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Contribution of the Diastolic Vortex Ring to Left Ventricular Filling

Pablo Martínez-Legazpi, MEng, PhD,* Javier Bermejo, MD, PhD,† Yolanda Benito, DCS, DVM,† Raquel Yotti, MD, PhD,† Candelas Pérez del Villar, MD,† Ana González-Mansilla, MD, PhD,† Alicia Barrio, DCS, MBiol,† Eduardo Villacorta, MD,† Pedro L. Sánchez, MD, PhD,† Francisco Fernández-Avilés, MD, PhD,† Juan C. del Álamo, AeEng, PhD*‡

ABSTRACT

BACKGROUND Intraventricular fluid dynamics can be assessed clinically using imaging. The contribution of vortex structures to left ventricular (LV) diastolic function has never been quantified in vivo.

OBJECTIVES This study sought to understand the impact of intraventricular flow patterns on filling and to assess whether impaired fluid dynamics may be a source of diastolic dysfunction.

METHODS Two-dimensional flow velocity fields from color Doppler echocardiographic sequences were obtained in 20 patients with nonischemic dilated cardiomyopathy (NIDCM), 20 patients with hypertrophic cardiomyopathy (HCM), and 20 control healthy volunteers. Using a flow decomposition method, we isolated the rotational velocity generated by the vortex ring from the surrounding flow in the left ventricle.

RESULTS The vortex was responsible for entering 13 ± 6% of filling volume in the control group and 19 ± 8% in the NIDCM group ($p = 0.004$), but only 5 ± 5% in the HCM group ($p < 0.0001$ vs. controls). Favorable vortical effects on intraventricular pressure gradients were observed in the control and NIDCM groups but not in HCM patients. Differences in chamber sphericity explained variations in the vortex contribution to filling between groups ($p < 0.005$).

CONCLUSIONS The diastolic vortex is responsible for entering a significant fraction of LV filling volume at no energetic or pressure cost. Thus, intraventricular fluid mechanics are an important determinant of global chamber LV operative stiffness. Reduced stiffness in NIDCM is partially related to enhanced vorticity. Conversely, impaired vortex generation is an unreported mechanism of diastolic dysfunction in HCM and probably other causes of concentric remodeling. (J Am Coll Cardiol 2014;64:1711–21) © 2014 by the American College of Cardiology Foundation.

Little is known about the relationship between diastolic function and the complex flow dynamics that occur during ventricular filling (1). The large vortical flow structures that develop during diastole appear particularly relevant. These vortices are a consequence of the heart's chiral geometry and the interaction of the filling jet with the walls and mitral valve of the left ventricle (LV), but their effects on chamber hemodynamics remain incompletely understood. Once generated, vortices are relatively longstanding inertial flow structures capable of entraining fluid and decreasing pressure in their vicinity without an energetic cost of the driving system. Thus, a leading vortex can transport

From the *Mechanical and Aerospace Engineering Department, University of California, San Diego, La Jolla, California; †Department of Cardiology, Hospital General Universitario Gregorio Marañón, Facultad de Medicina, Universidad Complutense de Madrid, and the Instituto de Investigación Sanitaria Gregorio Marañón, Madrid, Spain; and the ‡Institute for Engineering in Medicine, University of California, San Diego, La Jolla, California. This study was supported by grants PI12/02885, PIS09/02603, RD12/0042 (Red de Investigación Cardiovascular), and CM12/00273 (to Dr. Pérez del Villar) from the Instituto de Salud Carlos III-Ministerio de Economía y Competitividad, Spain, and National Institutes of Health grant 1R21 HL108268-01 (to Dr. del Álamo). Drs. Pérez del Villar and González-Mansilla have been partially supported by grants from the Fundación para Investigación Biomédica Gregorio Marañón, Madrid, Spain. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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ABBREVIATIONS AND ACRONYMS

2D+t = time-resolved 2-dimensional

ANOVA = analyses of variance

DIVPD(G) = diastolic intraventricular pressure difference (gradient)

HCM = hypertrophic cardiomyopathy

LV = left ventricle/ventricular

NIDCM = nonischemic dilated cardiomyopathy

more mass than an equivalent straight jet of fluid (2,3). In the LV, vortices conserve kinetic energy between the intermittent periods of the cardiac cycle (4,5). Therefore, we hypothesized that vortex rings could play a major role in cardiac diastolic function.

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Global LV chamber passive stiffness is determined by myocardial tissue material stiffness and thickness (6). In patients with hypertrophic cardiomyopathy (HCM), increased LV passive chamber stiffness is believed to be caused by stiffening and thickening of the cellular and extracellular components of the myocardium, whereas in nonischemic dilated cardiomyopathy (NIDCM), wall thinning contributes to typically seen reduced chamber stiffness (7). However, structural changes of the myocardium do not completely explain modifications in global chamber stiffness induced by chamber remodeling (8). Although chamber volume and shape additionally affect chamber stiffness, the mechanisms by which LV geometry influences such stiffness are not fully understood.

Given that vortex properties depend greatly on chamber geometry (5,9), we hypothesized that, in addition to structural changes in the myocardial compartment, modified flow dynamics, caused by abnormal chamber geometry, may contribute to the chamber stiffness characteristic of NIDCM and HCM. The present study was designed to assess the contribution of the intraventricular vortex to LV filling in normal and remodeled hearts. We used a previously validated algorithm to reconstruct the time-resolved 2-dimensional (2D+t) velocity field from color Doppler echocardiographic studies. By means of a velocity decomposition method, we were able to quantify, for the first time, the capacity of the diastolic vortex ring to transport LV filling volume in normal and abnormal ventricles.

TABLE 1 Demographic and Echocardiographic Data

	Control (n = 20)	NIDCM (n = 20)	HCM (n = 20)
Sex, M/F	4/16	10/10*	13/7*
Age, yrs	60 ± 10	63 ± 10	51 ± 13*
NYHA functional class			
I	20	6	12
II	0	10	6
III	0	4*	2*
Heart rate, beats/min	68 ± 17	67 ± 12	56 ± 9*
QRS duration >120 ms	0 (0)	11 (55)*	3 (15)
LV chamber			
End-diastolic volume, ml	78 ± 18	150 ± 71*	76 ± 22
End-systolic volume, ml	30 ± 8	114 ± 67*	25 ± 10
Stroke volume, ml	49 ± 11	37 ± 14*	51 ± 16
Ejection fraction, %	62 ± 5	26 ± 9*	69 ± 6*
LV mass, g	127 ± 37	223 ± 74*	441 ± 135*
LV diameter-to-posterior wall thickness ratio (dimensionless)	5.45 ± 1.09	7.13 ± 2.24*	3.38 ± 0.83*
LV sphericity (dimensionless)	0.51 ± 0.06	0.71 ± 0.12*	0.36 ± 0.09*
Conventional Doppler			
Mitral regurgitation 3+ or 4+, n	0	7*	1
IVRT, ms	99 ± 23	138 ± 32*	89 ± 30
E-wave velocity, m/s	0.67 ± 0.11	0.63 ± 0.21	0.75 ± 0.28
A-wave velocity, m/s	0.67 ± 0.19	0.75 ± 0.23	0.68 ± 0.3
E/A	1.07 ± 0.34	0.92 ± 0.46	1.24 ± 0.55
E-wave deceleration time, ms	204 ± 50	166 ± 51*	207 ± 59
e′ wave velocity, cm/s	9.4 ± 2.9	4.3 ± 2.3*	4.8 ± 1.9*
E/e′	7.8 ± 2.6	17.4 ± 7.9*	18 ± 9.8*
Myocardial deformation			
Peak systolic longitudinal strain, %	−18.9 ± 3.3	−8.8 ± 3.3*	−13.6 ± 3.7*
Peak systolic transversal strain, %	−18.8 ± 14.7	−12.4 ± 8.8	−16.5 ± 10.7
Peak systolic longitudinal strain rate, s^{-1}	−1.02 ± 0.12	−0.46 ± 0.18*	−0.77 ± 0.21*
Peak early diastolic longitudinal strain rate, s^{-1}	1.01 ± 0.28	0.41 ± 0.14*	0.64 ± 0.29*
Peak early diastolic transversal strain rate, s^{-1}	1.89 ± 1.09	0.95 ± 0.67*	1.39 ± 0.74
Peak late diastolic longitudinal strain rate, s^{-1}	1.07 ± 0.28	0.54 ± 0.25*	0.66 ± 0.28*
Peak late diastolic transversal strain rate, s^{-1}	1.14 ± 0.47	0.78 ± 0.41*	0.89 ± 0.47

Values are n, mean ± SD, or n (%). *p < 0.05 vs. control.

HCM = hypertrophic cardiomyopathy; IVRT = isovolumic relaxation time; LV = left ventricular; NIDCM = nonischemic dilated cardiomyopathy; NYHA = New York Heart Association.

METHODS

PATIENTS. Sixty subjects were prospectively selected (Table 1). Inclusion criteria for all study participants were: 1) the presence of sinus rhythm; 2) a suitable apical ultrasonic window; 3) absence of relevant aortic regurgitation (<2+); and 4) absence of E-A-wave fusion on the transmitral filling flow profile. Twenty patients with NIDCM were randomly selected from a large group recruited on the basis of angiographically proven absence of significant coronary artery disease and a stable clinical status (5). Another 20 patients with a firm diagnosis of HCM (based on clinical, familial, and genetic data) were selected from the outpatient clinic, 9 of which had obstructive disease (intraventricular pressure gradient of 51 ± 33 mm Hg in this subgroup). Twenty controls were also randomly selected from a large control population of normal subjects without known or suspected cardiovascular disease, with normal electrocardiographic and Doppler echocardiographic examinations, and with no history of hypertension or diabetes (5). The study was approved by the institutional review board, and all participants provided written informed consent.