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Suggested New Upper Limit of Physiologic Cardiac Hypertrophy Determined in Japanese Ultramarathon Runners Must Be Interpreted Cautiously

With great interest and surprise we read the study by Nagashima et al. (1). The researchers report that 11% of the participants (33 of 291) had a left ventricular end-diastolic diameter (Dd) larger than 70 mm. In addition, further extraordinary values for the end-Dd (mean value, 61.8 ± 6.9 mm; range, 42 to 75 mm), the end-systolic diameter (mean value, 39.6 ± 6.9 mm; range, 23 to 55 mm), and the intraventricular septal thickness (range, 5 to 19 mm) are presented, which are in great contrast to results reported previously (2–5).

The presented data are even more surprising when normalized to the body surface area (BSA; mean value, 1.66 ± 0.1 m²), resulting in an unbelievable mean Dd-to-BSA ratio of approximately 37 mm/m². Such a high value seems unrealistic, especially when compared with the data of Pelliccia et al. (3), who found the highest mean Dd-to-BSA ratio of about 31 mm/m² in healthy endurance athletes competing at national and international levels (59 track-and-field athletes, 49 cyclists, and 41 cross-country skiers). Also, the range of the intraventricular septal thickness (5 to 19 mm) is suspicious for at least some pathologically hypertrophied hearts. Consequently, based on the data of previous reports (2–6), hearts with an end-Dd ≥60 mm or an intraventricular septal thickness ≥13 mm in athletes with a BSA <1.82 m² are highly suspicious for pathologic hypertrophy (2) and would have required further cardiologic examinations to confirm the statement of a physiologic hypertrophy. Therefore, the data presented by Nagashima et al. (1) suggesting a new upper limit of physiologic cardiac hypertrophy may lead to confusion and to misinterpretations in daily routine (diagnosis of athlete’s heart instead of pathologic hypertrophy), and put athletes at an avoidable risk.

The main confounding variable of the study (1) seems to be the age of the subjects (mean age, 41.8 ± 9.7 years; range, 20 to 73 years). As it is well known that the incidence of cardiovascular diseases rises with age (especially above 35 to 40 years) (7), cardiovascular diseases in the participants should have been excluded by cardiologic examinations. It is not sufficient to assess the state of an athlete’s health by taking only the resting blood pressure or replies to questionnaires (1). Furthermore, relevant additional examinations should have been performed, and important parameters should have been given in the study: ejection fraction to describe the systolic function; E/A ratio to evaluate the diastolic function; Doppler-echocardiography to exclude valvular diseases resulting in pathologic ventricular overload and dilation; 12-lead electrocardiogram at rest and exercise (including blood pressure measurements) to detect possible coronary artery disease or exercise-related hypertension; and spiroergometry to obtain maximal oxygen consumption, which describes the state of physical fitness and is closely related to cardiac dimensions [8–10]. Moreover, the convention of echocardiographic measures is not given (Penn or American Society of Echocardiography convention [11,12]).

In conclusion, it seems that the study by Nagashima and co-workers does not define a new upper limit (which now would be 70 mm or even 75 mm, when the 95% confidence interval for the left ventricular end-Dd is calculated), but a wrong upper limit of physiologic cardiac hypertrophy.

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REPLY

The aim of our study (1) was that: 1) our cases include more of the larger cardiac chambers than had ever been previously reported; and 2) larger cardiac chambers are not significantly related to body surface area (BSA) but to the extent of the monthly running.

The main insistence of the comment from Dr. Whalley et al. seems to be in the inappropriateness of using BSA as an index of body size. We did not describe the left ventricular mass but the chamber size. This was done to compare previous reports on cardiac size. Lean body mass was not measured in our study because of the limitation of methodology. Milliken et al. (2) observed that the cardiomegaly observed in highly trained athletes was much higher than in non-athletes and its grade was above the level of correction by the body size. We agree about the importance of evaluation through accurate correction of body size among the non-athletic general population, but it seems that the choice of BSA or lean body mass is not critical for evaluating cardiomegaly in highly trained athletes. Our subjects included an exclusively homogeneous group of long-distance runners having lean body composition. Hence, the use of BSA as the index of body size could be justified, and it was concluded that the sole determining factor of cardiac size in athletes is the intensity of training. Whether this cardiomegaly is pathological or physiological remains unclarified, as described in our Study Limitations section. Further follow-up study is now being conducted.

We understood the questions raised by Drs. Kasikcioglu and Akhan as to 1) how many investigators participated in the study, and 2) that pathological hypertrophy could be included. Concerning the first question, both the examination and the measurement of echocardiograms were performed solely by J. Nagashima, MD, a certified Fellow of the Japanese Society of Ultrasound in Medicine. Gordon et al. (3) concluded that intraindividual variability was minimal, but interobserver and beat-to-beat variability levels were of sufficient magnitude to suggest that serial measurements on a given subject be made, ideally by a single person, and that several cycles be averaged for a given measurement.

Concerning the second question, our entry conditions in this study were exclusion of subjects with hypertension or a positive reply to Revised Physical Activity Readiness Questionnaire. We found six cases showed more than 16 mm in the inter-ventricular septal thickness; the minimum diameter of left ventricle was 58 mm, and the ejection fraction was above 62%. It seems different from the conventional type of hypertrophic cardiomyopathy (HCM) because of the absence of a lessered size of the left ventricular (LV) chamber and also different from the dilated phase of HCM because of the presence of normal LV contractility (4).

These might be considered to be HCM modified by the effect of intensive endurance training, but the precise determination of etiology remains undefined at the present time.

The questions raised by Dr. Scharhag et al. concern 1) end-diastolic diameter (Dd) to BSA ratios that are much larger than in previous reports. Hence, pathological hypertrophy might be included; and 2) the precise method of echocardiography is not described. We already addressed these issues in the reply to the query by Dr. Kasikcioglu et al. Regarding the Dd-to-BSA ratio, we already stated in our discussion that “Compared with the subjects of previous reports, the BSA of our subjects was significantly smaller, so a larger cardiac load per unit of body size may have been related to our findings.” One reason for obtaining so high a value of Dd-to-BSA ratio might be due to the significantly small BSA in our subjects compared to the previous reports. Dr. Scharhag’s criticism concerns the insufficient exclusion of cardiac abnormality. We could not completely exclude the presence of possible latent coronary artery disease, as he points out. However, all our subjects are apparently quite healthy individuals who could perform the highly severe daily training for a long time and finish the race within the time limit of 14 h, showing their high endurance ability.

Finally, besides the measurements described in the text, ejection fractions calculated using Teichholz’s method were within normal limits in all subjects. We believe that a small grade of coronary artery disease, which could not be detected by routine measurements, could not influence the results of our study. But we do recognize the importance of the questions raised, and hence we want to show the longitudinal follow-up study in the future.

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