Quantitative Relationship Between Severity of Pulmonary Hypertension and LV Diastolic Function Has Been Established

We read with interest the article entitled “Correlation of Left Ventricular Filling Characteristics With Right Ventricular Overload and Pulmonary Artery Pressure in Chronic Thromboembolic Pulmonary Hypertension” published in the Journal (1). Dr. Mahmud and his colleagues identified an impaired relaxation pattern of left ventricular (LV) filling in 39 patients with chronic thromboembolic pulmonary hypertension (CTPH), with a mean pulmonary artery pressure >30 mm Hg, which normalized after successful pulmonary thromboendarterectomy. The objective (e.g., finding a quantitative relationship between the right ventricular [RV] pressure overload and the type of LV diastolic dysfunction) and some of the results of this study (only patients with severe pulmonary hypertension have an altered LV filling) resemble those published by our group in 2001 (2). We were disappointed by the investigators’ claim in the Objectives section of their Abstract that “a quantitative relationship between RV pressure overload and LV diastolic function has not been established.”

We examined by Doppler-echocardiography 120 patients with chronic pulmonary hypertension (of whom 12 patients had CTPH), and we found that only in patients with a systolic pulmonary artery pressure (SPAP) ≥60 mm Hg is LV diastolic filling altered in the form of impaired relaxation pattern. In addition, we also found that the late systolic and early diastolic interventricular septum flattening occurs in 70% of patients with SPAP ≥60 mm Hg and only in 6% of those with SPAP <60 mm Hg. Therefore, a quantitative relationship between the severity of pulmonary hypertension and LV diastolic function was already established.

We would like to add that, in addition to abnormal geometrical configuration and motion of interventricular septum mentioned by Dr. Mahmud, other possible mechanisms of LV diastolic dysfunction in patients with severe pulmonary hypertension might be related to the presence of some degree of LV interstitial edema, which increases the LV wall stiffness and alters its normal diastolic filling (3) and the diastolic asynchrony found in the apical and lateral walls (4).

Finally, we believe that the findings of both reports complement each other and enlarge our understanding of the mechanisms of dyspnea in patients with severe pulmonary hypertension.

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REPLY

We would like to respond to the comments by Barasch et al. regarding our study “Correlation of Left Ventricular Filling Characteristics With Right Ventricular Overload and Pulmonary Artery Pressure in Chronic Thromboembolic Pulmonary Hypertension” recently published in the Journal (1). The study by Moustapha, Barasch, and colleagues (2) was published only a short time before we submitted our manuscript and so, regrettably, was not included in our references.

The objective of our study was to define a quantitative relationship between right ventricular (RV) pressure overload and left ventricular (LV) diastolic filling characteristics, measuring both as continuous variables (i.e., mean pulmonary artery pressure and E/A ratio). Furthermore, we only studied patients with chronic thromboembolic pulmonary hypertension (CTEPH) who were undergoing pulmonary thromboendarterectomy (PTE) so that we could assess the change in LV diastolic filling with resolution of an RV pressure overload state.

In the study by Moustapha et al. (2), the degree of pulmonary hypertension was defined only as a dichotomous variable (mild-moderate vs. severe). Pulmonary artery (PA) pressure was estimated by Doppler-echocardiography, not measured directly as in our study. Moustapha and colleagues reported that the group of patients with severe (systolic PA pressure >60 mm Hg) had lower E/A ratios than did the group with mild-moderate pulmonary hypertension (systolic PA pressure 40 to 60 mm Hg) (0.96 ± 0.37 vs. 1.34 ± 0.54, respectively [p < 0.05]). This relationship of low E/A ratio and severe pulmonary hypertension with associated interventricular septal distortion is, however, well recognized (3,4).
In contrast to these clearly semiquantitative results, we found a direct, logarithmic, and quantitative relationship between E/A ratio and PA pressure measured as continuous variables in patients with CTEPH. We also documented a similar direct relationship between E/A and cardiac output in the same group. The results of our study expand upon previous studies that have reported on abnormal LV diastolic function in patients with chronic pulmonary hypertension. However, it is still difficult to hypothesize that abnormal LV diastolic filling contributes significantly to dyspnea in these patients. In fact, pulmonary capillary wedge pressure increased slightly from 9.8 ± 5 to 11.5 ± 4.5 mm Hg (p = 0.04) after PTE. This suggests that dyspnea in patients with severe pulmonary hypertension is unlikely to be related to an elevated LV filling pressure.

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Detrimental Effects of Late Artery Opening

In reference to the recent study by Yousef et al. (1) in the Journal, we agree that percutaneous coronary interventions (PCI) for an occluded artery late after an acute myocardial infarction are not risk-free procedures, and even an immediate angiographic success may be harmful in the long term. The investigators attribute detrimental effects of late reperfusion—significantly greater one-year left ventricular (LV) end-systolic and end-diastolic volumes—to periprocedural microembolization of collateral vessels. However, as reported in the study limitations, the researchers did not routinely measure cardiac markers after PCI; therefore, no proof for such a causative mechanism exists.

An alternative explanation for this finding is that the adverse effect on remodelling of the invasive strategy is not related to the PCI procedure itself but to the extremely high rate of late events occurring in these patients in relation to a suboptimal treatment. Here the 50% rate of adverse event at one year is far higher than the 32% rate reported after stent implantation in occluded arteries, even using the now outdated Palmaz-Schatz stents and warfarin therapy (2). The use of NIR stents (3) and the suboptimal (two weeks) duration of clopidogrel therapy might be advocated as a cause of higher-than-expected restenosis and reocclusion rates in the study by Yousef et al. (1).

Moreover, LV volumes are certainly a better surrogate end point than exercise tolerance, but improvements in exercise duration and peak rate-pressure products in the intervention group are unexpectedly discordant from echocardiographic findings. Changes in mitral regurgitation severity may contribute to limitation of exercise capacity (4). The assessment of mitral regurgitation in this setting could be extremely helpful in clarifying whether detrimental effects on remodelling are uniformly found in the entire cohort of patients who underwent PCI or only in the subgroup of patients who experienced adverse events.

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REPLY

Zimarino et al. have based their conclusions on a comparison of events between the TOAT (1) and GISSOC (2) studies. This comparison is not justified as neither study was powered to examine clinical end points. Furthermore, the inclusion criteria of the trials differed. For example, only 50% of the stented patients within GISSOC had a prior myocardial infarction, whereas in TOAT this was 100%. Moreover, to increase the prevalence of adverse remodelling, the eligibility criteria of TOAT ensured that the stented vessel subtended a large volume of infarcted myocardium with a presumed high microvascular resistance and thus more disordered flow. In addition, 28% of events within TOAT comprised heart failure and stroke, end points that are unlikely to be related to reocclusion. Omitting these end points results in a one-year event rate of 24% in those randomized to intervention,