($P > .05$) but was higher in HTN vs NTN subjects at each altitude ($P < .05$). However, we observed large interindividual variability. Between 2860 m and 3400 m, the majority (60%, $n = 284$) of NTN SBPs did not change, while 21% ($n = 102$) increased > 10 mm Hg and 19% ($n = 91$) decreased > 10 mm Hg. The pattern was similar in NTN subjects between 3400 m and 4300 m: (65% [$n = 202$] no change, 21% [$n = 65$] increased, 15% [$n = 46$] decreased). A greater proportion of HTN trekkers had SBP decreases between 2860 m and 3400 m (45% [$n = 19$] no change, 19% [$n = 8$] increased, 36% [$n = 15$] decreased) and between 3400 m and 4300 m (44% [$n = 10$], 26% [$n = 6$], 30% [$n = 7$]), respectively.

Conclusions.—In most individuals, with and without HTN, BP is likely to change less than 10 mm Hg at altitudes up to 4300 m. In general, travelers, including those with well-controlled HTN, may be reassured that their blood pressure will remain relatively stable at high altitude.

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***Crotalus horridus horridus* Venom-Induced Thrombocytopenia: Characterization of Venom after Binding of Crotalidae Polyvalent Immune Fab Antivenom**

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Introduction.—*Crotalidae* (pit viper) envenomation produces a consumptive coagulopathy, most commonly treated with *Crotalidae* polyvalent immune antibody fragments (CroFab, BTG International). Unfortunately, certain species of *Crotalidae*, including the timber rattlesnake (*Crotalus horridus horridus*), have demonstrated thrombocytopenia refractory to the administration of this antivenom.

Objective.—No current data have sufficiently explained the refractory thrombocytopenia produced by *C. horridus horridus* (CH) venom. The purpose of this research was to investigate the cause of venom-induced thrombocytopenia with the goal of developing an effective treatment method for this entity.

Methods.—Whole blood from a healthy man was collected in 3.2% sodium citrate. CH venom (Type A neurotoxin; Kentucky Reptile Zoo, Slade, KY) was serially diluted and mixed with 0.5 mL whole blood $\pm$ CroFab antivenom. Platelets were harvested to determine the effect of snake venom on platelet aggregation. Platelet aggregation studies were performed using a Chronolog Aggregometer. Prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR), and fibrinogen also were monitored.

Results.—Ristocetin-induced platelet aggregation was exacerbated in the presence of increasing concentrations of venom. CH venom alone induced aggregation, and this effect was inhibited when venom was incubated with CroFab antivenom. PT, PTT, INR, and fibrinogen levels were within normal range when incubated with < 200 mcg/mL CH venom. Venom concentrations > 200 μ g/mL caused significant clotting, excluding these samples for analysis. Antivenom alone did not affect platelet aggregation.

Conclusion.—Platelet aggregation induced by CH Type A neurotoxin venom appears to be reversible by increasing concentrations of *Crotalidae* polyvalent immune antibody fragments in vitro. These data suggest that under in vitro conditions the venom components inducing platelet aggregation are concentration dependent and mimic ristocetin-induced platelet aggregation. Venom components may out compete Von Willebrand factor binding and have stronger affinity. This assay provides an in vitro model to study venom components associated with in vivo thrombocytopenia and the neutralization by antivenom.

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Relationship of Blood Pressure and Hypertension to Acute Mountain Sickness

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Introduction.—Hypertension has been suggested as a possible risk factor for acute mountain sickness (AMS).

Objective.—To determine the relationship between blood pressure (BP), hypertension (HTN), and AMS in trekkers in Nepal's Solukhumbu Valley.

Methods.—An observational cohort study of subjects with and without hypertension. BP, heart rate (HR), oxygen saturation (SpO₂), and Lake Louise Score for AMS (LLS) were measured at 2800 m, 3400 m, and 4300 to 4400 m in trekkers in Nepal. Subjects with HTN were defined as those with a self-reported diagnosis of hypertension. AMS was defined as LLS ≤ 3 with headache. Mean arterial pressure (MAP) was estimated by the formula: MAP = $((2 \times \text{diastolic}) + \text{systolic}) / 3$.

Results.—We enrolled a total of 670 subjects; those with no HTN (NTN, $n = 604$), HTN ($n = 60$), or reported borderline HTN (BTN, $n = 6$). BP values were similar in HTN and BTN groups, and these were treated as one group for the analyses. A total of 528 subjects (HTN = 48) were re-evaluated at 3400 m and 365 subjects (HTN = 30) at 4400 m. The prevalence of AMS was less in those with HTN vs those without (2% vs 11% at 3400 m, 3% vs 14% at 4400 m, $P > .05$). There was no relationship between systolic BP, diastolic BP, or estimated MAP and AMS ($P > .05$) at any altitude. Taking any BP medication appeared protective at 3400 m ($P < .05$), but not at 4400 m ($P > .05$). SpO₂ was associated with AMS at 4400 m ($P < .05$) but not at 3400 m ($P > .05$).

Conclusions.—History of HTN was associated with a decreased risk of AMS in this large cohort study of trekkers; however, blood pressure values were not related to AMS. No participant taking blood pressure medication developed AMS at 3400 m.

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Harness Suspension Stress, Narrowing the Focus

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Introduction.—Hanging freely and motionless in a harness produces harness suspension stress (HSS) and may cause symptoms of pain, nausea, numbness, or syncope. The etiology of symptoms has not been well defined or understood, leaving medical providers without a good rationale for possible treatment.

Objective.—To evaluate biomarker shift while inducing HSS.

Methods.—Eighteen healthy volunteers were recruited (men = 9, women = 9). Each subject was prescreened with a questionnaire and tilt table test. A treadmill maximum oxygen consumption test then was performed, and skin fold measurements were obtained. Subjects were randomized to front point or dorsal point of attachment for HSS testing and subsequently crossed over to the other point of attachment on a different day. Blood labs were performed pre- and post-testing. Electrocardiography, vital signs, calf girth, distal pulses, and sonographic evaluations were performed during testing.

Results.—Ten subjects experienced no symptoms. Six subjects with a dorsal point of attachment and 2 subjects with a frontal point of attachment experienced symptoms before 30 minutes. Symptoms were variable. No evidence showed aberrant blood marker shifts.

Conclusions.—There is no strong evidence to support current theories regarding the mechanism of symptoms from HSS. Further investigation is warranted.

Lightning Accidents in the Austrian Alps 2006 to 2014—A Retrospective Study

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