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Increasing afterload induces myocardial diastolic dysfunction

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Keywords

Afterload, diastole, experimental, haemodynamics, heart, heart failure, pathophysiology, ventricular function

Comments

This is an interesting paper that suggests that changes in afterload produce significant changes in diastolic performance and the authors comment that this may be further exacerbated in diseased heart. This has obvious importance to ICU patients and emphasises the need for cardiovascular stability in patients, many of whom have a chronically or acutely impaired myocardium. This experimental finding could be investigated clinically using echocardiographic parameters of diastolic dysfunction.

Introduction

Systolic dysfunction cause elevated filling pressures due to a shift along an unchanged end-diastolic pressure-volume relation. Diastolic dysfunction corresponds to an upward displacement of the end-diastolic pressure-volume relation, with a higher pressure at a given volume. This is related to myocardial relaxation which is usually completed during rapid filling near minimum left ventricular pressure (LVP). In healthy hearts, small elevations of afterload may accelerate the fall in LVP, whereas larger elevations of afterload slow this fall. In failing hearts this slowing of LVP fall can be seen with only minor elevations of afterload. These findings lead to the concept of relative load, the ratio of systolic LVP to isovolumic LVP, ie when the relative load is low there is afterload reserve to allow the heart to face increases in afterload without slowing the LVP fall.

Aims

To investigate the hypothesis that an afterload elevation which increases relative load and markedly slows the rate of LVP fall would result in incomplete myocardial relaxation and in the upward shift of the end-diastolic pressure-volume relation, ie diastolic dysfunction.

Methods

New Zealand White rabbits (n=9) were anaesthetised (with ketamine) and a silk suture placed around the ascending aorta. A micromanometer was placed in the left ventricle to measure LVP. LV dimensions (ID) were measured with ultrasonic gauges. Multiple graded aortic occlusions were performed. The heart beat prior to occlusion was used as the control and the heart after occlusion was used as the test beat. Peak rates of LVP rise and fall were measured as were LV dimensions. Afterload levels were described as relative load, ie max LVP/peak isovolumic LVP. The curve was fitted to either a monoexponential curve or logistic model to give a time constant of LVP fall. These data were then compared to similar retrospective data in open chested anaesthetised dogs.

Results

The resting relative load was 60% and this was increased step wise to 70, 80, 90 and 100%. Fractional shortening progressively decreased as afterload increased. At 70%, the LVP fall time constant slightly (but significantly) decreased indicating accelerated LVP fall. At 80%, relative loads and higher LVP fall was significantly slowed. When plotted, the LVP-ID loops showed elevation of the diastolic LVP with increasing afterload. The ID at the start of the test beat was similar at all load levels suggesting there was no increase in filling and so the increased diastolic LVP reflected true diastolic dysfunction. The slowing of LVP fall appeared to be predictive of the magnitude of diastolic failure induced by afterload changes. From the dog data heavily afterloaded and isovolumetric heart beats induced an upward shift of the end-diastolic LVP segment length loops.

Discussion

Elevated afterloads prolonged the half-life of LVP fall and caused an upward shift of the diastolic LVP-ID relation. This was associated with a decrease in fractional shortening. This represents abnormal systolic function in the face of increased afterload. Although slight afterload increases caused an accelerated LVP fall, further increases caused slowing of relaxation. This transition from acceleration to deceleration occurred at lower loads in the rabbit relative to the dog suggesting there may be species-specific differences (possibly related to calcium handling). In this experiment, only one heart beat was used as the test, after which the afterload was reduced which should have eliminated any neuro humoral or global haemodynamic changes. Relative loads of greater than 80% produced frank diastolic dysfunction. This was probably exacerbated by the decreased duration for LV filling due to the proportion of the cardiac cycle taken up with relaxation. These data suggest that afterload mismatch is a causal mechanism in heart failure. This might explain why vasodilators and inotropic agents (which accelerate relaxation and decrease relative load) may lower filling pressures in the diseased heart.

References

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