Cardiac function in the chronically volume-overloaded canine heart

Experimentelle Untersuchungen zur Funktion des chronisch volumenbelasteten Herzens

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With 3 figures

Summary

In the chronically volume-overloaded canine heart due to AV-block evaluations of cardiac function were performed during the development of hypertrophy and at stable hypertrophy. In an early stage (1 and 2 weeks of AV-block) when no or only a slight increase of cardiac muscle occurred, contractility measured from $\frac{dP}{dt_{\text{max}}}$ at comparable load is elevated, while later (10 weeks of AV-block) when stable hypertrophy is present, contractility becomes again normal. In the hypertrophied heart a non-depressed cardiac performance and contractility and functional reserve was established from in-situ experiments and from evaluations in the isolated heart.

From clinical and experimental studies it is well known that chronic overload of the heart results in myocardial hypertrophy. The increase in myocardial mass provides a fundamental adaptive response that permits the heart to compensate for augmented load. During the development of hypertrophy, cellular and subcellular alterations of the myocardium have been reported, the primary trigger mechanism leading to cardiac enlargement remains, however, unknown. From a mechanical point of view wall stress was found to be increased during augmented load. Cardiac hypertrophy normalizes wall stress by an increase in wall thickness (1, 2) suggesting that hypertrophy is an adaptive mechanism to maintain stress at a constant value (3). The effect of hypertrophy on the contractile and functional performance of the heart is still a matter of dispute (4). Different results may be caused by several factors, such as: the type of chronic overload, pressure or volume; the degree of hypertrophy, and the type of experimental preparation. Some investigators have used the isolated papillary muscle, while others employed the intact ventricle. The interpretations of results obtained from intact hearts are difficult when data are not corrected for changes in pre- and afterload (5). It is, however, important to study hypertrophy in the intact heart because of the clinical relevance. In the present study cardiac performance and contractility of
the intact left ventricle were investigated in the chronically volume-overloaded heart during the development of hypertrophy and in stable hypertrophy.

**Methods**

Chronic volume overload was produced by complete atrio-ventricular block in mongrel dogs as described by Turina and Krayenbuehl (6). Experimental evaluations were carried out in normal dogs, in sham-operated ones and in dogs after 1 week, 2 weeks and 10 weeks of chronic AV-block. Hemodynamic studies were done in the anesthetized animals by right and left heart catheterization at closed-chest preparation in normals and sham-operated dogs during ventricular pacing and in all groups of animals with AV-block during idioventricular rhythm and pacing. Normal hearts and chronically volume-overloaded hearts after 10 weeks of AV-block were investigated in an in-situ preparation during open-chest and in an isolated-heart preparation. In the in-situ experiments the contractile reserve and maximal cardiac performance was measured during paired pacing, arterenol infusion and cross clamping of the aorta. In the isolated-heart experiments the hearts were perfused at constant pressure with blood from the support dog, and cardiac performance was measured during isovolumetric beating over a wide range of ventricular load.

**Results**

After 2 weeks of chronic AV-block an 18% increase of the left ventricular weight became statistically signifikant. After 10 weeks of AV-block ventricular weight increased further to 41% for the left and 71% for the right ventricle. In a few hearts with 5 to 6 months of AV-block, cardiac weight was not different from the 10 weeks group. Signs of heart failure were not observed in any of the animals with cardiac overload, not even in those which died suddenly, probably because of cardiac arrhythmia.

![Graphs showing hemodynamics](Image)

Fig. 1. Hemodynamics of control dogs and animals after 1 week, 2 weeks and 10 weeks of chronic AV-block (for further explanation see text).