Reply to Pancheva, Panchev, and Pancheva

N. Edward Robinson, Kurt J. Williams, Alice Stack, Frederik J. Derksen, Joseph Hauptman, Melissa Millerick-May, and Heather DeFeijter-Rupp

Department of Large Animal Clinical Sciences, Michigan State University, East Lansing, Michigan

TO THE EDITOR: We are pleased that our paper “Distribution of venous remodeling in exercise-induced pulmonary hemorrhage of horses follows reported blood flow distribution in the equine lung” (5) has stimulated critical thought on the role of inertia as a cause of the venous remodeling by Pancheva and coworkers (3). Our group has extensively considered the forces being imposed on the vein wall and their role in remodeling, and we acknowledge that we never considered the inertial forces resulting from the movement of the horse as a major factor determining venous wall stress. We are aware that in the high-velocity flow regime of large arteries, inertial forces outweigh viscous forces (1). Furthermore, inertial forces contribute to the flow and pulse wave form in the pulmonary veins (2). Certainly in the galloping horse that has a cardiac output in excess of 300 l/min, these inertial forces associated with the high flow velocity in large-bore pulmonary arteries and veins are likely to be considerable. However, in the very small veins in which remodeling is occurring, flow velocity is low and inertial forces arising from the movement of the blood are likely to be negligible (1).

Pancheva and coworkers are not the first to suggest that forces from the limbs play an etiological role in exercise-induced pulmonary hemorrhage (EIPH). In 1998, Schroter and coworkers (4) suggested that pressure waves resulting from forelimb impact with the ground transmitted through the lung parenchyma are responsible for the dorsocaudal location of EIPH lesions. It is also likely that inertial forces provided by the acceleration of the horse around its fore- and hindlimbs have effects on pressure waves within the large vessels of the pulmonary circulation where blood mass is greatest. Predicting and measuring the interactions of these limb forces with those imposed by the heart will be difficult, in part because of phase variations between the heart (~220 beats/min) and stride rate (~120/min) of the exercising horse. Even if that could be done in the large vessels, understanding the role of limb acceleration on vein wall stress in complexly branching very small pulmonary veins will be confounded by their varying geometric relationships to the force vectors arising from the motion of the limbs.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES