greater volume overload, such as isometric exercises (e.g. marathon runners), develop an increase of all cardiac chambers.\(^{16}\) Therefore, it is plausible to conjecture that the type training overload (volume and / or resistance) and the position in which the athlete usually trains are variables that may influence the type of cardiac adaptation, that is, different autonomic or non-autonomic adaptations may result from different training overloads and training phases. The study by Kaur\(^{19}\) further states that training involving mostly strength results in changes in cardiac dimensions. This study reveals a significant increase of the interventricular and posterior septal walls in wrestlers athletes.

Still on cardiac structure, Zakynthinos et al.\(^{22}\) found that athletes who practiced water polo had dilatation of the cardiac chambers, combined with hypertrophy, but preservation of normal left ventricular function. Belotserkovskii et al.\(^{18}\) suggest that athletic activity has a distinct effect on the morphology and function of the cardiovascular system.

In the studies by Miki, Nishimura and Urhausen,\(^{30-32}\) evaluating athletic modalities with exercises causing both volume and resistance overload, both left ventricular dilation and hypertrophy were observed. However, no association analysis was performed with cardiac rhythm or bradycardia mechanism. This could be pointed out as a possible limitation of studies for the evaluation of cardiac adaptations to exercise.

The findings of this research suggest that the bradycardia found in athletes can be explained by non-autonomic and autonomic mechanisms, depending on the type of effort or athletic modality. Still in this regard, another factor that could influence cardiac adaptation is the training phase. The recent study by Azevedo et al.,\(^{21}\) published in 2016, when evaluating professional cyclists, showed differences between the transitional and the competitive phases of training, in addition to their influence on resting heart rate. Thus, there is evidence that, in addition to athletic modality, the training phase may modify the magnitude of heart rate control mechanisms, even if temporarily.

**CONCLUSION**

Studies show that bradycardia may be related to not only athletic modality but also type of training. The findings of the present study suggest that athletic modality and type of training produce different cardiac adaptations that result in structural alterations of diverse magnitudes, which can influence the sinusal rhythm.

The mechanisms involved in each athletic modality are still inconclusive and, although there is some research on the mechanisms of bradycardia, this review reinforces the need for other studies on the subject. Our findings suggest that new studies addressed to better understand the mechanism of athlete's bradycardia should compose more homogenic groups of volunteers, in terms of the sport modality, type and phase of exercise training. In other words, it seems that the comparison of athletes vs non-athletes it is a comprehensive approach.

The understanding of the possible mechanisms and their relationship with the athletic modality or the type of training can help health professionals to understand the phenomenon and its relationship with sports, mainly in the evaluation and monitoring of professional athletes.

**Table 2. Descriptive results (continued).**

| Authors | Age (years) | Sex | Athletic modality (Groups) | Bradycardia Evaluation form | Findings |
|---------|-------------|-----|---------------------------|----------------------------|---------|
| belotserkovskii et al, 2009(16) | 17-29 | unspecified | 527 basketball, water polo, boxing and soccer athletes | ECG Polycardiography Echocardiogram | Sport activity alters the functional and morphological cardiovascular characteristics. Thus, athletes present complex characteristics to maintain cardiovascular function. |
| Kaur, 2013(19) | 18-24 | unspecified | 30 wrestlers athletes 30 healthy individuals | ECG Echocardiogram | Resistance-predominant sports result in changes in cardiac dimensions (left ventricle concentric hypertrophy). |
| Molina et al, 2013(10) | 22-31 | male | 12 cyclists 11 healthy individuals | ECG HRV cardiopulmonary testing | Bradycardia is not dependent on autonomic modulation, rather, it suggests intrinsic changes. |
| Azevedo et al, 2014(17) | 22-36 | male | 13 runners 11 cyclists | ECG Pharmacological blockade Echocardiogram | Bradycardia observed in cyclists seems to be associated with reduction on sinus node frequency and structural cardiac changes (septal and posterior wall). On the other hand, bradycardia observed in runners are related to higher parasympathetic. |
| Azevedo et al, 2016(21) | 25-27 | male | 10 cyclists | ECG Pharmacological blockade Echocardiogram | Bradycardia can be explained by autonomic and non-autonomic mechanisms according to the training phase |

ECG: electrocardiogram.
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