Twisting, untwisting and diastolic function in aortic valve disease

See page 582 for the article to which this Editorial refers

Global and regional deformation of left ventricular muscle tissue is sensitive to a variety of cardiac disorders such as coronary artery disease and pressure–load hypertrophy. In the domain of coronary artery disease, changes in local wall motion and thickening may occur in acute and chronic myocardial infarction, and during stress-induced myocardial ischaemia. Under conditions of pressure overload, the development of left ventricular hypertrophy may result in delayed and incomplete relaxation of the left ventricular wall\[1\]. Consequently, diastolic dysfunction may occur, which may precede the occurrence of systolic dysfunction ultimately leading to overt heart failure\[2\].

In recent years, magnetic resonance imaging methods have been described that allow the determination of absolute motion and thickening of specific myocardial segments. The use of myocardial tagging has been a major breakthrough in the kinematic analysis of cardiac muscle properties\[3–5\]. Although multiple imaging modalities exist for the assessment of global and regional wall motion and thickening, myocardial tagging offers the advantage of being completely non-invasive and without ionizing radiation. Myocardial tagging involves localized radiofrequency saturation of myocardial tissue before acquiring images, which allows monitoring of the progressive distortion of the myocardium during the cardiac cycle. One of the most advanced tagging sequences in called complementary spatial modulation of magnetization (CSPAMM), which produces cardiac images with a regular (‘grid’-like) pattern of stripes that move with the cardiac wall throughout the complete cardiac cycle\[6\]. The CSPAMM technique provides therefore a unique three-dimensional method to evaluate myocardial motion and thickening, to assess ventricular non-uniformity, and to study rotational deformation during systole (‘twisting’) and diastole (‘untwisting’)\[7\]. Needless to say, myocardial tagging is very well suited to the study of cardiac torsion and diastolic relaxation under conditions of pressure–load hypertrophy.

Nagel et al.\[8\] have addressed this issue by studying the time course of rotational motion of the left ventricle using the CSPAMM technique in 13 patients with severe aortic valve stenosis. Compared to 12 healthy controls, the authors clearly showed that left ventricular pressure overload was associated with a reduction in basal rotation and an increase in apical rotation of the heart, indicating increased torsion of the left ventricle in severe aortic valve stenosis. In addition, it was shown that diastolic untwisting was delayed and prolonged in the patients with aortic valve stenosis. These findings explained the occurrence of diastolic dysfunction in these patients.

One major clinical important implication of the findings reported by Nagel et al.\[8\], as already alluded to by the authors themselves, might be the differentiation of physiological from pathological hypertrophy. The accurate distinction of these two conditions holds particularly for elite athletes in whom severe left ventricular hypertrophy is usually present. The issue of physiological vs pathological hypertrophy has been previously evaluated by magnetic resonance imaging and spectroscopy, which showed physiological hypertrophy in athletes at the levels of cardiac anatomy, left ventricular function (both systolic and diastolic), and myocardial metabolism\[9,10\]. Consequently, it would be reassuring if myocardial tagging could corroborate these findings at the level of intrinsic cardiac muscle properties. Fortunately, in a subsequent recent article from the same group\[11\], it was demonstrated that in the athlete’s heart, torsion and untwisting remained unchanged compared with those of the control subjects, whereas patients with aortic valve stenosis clearly showed diastolic dysfunction. These findings emphasize the ability of myocardial tagging to distinguish normal from pathological conditions, and underscore the value of myocardial tagging as an independent reliable marker of myocardial function.

Another implication of the present study, not addressed by the authors, could be the use of myocardial tagging in the timing of aortic valve surgery. Generally, prophylactic aortic valve surgery is recommended in selected asymptomatic patients with severe aortic valve disease (aortic valve area ≤1·0 cm\(^2\)), whereas surgery is not indicated in patients with mild disease (aortic valve disease >1·5 cm\(^2\))\[12\]. The present study cannot answer this question mainly because of the small sample size and the wide range of aortic valve areas from 0·32–1·5 cm\(^2\), including both patients with severe and moderate aortic valve disease. Certainly when early diastolic dysfunction can be easily recognized by the use of myocardial tagging,
it might be worthwhile to investigate these issues in larger populations. The present study forms the basis to encourage and initiate such studies.

E. E. VAN DER WALL
Leiden University Medical Center,
The Netherlands,

References


Blood viscosity and the risk of death from coronary heart disease

See page 515 for the article to which this Editorial refers

In this issue Danesh et al. pooled data from prospective studies (meta-analysis) to determine if there is an association between haematocrit, viscosity and erythrocyte sedimentation rate and the incidence of coronary heart disease events[1]. They combined results from 18 studies on haematocrit and found that patients in the top third (usual values >46%) had a 30% increase in coronary heart disease events compared to those in the lower third (<42%). Pooled data from a smaller number of studies also demonstrated an increased incidence of coronary heart disease in patients with elevated plasma viscosity, total blood viscosity and elevated erythrocyte sedimentation rate. The reason for reporting these analyses together is that both haematocrit and erythrocyte sedimentation rate are related to blood viscosity.

Ideally, meta-analyses analyse combine data from randomized, controlled trials. Even then, critics emphasize the intrinsic weaknesses of such an approach, primarily because pooled results incorporate biases from each of the individual studies. New sources of bias are introduced from selection of the studies and from the inevitable heterogeneity among them[2]. In fact meta-analyses fail to predict the results of large, randomized, controlled trials (the gold standard for evaluations of efficacy of clinical interventions), 35% of the time[3]. Errors are more likely when meta-analyses are used to analyse observational studies because comparison groups have not undergone randomization and the degree and direction of biases within each study might not have been identified and are apt to lead to a false-positive association[3]. This is especially true for studies where the strength of association is less than 2; one of the most important criteria for causality.

Results from the three largest studies on haematocrit were heterogeneous. The Framingham study compared the highest fifth to the middle third of haematocrit values for the risk of dying from cardiovascular disease or coronary heart disease over a 34-year follow-up period[5]. They found that

© 2000 The European Society of Cardiology