Peak pulse pressure during exercise and left ventricular hypertrophy in athletes

Sporcularda egzersiz sırasında doruk nabız basıncı ve sol ventrikül hipertrofisi

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The differentiation of physiologic and pathologic hypertrophy can be difficult, and is important in determining the presence or absence of cardiac disease in athletes in order to prevent exercise-related sudden cardiac death (1). In cross-sectional studies, pulse pressure (PP) showed a direct association with left ventricular (LV) mass in hypertensive patients (2). However, the relation of the systemic arterial PP during exercise and LV mass is not described in athletes. We evaluated the relationship between PP and LV mass in endurance-trained athletes.

Twenty Caucasian male middle-distance runners and 20 age-matched healthy Caucasian male controls were included in the study. All subjects underwent resting echocardiography (System Five, GE Vingmed Ultrasound, Horten, Norway) and graded treadmill exercise for measuring of maximal oxygen consumption (VO_{2max}) (2900C BxB, Sensormedics, CL, USA) at the same day. We regarded the subjects as runners when they had trained at least 10 hours per week (averaging 25 miles/week) for at least 5 years. Subjects were regarded as sedentary controls, when they exercised (walking) for less than 3 hours per week. All subjects were free of cardiovascular disease as determined with detailed history and physical examination. Systolic and diastolic blood pressures were measured simultaneously with mercury sphygmomanometer at the brachial artery. Pulse pressure was calculated as systolic minus diastolic blood pressure. Mann-Whitney U test was used for statistical analysis. Correlations were assessed by Pearson's coefficients.

Mean VO_{2max} in athletes was higher than that of control group (60.1±3.7 vs. 41.5 ± 5.2 ml/kg/min, respectively, $p<0.001$). Left ventricular mass index in athletes was also significantly greater than in control subjects (122.4 ± 30.8 vs. 85.4 ±6.8 g/m^2, respectively, $p<0.001$). Although PP at rest was similar in two groups, peak PP during exercise in athlete group was higher than in control group (117±15 vs.101±12 mmHg, respectively, $p<0.05$). The LV diastolic parameters in athletes were higher than those of controls. There were significantly positive correlations between VO_{2max} and peak PP ($r=0.41$, $p<0.05$), and between peak PP and LV mass index ($r=0.53$, $p<0.01$).

The factors involved in the development of physiological hypertrophy in humans are still unknown and it is felt that once these factors are determined, their knowledge might also be relevant for better understanding the mechanisms involved in the cardiac adaptive response to the pathological increase in hemodynamic workload (3) The current study shows that PP during maximal exercise is more closely associated with LV mass index and predictive of LV hypertrophy.

An important basic mechanism of the rise in PP with age is believed to be the progressive stiffening of large arteries in hypertensive patients at rest. A high PP may reflect already diseased arterial walls, with several adverse cardiac implications of potential prognostic value (4). On the other hand, it is known that stiffness of large arteries in athletes is lower than in seden-

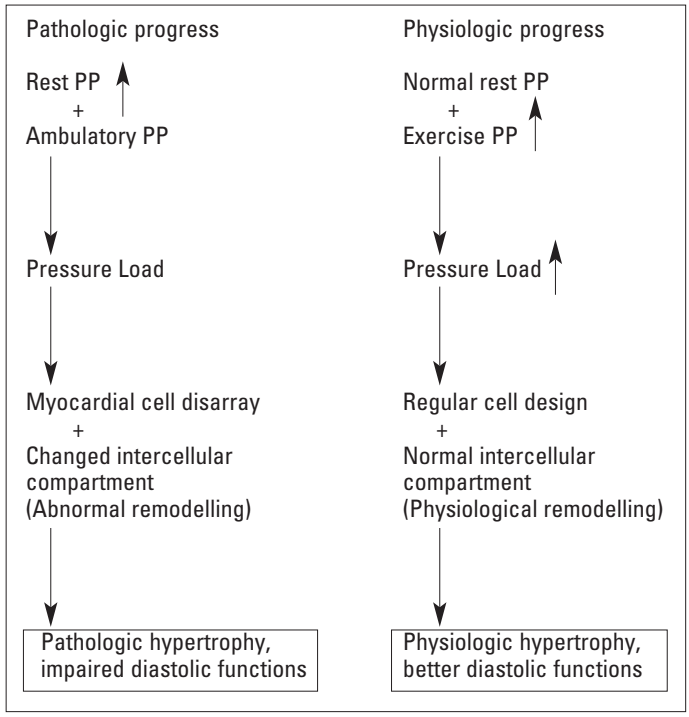

Figure 1. Possible mechanism of pulse pressure (PP) and cardiac remodelling response in physiologic and pathologic conditions.

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tary subjects (5). We believed that increased PP during exercise in athletes did not cause stiffness of large arteries and abnormal ventricular remodelling, contrary to pathologic conditions (Fig. 1). Major evidence of this hypothesis is better diastolic functions in athletes compared with control subjects.

In conclusion, LV mass appears to be more strongly related to the peripheral pulse pressure, measurements of hemodynamic pulsatile load during exercise in athletes.

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