Coronary haemodynamics associated with left ventricular hypertrophy in aortic stenosis and hypertension

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Background: Left ventricular hypertrophy (LVH) occurs in both aortic stenosis (AS) and hypertension (HT) due to an increase in afterload. However, in AS there is an increase in resting coronary flow (per gram of LV) while in HT it is reduced.

Wave intensity analysis (WIA) is a well-established method of characterising and quantifying the energies that drive coronary flow. Energies propagating from the proximal vessel (aorta and systemic arteries) interact with energies travelling from the distal end (myocardial microcirculation). WIA allows the separation of these energies into the waves that drive cyclic changes in coronary flow.

Purpose: We aimed to compare coronary flow patterns in LVH secondary to AS with coronary flow patterns in LVH secondary to HT.

Methods: Thirty-one participants were recruited (mean age 63, 18 female), 10 with LVH and severe AS, 11 with LVH and HT, and 10 with no LVH and no AS. Participants underwent invasive pressure and Doppler velocity measurements in each of the left coronary arteries and echocardiography. We applied WIA.

Results: Mean resting coronary flow per gram of LV tissue (Fig. 1) was increased in participants with LVH secondary to AS (1.62±0.60 ml/min/g) and reduced in participants with LVH secondary to HT (0.49±0.27 ml/min/g), compared to participants with no LVH and no AS (1.47±0.73 ml/min/g).

We observed marked differences between the magnitudes of the waves driving coronary flow in the three groups (Fig. 2). Forward and backward travelling waves are shown above and below the line respectively. Waves causing acceleration of coronary forward flow are shown as black and waves causing deceleration are shown in white.

Wave 6, the backwards decompression wave (BDW), is particularly important for myocardial perfusion. The BDW corresponds to the diastolic 'suction' of blood down the coronary arteries during myocardial relaxation. The energy of the BDW was increased in LVH secondary to AS ($31.1x10^3$ W m^{-2} s^{-2}) but was reduced in LVH secondary to HT ($12.3x10^3$ W m^{-2} s^{-2}) ($p<0.05$), compared to participants with no LVH and no AS ($14.3x10^3$ W m^{-2} s^{-2}).

The energy of the BDW correlated with LV cavity pressure ($r=0.84$, $p<0.001$) and diastolic time ($r=-0.62$, $p<0.001$) only in LVH secondary to AS participants. In contrast, the BDW correlated with LV mass ($r=-0.49$, $p=0.03$) in participants with LVH secondary to HT and with no LVH and no AS, but not in participants with LVH secondary to AS.

Conclusions: In hypertension, LVH is associated with reduced mean coronary flow and reduced myocardial "suction" during diastole, presumably by the reduction in myocardial compliance associated with diastolic dysfunction.

However, in AS the large pressure gradient between the LV cavity and the aorta results in a large contractile force which is generated in systole and then released in diastole. This large diastolic force overwhelms any local impairment caused by the hypertrophied myocardium and contributes to high resting coronary flow in AS.

Figure 1: Mean coronary flow per gram of LV for each group

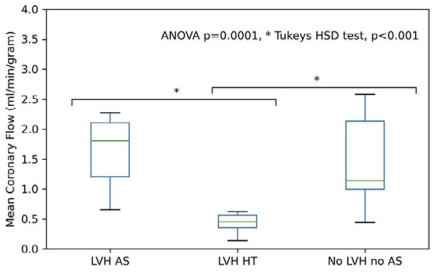

Figure 2: Intracoronary wave intensity in a participant with LVH secondary to hypertension (left) and in a participant with LVH secondary to aortic stenosis (right)