Coronary vasodilator reserve in left ventricular hypertrophy

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The coronary vasodilator reserve (CVR), defined as the ratio of myocardial blood flow (MBF) during near maximal hyperaemia to baseline MBF, is reduced in LVH due to hypertension, aortic stenosis, aortic regurgitation, supravalvar aortic stenosis and hypertrophic cardiomyopathy. Coronary vasodilator reserve (CVR) in experimental models of LVH has been impaired (Fig. 1). The reduced CVR limits the ability of hypertrophied hearts to meet the metabolic requirements when demand is increased. Despite normal myocardial oxygen consumption and myocardial perfusion per unit mass, at rest, the hypertrophied heart is more vulnerable to ischaemia.

Experiments in animals have demonstrated that the impairment of CVR is more marked in the subendocardial layers of the left ventricle, with some evidence of an alteration in the normal subendocardial-subepicardial distribution of perfusion during near maximal vasodilatation in patients with hypertrophic cardiomyopathy. More recently, we have demonstrated that CVR is impaired in patients with LVH secondary to aortic stenosis and that the impairment correlates with the severity of the transvalvular gradient. In addition, as the gradient increases, subendocardial CVR becomes progressively more impaired than subepicardial CVR (Fig. 2).

Effect of left ventricular hypertrophy regression on the impairment of coronary vasodilator reserve

Experimental studies have reported that antihypertensive treatment of spontaneously hypertensive rats reduces LVH with improvements in left ventricular compliance and reduced vulnerability to ischaemia although CVR remained impaired in LVH. In contrast, other studies have demonstrated some recovery in coronary vascular morphology and CVR with regression of experimental hypertrophy. Reduction in echocardiographically measured left ventricular mass has been observed clinically after treatment in patients with hypertension. Regression of LVH does occur following aortic valve replacement but may remain incomplete in 50% of such patients. In contrast to the intense interest in regression of left ventricular mass, few studies have examined whether this is accompanied by reversal of the pathophysiological effects of LVH. Pre-
liminary results from our study in patients with aortic stenosis and LVH show that reduction in left ventricular mass following aortic valve replacement is associated with a marked improvement of overall CVR. It is unclear whether similar changes occur with regression of LVH in hypertension. One study has demonstrated an improvement in CVR with antihypertensive treatment, although it remains to be established whether regression of LVH correlates with improvement in CVR.

References