Vortex imaging: new information gain from tracking cardiac energy loss

Gianni Pedrizzetti1 and Partho P. Sengupta2*

1Department of Engineering and Architecture, University of Trieste, Trieste, Italy; and 2Zena and Michael A. Wiener Cardiovascular Institute, School of Medicine at Mount Sinai, One Gustave L. Levy Place, PO Box 1030, New York, NY 10029, USA

During a cardiac cycle, the filling and emptying of the left ventricle (LV) are associated with characteristic patterns of blood flow motion. Diastolic filling shows an initial rapidly flowing transmitral jet, which decelerates and is subsequently redirected towards the LV outflow track. During this process of flow redirection, the formation of vortices store part of the kinetic energy of the incoming flow in a rotary motion.1–3 However, vortices are dynamical entities and therefore inherently also show marked, intrinsic instability. When they develop under unfavourable conditions they can give rise to turbulence with loss of kinetic energy due to increased friction.

The development of LV diastolic vortical intraventricular blood motion has captured the imagination of researchers in biomechanics and cardiovascular science. Numerous recent studies have demonstrated that anomalies in cardiac function are related to the changes in LV flow.3–7 In this issue of the journal, the article by Stugaard et al.8 analysed the alterations in intraventricular flow at patients with varying severity of aortic regurgitation (AR). The presence of an AR jet is expected to alter the natural swirling motion of blood within the LV and increase turbulence. Authors specifically illustrate this by tracking the two-dimensional (2D) intraventricular flow velocities using a novel echocardiographic technique of Vector Flow Mapping (VFM), which is based on the processing of color Doppler acquisitions. VFM provides the velocity component along the scan line and estimates the transversal motion with the aid of mass conservation and is therefore useful for deriving the amount of kinetic energy that is lost during the disturbed flow motion.

Energy loss is expected to increase with worsening AR and therefore may provide a novel index to grade the severity of AR. The study by Stugaard et al.8 in this issue explores this hypothesis by an experimental design in a canine model with further clinical verification in patients. The results confirm the hypothesis with highly comparable data from both experimental dog model and observations in the human subjects. The visualizations of blood flow quite remarkably illustrate the orientation of AR jet that is directed towards the posterior wall and therefore collides with the diastolic inflow coming from the mitral valve. This interference modifies the LV flow pattern and inhibits a normal vortex formation process. Figure 1 illustrates the flow pattern corresponding to an isolated mitral jet9 and the same pattern in the presence of a smaller AR jet colliding with it. Energy loss appears to be the consequence of the large shear stresses provoked by high velocities in the small regurgitant jet which creates turbulence in the LV cavity and disturbs the normal smooth swirling motion of blood.

The results of Stugaard et al.8 are however largely descriptive; therefore, their incremental clinical value may not be immediately apparent. However, a clinical interest emerges from the discussion in the article and suggests that the evaluation of AR in terms of ventricular efficiency could be useful with respect to the long-term outcome as previously hypothesized.5–7 Patients with reduced LV flow efficiency and higher energy loss may be more prone to develop maladaptive mechanisms that may lead to varying degree of eccentric hypertrophy and LV remodelling as seen in patients with AR. The progression of LV kinetic energy dissipation was previously reported in patients with acute myocardial infarction,10 suggesting a relationship between LV remodelling and energy efficiency. Indeed, the alteration of the natural vortex formation process and the generation of turbulence, as shown by Stugaard et al.8 could generate abnormal shear stress and pressure gradients that may be sensed by cardiac tissue for inducing cellular signalling pathways for initiating the process of cardiac remodelling.2,11 Future studies therefore need to assess whether patients with AR have differences in cardiac efficiencies and if correction of AR with or without reversal of the hemodynamic inefficiency are correlated with reversal or persistence of LV remodelling. Such data would eventually help us parse out the relative significance and incremental value of fluid mechanics parameters.

The merits and pitfalls of VFM technology8 also deserve further discussion. Although partly validated,12 VFM has theoretical limits of applicability. It assumes a primary direction of flow and Doppler measurements could underestimate such flow if not aligned in the same direction. The assumptions in VFM treat differently the different velocity components (anisotropy), this may lead to measurements that are not physically affected by changes in the orientation of blood motion. Moreover, the computation of the transversal velocity component is based on mass conservation on the 2D plane; however,
LV flow is three-dimensional, especially in the presence of turbulence, and energy dissipation may occur in another plane and may be underestimated in 2D. For example, an AR jet that slightly deviates out of the scan-plane may be captured only along its initial trajectory, resulting in underestimation of energy dissipation. The evaluation of energy dissipation depends crucially on the velocity gradient along multiple directions; their indisputable accurate evaluation would require much higher spatial resolution than what is currently available in color Doppler and VFM modalities. In addition, despite the use of a sophisticated technology, the need for manual adjustments during the post processing may introduce data variability.

The heart is an adaptive system where haemodynamic forces are exchanged continuously between the flowing blood and the surrounding tissues. Studies like Stugaard et al. 8 are symbolic of the burgeoning interest in the field to explore the contribution of vortices to cardiac function. It is a current belief that vortices form the primary haemodynamic link between the flowing blood and the function of surrounding tissue. Future investigations will need to specifically focus on the incremental value of cardiac fluid dynamics over known metrics of cardiac function for assessing disease severity and predicting the development of clinical end points. The evolution of such novel fluid dynamic parameters may be pivotal for optimizing the timing of therapeutic interventions and designing repair strategies for avoiding irreversible changes in cardiac function.

**Conflict of interest:** None declared.

**References**


