do not comment on the need for concordance between target vessel PCI and territories of ischemia, clinical practice often relies on localizing ischemia to guide revascularization (4).

As seen in this single-center study of patients treated before the formation of a dedicated CTO PCI program, operators treating patients with CTOs often revascularized coronary vessels that were not supplying areas of ischemia detected on stress imaging. Although non-CTO PCI may potentially augment collateral flow to the CTO territory, non-CTO PCI may be insufficient in alleviating ischemia and anginal symptoms due to the CTO. Resistance to treat the CTO lesion may, in part, reflect operator perceptions about the higher risks associated with CTO PCI, a lack of availability of skills and equipment for technically more complex procedures, and the greater investment of time and resources required for successful treatment.

In conclusion, among patients with CTOs, revascularization of lesions unrelated to areas of ischemia detected on stress imaging is common and occurs more frequently among those undergoing non-CTO PCI. These results may motivate further discussion of the optimal revascularization strategy, including PCI or surgery, for patients with a CTO to ensure that revascularization has the greatest opportunity to abate symptoms and reduce ischemic burden. In addition, CTO patients receiving non-CTO PCI may need closer follow-up to determine if sufficient ischemia reduction has occurred.

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Issues With Estimating "Diastolic Function" and Left Ventricular Filling Pressure Using the New Guidelines

Andersen et al. (1) are to be congratulated for their work on testing recent guidelines (2) to estimate left ventricular (LV) filling pressure using echocardiography. I have several concerns about the utility of this approach.

First, E/e' (the ratio between early mitral inflow velocity and mitral annular early diastolic velocity) is clearly the most useful predictor of LV filling pressure. In fact, if E/e' is <14 even with tricuspid regurgitant velocity elevated and left atrial volume index high, only 11 of 19 patients had left ventricular elevated filling pressure (LVEFP). Thus, the algorithm in this case would have one reporting out "Grade II, diastolic dysfunction, high left atrial pressure" on a patient in whom 50% of the time the LVEFP is normal.

Second, the authors state that "the algorithm is based on the interpretation of 2D and Doppler signals in patients with cardiovascular diseases and not in patients without cardiac diseases who are explicitly excluded from the algorithm" (1). It is not clear from the paper what qualified patients as having cardiovascular disease. Indeed, deciding which patients qualify for this algorithm is quite problematic because most echocardiograms in clinical practice are read without complete knowledge of the patient's clinical history. For instance, a patient with significant coronary artery disease could have normal appearing LV size and function by 2-dimensional echocardiography: a reader of the echocardiogram would call "normal diastolic function" if unaware of that history and

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call "abnormal diastolic function" if aware of the coronary artery disease. Similarly, presumably LV hypertrophy puts the patient in the category of "myocardial disease." Most clinical echocardiography laboratories do not routinely or accurately measure LV mass index, the gold standard of LV hypertrophy.

Third, mitral annular calcification has a profound effect on mitral inflow independent of LVEFP. It would be instructive to know how many study patients had severe mitral annular calcification and whether this group skewed the overall results.

In an accompanying editorial (3), the authors note that the 2009 American Society of Echocardiography/European Association of Cardiovascular Imaging guidelines were widely criticized for being "awkward, ambiguous and frequently wrong." It does not appear that the 2016 American Society of Echocardiography/European Association of Cardiovascular Imaging guidelines will be able to eliminate those criticisms.

Finally, I would agree that diastolic function assessment from noninvasive data is "an impossible mission" and question why guidelines and echocardiographers continue to refer to grades of "diastolic dysfunction" when LVEFP is dependent on factors other than diastolic dysfunction.

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REPLY: Issues With Estimating "Diastolic Function" and Left Ventricular Filling Pressure Using the New Guidelines

We appreciate the interest of Dr. Pearson in our paper. In his first comment, he selected 19 patients from the 450 patients included in the study to comment on, rather than assessing all the data presented. The conclusion Dr. Pearson draws later on about the overall utility of the approach is based on these 19 patients only. We would point out that in the vast majority of patients in our study, and also in the subset with ≥2 abnormal findings, the algorithm worked well, with a high feasibility and good accuracy. With respect to the specific point about patients with peak tricuspid regurgitant velocity >2.8 m/s and increased left atrial maximum volume index, we view it as an observation that merits additional evaluation but cannot draw conclusions given the very small number of patients in this subset.

In our paper (1) and in the guidelines (2), we discuss how patients can be identified as having cardiovascular disease based on clinical data, 2-dimensional findings, and specific Doppler signals. Dr. Pearson brings up important points about the clinical practice of some, where they interpret left ventricular (LV) diastolic function in the absence of clinical data and 2-dimensional echocardiography findings, including ignoring the presence of LV hypertrophy. These practices are discouraged. There are no good reasons why a physician would not seek clinical data before reading an echocardiogram or why LV wall thickness or LV mass are not measured. This is important because, as discussed in our paper, the accuracy of the algorithm for estimating LV filling pressure is likely to be lower for populations with lower prevalence of cardiac disease.

Patients with severe mitral annular calcification were not targeted in this investigation. However, there is a recent study showing the good accuracy of Doppler in these patients (3,4).

We would like to correct Dr. Pearson regarding the editorial referenced. This was written by Drs. Flachskampf and Baron, who are not coauthors of our paper.

In his last comment, Dr. Pearson questions the value of grading diastolic dysfunction at all. We would point out that diastolic dysfunction grade, as well as its progression or regression, is an independent predictor of outcome in several populations with different etiologies and severities of cardiovascular disease (5), let alone its utility in detecting early myocardial disease. Thus, ignoring the grading of diastolic dysfunction would remove a powerful tool in assessment and prognostication of cardiac patients.

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