Functional Assessment of the Hypertrophic Right Ventricle in the Rat Heart

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Introduction
Whereas a number of studies can be found dealing with the function of the left ventricle (LV) in experimental cardiac hypertrophy, considerably less attention has been paid to the right ventricular (RV) performance. This is despite the fact that many pathogenetic factors such as chronic hypoxia (e.g. 1), strenuous exercise (2), excess thyroid hormone or catecholamines (3, 4) are known to induce cardiac hypertrophy with the right ventricular dominance. One of the reasons for this may consist in methodical difficulties associated with the assessment of the RV function, especially in small laboratory animals. Measurements of force/length/velocity relationships on isolated thin preparations of ventricular muscle represent a frequently used approach. Problems arise, however, when the mechanical and pump functions of the whole intact RV are to be assessed under conditions of controlled preload and afterload.

In this study we compared two methods which originally had been developed and routinely employed for evaluation of the LV function. Recently, their modifications were introduced which enable us to study the RV performance. The first method is based on the "in vitro" perfused heart with the RV performing pressure/volume work (5, 6). It represents a modification of the classic (left ventricular)
working heart (7) and makes it possible to measure the RV contractile and pump activities under controlled loading conditions. The other method makes it possible to estimate the maximum RV mechanical performance (or reserve) in the "in situ" heart as a response to acute pressure load after a ligation of the pulmonary artery (4). Also this technique is a modification of the procedure used for the measurement of the LV function (8, 9). The rat heart with the RV hypertrophy induced by chronic hypoxic hypoxia was employed as an experimental model.

Animal Model
60-day-old male rats of Wistar strain were acclimatized to intermittent high altitude (IHA) hypoxia simulated in a low pressure chamber for 8 h/day, 5 days/week. Barometric pressure was reduced gradually during 13 exposures up to the level of 306.8 mm Hg which corresponds to an altitude of 7000 m; the total number of exposures was 24-26. Half of the IHA-acclimatized group was employed for the functional study 24 hours after the last hypoxic exposure (hypertrophic group); the remaining animals were transferred into the normobaric conditions equivalent to an altitude of 200 m and kept for a further 5 weeks until employed (regression group). The control group of rats was kept at the altitude of 200 m for the whole investigated period. All animals had free access to tap water and a standard laboratory diet.

Body weight and heart weight parameters of rats acclimatized to IHA and those examined 5 weeks after the termination of hypoxic exposure are compared with controls in Table 1. The IHA hypoxia induced absolute as well as relative right ventricular enlargement; the complete regression of weight occurred 5 weeks after withdrawal of rats from hypoxic environment.

Right Ventricular Function in Vitro
The function of the RV was assessed using a modified isolated perfused heart with the RV performing pressure/volume work (5, 6). The perfusion scheme of this preparation is illustrated on Fig. 1. The animals were anesthetized with sodium pentobarbital