

## Author Response: Subfoveal Choroidal Thickness in High-Altitude Mountaineers

Our research group thanks Jonas and Panda-Jonas<sup>1</sup> for their comments about our recent report entitled “Reversible Increase of Central Choroidal Thickness During High Altitude Exposure.”<sup>2</sup> The question was raised whether the observed increase in subfoveal choroidal thickness may have been associated with, or influenced by, an increased cerebrospinal fluid pressure.<sup>3</sup>

This is an interesting and relevant question as the venous choroidal blood drains into the intracranial cavernous sinus and cerebrospinal fluid pressure indeed may influence the choroidal thickness. However, as our group did not measure cerebrospinal fluid pressure, we can only hypothesize on its role on choroidal thickness. Jonas and Panda-Jonas<sup>1</sup> have suggested taking the systemic blood pressure as a surrogate marker for cerebrospinal fluid pressure, but our study design does not allow such an analysis (no such data at altitude).

The pathophysiological basis of acute mountain sickness (AMS) is still unresolved and remains controversial. Current concepts for the development of acute mountain sickness include elevated brain water due to vasogenic edema with consequent brain swelling, activation of the trigeminal vascular system, or dynamic cerebral spinal fluid flux into the brain.<sup>4</sup>

We agree that the number of study participants in our study was too small to allow a definite statement for a missing association between increased subfoveal choroidal thickness and acute mountain sickness. However, we disagree that none of our subjects developed marked acute mountain sickness.

Furthermore, the frequent occurrence of retinal vascular leakage (representing vasogenic edema) in both healthy subjects and those suffering from acute mountain sickness indicates that increased cerebrospinal fluid pressure may not be the underlying mechanism for the occurrence of acute mountain sickness.<sup>5</sup>

Thus, we would like to stress that hypoxia-induced choroidal hyperperfusion remains important for a steady state of oxygen supply to the retinal photoreceptors. In addition, this may be influenced by cerebrospinal fluid pressure. Future studies investigating the choroid might be able to shed more light on the role of cerebrospinal fluid pressure in the development of AMS as well as changes in the visual system.

M. Dominik Fischer<sup>1</sup>  
 Andreas Schatz<sup>1,2</sup>  
 Florian Gekeler<sup>1,2</sup>  
 Gabriel Willmann<sup>1,2</sup>

<sup>1</sup>Centre for Ophthalmology, University of Tübingen, Tübingen, Germany; and the <sup>2</sup>Department of Ophthalmology, Katharinen-hospital, Stuttgart, Germany.

E-mail: gabriel.willmann@googlemail.com

### References

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