

PHYSIOLOGICAL LIMITATIONS OF THE LEFT VENTRICLE WALL STRUCTURE & AGEING: A POSSIBLE ROLE FOR THEBESIAN VEINS

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Main causes of human premature deaths are consequences of altered tissue structures due to atherosclerosis. Despite the controversy regarding the origin of essential hypertension and accompanying vascular changes, the left ventricle (LV) thickness is among limiting factors of optimal heart size and strength. Atrial muscle and the right ventricle are perfused during the entire heart cycle. The LV systolic pressure forces blood vessels in the muscle wall to collapse, particularly in the subendocardial muscle layer. Due to evolutionary disappearance of features that compromise the LV compliance, mammalian heart has decreased vascular density and limited coronary vessel diameters. Coronary circulation is helped by Thebesian veins that drain blood from heart muscle into the chambers. These veins are reported numerous in atria and right ventricle, but scarce in the left ventricle. Based on reports that Thebesian veins drains the ventricle apex and papillary muscles base, regions remote from the coronary sinus, an interpretation is here proposed that the Thebesian vein role is to act as local reducers of venous hydrostatic pressure that correct regional differences in fluid filtration, thus preventing myocardial edema and maintaining contractility. Described features make LV muscle prone to hypoxia. During ageing, LV perfusion can become dependent on age related hypertension that increases diastolic aortic and coronary pressures, until atherosclerosis turns this advantage into a premature death risk factor.