Reply to the letter to the editor

Dr Callejas Rubio contends that the prevalence of mild pulmonary arterial hypertension (PAH) in patients with autoimmune disease (PAH) is excessively high based on the parameters used in our assessment.

Assessment of pulmonary artery pressure (PAP) remains an emerging utility for stress echocardiography. There are relatively few, small studies having been performed in this area, and we agree with Dr Callejas Rubio that normal values are not clearly defined.

Bossone et al. noted small changes in pulmonary artery pressures during exercise. These changes were less than those noted in our population, which would be independent of differences in assumed right atrial pressure. Of note, the most marked changes were observed in athletes; a population significantly different to our screened population. It is difficult, therefore, to infer conclusions about normal subjects based on the small number of normal patients included, and their less dramatic increase in PAP.

Grunig et al. assessed family members of patients with familial primary pulmonary hypertension, which may represent an at risk population similar to our cohort. This study demonstrated a rise in pulmonary artery pressure among all patients, with the authors concluding this represented an aberrant response to exercise. While their threshold for diagnosis was lower, the nature of the population again makes it difficult to make inferences about normal values. Given our acknowledged difficulties in assessment of normal patients, we based our parameters for measurement and severity on previously published echocardiographic data in this specific patient population.

Our aim was to screen an at risk population to potentially identify early disease in those who may then warrant further follow up and closer observation. Given the unclear nature of disease progression in this patient population with mild elevation in PAP with exercise, it is difficult to speculate on future changes in echocardiographic parameters. As such, it would be unwise to dismiss even minor changes in PAP with exercise without further investigation and ongoing follow up.

Indeed, as our experience with this modality grows, and with the benefit of longitudinal assessment, we may be able to make an educated assessment of abnormal pulmonary artery hypertension as measured by Doppler echocardiography during exercise. A higher PAP may be more appropriate to define an abnormal response to exercise as suggested by Dr Callejas Rubio. However, until we have accumulated this experience, in conjunction with observation of the natural history of PAP as measured by stress echocardiography, to consider the currently utilised parameters, and subsequently determined prevalence, "inadequate" is premature.

References


Nicholas John Collins
Cardiovascular Unit, John Hunter Hospital, Lookout Rd, New Lambton, NSW 2305, Australia
Tel.: +1 416 726 9452.
E-mail address: nandl_collins@yahoo.com.au

19 March 2006

Available online 2 May 2006