Title
Comments on Defining the Contribution of Diastolic Vortex Ring to Left Ventricular Filling

Journal
Journal of the American College of Cardiology, 65(23)

ISSN
0735-1097

Authors
Pedrizzetti, Gianni
Vlachos, Pavlos P
Little, William C
et al.

Publication Date
2015-06-01

DOI
10.1016/j.jacc.2014.12.076

Peer reviewed

*Olle Melander, MD, PhD
Dov Shiffman, PhD
Michael P. Caulfield, PhD
Judy Z. Louie, MsC
Charles M. Rowland, MsC
James J. Devlin, PhD
Ronald M. Krauss, MD

*Skåne University Hospital Clinical Research Center
Ent 72, Building 91
Floor 12
Skåne University Hospital
Malmö SE 20502
Malmö
Sweden
E-mail: olle.melander@med.lu.se

http://dx.doi.org/10.1016/j.jacc.2015.02.077

Please note: Dr. Shiffman, Dr. Caulfield, Ms. Louie, Mr. Rowland, and Dr. Devlin are employees of Quest Diagnostics. Dr. Caulfield, Dr. Rowland, and Dr. Devlin report stock ownership in Quest Diagnostics. Dr. Krauss is a recipient of a research grant from and serves as a consultant for Quest Diagnostics. Dr. Melander has reported that he has no relationships relevant to the contents of this paper to disclose.

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Comments on Defining the Contribution of Diastolic Vortex Ring to Left Ventricular Filling

With much interest, we read the recent article by Martínez-Legazpi et al. (1), which suggests insights into the role of diastolic vortex formation in filling of the left ventricle. We are delighted to see that fluid dynamics is increasing its presence in the clinical cardiology community and contributes to an improved understanding of cardiac function as underlined in the editorial comment (2).

The article by Martínez-Legazpi et al. (1) introduces a technique to evaluate the contribution of the left ventricular (LV) vortex to diastolic filling using intraventricular velocity estimated by 2-dimensional color Doppler echocardiography. They aimed to quantify the percentage of diastolic filling volume related to the LV vortex and tried to distinguish between normal and abnormal LV function. Their method is based on dividing the intraventricular velocity field into 2 components where one is the rotational flow, directly related to the presence of the LV vortex, and the other is irrotational, a mathematical approach known as Stokes decomposition widely applied in fluid dynamics.

Accordingly, Martínez-Legazpi et al. (1) first isolated the LV vortex and subsequently tried to adjust the corresponding velocity to account for the presence of the LV wall and mitral leaflets. Based also on the communication that we had with some of the authors, we understood that the method is initiated by computing the rotational velocity under the assumption that the LV wall is rigid, which should yield a zero volume for transmitral inflow. However, this results in an estimate of a new transmitral inflow volume that is nonzero. Then the method repeats the same procedure that always results in an inflow volume that is different from the expansion volume, and this process continues until the solution converges to a value.

One major concern with this method is that once the LV wall is assumed to be rigid and the ventricle is full of blood, it is physically impossible to achieve a nonzero transmitral inflow volume. Similarly, when the LV wall expands, its volume increase cannot differ from the transmitral inflow volume. The fact that these volumes differ during the individual iterative steps is a direct violation of the fundamental law of the conservation of mass, and reveals a questionable calculation in their method. This flaw calls into question the study's methodology and subsequent results.

Moreover, according to the fluid dynamics conservation laws, the role of the LV vortex in diastolic filling volume cannot be evaluated merely by volumetric balances, but needs to be considered in conjunction with the corresponding momentum balances and intraventricular pressure gradients. Here, the second controversial notion introduced by Martínez-Legazpi et al. (1) stems from the decomposition of the pressure gradient into a rotational and an irrotational component. The analysis did not take into consideration that every gradient field is a conservative field by definition; hence, it has no rotational

component. Therefore, the pressure decomposition presented in their analysis is rather improper for fluid dynamics, and we do not believe that it should be used for such a purpose.

In conclusion, this letter aims to bring attention to the analyses reported by Martínez-Legazpi et al. (1) and the supporting editorial views on the article (2). Although the concerns raised here are highly technical, we believe that they may have an impact on the validity of the reported methodology and results, and consequently, their clinical implications. We hope that the present letter clarifies these technical, albeit fundamentally significant, points on this published study (1).

Gianni Pedrizzetti, PhD
Pavlos P. Vlachos, PhD
William C. Little, MD
Fotis Sotiropoulos, PhD
Morteza Gharib, PhD
***Arash Kheradvar, MD, PhD**

*Department of Biomedical Engineering, and Medicine
University of California Irvine
2410 Engineering Hall
Irvine, California 92697
E-mail: arashkh@uci.edu

http://dx.doi.org/10.1016/j.jacc.2014.12.076

Please note: Dr. Little is a consultant for Medtronic, CVRx, CorNovus, and CorAssist. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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REPLY: Comments on Defining the Contribution of Diastolic Vortex Ring to Left Ventricular Filling

We read with great interest the letter by Pedrizzetti and colleagues Because our study (1) has raised some concerns among these investigators, here we are pleased to reassure the readers of *JACC* about the validity of our findings.

The impact of a vortex on the intraventricular flow is conditioned by its effects on the chamber's wall movements, which cannot be predicted a priori without considering the mechanical properties of the walls and the momentum exchange between the walls and the fluid. It is true that uncoupling these effects from the rest of the flow is a challenging task that must be accomplished while enforcing fluid dynamics conservation laws. We were aware of this issue from the very onset of our study design. Thus, we defined that a fraction of wall velocity is caused by the vortex. This fraction was entered as a single free parameter (lambda, in our Online Appendix) in our calculations.

Making use of a novel iterative method, we updated lambda until mass conservation was ensured. Briefly, we started the calculation of the vortex flow with the assumption of rigid walls and thus null lambda. This resulted in values of flow velocity between the tips of the mitral valve, which were not null. Inflow volume was measured by time integrating this localized velocity under a 1-dimensional flow assumption. Note that this 1-dimensional approximation has been routinely used by the authors of the letter (2,3) and by others (4) for measuring inflow and has provided important new insight into left ventricular physiology. Finally, we updated lambda and repeated the procedure until the new transmitral inflow volume matched the expansion volume dictated by lambda. Importantly, convergence was achieved in fewer than 5 iterations in all cases. Furthermore, convergence to the same value of lambda was achieved regardless of the initial assumption about wall motion (i.e., the same result was obtained for any initial value of lambda), and the measured inflow volume was only slightly altered by the change in boundary conditions.

By using this method we were able to demonstrate some of the physiological implications of intraventricular vorticity in a clinical scenario using clinical measurements. This was one of the major strengths of our paper, as emphasized by the reviewers and the accompanying editorial (5).

Regarding the second concern of Pedrizzetti and colleagues, which related to the conservation of momentum, we estimated the rotational velocity field directly from the measured velocity field. The latter is the result of the balance of fluid momentum pressure and the fluid-structure interaction with the ventricular walls. It is from the resultant velocities that we reconstructed the pressure gradient fields and not vice versa. This approach has also been used before by the authors of the letter to the editor (3).

Thus, no physical law was inappropriately used in our study. Although the numerical values reported are approximate, we would like to reinforce the validity of our results and their clinical implications.

Pablo Martínez-Legazpi, MEng, PhD
***Javier Bermejo, MD, PhD**
Yolanda Benito, DCS, DVM