Athlete’s bradycardia may be a multifactorial mechanism

David Matelot,1 Frédéric Schnell,1 Gaelle Kervio,2 Nathalie Thillaye du Boullay,2 and François Carr1

1Laboratoire de Traitement du Signal et de l’Image, INSERM U1099, Rennes, France; and 2Centre d’Investigation Clinique et d’Innovation Technologique, INSERM 804, Rennes, France

TO THE EDITOR: Boyett et al. (1) provides interesting evidences, arguing that endurance training-induced bradycardia seems mostly related to a remodeling of the sinoatrial node. However, we would like to add four complementary elements to extend this paper.

About intrinsic heart rate. In Boyett et al.’s Table 1, seven studies explored long-time endurance athletes and three studies explored subjects after few months of training. There is confusion between the effects of endurance training in previously nontrained subjects and the mechanisms leading to training-induced bradycardia in long-term athletes. However, it seems that endurance-related adaptations differ in time. Indeed, cross-sectional studies in long-term athletes suggested that training bradycardia was almost due to a lower intrinsic heart rate (HR) (5, 8, 13). But two longitudinal studies, inducing resting bradycardia in previously untrained men, showed no change in intrinsic HR (7, 12). To conclude about intrinsic HR, we would like to highlight that pharmacological blockade, always used in the studies, also has some limitations (2).

Athlete’s bradycardia definition. We believe that a training bradycardia defined as a resting HR < 50 beats/min should have been more relevant. Indeed, a resting HR < 60 beats/min in an athlete is trivial (10), whereas it becomes clinically significant when < 50 beats/min (11). However, all the studies in Table 1 reported mean resting HR < 60 beats/min but > 50 beats/min. Furthermore, Table 2 proposed a review of animal studies, for which extrapolation in humans must be done with caution, because training-induced bradycardia is not well defined in animals.

Autonomic balance. First, standard deviation of the normal beat-to-beat interval (SDNN) is presented as an indicator of the parasympathetic tone; however, SDNN is usually used as an index of global heart rate variability (HRV) (15). Other indices such as proportion of pairs of successive normal beat-to-beat intervals or square root of mean squared difference of successive normal beat-to-beat intervals (pNN50 or RMSSD, respectively) are more relevant to quantify the vagal tone. Second, overstimulation of the parasympathetic system on the sinus node is not always associated with an increase in the high-frequency component of HRV (9). So, low parasympathetic HRV indices do not mean low parasympathetic nervous tone to the sinus node. Third, during competitive periods, autonomic balance can present a conversion from vagal to sympathetic predominance (4). This factor has not been controlled. Last, some proposed that a lower sympathetic tone could also explain training-induced bradycardia (13). This other alteration of the autonomic balance should be taken into account concerning athlete’s bradycardia mechanisms.

A multifactorial mechanism. Boyett et al. considered that because autonomic balance and intrinsic HR are not altered, it must be a remodeling of the sinoatrial node that explains training-induced bradycardia. However, as suggested by others studies, cardiac hypertrophy (3), baroreflex alteration (14), and genetics factors (6) could also be involved in an athlete’s bradycardia. These factors should also be investigated, because training-induced bradycardia may be not related to one single cause, but it could be a multifactorial mechanism.

To conclude, Lewis et al. (8) have already proposed that “a training-induced alteration in the electrophysiology of the sinoatrial node could reset both the resting and maximal HR at a lower level.” This hypothesis seems really valuable; however, evidence is still lacking.

DISCLOSURES
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