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The slowing pulse: a history of research on training-induced resting bradycardia

Marcin Grandys[1], Miroslaw Wozniak[1,2], Jerzy A. Zoladz[1]

[1] Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Kraków, Poland
[2] Department of Angiology, Jagiellonian University Medical College, Kraków, Poland

Corresponding author: Jerzy A. Zoladz, Ph.D., D.Sc.
Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences
Jagiellonian University Medical College
ul. Skawińska 8, 31-066 Kraków, Poland
Phone: +48 12 433 28 53; E-mail: j.zoladz@uj.edu.pl

Abstract: Training-induced resting bradycardia is characteristic of endurance athletes, but it also often occurs in less trained, physically active people. It is commonly believed that slowing of the heart rate is a positive sign of systemic adaptation to physical training, but severe bradycardia phenomenon (below 40 beats per minute) could be a matter of concern for athletes' health. In this review, we present the fascinating history of research into this one of the most fundamental adaptive responses of the cardiovascular system to exercise training, which teaches us an important scientific truth — that nothing that has not been questioned has truly been proven. In the context of training-induced resting bradycardia, this refers to many aspects of the phenomenon, from its name and meaning to its physiological mechanisms.

Keywords: heart rate, athletes, autonomic control, endurance training, hyperpolarization-activated cyclic nucleotide-gated channel 4.

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Introduction

Exercise training-induced adaptive responses are associated with changes in both the structure and function of many tissues and organs (see e.g. [1–6]). The changes that are most readily observed and easiest to measure are those related to the cardiovascular system. This is also evidenced by the fact that one of the earliest documented training-induced changes is cardiac enlargement [7] and resting bradycardia [8].

Training-induced resting bradycardia is a form of sinus bradycardia that is defined as a heart rate lower than 60 bpm (beats per minute) [9], but a lower limit for sinus bradycardia of 50 bpm

is also often used in various studies (see e.g. [10, 11]). It is worth noting that the resting heart rate (RHR) in endurance trained athletes can often be lower than 40 bpm and the lowest RHR ever recorded in athlete was 17 bpm during sleep [12]. It should be also noted, that sinus bradycardia is part of the broader phenomenon of bradycardia, which is defined differently depending on its nature and origin (see Table 1).

Table 1. Terms related to bradycardia phenomenon.

Bradyarrhythmias	*"Any disturbance of cardiac rhythm in which the mean heart rate is <than 60 beats per minute"* Mooren [13]
Sinus node dysfunction	*"Sinus node dysfunction (SND) encompasses a broad array of disturbances of sinus node or sinoatrial function that result in chronic or intermittent periods of inappropriate slow or fast heart beating".* Benditt *et al.* [11]
Sinus bradycardia (resting bradycardia)	*"If the heart rate is lower than 60 beats/min but originates from the sinus node and is normally conducted, it is termed sinus bradycardia"* Smith and Fernhall [9]
Relative bradycardia	*"The inability of the heart to respond to shock with tachycardia has been described as relative bradycardia, paradoxical bradycardia, or absence of tachycardic response".* Demetriades *et al.* [14]
Exercise bradycardia	*"…a decreased heart rate at any absolute level of submaximal oxygen uptake…"* Blomqvist and Saltin [15]
Reversed bradycardia	*"…elevation (>10%) in resting heart rate (HR) with training overstress, (…) that negatively affects running performance"* Dressendorfer *et al.* [16]
Diving bradycardia	*"Diving bradycardia is a primordial oxygen-conserving reflex by which the heart rate of air-breathing vertebrates, including humans, slows down in response to water immersion"* Vega *et al.* [17]
Chronotropic incompetence	*"Inappropriate heart rate response (i.e. chronotropic incompetence, inappropriate sinus tachycardia) such as might be observed during or after physical exertion, while at rest, or during activities of daily living, has been the most recently recognized component of the many features of sinus node dysfunction"* Benditt *et al.* [11]

Discovery of the phenomenon of the training-induced resting bradycardia is commonly referred to the work of eminent Boston cardiologist Paul D. White dated back to 1942 (see [18, 19]), but Pedoe [20] also pointed to A.V. Hill's lectures from 1927 as the first mention of lower RHR as a symptom of training in athletes. It seems that the earliest publication by Fendick *et al.* [21] with the case of severe bradycardia (28 bpm) in "*person who otherwise enjoy good health*" is forgotten. The same applies to the great work by Buchanan who recorded in 1909 by means of electrocardiography a pulse rate of 44 bpm in an athlete (see [8, 22]) and to the paper by Hoogerwerf [23] who observed a record (at that time) low pulse rate of 30 bpm in an Olympic athlete. Evidence

that training-induced resting bradycardia was already a well-known phenomenon in those years, comes also from the paper by Cotton [24] in which he summarized the experiments on RHR that had appeared in the literature since the beginning of the twentieth century concluding that a slow heart rate is a feature of highly trained athletes.

Training-induced resting bradycardia is a very common not only in highly trained athletes, but also in physically active individuals [25]. It has been reported that its prevalence is up to 90% in athlete populations [26], but we are aware of a study in which 100% of subjects (20 endurance trained track and field and cross-country female runners) showed sinus bradycardia (see Table 2). From the data presented in Table 2 it can be argued that occurrence of resting bradycardia is the higher the higher the endurance component of training. This is consistent with findings that most cases of profound resting bradycardia involve endurance athletes with long training experience (see [10, 12]). This type of athletes is also most likely to experience adverse effects related to a signs of sick sinus syndrome [27].

Table 2. Prevalence of sinus bradycardia in different populations of athletes.

Athletes	n	Prevalence	Reference
Endurance track and field or cross-country runners (women)	20	100%	George *et al.* [28]
Highly trained endurance athletes	13	77%	Azevedo *et al.* [29]
Highly trained long-distance runners	37	65%*	Ector *et al.* [10]
Triathletes	22	68%	Climstein *et al.* [30]
Elite soccer players (women)	81	65%	Morrison *et al.* [31]
Road cyclists	11	55%	Azevedo *et al.* [29]
Soccer players	2484	54%	Huttin *et al.* [32]
College athletes (men and women)	1229	36%	Bessem *et al.* [33]

* bradycardia defined as RHR <50 bpm. Data are for men, unless otherwise noted.

It is obvious that more than 100 years of research on training-induced resting bradycardia have given us insight into the characteristics of this phenomenon, but answers to some of its aspects are still being sought. In this review we will focus on the fascinating history of research on the training-induced resting bradycardia, starting with ancient observations of pulse and ending with contemporary discoveries concerning the mechanisms of this phenomenon.

Methods

In order to identify the broadest possible list of publications related to history of research on training-induced resting bradycardia, a literature search was performed using the PubMed/MEDLINE database with the following terms: "bradycardia" and "exercise" with no restriction regarding publication year. To find the earliest papers that deal with the bradycardia phenomenon, PubMed/Medline database was also searched within the publication years 1895–1960 using only the keyword "bradycardia" (1895 was the earliest year in which records related to this term appeared). At

the end, this database was searched using the keyword "pulse" within the publication years 1815–1895 (again, 1815 was the earliest year in which records for the term "pulse" occurred). In addition, a manual electronic search was conducted in the following databases: Google Scholar and Wellcome Collection (https://wellcomecollection.org), when a specific historical source related to the topic of pulse measurement was sought (e.g. "Sir John Floyer" or "The writings of Galen").

History of pulse measurement

Pulse palpation has been a fundamental part of medical examination since ancient times. According to Hajar [34], the earliest reference indicating that a heartbeat was considered a major vital sign and that it could be palpated dates back to around 2600 BC. In the ancient Egyptian, Chinese and Indian medical systems, examination of the pulse (i.e. its rate, strength, rhythm, character) has become a highly sophisticated method of assessing a patient's health, taking the form of an advanced medical art [35, 36]. The interest in studying the pulse was continued in ancient Greek medicine by all the famous physicians, including Hippocrates, Praxagoras, Herophilus, Erasistratus and then by Galen who described his findings about the pulse in eighteen essays (see [35–37]). These works dealt with the classification of pulse types, the causes of its formation, diagnostic possibilities and the prognostic value of the pulse. Galen's achievements in circulatory physiology influenced all of medieval medicine and remained largely relevant until the mid-17th century, when William Harvey described the circulatory system and discovered the principle of blood circulation in the human body [38]. In turn, the works of Iosephus Struthius (see [37, 39]), Santorio Santori (see [40]), Johannes Kepler (see [41]), and especially Sir John Floyer [42]), who invented a "phisician's pulse-watch" to measure it for sixty seconds in the way we determine pulse rate today, contributed to the expansion of knowledge related to pulse measurement.

Introducing the term "bradycardia"

The first documented description of the extremely slow pulse rate was presented in 1717 by Marcus Gerbezius, a famous Slovenian physician (see [43]), who found it in a patient with clinical signs of bradycardia (between 20–25 bpm). For the next hundred years, cases of very slow pulse rate, manifested also by syncope and seizures, were regularly described by several eminent physicians of the time — Morgagni in 1761, Spens in 1793, Adams in 1827; Burnett in 1827 and Stokes in 1846 (see in [43–46]). Taking this into account, it is becoming clear that referring to the condition of sudden, transient loss of consciousness due to profound bradycardia as Adams-Stokes syndrome ignores at least some of the researchers who contributed to its description. Despite the growth of interest in the phenomenon of slow pulse rate in the 18th and 19th centuries, it was not until 1888 that the term we now know as "bradycardia" was first used by Fritz Grob and Hermann Eichhorst [47] to describe a low pulse rate. Grob found 82 cases of slow pulse rate out of 3578 patients (2.29%) during a three-year follow-up at the Zurich Medical Clinic and defined the presence of bradycardia in those patients whose pulse rate did not reach 60 beats per minute at least once during consecutive days of hospitalization [47]. He wrote: "*Auf Vorschlag des Herrn Prof. Eichhorst wählte ich der Kürze halber für das in Frage kommende Phänomen die Bezeichnung, 'Bradycardie', als Gegensatz der Tachycardie*" (Eng. "*As suggested by Prof. Eichhorst, for the sake of brevity I have chosen the term 'bradycardia' for the phenomenon in question, as the opposite of tachycardia*").

The correctness of the term proposed by Grob was questioned just a few years later. Landois [48] claimed that the prefix "brady" refers to the Greek word "*βραδυς*" (phonetically "bradys") meaning "slow", and should refer to the word rare, from the Greek "*σπανιος*" (phonetically "spanios"), therefore the correct term would be "spanicardia". In the same way, it was argued that the correct term for a frequent heart rate is "pyknocardia" instead of "tachycardia", because Greek word "*πυκνος*" (phonetically "pyknos") means "frequent" and "*ταχυς*" (phonetically "tahys") means "quick" [48]. However, the previously used phrases "bradycardia" and "tachycardia" have spread so much in the scientific literature that even these legitimate proposals to restore the proper nomenclature to describe infrequent and frequent heart rhythms have not entered the common usage.

Resting bradycardia as a manifestation of training adaptation

At that time pulse rate also became a subject of interest in the field of exercise and sports-related research. In 1899 Fendick *et al.* [21], in their case report related to extreme bradycardia in older patient, stated that a low pulse rate may be the normal condition in healthy persons and reported about "*a man whose pulse for some time remained about 28 bpm.*" At the same time, a work was published that documented (for the first time to our knowledge) cardiac training adaptation to athletic training by demonstrating enlargement of the heart in trained cross-country skiers [7]. Another important step in the study of circulatory responses to exercise was the study of Florence Buchanan on the resting and exercise-induced changes in the pulse rate by means of electrocardiogram [8]. She demonstrated that the pulse rate vary between 44 and 80 per min in young "*chiefly athletic men*" and was able to demonstrate that the pulse rate is increased at the very beginning of exercise. It is interesting that Buchanan provided data for the seminal paper of Krogh and Linhard [22] who wrote in that paper: "*We have never been able to count the pulse during the first seconds of bicycling on the ergometer, but it is known from the investigations of Miss Buchanan' that the pulse rate is increased from the first beat occurring after the beginning of work. Miss Buchanan has shown us the very great kindness to take some electrocardiograms on subjects starting work on a stationary tricycle.*" It became clear for the scientists interested in exercise physiology that examination of the pulse rate is the easiest way to test the effects of muscular exercise in humans [49]. These authors stressed that pulse rate should be measured before, during and after exercise. They also concluded that the trained man has a slower pulse rate at rest and recovers faster after exercise and presented an original thesis that fast return of pulse rate to its resting frequency appears to be the best indicator of adaptation to training.

Early studies on training-induced resting bradycardia in athletes

A manifestation of the growing interest in human adaptation to physical training was the scientific project under the name of "*Stadionlaboratorium*" during Olympic Games in Amsterdam in 1928. A group of more that 20 scientists from different countries and different disciplines under direction of Prof. Buytendijk, had great opportunity to investigate the Olympic athletes, also in research related to "sports bradycardia" phenomenon. Bramwell and Ellis [50] took extensive examinations on pulse rate and blood pressure whereas Hoogerwerf [23] performed electrocardiogram measurements. Based on their findings, it can be postulated that Bramwell and Ellis [50] were the first to attempt to organize runners according to the increasing duration of their running events in

relation to their resting pulse rate — from sprinters with the highest average pulse rate of 66 bpm to marathon runners with an average lowest pulse rate of 58 bpm. On the other hand, Hoogerwerf [23], in addition to describing the characteristics of ECG recording in athletes, showed that the average heart rate in Olympic athletes was 50 bpm, with the lowest value ever presented in athletes — being 30 bpm. The result of increasing knowledge related to the cardiovascular response to exercise and training was the paper by Cotton [24] who summarized the experiments on RHR that appeared in the literature since the beginning of XX century and provided his own results that both lead him to conclusion that slow pulse rate is characteristic of highly trained athletes. He conducted his own measurements on champion swimmers (including world record holders and Olympic medalists) and reported that the baseline pulse rate was 44.6 bpm, the lowest value in the group to date. He also tried to establish the relation between the slowing of the pulse rate and the athletic training background and with the conclusion: "*it is striking how the basal pulse rate invariably decreases (…) with increasing intensity and duration of athletic training*," he was the first to describe this relationship.

Studies on mechanisms of training-induced resting bradycardia

The 1920s and 1930s were also the beginning of research into the mechanisms of sports bradycardia. Of great importance for the development of knowledge in this field was Herbert Herxheimer, who as early as 1921 studied the vagal effects on heart rate using atropine administration in athletes [51]. Moreover, in a subsequent study, he demonstrated that the transverse diameters of the heart was the highest in athletes participating in long-endurance sports (comparing to boxers, swimmers and middle-distance runners) and concluded that higher heart diameter would lead to higher stroke volume and results in the corresponding resting bradycardia [52]. This was one of the first attempts to explain resting bradycardia in athletes, but since then, efforts to unravel the causes of training-induced bradycardia have dominated the next nearly 100 years of research on the subject. In this time, several underlying mechanisms have been proposed, i.e. (i) cardiac hypertrophy and increased myocardial contractility [51]; (ii) increased parasympathetic tone and decreased adrenergic influence [53]; (iii) decreased responsiveness to beta-adrenergic stimulation and reduction in adrenergic receptor density [54]; (iv) decreased intrinsic heart rate [55]; or (v) combination of these factors [56]. The most comprehensive discussion concerned the significance of the increased parasympathetic regulation and changes in intrinsic pacemaking activity of the sinus node as the main mechanisms of bradycardia. The culmination of this topic can be found in four widely reported and cited scientific debates proposed in the pages of such esteemed physiological journals as *The Journal of Physiology* and *Journal of Applied Physiology (1985)* in the special sections called "CrossTalk proposal & opposing view" and "Point:Counterpoint" series, respectively [57–64]. The most important discoveries confirming both concepts have been published in the last decade — one relates to a downregulation of hyperpolarization-activated cyclic nucleotide-gated channel 4 (HCN4) and related "funny" current (I_f) in athletes [65] and second to an increase in resting activity of rat vagal preganglionic neurons after exercise training [66]. This shows that the full understanding of the phenomenon of training-induced resting bradycardia, which has been known for over 100 years, requires further research. Experiments determining the probable interactions between the parasympathetic and the sinus node activity would be particularly valuable.

Conclusions

The pulse measurement, as the basis for diagnosing bradycardia phenomenon, has a long history dating back to ancient times. Pulse slowing below 60 bpm was defined as "bradycardia" and introduced by Fritz Grob and Hermann Eichhorst in 1888 [47]. This fact was however preceded by observations of low or even very slow pulse rate by various physician over the previous two centuries. From the beginning of the XX century bradycardia began to be documented in an athletes as an effect of physical training. It is interesting that after more than one hundred years of research on the mechanisms of training-induced resting bradycardia, we still don't have a full understanding of this phenomenon. Further studies aimed at investigating the interaction between autonomic signaling and sinus node activity may ultimately clarify the remaining uncertainties.

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Authors contributions

Jerzy A. Zoladz conceived the idea for this review. Marcin Grandys and Miroslaw Wozniak conducted the literature search and wrote the first draft of the manuscript. All authors have read, revised for important intellectual content, approved the final version, and agreed to be accountable for the work.

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