The Impact of Function-Flow Interaction on Left Ventricular Efficiency in Patients with Conduction Abnormalities: A Particle Image Velocimetry and Tissue Doppler Study

Emre Gürel, MD, Christian Prinz, MD, PhD, Lieve Van Casteren, MD, Hang Gao, PhD, Rik Willems, MD, PhD, and Jens-Uwe Voigt, MD, PhD, *Leuven, Belgium*

Background: The aims of this study were to assess the influence of left bundle branch block (LBBB)–like conduction abnormalities on left ventricular (LV) blood flow patterns and to characterize their potential impact on LV efficiency by measuring the changes in vortex formation and energy dissipation in the left ventricle using echocardiographic particle image velocimetry.

Methods: Thirty-six subjects were prospectively studied, including 20 patients with pacemakers, six patients with LBBB, and 10 healthy control subjects, all of whom had normal ejection fractions (>50%). In patients with pacemakers, data were acquired in both DDD and AAI modes. Standard grayscale, tissue Doppler myocardial imaging, and contrast-enhanced echocardiographic particle image velocimetric data were acquired, and LV flow patterns were analyzed using dedicated software. Dyssynchrony was quantified by measuring apical transverse motion.

Results: Apical transverse motion was significantly higher in patients with LBBB compared with normal control subjects (mean, 4.9 ± 1.9 vs 1.0 ± 0.7 mm; $P < .001$). Quantitative measures of vortex energy dissipation (relative strength, vortex relative strength, and vortex pulsation correlation) were significantly higher in patients with LBBB (2.05 ± 0.54, 0.53 ± 0.13, and 0.87 ± 0.47, respectively) compared with control subjects (1.48 ± 0.28, 0.33 ± 0.05, and 0.24 ± 0.51, respectively) ($P < .02$ for all). Vortex duration time in relation to the entire cardiac cycle was shorter in patients with LBBB than in control subjects (28% vs 44%). All findings in both groups were comparable with DDD and AAI.

Conclusion: LV flow pattern analysis by echocardiographic particle image velocimetry reveals that conduction delay due to LBBB or pacemaker stimulation in the right ventricle (DDD) disturbs the transfer of kinetic energy during the cardiac cycle and causes less efficient LV function. These data contribute to a better understanding of hemodynamic consequences of conduction delays and may help in the optimization of therapeutic approaches. (J Am Soc Echocardiogr 2016;■:■-■.)

Keywords: Particle imaging velocimetry, LV blood flow, LBBB, Apical rocking, Ventricular efficiency

Vortices are complex flow structures that contain kinetic energy due to rotating fluid.[1,2] In two dimensions, vortical flow can be quantified by the parameter vorticity (ω), which describes the difference of the gradient of the y component of the flow velocity in the x direction ($\partial V_y \partial x$) and the gradient of the x component of the flow velocity in the y direction ($\partial V_x \partial y$) according to the following formula:

$$\omega = \frac{\partial V_y}{\partial V_x} - \frac{\partial V_x}{\partial V_y}.$$

Accordingly, a counterclockwise vortex has positive vorticity and a clockwise vortex has negative vorticity, while laminar flow has zero vorticity.[3]

Vortices occur when laminar flow detaches from a sharp edge. In the human left ventricle, diastolic filling triggers vortex formation at the tips of the mitral valve leaflets.[4,5] As the moving blood of a vortex stores kinetic energy, it thereby facilitates its transmission

From the Department of Cardiovascular Diseases, University Hospital Gasthuisberg, Catholic University Leuven, Leuven, Belgium.

Dr Gürel is currently affiliated with the Department of Cardiology, Pendik State Hospital, Istanbul, Turkey. Dr Prinz is currently working in private practice in Sögel, Germany. Dr Van Casteren is currently affiliated with the Department of Rhythmology, University Hospital Liege, Liege, Belgium.

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Reprint requests: Jens-Uwe Voigt, MD, PhD, University Hospital Gasthuisberg, Department of Cardiovascular Diseases, Herestraat 49, 3000, Leuven, Belgium (E-mail: *jens-uwe.voigt@uzleuven.be*).

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Abbreviations

ATM = Apical transverse motion

ATM_{4CV} = Apical transverse motion in the four-chamber view

ε_{et} = Strain during ejection time

ε_{tot} = Strain during the entire cardiac cycle

LBBB = Left bundle branch block

LV = Left ventricular

PIV = Particle image velocimetry

RS = Relative strength

VA_{rel} = Vortex area relative to left ventricular cavity area

VD = Vortex depth

VL = Vortex length

VPC = Vortex pulsation correlation

VRS = Vortex relative strength

from diastolic inflow to systolic outflow and reduces the need for reacceleration of the blood before ejection, which increases left ventricular (LV) efficiency. Any pathology that disturbs the normal interplay of chamber mechanics and hemodynamics by preventing vortex formation might therefore impair ventricular efficiency.[6,7]

Although a singular, large vortex is a relatively steady structure, turbulence is characterized by many fast changing vortices that cause a rapid dissipation of kinetic energy. The amount of regional change in vortex structures has been referred to as "pulsatility" and can be described for the entire left ventricle as relative pulsatile vorticity strength or, for the vortex only, vortex relative pulsatile vorticity strength and the correlation between steady and pulsatile vorticity. All three parameters have been proposed as indicators of energy dissipation in the human left ventricle.[3]

Particle image velocimetry (PIV) is an image analysis method that enables the determination of flow patterns by tracking particles that move together with the fluid. Recently, this method has been successfully applied to contrast enhanced echocardiographic images. Initial studies showed that contrast tracking delivers sufficiently accurate information for the detection of vortex structures and the calculation of vorticity and other derived parameters.[8,9]

Electrical conduction abnormalities such as left bundle branch block (LBBB) patterns are common in patients with heart failure. Similarly, therapeutic right ventricular apical pacing can induce conduction abnormalities comparable with LBBB and lead to systolic dyssynchrony.[10] In both cases, the sequentially activated LV myocardial regions prestretch each other, which results in inefficient ventricular work. Consequently, hearts with LBBB-like conduction delays generate less stroke volume, have prolonged isovolumetric time intervals, and have disturbed filling.[11,12] It is unknown, however, to what extent the abnormal sequence of LV wall contraction interacts with vortex formation and the transfer of kinetic energy from diastole to systole.

The aims of this study were therefore to investigate the influence of LBBB-like conduction abnormalities on LV blood flow patterns and to characterize their potential impact on LV efficiency by measuring the changes in vortex formation and energy dissipation in the left ventricle by means of echocardiographic PIV.

METHODS

Study Population

A total of 54 subjects were prospectively screened for this study, including 23 patients with pacemakers, 21 patients with LBBB, and 10 volunteers. Bad echogenicity, reduced LV ejection fraction (<50%), regional dysfunction, more than mild valvular disease, pulmonary hypertension, previous myocardial infarction, percutaneous coronary intervention, cardiac surgery, atrial or ventricular arrhythmia, and any unstable cardiovascular condition were exclusion criteria.

Pacemaker Group. This group was recruited from the pacemaker clinic of our department (University Hospital Gasthuisberg, Leuven, Belgium). All patients underwent pacemaker testing and screening echocardiography before inclusion. We selected patients with dual-chamber pacemakers capable of working in AAI and DDD modes, with the ventricular lead placed at the right ventricular apex. Care was taken that all atrioventricular or ventricular conduction delays were intermittent and not present at the time of the investigation. Patients with permanent atrioventricular block, any intrinsic bundle branch block, or inadequate echocardiographic image quality were excluded. From 23 screened patients, three were excluded because of bad echocardiographic image quality.

LBBB Group. Patients were recruited by screening electrocardiograms from the electrocardiography service of our department. As in pacemaker patients, all patients with LBBB had undergone screening echocardiography before inclusion to rule out structural heart disease and to check for adequate echocardiographic image quality. Six patients with idiopathic LBBB and normal global LV function were included. All were in sinus rhythm, without any atrial or ventricular arrhythmia. The absence of coronary artery disease was confirmed by coronary angiography in all patients. Among 21 screened patients, 15 had to be excluded because of LV dysfunction ($n = 3$), coronary artery disease ($n = 3$), atrial fibrillation ($n = 5$), and bad image quality ($n = 4$).

Normal Control Subjects. Finally, 10 healthy volunteers without any histories of cardiovascular disease and with normal findings on electrocardiography, physical examination, and echocardiography were recruited to participate in this study. All screened volunteers could be included.

All participants gave written informed consent before inclusion. The study was approved by the local ethics committee.

Pacing Protocol

Pacemakers were programmed to a heart rate slightly higher than the intrinsic sinus activity to allow constant atrial pacing. All echocardiographic data were acquired twice, in both AAI and DDD pacing modes. Five minutes of hemodynamic adaptation were allowed after mode change. The order of modes was random. For DDD pacing, the atrioventricular delay was adjusted so that full ventricular capture was achieved, but no A-wave truncation occurred.

Echocardiographic Image Acquisition

A commercially available Vivid 7 ultrasound scanner was used (GE Vingmed Ultrasound AS, Horten, Norway) to acquire complete standard transthoracic echocardiograms. To characterize regional myocardial function, color tissue Doppler myocardial imaging data were acquired from parasternal (parasternal long-axis) and apical (four-, three-, and two-chamber view) windows with optimized sector and depth settings to achieve high frame rates.

For echocardiographic PIV, dedicated two-dimensional grayscale images with the highest possible frame rate (95–110 frames/sec) were acquired from three apical planes using a Siemens Acuson Sequoia ultrasound system (Siemens Medical Solutions USA, Mountain View, CA). We used SonoVue (Bracco, Milan, Italy) as a contrast agent, which contains microbubbles in a concentration of 1×10^8 to 5×10^8 per milliliter.[13] This suspension was injected