

Commentaries on Viewpoint: Is the resting bradycardia in athletes the result of remodeling of the sinoatrial node rather than high vagal tone?

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CHANGES IN PACEMAKER CELLS DETERMINE RESTING BRADYCARDIA AFTER EXERCISE TRAINING

TO THE EDITOR: Resting bradycardia is an unquestionable marker of exercise training adaptation (1) and has been consistently documented in different species (2, 4). The question is whether this cardiac adaptation is due to increased vagal tone, sympathetic tone reduction, or decrease in intrinsic heart rate. In our investigations, exercise training reduces vagal and sympathetic tone in a similar manner (5), which implies that no change in resting heart rate should be expected. However, we found a reduced resting heart rate, as extensively demonstrated by other investigators. These findings strongly suggest that the bradycardia provoked by exercise training is not simply a result of changes in the vagal and sympathetic balance. In fact, further investigation with double blockade with methylatropine and propranolol showed that intrinsic heart rate was significantly reduced in exercise-training animals compared with untrained animals. Our studies provide no evidence for increased vagal function after exercise training. On the contrary, exercise training virtually decreases vagal function. Baroreflex bradycardia provoked by vasoconstrictor drug, bradycardic responses caused by electrical stimulation of the vagal nerve, and responses to methacholine injection are all depressed after exercise training. These findings lead us to strongly agree with Boyett and collaborators (3) that “resting bradycardia cannot be explained by high vagal tone.” The resting bradycardia is a consequence of pacemaker cell alterations and intrinsic heart rate reduction.

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RESTING BRADYCARDIA IN ATHLETES: COULD THE CURRENT TECHNIQUES LEAD TO MISINTERPRETATION?

TO THE EDITOR: Trained athletes have been reported to have resting bradycardia, which was initially attributed to increases in cardiac vagal tone (2). More recently, this hypothesis has been questioned based on experimental evidences that shifted the focus to the sinoatrial node suggesting a reduction in the intrinsic heart rate (3). In their Viewpoint, Dr. Boyett and

colleagues (1) took a critical look at the two lines of evidence favoring the high cardiac vagal tone hypothesis: 1) little or no change in the intrinsic heart rate in athletes and 2) an increase in heart rate variability in athletes. Although authors suggest that intrinsic remodeling of the sinoatrial node would play a major role in the bradycardia seen in athletes, we believe that the limitations of the currently employed techniques, such as drug-induced autonomic blockade with propranolol and atropine as well as heart rate variability, for indirect measuring cardiac vagal activity, compromised the conclusions and could lead to misassumptions. Therefore, we believe that combining direct recording of the cardiac vagal tone in athletes and/or experimental animals to cellular approaches ranging from enzymes and other cellular mediators to electrophysiological properties of the sinoatrial node cells would help to unravel the precise role played by the parasympathetic activity and by the cells in the sinoatrial node in producing resting bradycardia in trained athletes. However, there is still need for refining the experimental approaches to investigate such mechanisms.

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A STALKING HORSE FOR RESTING HEART RATE IN ATHLETES

TO THE EDITOR: The paper by Boyett et al. (3) makes an argument in a theoretical analysis for a decrease in intrinsic pacemaker rate. This idea is not new. Boyett et al. (3) provide some further “food for thought.” I have some criticisms of this analysis. It is poor science to cross-compare data between the studies in parallel groups 2–11 (Table 1) that are not biologically similar. This is only valid in a longitudinal study such as one in which the conclusion could be different to that of Boyett's. The arguments take no account of changes in the vagal mediated baro-heart rate reflex sensitivity following training in animal and human that also have been shown to be protective against ventricular fibrillation (5). Appraisal of heart rate variability (HVR) is superficial and there is no comment on its vagal dependence. Al-Ani et al. (1) based conclusions on several indices and did not use SDNN. Their data of HF peak in PSA and the larger initial changes in R-R interval in response to muscle contraction in trained vs. untrained subject are well proven indices of vagal tone (2, 4). Changes in peripheral signaling involving nNOS are not considered. I accept that long-term exercise is likely to result in a remodeling

of pacemaker currents but the influence of autonomic innervation should not be dismissed.

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COMMENT ON “IS THE RESTING BRADYCARDIA IN ATHLETES THE RESULT OF REMODELING OF THE SINOATRIAL NODE RATHER THAN HIGH VAGAL TONE?”

TO THE EDITOR: The basal heart rate (HR) has always been considered an indirect index of fitness, often used as a parameter in individuals undergoing physical training. However, where was discussed the origin of prominent bradycardia at rest observed in trained individuals, especially in high level athletes? The study by Boyett et al. (1) makes a systematic review of the findings of several authors, both clinical and experimental, and discusses the possible mechanisms responsible for bradycardia, with a focus on cardiac autonomic

control and reduction of intrinsic HR (iHR). In fact, the autonomic nervous system has been touted as a major contributor to the bradycardia at rest. This bradycardia was due to adjustments in cardiac autonomic balance characterized by increased vagal autonomic drive and/or reduction of sympathetic autonomic drive (2). In this case, these adjustments may result from central or peripheral adaptations of structures involved with autonomic nervous system, i.e., afferent, central nuclei, efferent, and cardiac autonomic receptors. However, in experimental studies with rats, the reduction in basal HR, due to the physical training, seems to depend on the two mechanisms, i.e., reduction in iHR and adaptations in cardiac autonomic balance (3, 4). Moreover, the study of Tezini et al. (4) also shows that the bradycardia at rest in young animals is more dependent on iHR reduction, whereas in older animals it is more dependent on the autonomic balance adjustments. Finally, studies in humans are needed to confirm these findings.

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