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Author's reply

The valuable comments made by Babu et al. are very much appreciated. Babu et al. stress the value of exercise oxygen consumption and oxygen pulse to assess mortality risk.[1,2] Indeed, both CPET parameters were reduced in the presented patient and remained reduced after the start of therapy.[3] Babu et al. hypothesize that the lack of normalization in CPET parameters may have been explained by a short duration (4 months) of therapy. However, during her recent 1 year follow-up evaluation, we observed no further improvements in maximal exercise capacity (from 167 to 162 W), maximal oxygen consumption (from 2.11 to 2.03 l/min), or oxygen pulse (from 12 to 11 ml/beat). It is difficult to determine whether the lack of normalization of CPET parameters translates into a poor prognosis. The prognostic value of changes in CPET parameters after the start of therapy remains unclear. Studies examining the prognostic utility of CPET in pulmonary arterial hypertension (PAH) were performed in a relatively small number of patients,[4] and in these studies, measurements were only done at baseline.

Babu et al. make valuable comments on the usefulness of CPET as a screening tool for patients with a family history of PAH. They suggest that in the presented case, it may have been appropriate to perform yearly CPET along with a blood workup and echocardiography. They hypothesize that the patient may have had steady decrements in CPET parameters between 2009 (no PAH) and 2012 (diagnosis of PAH). Although we agree that steady decrements may have been visualized by repeated tests, we question if performance of CPET would have altered this patient's management. She was seen by a cardiologist between 2009 and 2012 and repeated echocardiography did not reveal any signs of PH. In addition, the patient only started to experience physical complaints in November of 2011. We would probably not have performed a right heart catheterization in a patient with no physical complaints or signs of PH on echocardiography, even if CPET would have shown some subtle abnormalities. We agree with Babu et al. that only a prospective follow-up study in offspring of heritable PAH patients will determine the value of CPET as a screening tool for PAH.

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