Pulmonary capillary recruitment in exercise and pulmonary hypertension

To the Editor:

We read with interest the excellent European Respiratory Society statement on exercise pulmonary hypertension (PH) by Kovacs *et al.* [1] in the *European Respiratory Journal*. Fundamentally, exercise PH, especially with precapillary causes such as pulmonary arterial hypertension (PAH), is an inability of the lung circulation to accommodate increased blood flow during exercise. Although the authors mention "distention" of the vasculature, implying stretching of already perfused vessels, the increased pulmonary blood flow is primarily accommodated in the normal lung by recruitment of unused capillaries, allowing the pulmonary artery pressure to change minimally during exercise [2, 3]. Further evidence of this recruitment is found in true vasoresponders during an acute vasodilator challenge for evaluation of idiopathic PAH [4]. By contrast, because it is caused by precapillary vascular obstruction and not vasoconstriction, vasodilator-nonresponsive PAH accommodates any increased cardiac output *via* distention and not *via* recruitment [5]. We have also observed pulmonary capillary recruitment in normal humans during exercise (unpublished data). Recruitment is a normal physiological process, and it is impaired in many types of PH. As we move forward in our understanding of exercise PH and its physiology, precise definitions and semantics will be critical.

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David Langleben[1] and Stylianos E. Orfanos[2]
[1]Center for Pulmonary Vascular Disease, Cardiology Division, Jewish General Hospital and McGill University, Montreal, QC, Canada. [2]Pulmonary Hypertension Clinic, Attikon Hospital and 2nd Dept of Critical Care, National and Kapodistrian University of Athens, Athens, Greece.

Correspondence: David Langleben, Jewish General Hospital, E-206, 3755 Cote Ste Catherine, Montreal, QC, H3T 1E2, Canada. E-mail: david.langleben@mcgill.ca

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From the authors:

We appreciate the comments by Langleben and Orfanos. Indeed, there has been a controversial discussion on recruitment of perfused vessels, distensibility of perfused vessels and even active vasodilatation of the pulmonary circulation during exercise. Shortly after introduction of right heart catheterisation as a

diagnostic method, some reports suggested that during exercise, cardiac output may increase considerably with little or no change in pulmonary arterial pressure [1, 2]. Subsequent studies in healthy adults of different ages and sexes, performed in experienced high-volume centres, could not reproduce these results [3]. Indeed it seemed that the increase in pulmonary arterial pressure was always linearly related to the increase in cardiac output, although the steepness of this relationship was strongly age dependent.

One of the key questions is the amount of pulmonary vascular resistance (PVR) change from rest to exercise. Experimental models using isolated perfused lungs or open chest preparations with positive pressure ventilation found a profound PVR decrease from low to normal pulmonary perfusion pressures [4], but these experimental conditions may not resemble physiological conditions in healthy volunteers. In our literature research of all published data with the use of strict quality criteria, we found no evidence of a strong decrease of PVR during supine exercise, even in young healthy adults [3, 5]. These results have later been confirmed by others [6, 7].

Therefore, we agree with Langleben and Orfanos that it is important to use exact terminology and to carefully analyse the subtle changes in pulmonary haemodynamics during exercise. However, it would be premature to judge the recruitment capacity of the pulmonary vasculature in healthy individuals based on the published literature, or to draw any final conclusions. Much more effort is needed to solve the old physiological questions about the regulation of pulmonary blood flow in humans.

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Horst Olschewski[1,2], Gabor Kovacs[1,2] and Philippe Herve[3]
[1]Medical University of Graz, Dept of Internal Medicine, Division of Pulmonology, Graz, Austria. [2]Ludwig Boltzmann Institute for Lung Vascular Research, Graz, Austria. [3]Centre Chirugical Marie Lannelongue, Thoracic and Vascular Surgery, Le Plessis-Robinson, France.

Correspondence: Gabor Kovacs, Medical University of Graz, Auenbruggerplatz 20, 8036 Graz, Austria.
E-mail: gabor.kovacs@klinikum-graz.at

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