Circulation: Cardiovascular Imaging

ORIGINAL ARTICLE

Interobserver Variability in Applying American Society of Echocardiography/European Association of Cardiovascular Imaging 2016 Guidelines for Estimation of Left Ventricular Filling Pressure

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BACKGROUND: Assessment of left ventricular (LV) filling pressure is among the important components of a comprehensive echocardiographic report. Previous studies noted wide limits of agreement using 2009 American Society of Echocardiography/European Association of Echocardiography guidelines, but reproducibility of 2016 guidelines update in estimating LV filling pressure is unknown.

METHODS: Echocardiographic and hemodynamic data were obtained from 50 patients undergoing cardiac catheterization for clinical indications. Clinical and echocardiographic findings but not invasive hemodynamics were provided to 4 groups of observers, including experienced echocardiographers and cardiology fellows. Invasively acquired LV filling pressure was the gold standard.

RESULTS: In group I of 8 experienced echocardiographers from the guidelines writing committee, sensitivity for elevated LV filling pressure was 92% for all observers, and specificity was 93±6%. Fleiss κ-value for the agreement in group I was 0.80. In group II of 4 fellows in training, sensitivity was 91±2%, and specificity was 95±2%. Fleiss κ-value for the agreement in group II was 0.94. In group III of 9 experienced echocardiographers who had not participated in drafting the guidelines, sensitivity was 88±5%, and specificity was 91±7%. Fleiss κ-value for the agreement in group III was 0.76. In group IV of 7 other fellows, sensitivity was 91±3%, and specificity was 92±5%. Fleiss κ-value for the agreement in group IV was 0.89.

CONCLUSIONS: There is a good level of agreement and accuracy in the estimation of LV filling pressure using the American Society of Echocardiography/European Association of Cardiovascular Imaging 2016 recommendations update, irrespective of the experience level of the observer.

Key Words: diastole ■ echocardiography ■ echocardiography, Doppler ■ guideline ■ humans

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CLINICAL PERSPECTIVE

Assessment of left ventricular diastolic function is important for determination of underlying cause of dyspnea. Echocardiography is a readily available, versatile technique that can accomplish this objective. American Society of Echocardiography/European Association of Cardiovascular Imaging guidelines for the evaluation of left ventricular diastolic function have good accuracy and good agreement when variability is tested among observers with different levels of background and experience. The routine assessment and reporting of left ventricular diastolic function and filling pressure is, therefore, encouraged. Additional studies are of value in looking at more novel indices of cardiac function, their variability, and what incremental information they can add in different clinical settings.

Elevated left ventricular (LV) filling pressure is one of the important reasons behind shortness of breath in patients with heart failure. The diagnosis is most conveniently established by echocardiography. ASE (American Society of Echocardiography) and European Association of Echocardiography published the first set of guidelines about echocardiographic evaluation of LV diastolic function in 2009.[1] In these guidelines, the writing group recommended consideration of several measurements but allowed for each reader to judge the hierarchal importance of individual variables based on the clinical setting, as well as the technical and the pathophysiologic limitations and applications. When the 2009 guidelines were tested among a group of senior echocardiographers, 1 study reported wide interobserver variability, which was attributed, in part, to the inclusion of numerous measurements and the lack of clear recommendation on the number of abnormal variables needed to establish the diagnosis and their hierarchy.[2] In the latter study, the investigators used imaging data from 20 patients only and did not compare the accuracy of the assessment by different observers against invasive LV filling pressure as the gold standard.[2] More recently in 2016, ASE and EACVI (European Association of Cardiovascular Imaging) updated LV diastolic function evaluation guidelines.[3] To our knowledge, there are no data validating the reproducibility and accuracy of applying the most recent guidelines by different observers with variable experience, which is important because the ultimate utility depends on the correct application of the guidelines. Thus, we sought to address this question in a sample of 50 patients who were randomly selected from a study group undergoing echocardiographic imaging and cardiac catheterization aimed at looking at the accuracy of the 2016 ASE/EACVI updated guidelines for estimating LV filling pressure.

METHODS

The data and study material will not be made available in public to other researchers. Echocardiographic and hemodynamic data were obtained from 50 patients who underwent cardiac catheterization for clinical indications. The study was approved by institutional review board, and patients provided written informed consent. The only exclusions were the presence of mitral valve stenosis, mitral valve repair, prosthetic mitral valves, heart transplants, and complex congenital heart disease. In each of these diseases, there are limitations to several of the echocardiographic parameters that are used to draw conclusions about LV filling pressure and hence the exclusion of patients with these findings. Patients were selected at random provided they did not meet the exclusion criteria. The presence of a complete data set was not a requisite for inclusion because the goal was looking at the day-to-day application of the 2016 ASE/EACVI guideline update, including feasibility and not only accuracy. Echocardiography was performed either during or immediately post-cardiac catheterization and without intervening clinical change or administration of medications.

Echocardiographic Studies and Cardiac Catheterization

Patients were imaged, and measurements were performed according to standard guidelines.[3,4] Measurements included biplane LV volumes and ejection fraction (EF), biplane left atrium (LA) maximum volume index, mitral inflow velocities (early diastolic velocity or E and late diastolic velocity or A, and E/A ratio), tissue Doppler mitral annulus velocities (septal, lateral, average e′ velocity, and E/e′ ratio) by pulse-wave Doppler, peak tricuspid regurgitation velocity from multiple windows, and when satisfactorily recorded, pulmonary vein velocities. Severity of mitral regurgitation was determined per guideline criteria.[5] Echocardiographic measurements, heart rate, blood pressure, and clinical findings were communicated to the reviewers who were blinded to patient identity. The consideration of clinical status is in keeping with the 2016 update recommendations, which emphasize the need to consider clinical status as the first step in the application of the guidelines.[3] The findings simulate the day-to-day practice in a busy laboratory because not all signals were available, and several patients had merging of mitral E and A velocities, were in a paced rhythm, or atrial fibrillation.

LV filling pressure was mean wedge pressure in 34 patients obtained by right heart catheterization and LV pre-A pressure in 16 patients obtained during left heart catheterization. Mean wedge pressure (pulmonary capillary wedge pressure) and LV pre-A pressure are closely related,[6] and thus LV pre-A pressure was used as a surrogate for mean wedge pressure, which is also consistent with recommendations of the 2016 guidelines.[3] An LV filling pressure >12 mm Hg was considered elevated based on recommendations for diagnosis of heart failure with normal LV EF.[7]

Analysis of Interobserver Variability

The main objective was to look at variability in applying the guidelines and not in performing actual measurements. Thus, we used a similar design to a previous study[2] addressing this objective, which did not evaluate variability in measurements performed by different observers. The tabulated data of each patient, including clinical and echocardiographic findings but not invasive hemodynamics, were provided to 4 groups of observers. In the application of the guidelines, the different observers took notice of clinical status and 2-dimensional (2D) and Doppler findings as recommended in the guidelines.[3] In patients with normal LV EF, the presence of clinical diagnoses associated with diastolic dysfunction and heart failure with preserved EF was considered as hypertensive cardiovascular disease (hypertension plus pathological LV hypertrophy based on LV mass index or hypertension plus LA enlargement based on LA maximum volume index), diabetes mellitus, chronic kidney disease, and coronary artery disease with segmental dysfunction. In many laboratories, these data are routinely obtained and are available to the interpreting physicians being readily accessible from electronic health records. After consideration of clinical and 2D data, patients were determined to have one of the following: myocardial disease with normal EF, depressed EF, or none. If the answer was affirmative (depressed EF or myocardial disease with normal EF), then estimation of LV filling pressure proceeded as recommended in the specific algorithm in the 2016 guidelines (see Figure 8B in the guidelines by Nagueh et al[3]). On the contrary, if there was no evidence of myocardial disease in patients with normal EF, then the 4 variables (mitral annulus velocities, average E/e′ ratio, LA maximum volume index, and peak tricuspid regurgitation velocity by continuous wave Doppler from multiple windows) were evaluated to arrive at a conclusion of whether diastolic dysfunction was present or absent (see Figure 8A in the guidelines by Nagueh et al[3]). If 2 of 3 or 3 of 4 signals were abnormal, diastolic dysfunction was deemed present and the exercise of LV filling pressure estimation followed (see Figure 8B in the guidelines by Nagueh et al[3]).

We sought to include investigators from the diastolic guidelines writing group members, as well as other investigators who did not participate in drafting the guidelines. This would allow for comparison between investigators who are the most familiar with the guidelines and other investigators who would be applying them the way they interpret the guidelines. In addition, we wanted to compare the performance of experts and trainees. Thus, 1 group was comprised of 8 experienced echocardiographers who were members of the 2016 diastolic function guidelines writing group. They included physicians from Belgium, Italy, Norway, Romania, Sweden, and the United States. The second group was comprised of 4 fellows undergoing training in cardiology from 2 institutions in the United States who had members in the guidelines writing group (M.A.C.-p., K.C.E.t., S.C.H., J.G.). The third group was comprised of echocardiographers who were not members of the writing group. They included physicians from France, Italy, the Netherlands, and the United States. The fourth group was comprised of 7 fellows from the same institutions of group 3 observers (A.B., A.H., G.D., R.E., R.Z.M., M.P., S.R.S.).

The trainees participating in the analysis were designated by the lab head, but there was no a priori testing of their accuracy or agreement with the experienced echocardiographer. The invasively acquired LV filling pressure was the gold standard against which the accuracy of the different observers was tested.

Statistical Analysis

Sensitivity and specificity of the different observers in detecting elevated LV filling pressure were calculated, and their mean and SD values, as well as median and range were obtained. Agreement between the different members of each group of observers was assessed by Fleiss κ-statistics, and the 95% CIs are presented in the results. The sample size was based on an expected margin of error at 12% to 15% of physicians agreeing with a statement pertaining to status of LV filling pressure. At a sample size of 50, if 70% of reviewers agree to the statement, then between 57% and 83% (expected margin of error is ≈13%) of all physicians will agree to the same statement. All analyses were performed with STATA, version 14 (Stata Corp LP, College Station, TX). Sherif F. Nagueh, MD, had full access to all the data in the study and takes responsibility for its integrity and the data analysis.

RESULTS

The diagnoses, heart rate and rhythm, presence and severity of mitral regurgitation, and 2D findings for each patient are shown in Tables 1 and 2. All patients were in sinus rhythm except 4 patients with a paced rhythm and 1 patient in atrial fibrillation. There were 22 patients with EF <50%. None of the patients had more than mild mitral annular calcification. Table 2 presents the echocardiographic measurements and invasive LV filling pressure. Figures 1 and 2 show examples from 2 patients. Movies I through III in the Data Supplement show the 2D findings from the patient with Doppler data in Figure 2. Mitral inflow (E/A ratio and peak E velocity) was sufficient to estimate LV filling pressure in 14 patients, and other parameters were needed in the remaining patients. Two patients had indeterminate LV filling pressure by the 2016 ASE/EACVI guidelines.

Agreement Between Group I (Guidelines Writing Group Members) Experienced Observers in the Estimation of LV Filling Pressure

Sensitivity for elevated LV filling pressure was identical for all 8 experienced observers at 92% using the invasive measurement as the gold standard (Table 3). Specificity was 93±6% (range, 84%–100%). Fleiss κ-value for the agreement between the different observers was 0.80 with 95% CIs of 0.73 to 0.88.

Table 1. Clinical Diagnosis of the 50 Patients

Patient No.	Clinical Diagnosis	Patient No.	Clinical Diagnosis
1	DCM on milrinone	26	CAD, PVD, CKD, AS, CABG, on dobutamine
2	CAD, CKD, DM	27	HTN CVD, PVD
3	DCM on IV Lasix	28	CAD, HTN CVD
4	HFpEF, HTN CVD, CKD	29	HFpEF, HTN CVD
5	CAD, CABG, HTN	30	HTN, CAD, DM, HFrEF
6	HTN CVD	31	HTN CVD, CAD, CABG, COPD
7	HTN CVD, CKD	32	HFpEF, HTN CVD
8	Peripartum cardiomyopathy	33	Dilated cardiomyopathy
9	Nonischemic cardiomyopathy	34	CAD
10	HTN CVD, CKD	35	CAD
11	COPD	36	CAD
12	PCI to proximal LAD	37	HTN, pulmonary disease
13	PCI to proximal LAD	38	HTN, paroxysmal atrial fibrillation
14	PCI to proximal and mid-LAD	39	COPD
15	HFpEF, HTN CVD	40	Dilated cardiomyopathy
16	HFpEF, HTN CVD	41	HFrEF
17	CAD, ischemic cardiomyopathy	42	HTN CVD
18	Severe AS, CAD, CKD	43	HTN CVD
19	HFpEF, HTN CVD, CAD, DM	44	Dilated cardiomyopathy
20	HFpEF, HTN CVD	45	Dilated cardiomyopathy
21	CAD	46	HTN CVD, CKD
22	HFpEF, HTN CVD	47	Dilated cardiomyopathy
23	HFrEF	48	CAD, persistent atrial fibrillation
24	CAD, acute MI, HTN	49	Precapillary pulmonary HTN
25	CAD, CABG, DM, HTN, CKD	50	CAD, severe tricuspid regurgitation

HTN CVD was diagnosed in the presence of HTN and LA enlargement or LV hypertrophy as determined by LV mass index. AS indicates aortic stenosis; CABG, coronary artery bypass graft surgery; CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; DCM, dilated cardiomyopathy; DM, diabetes mellitus; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HTN, hypertension; HTN CVD, hypertensive cardiovascular disease; IV, intravenous; LA, left atrium; LAD, left anterior descending coronary artery; LV, left ventricle; MI, myocardial infarction; PCI, percutaneous coronary intervention; and PVD, peripheral vascular disease.

Agreement Between Group II (Fellows in Training) in the Estimation of LV Filling Pressure

Sensitivity for elevated LV filling pressure was 91±2% and had a range between 88% and 92%, using the invasive measurement as the gold standard. Likewise, specificity was excellent at 95±2% and had a range between 92% and 96% (Table 3). Fleiss κ-value for the agreement between the 4 trainees was 0.94 (95% CI, 0.88–1.00).

Agreement Between Group III Experienced Observers (Not Members of the Guidelines Writing Group) in the Estimation of LV Filling Pressure

Sensitivity for elevated LV filling pressure was 88±5% and had a range between 80% and 92%, using the invasive measurement as the gold standard (Table 3). Specificity was 91±7% (range, 80–100). Fleiss κ-value for the agreement was 0.76 (95% CI, 0.68–0.84).

Agreement Between Group IV (Fellows in Training) in the Estimation of LV Filling Pressure

Sensitivity for elevated LV filling pressure was 91±3% and had a range between 84% and 92%, using the invasive measurement as the gold standard. Specificity was 92±5%, and had a range between 88% and 100% (Table 3). Fleiss κ-value for the agreement was 0.89 (95% CI, 0.83–0.96).

Agreement in Grading LV Diastolic Dysfunction

Good agreement was present in grading LV diastolic dysfunction. Fleiss κ-value for the agreement between

Table 2. Summary Statistics of Clinical, 2-Dimensional, and Doppler Findings in the 50 Patients

Parameter	
Age, y	58±13
Heart rate (per min)	73±16
Rhythm	Sinus in 42 patients, paced rhythm in 4, sinus tachycardia in 3, and atrial fibrillation in 1 patient
MR severity	No MR in 19, trivial MR in 6, mild MR in 13, moderate MR in 10, and severe MR in 2 patients
LV EF, %	51±19.7
LA maximum volume index, mL/m^2	44±21
Peak E velocity, cm/s	84.6±28.5
E/A ratio	1.6±1.1
Peak TR velocity, m/s	2.8±0.6
Average E/e′ ratio	13.5±6.7
Invasive LV filling pressure, mm Hg	16±10

A indicates peak mitral late diastolic velocity; E, peak early diastolic velocity; e′, early diastolic velocity by tissue Doppler; EF, ejection fraction; LV, left ventricle; MR, mitral regurgitation; and TR, tricuspid regurgitation.

the 8 observers in group I was 0.82 (95% CI, 0.74–0.91). Fleiss κ-value for the agreement between the 4 trainees in group II was 0.96 (95% CI, 0.91–1.00). Fleiss κ-value for the agreement between the observers in group III was 0.75 (95% CI, 0.66–0.84). Fleiss κ-value for the agreement between the observers in group IV was 0.80 (95% CI, 0.72–0.88).

DISCUSSION

To our knowledge, this is the first report looking at the interobserver variability in applying ASE/EACVI 2016 guidelines for the evaluation of LV filling pressure. Sensitivity and specificity for detecting elevated LV filling pressure were good irrespective of the experience level.

Sources of Variability in Evaluating LV Diastolic Function

There are several reasons that can lead to increased variability in the assessment of LV diastolic function. These include technical factors, as well as analysis factors. Although technical aspects were not evaluated in this study, suboptimal signals if acquired and used to draw inferences about LV filling pressure can lead to erroneous conclusions not only because of their spurious values but also because of large variation in the

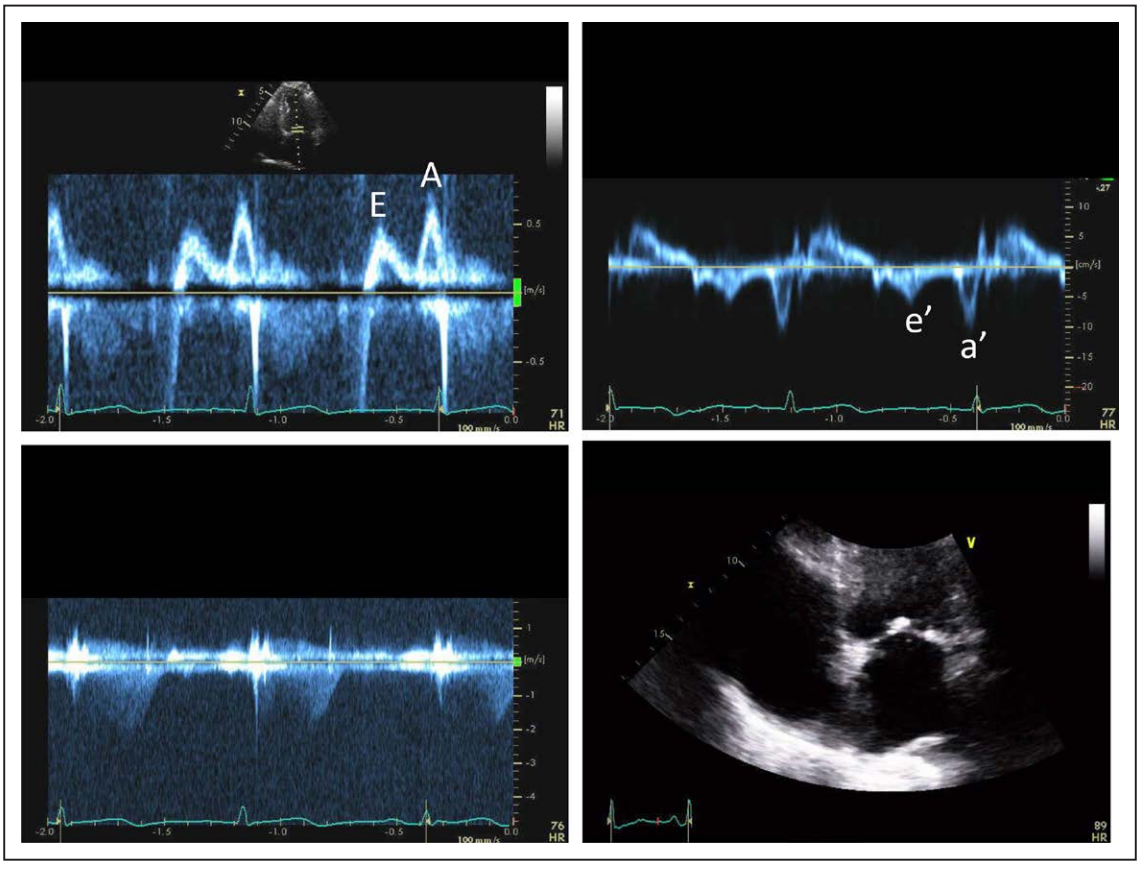

Figure 1. Mitral inflow (upper left), lateral tissue Doppler (TD) velocities (upper right), tricuspid regurgitation velocity by continuous-wave Doppler (lower left), and zoomed view for the left atrium in apical 4-chamber view (lower right) from a patient with hyperdynamic LVEF.
Mitral inflow shows impaired relaxation pattern with mitral peak early diastolic velocity (E)/peak mitral late diastolic velocity (A) ratio <0.8 and peak E velocity <50 cm/s. Lateral mitral annulus TD mitral annulus early diastolic velocity by TD (e′) velocity is reduced at 6 cm/s, with average E/e′ ratio at 9.5. Left atrium maximum volume index was normal, and peak tricuspid regurgitation velocity was rather incomplete. LV filling pressure was normal at 8 mm Hg. a′ indicates mitral annulus late diastolic velocity by TD; and LVEF, left ventricular ejection fraction.

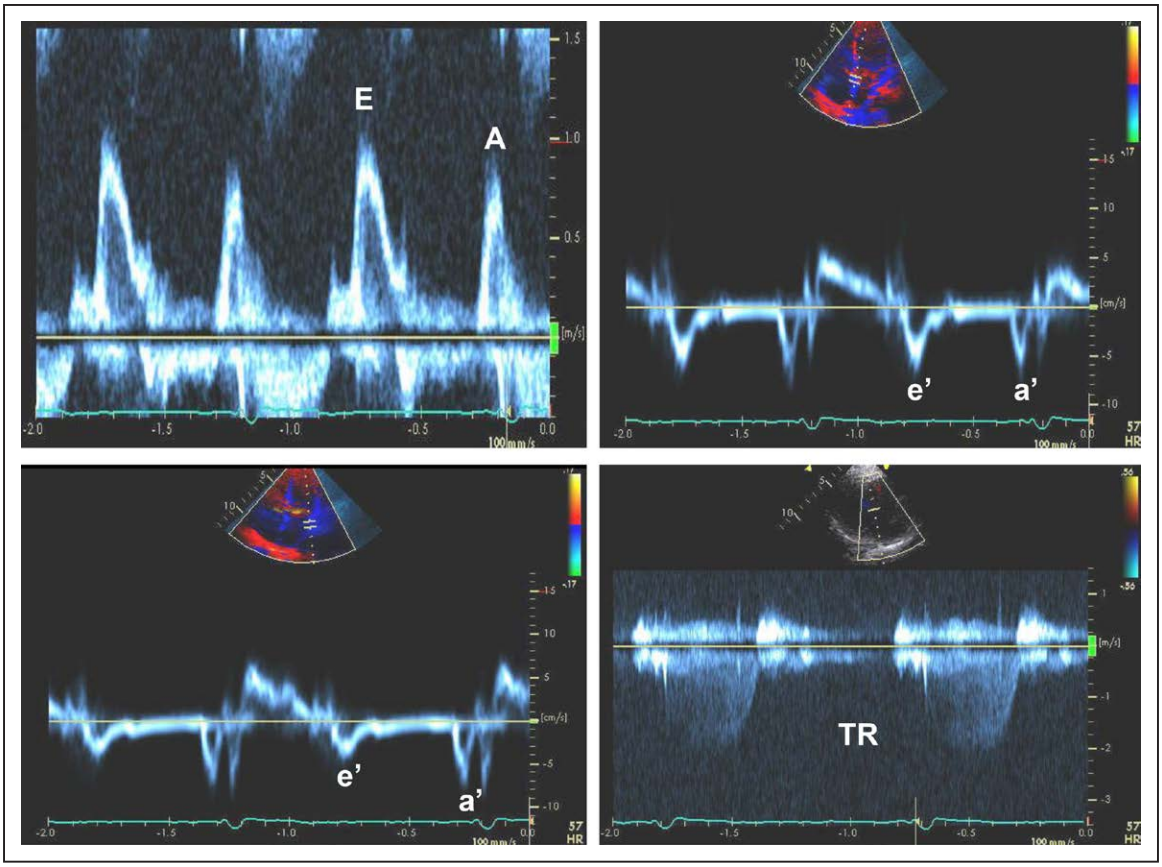

Figure 2. Mitral inflow (upper left), septal tissue Doppler (TD) velocities (upper right), lateral TD velocities (lower left), and peak tricuspid regurgitation (TR) velocity (lower right) from an 81-y-old woman with history of hypertension.
LV size and function are normal with preserved ejection fraction and normal LA volume (Movies I through III in the Data Supplement). Peak tricuspid regurgitation velocity is 2 m/s, mitral peak early diastolic velocity (E)/peak mitral late diastolic velocity (A) ratio is 1.3, septal and lateral annular velocities are reduced. Average E/mitral annulus early diastolic velocity by TD (e′) ratio is >14. LV global longitudinal strain was normal at −21.1%. Accordingly, there is no evidence of myocardial disease, and one has to rely on the 4 variables (annular e′ velocities, average E/e′ ratio, LA maximum volume index, and peak tricuspid regurgitation velocity). Because only 2 of the 4 variables are abnormal (e′ velocities and average E/e′ ratio), LV diastolic function is indeterminate per the 2016 guidelines. a′ indicates mitral annulus late diastolic velocity by TD.

measurement of suboptimal signals by different observers. For example, when mitral annulus velocities are acquired with suboptimal gain and filter settings, there is often difficulty in determining the onset of the e′ velocity and its peak value. A foreshortened LA will result in underestimation of the true LA maximum volume, no matter how carefully the LA endocardium is traced. In each of the above examples, the observer should not rely on the suboptimal signal in drawing conclusions about LV filling pressure.

Likewise, satisfactory signals, if incorrectly measured, can lead to wrong conclusions. Training plays an important role in obtaining correct measurements and increasing interobserver agreement for the assessment of LV diastolic function as has been recently shown.[8]

On the analysis side, incorrect application of the guidelines can be a challenging hurdle to contend with in trying to improve interobserver agreement. In that regard, the 2016 guidelines are explicit in recommending consideration of clinical findings, 2D data, as well as Doppler signals in trying to reach conclusions about LV diastolic function. The appropriate consideration of the presence of myocardial disease, which involves clinical assessment, as well as 2D and Doppler findings,

Table 3. Sensitivity and Specificity of the 4 Groups Against Invasively Measured Left Ventricular Filling Pressure as the Gold Standard

Group	Sensitivity (Mean±SD), %	Sensitivity (Median), %	Sensitivity (Range), %	Specificity (Mean±SD), %	Specificity (Median), %	Specificity (Range), %
I	92±0.00	92	92–92	93±6	94	84–100
II	91±2	92	88–92	95±2	96	92–96
III	88±5	88	80–92	91±7	92	80–100
IV	91±3	92	84–92	92±5	92	88–100

reduces the number of cases with indeterminate LV filing pressure. Furthermore, there is a clear hierarchy with respect to the sequence of signals considered and the number of abnormal findings that should be present before concluding that LV filling pressure is elevated. Accordingly, the overall agreement we noticed in our study is much higher than that reported previously.[2] Importantly, the satisfactory agreement was noticed for experienced observers irrespective of whether they were members of the guidelines writing group or not, as well as cardiology fellows in training.

Implications for Clinical Application of the 2016 ASE/EACVI Guidelines

The ultimate goal of any set of guidelines is to improve patient care, thus potentially contributing to better outcomes. The 2016 ASE/EACVI diastolic function guidelines render the assessment of LV filling pressure more consistent among different levels of observers as shown in this study. Further, they have good accuracy in identifying patients with elevated LV filling pressure. As a result of higher reproducibility and high accuracy, the guidelines are useful in drawing the attention of referring clinicians to the status of LV filling pressure, which in turn can help confirm the diagnosis of heart failure, as well as inform fluid management decisions.

Limitations

The study design did not evaluate the interobserver variability in measuring 2D and Doppler parameters. We did not seek to assess measurement variability because the main objective of our study was to evaluate the agreement of different observers in reaching the correct conclusion about LV filling pressure when applying the 2016 guidelines given the wide variability reported with the application of the 2009 guidelines.[2] In that regard, the current study design with confined analysis to guideline application as opposed to measurement variability is similar to a previous study looking at agreement between different observers in evaluating LV diastolic function.[2] Although there were 50 patients, there were 1400 assessments in total by the observers. One of the reasons behind the results observed is the availability of a satisfactory tricuspid regurgitation jet in 34 of the 50 patients (68%), and it is possible that lower and higher feasibility rates can affect the results. Notwithstanding, tricuspid regurgitation is one of the important signals that is endorsed in several guideline recommendations for the estimation of pulmonary artery systolic pressure and should be routinely acquired. Patients who undergo cardiac catheterization are more likely to have cardiac disease and diastolic dysfunction. However, if one is to test the accuracy of the guidelines, the gold standard of invasive LV filling pressure is needed. The accuracy of the guidelines in general, as a separate issue aside from variability, is likely lower in populations with lower prevalence of cardiovascular disease and in asymptomatic subjects. With respect to patients not undergoing cardiac catheterization, outcome events as incidence of heart failure and cardiac mortality may be used to gain insight into the ability of the guidelines to predict outcomes.[9] There were few patients with a paced rhythm or atrial fibrillation, and additional studies are needed to evaluate the variability of the application of the guidelines in patients with these rhythms.

Conclusions

There is a good interobserver agreement in the evaluation of LV filling pressure using the ASE/EACVI 2016 update irrespective of the experience level of the observer. This conclusion is the most applicable when satisfactory quality signals are acquired and carefully measured.

ARTICLE INFORMATION

Received July 5, 2018; accepted November 27, 2018.

The Data Supplement is available at https://www.ahajournals.org/doi/suppl/10.1161/CIRCIMAGING.118.008122.

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Disclosures

None.

APPENDIX

Diastolic Function Assessment Collaborators: Methodist DeBakey Heart and Vascular Center and Center for Outcomes Research, Houston Methodist Research Institute, Houston, Texas (Sherif F. Nagueh, MD, Mohammed A. Chamsi-pasha, MD, Kinan Carlos El tallawi, MD, Jiaqiong Xu, PhD); University of Oslo, Oslo, Norway (Otto A. Smiseth, MD, PhD, Thor Edvardsen, MD, PhD); Department of Medical Sciences, Uppsala University, Clinical Physiology and Cardiology, Akademiska, Uppsala, Sweden (Frank A. Flachskampf, MD, PhD); Cleveland Clinic, Cleveland, Ohio (James Gentry, MD, Serge C. Harb, MD, Allan L. Klein, MD); University of Liège Hospital, Liège, Belgium (Patrizio Lancellotti, MD, PhD); Universita Piemonte Orientale, Novara, Italy (Paolo Marino, MD); University of Medicine and Pharmacy "Carol Davila," Euroecolab, Institute of Cardiovascular Diseases, Bucharest, Romania (Bogdan A. Popescu, MD, PhD, Carmen Beladan, MD, PhD); Service de Cardiologie et Maladies Vasculaires, LTSI INSERM 1099, Universite Rennes-1, CHU Rennes, Rennes, France (Erwan Donal, MD, PhD, Arnaud Hubert, MD); Department of Advanced Biomedical Sciences, Federico II University Hospital, Naples, Italy (Maurizio Galderisi, MD, Roberta Esposito, MD, PhD); Physiology Department, DHU Ageing-Thorax-Vessel-Blood, Henri Mondor Hospital, Assistance Publique Hôpitaux de Paris, Créteil, France (Genevieve Derumeaux, MD, PhD, Laura Ernande, MD, PhD, Martina Previato, MD); Department of Medicine, University of Massachusetts Medical School, Worcester, MA (Gerard P. Aurigemma, MD, Saket R. Sanghai, MBBS); Department of Cardiology, MedStar Heart and Vascular Institute, Washington, District of Columbia (Steven A. Goldstein, MD, Redah Z. Mahmood, MD); Division of Cardiology and Corrigan Minehan Heart Center, Massachusetts General Hospital, Harvard Medical School, Boston, MA (Judy Hung, MD); Department of Cardiology, Leiden University Medical Center, Leiden, the Netherlands (Jeroen J. Bax, MD, PhD, Victoria Delgado, MD, PhD, Giulia Dolci, MD); Division of Cardiology, University of California at San Francisco, San Francisco, California (Theodore P. Abraham, MD, Alyssa Browning, MD).

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