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In Reply to Dr Sikri et al

To the Editor:

Thanks to Sikri et al.¹ for showing interest in our research article.²

High altitude pulmonary edema (HAPE), a potentially fatal condition, is a rare form of altitude sickness that affects healthy lowlanders or nonacclimatized individuals who travel to high altitudes greater than 2500 m. The unrelated subjects selected for this study belong to different ethnic groups of an Indian population. All subjects with HAPE selected for the study were serving Army personnel who were acutely inducted to high altitude. A comparable number of control subjects selected for the study were also serving Army personnel who were acutely inducted and faced similar environmental challenges. Patients were admitted to Army Hospital of Leh, and after diagnostic confirmation of HAPE, blood samples were collected before the start of treatment. This procedure was well defined and uniformly followed throughout the study protocol. The present study purely aimed to seek the involvement of renin-angiotensin-aldosterone system pathway in genetic susceptibility to HAPE.³

Figure 1 of our article² shows arterial oxygen saturation (SaO₂) compared with genotypic profile of the angiotensin-converting enzyme gene (ACE) I/D polymorphism in HAPE and acclimatized control subjects groups. As Sikri et al. rightly pointed out in their letter, a significant amount of variation in SaO₂ is likely to occur during evacuation from remote locations to medical facilities; however, in this study, the setup of Army Hospital in Leh, in the Ladakh region of India, was in close proximity to that of the Army camp area, and no evacuation was required to provide medical help to the HAPE patients. When the volunteers complained about dyspnea at rest, cough, and breathlessness, they were immediately taken to the hospital. Also, both heart rate and SaO₂ levels were measured in seated volunteers via finger pulse oximeter. Hence, to the best of our understanding, the study of association between the alleles of the ACE gene and SaO₂ has given a fair idea about the involvement of renin-angiotensin-aldosterone system pathway genes in HAPE susceptibility. The idea of studying the association of HAPE with other physiological parameters (eg, heart rate and respiratory rate) included in the Lake Louise criteria for HAPE as suggested by Sikri et al in their letter is noted.

The present study indicated that the D/D genotype of the ACE gene appears to be associated with an increased

risk of HAPE in the Indian population. This finding could be an initial step toward the larger goal of designing screening criteria for early identification of HAPE-susceptible individuals in the community. This would not be a genetic discrimination, but a preventive step for the health of Army Jawans in the interest of the country. Since susceptibility to HAPE and other high altitude illnesses largely depends on the altitude attained, rate of ascent, and preacclimatization, a well-designed acclimatization schedule is always followed by Army personnel. Studies on different populations have suggested variability in individual susceptibility to altitude sickness depending on genetic makeup.⁴ Studies like the present one aim to understand the genetic basis of susceptibility to HAPE.

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Pitviper Envenomation Guidelines Should Address Choice Between FDA-approved Treatments for Cases at Risk of Late Coagulopathy

To the Editor:

We salute Dr. Kanaan and colleagues for their recent contribution, “The Wilderness Medical Society Practice Guidelines for the Treatment of Pitviper Envenomations in the United States and Canada,” an excellent paper encompassing many aspects of a complex disease.¹ Given the otherwise thorough treatment of the subject, it was a surprise to find that these guidelines contained such a minimal treatment of late coagulopathy, which we feel is among the most significant and challenging aspects of North American rattlesnake envenomation management