Passive Pulmonary Hypertension

More Than Hydrostatics

To the Editor:

Pulmonary hypertension is increasingly recognized as an important clinical condition, and the review article by Hansdottir et al in CHEST (August 2013) on pulmonary hypertension caused by cardiac disease is very timely. We agree with the statement of the authors that pulmonary hypertension due to left-side heart disease is probably the most common cause for elevated pulmonary pressure; however, comparative epidemiologic studies between the different clinical groups are lacking, and we guess that the prevalence of pulmonary hypertension in group 3 (pulmonary hypertension due to lung diseases or hypoxic conditions) is commonly underestimated. In addition to the remarks of the authors, we would like to emphasize one point that is commonly encountered as a misconception: While up to two-thirds of patients with aortic stenosis show increased pulmonary pressures, the prevalence and severity of pulmonary hypertension due to aortic stenosis is unrelated to the severity of the valve disease as shown in an echocardiographic study in 388 patients by Faggiano and coworkers. Of note, this conclusion is also true for patients with systolic heart failure, in which the extent of reduced left ventricular ejection fraction is unrelated to the severity of pulmonary pressure and is also at least partially true for diastolic heart failure, where the postcapillary hypertension related to diastolic dysfunction is only one component of the global severity of pulmonary hypertension.

Passive backpressure in the pulmonary circulation was found to result in active vasoconstriction of the pulmonary arterioles to protect the low-pressure pulmonary vascular bed against the potentially damaging increase of the capillary pressure. This phenomenon is known as Kitaev reflex or Hemo-Weiler reflex (reviewed in Arrigo and Huber). Whether individual differences in the activity of this reflex are responsible for the severity of pulmonary hypertension secondary, but unrelated, to severity of the heart disease is unknown but might provide a probable explanation.

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Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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REFERENCES

Response

To the Editor:

I thank Drs Arrigo and Huber for their comments on our review in CHEST. As Drs Arrigo and Huber point out, comparative epidemiologic studies looking at the prevalence of different World Health Organization groups of pulmonary hypertension (PH) are not available. A community-based, retrospective echocardiographic study out of Australia found that the minimum “indicative” prevalence of all groups of PH was 326 in 100,000. In this study, 68% of patients had PH due to left-side heart disease (LHD), and 9% had PH due to respiratory disease or sleep-disordered breathing. Patients with PH due to LHD may have been overrepresented in this study, however, as they may be more likely than patients with lung disease to undergo echocardiography. Therefore, we agree with Drs Arrigo and Huber that the prevalence of PH in lung disease may be underestimated. It is also worth noting that many common heart and lung diseases have mutual risk factors, and it may not always be clear whether the cause of PH is one or the other or both.

We also agree with Drs Arrigo and Huber that the pathophysiology of PH due to LHD is much more complicated than simple hydrostatics. Indeed, there is not a linear relationship between the severity of LHD and the development and severity of PH. Similarly, it has been shown that the correlation between pulmonary arterial pressure and lung function is weak. Why some patients