Impaired left ventricular systolic function in patients with left ventricular hypertrophy
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The incidence of congestive heart failure especially with preserved left ventricular ejection fraction is increasing in incidence. The prevalence of hypertension is also increasing, supporting that hypertension is a major contributor to the increasing incidence of congestive heart failure.

Impaired left ventricular systolic function may be caused by myocardial ischemia either due to increased myocardial oxygen consumption induced by hypertension and left ventricular hypertrophy or due to reduced oxygen supply caused by structural or functional epicardial changes, small vessel disease or increased oxygen diffusion distance due left ventricular hypertrophy. Furthermore, left ventricular ejection fraction, the most commonly used measure of left ventricular systolic function is a somewhat crude measure, especially in patients with left ventricular hypertrophy because several of the assumptions for its calculation are not present. Two studies in this issue of the journal give us insight to reasons for left ventricular dysfunction even in the presence of normal left ventricular ejection fraction.

We and others have shown data suggesting that patients with hypertension not only have a high prevalence of impaired left ventricular diastolic function [1], but especially when left ventricular hypertrophy is present also have high prevalence of impaired left ventricular myocardial function [2] that may progress to congestive heart failure. Furthermore, there are also data suggesting even more complex relationships between left ventricular systolic and diastolic function and that these conditions are in fact closely interrelated [3].

We and others have shown that blood pressure reduction does in fact improve measures of left ventricular diastolic [4] and systolic [5] function. Furthermore, it has also been shown that improvement of electrocardiographic strain pattern during antihypertensive treatment does reduce the risk of new-onset heart failure [6].

The study by Goebel et al. [11] in this issue of the journal investigated myocardial function and left ventricular twist in 86 hypertensive patients with so-called normal coronary angiograms. They found that left ventricular hypertrophy influences longitudinal and circumferential deformation rate and that left ventricular hypertrophy delays systolic contraction and reduces systolic twist rate also indicating a relationship between left ventricular hypertrophy and left ventricular systolic dysfunction.

Goebel et al. [11] included patients with suspected ischemic heart disease but with normal coronary angiograms and evaluated left ventricular systolic function. However, following the current guidelines, indication for an angiogram would generally require typical chest pain and some form of noninvasive documentation that the chest-pain actually derives from the heart or in absence of...
the latter at least multiple clinical risk factors (hypertension, hypercholesterolaemia, smoking, family history of ischemic heart disease, prior stroke, claudication and/or prior myocardial infarction). As a result, it is likely that the population selected had some degree of atherosclerosis in the coronary arteries not visible on angiography and/or some degree of small vessel disease both of which lead to impaired vasodilatory responses reducing oxygen supply to the hypertrophied left ventricle resulting in ischemia, angina and impaired regional left ventricular mechanics. Therefore, the observed impaired regional left ventricular mechanics may be due to impaired vasodilatory responses and/or left ventricular hypertrophy. The relative impact of impaired vasodilatory responses would have been better assessed if the authors had done noninvasive objective measurements like exercise testing with normal nuclear perfusion scans to ensure that patients were free of myocardial ischemia. Coronary angiograms are notorious for underestimating the burden of atherosclerosis in the coronary arteries because injecting contrast into a coronary artery is a structural measurement that only visualizes lumen (not the wall) and only to a minor degree evaluates the coronary artery function. Therefore, the informative value of a coronary angiogram is increasingly often improved by measuring either fractional flow reserve during adenosine infusion (i.e. the blood pressure at both ends of the epicardial coronary artery) in order to estimate the burden of diffuse coronary artery disease or the composite effects of several non-hemodynamic-significant stenoses in series resulting in myocardial ischemia.

In the report by Goebel et al. [11], the most likely scenario is that the myocardial vascular bed is in fact not normal in the patients included in the study. Furthermore, patients with left ventricular hypertrophy have probably even more impaired vasodilatory responses and in addition higher myocardial oxygen consumption leading to myocardial ischemia and myocardial dysfunction.

That said, the study by Goebel et al. [11] emphasizes the need for further research in hypertensive patients with angina pectoris and normal coronary arteries with estimation of the burden of myocardial ischemia especially when left ventricular hypertrophy is present.

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Conflicts of interest
There are no conflicts of interest.

References