

# Vascular Pathophysiology of Acute Mountain Sickness

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**Objective:** To elucidate how cerebrovascular performance relates to circulating levels of vascular endothelial growth factor (VEGF) and altitude sickness symptoms.

**Background:** One possible cause of Acute Mountain Sickness (AMS) is impairment of endothelial function from hypoxemia, characterized by increased permeability to macromolecules (1-3) increasing interstitial free water, resulting in High Altitude Cerebral Edema (HACE) (4) and High Altitude Pulmonary Edema (HAPE) (5).

**Methods:** 9 volunteers underwent a Transcranial Doppler (TCD) study with Dynamic Vascular Analysis (DVA), which is an advanced analysis of TCD (6). Blood samples were taken from each subject from sea level to the summit of Pike's Peak. Lake Louise, Spielberg, SEES Positive, & Symptom Checklist scores were given at each altitude.

**Results:** Mean Flow Velocity increased with increasing altitude, with a median level increasing from 46.64 cm/sec at sea level to a median of 55.32 cm/sec at altitude. Subjects whose Pulsatility Index (PI) increased with increasing elevation had fewer symptoms than those subjects whose PI decreased with increasing elevation. Subjects with lower DVA velocity-impedance ratios had fewer symptoms than subjects with higher DVA velocity-impedance ratios. Subjects with higher Systolic Acceleration values at all elevations reported fewer symptoms. Subjects with higher VEGF levels at elevations below altitude had more symptoms at altitude, and subjects with higher VEGF levels at altitude reported fewer symptoms at altitude.

**Conclusion:** Our study suggests symptomatic altitude illness implicates a dysfunction of the vascular endothelium resulting from increased hypoxemia with increasing vascular permeability as endothelial gap junctions open in capacitance vessels, resulting in dramatically diminished vascular impedance and serum VEGF levels. As this phenomenon advances, it results in HACE and HAPE. DVA proved to be a sensitive and reproducible method to elucidate and monitor the vascular pathophysiology of AMS.