Correspondence

Pulmonary Hypertension in HIV-Infected Individuals

To the Editor—We read the recently published study by Mondy et al [1] with interest and find that there are several methodological issues worth noting. The most frequent abnormality revealed in their study was pulmonary hypertension reported at a prevalence of 57%, which is much higher than that found in prior reports [2]. A closer examination of the methods for ascertaining pulmonary hypertension as assessed by right ventricular systolic pressure (RVSP) shows 2 important reasons why the percentage reported may be spuriously high. First, the value of 57% is obtained by using 322 individuals with a detectable tricuspid regurgitation (TR) jet as the denominator and excluding the remainder of individuals (334 [51%] of a total of 656 patients), who had no detectable TR jet. However, we would argue that the denominator for prevalence should be the 656 total patients studied, for a prevalence of 28%, because most individuals who have substantial pulmonary hypertension have measurable tricuspid regurgitation at echocardiography [3] and prior studies have assumed that individuals with trace or no tricuspid regurgitation have normal pulmonary artery pressure [4]. The majority of the individuals in their study who were excluded with no detectable TR jet likely have normal pulmonary artery systolic pressure, especially in the absence of symptoms.

Second, the formula used for pulmonary hypertension in this study was RVSP = 4v² + 10 mm Hg. This formula is not standard and assumes that the right atrial pressure is 10 mm Hg in every study participant. The standard assessment of right atrial pressure is estimated by diameter and degree of collapse of the inferior vena cava[5], which can range from 0 to 5 mm Hg in the majority of individuals, depending on volume status. By not assessing right atrial pressure by the inferior vena cava and by simply adding 10 mm Hg to every individual measurement, investigators are most likely overestimating the degree of pulmonary hypertension in their study. Indeed, the majority of individuals in their study had only mildly elevated RVSP of 31–35 mm Hg.

While the adjustments we describe above will certainly result in a downward estimate of the prevalence of pulmonary hypertension among human immunodeficiency virus (HIV)–infected individuals, the resultant estimate is still likely to be higher than that for the general HIV-uninfected population. Formally determining this corrected estimate would allow for pooling with other studies to better determine the influence of HIV infection in causing pulmonary hypertension.

Acknowledgments

Financial support. This work was supported by a grant from the National Institutes of Health to P. Y. H. (5R01HL091526).

Potential conflicts of interest. All authors: no conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed in the Acknowledgments section.

Priscilla Y. Hsue,1 Ann F. Bolger,1 and Jeffrey N. Martin2

1Division of Cardiology, and, 2Department of Epidemiology and Biostatistics and the Positive Health Program, University of California, San Francisco, California

References