restriction does not apply to the occasional marijuana smoker.

In conclusion, lung donation after fatal acute poisoning, although difficult, could be considered in selected cases of poisoning by pharmaceutical drugs that are not associated with lung injury and in selected cases of pure carbon monoxide intoxication. Oral ecstasy poisoning, as well as occasional marijuana smoking, do not appear to be an absolute contraindication.

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End points for pulmonary arterial hypertension: a way backward

To the Editors:

In the June 2004 issue of the *European Respiratory Journal*, PEACOCK *et al.* [1] recognised the need for end points, other than the assessment of functional capacity with the 6 min walking test, in pulmonary arterial hypertension (PAH) clinical trials. In their looking forward, the authors did not mention any role of the diffusing lung capacity for carbon monoxide (*D*L,CO) test, which was originally devised in 1909–1915 [2]. However, as recently reviewed [2, 3], the measurement of lung gas transfer for CO (*T*L,CO) holds some premise into the evaluation of patients with PAH. In daily clinical practice, *T*L,CO is derived from the product of constant rate of alveolar to blood CO uptake and the accessible alveolar volume, which is usually preserved in PAH; the examination of these two components allows exploration of the pathophysiological mechanism of *T*L,CO decrease at any time, due to pulmonary vascular abnormalities. Furthermore, the physiology of CO transfer is governed by the Roughton-Forster equation [4], partitioning the resistances to CO into membrane and red cell contributions, the latter accounting with appropriate calculations for 70–80% of total resistance [3]. Moreover, the *D*L for CO or another suitable gas with greater haemoglobin affinity, such as nitric oxide, could be assessed at rest and during exercise, in combination with noninvasive measurement of cardiac output, providing sensitive indicators of the diffusive oxygen transport effectiveness, and structural alteration of the alveolar-capillary barrier [5, 6]. Therefore, it seems logical to assume abnormalities in *D*L recruitment (or *D*L/cardiac output ratio) occurring in PAH before chronic cardiac consequences would be detected during echocardiography. Accordingly, *D*L,CO decrease at rest is present in 80% of PAH patients [7], and it is significantly related to the main cardiopulmonary exercise test parameters of aerobic function [8], which, in turn, are relevant to the prognosis of PAH [9].

In conclusion, following the authors' suggestions, we believe that diffusing lung capacity tests, combined in parallel with other markers, should receive consideration for as broader an application as possible for markers of pulmonary arterial hypertension. Hopefully, a view backward may widen the way forward.

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