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ORIGINAL RESEARCH

Field Ultrasound Evaluation of Central Volume Status and Acute Mountain Sickness

Justin T. Pitman, MD; Ghan B. Thapa, MD; N. Stuart Harris, MD, MFA

From the Department of Emergency Medicine, Massachusetts General Hospital, Boston, MA (Drs Pitman and Harris); and the Mountain Medicine Society of Nepal, Maharajgunj, Kathmandu, Bagmati, Nepal (Dr Thapa).

Objective.—To investigate whether ultrasonography can be used for field volume status assessment and to determine whether a detectable difference in intravascular volume exists in individuals with acute mountain sickness (AMS) compared with those without.

Methods.—Study was performed at the Himalayan Rescue Association Clinic in Manang, Nepal, located on the Annapurna trekking circuit at an altitude of 3519 m (11545 feet). A convenience sample was taken from individuals trekking over 5 to 8 days from 760 m (2490 feet) to 3519 m (11,545 feet), comparing asymptomatic trekkers vs those who experienced AMS. Subjects were evaluated for AMS based on the Lake Louise AMS Questionnaire (LLS $\geq$ 3 indicates AMS). After medical screening examination, both groups (control, n = 51; AMS, n = 18) underwent ultrasonography to obtain measurements of inferior vena cava collapsibility index (IVC CI) and left ventricular outflow tract velocity–time integral (LVOT VTI) before and after a passive leg raise (PLR) maneuver.

Results.—There was no statistically significant difference between groups regarding change in heart rate before and after PLR, or IVC CI; however, there was a statistically significant greater increase in LVOT VTI after PLR maneuver in control group subjects compared with those with AMS (18.96% control vs 11.71% AMS; $P < .01$).

Conclusions.—Ultrasonography is a useful tool in the assessment of intravascular volume at altitude. In this sample, we found ultrasonographic evidence that subjects with AMS have a higher intravascular volume than asymptomatic individuals. These data support the hypothesis that individuals with AMS have decreased altitude-related diuresis compared with asymptomatic individuals.

Key words: acute mountain sickness, intravascular volume

Introduction

Acute mountain sickness (AMS) is a common clinical syndrome that occurs at altitudes above 2500 m (8000 feet). The clinical symptoms of AMS include headache and one of the following: gastrointestinal upset, fatigue or weakness, dizziness, or sleep disturbance.[1] Severity and incidence of these conditions depend on the rate of ascent, elevation obtained (especially sleeping altitude), duration of altitude exposure, physical exertion, and inherent genetic susceptibility.[2–4] On acute exposure to 2500 m (8000 feet), approximately 25% of travelers experience symptoms of AMS, and 1% to 2% of people exhibit the life-threatening conditions of high altitude cerebral or pulmonary edema (HACE, HAPE, respectively).[5–8] The underlying pathophysiology of AMS remains poorly defined, but some data support the role of increased fluid retention, which may contribute to cerebral overperfusion, endothelial leakage, and eventual subclinical cerebral edema.[3]

Although the process of acclimatization to altitude is multifactorial and complex, data suggest individuals who experience AMS undergo a blunted bicarbonate [$HCO3^-$] diuretic response on exposure to a high altitude environment.[9,10] Various mechanisms have been proposed for this, including blunted hypoxic ventilatory response[6,9,11,12] and diminished renal perfusion secondary to sympathetic overstimulation.[13,14] It has been demonstrated that individuals with AMS have a neutral or net gain of intravascular volume compared with a net loss of intravascular volume in individuals who remain asymptomatic.[10,11,14]

Corresponding author: Justin T. Pitman, MD, Department of Emergency Medicine, Massachusetts General Hospital, 55 Fruit Street, Zero Emerson, Suite 3B, Boston, MA 02114 (e-mail: jpitman@mgh.harvard.edu).

Numerous studies have been published using bedside ultrasonography (US) to assess intravascular volume status in patients in emergency department and intensive care unit settings.[15–22] Two of the described techniques are of particular interest for volume status assessment: the inferior vena cava collapsibility index (IVC CI), and left ventricular outflow tract velocity–time integral (LVOT VTI) before and after a passive leg raise (PLR) maneuver. The IVC CI measures the change in the IVC diameter during the patient's respiratory cycle. Negative intrathoracic pressure generated during inspiration improves cardiac venous return, which leads to a collapse of the thin-walled IVC and increases right ventricular preload and pulmonary venous capacitance. The degree of IVC collapse with inspiration varies with the intravascular volume state of the individual: hypovolemic individuals have large degrees of collapse, euvolemic subjects have moderate collapse, and hypervolemic subjects have minimal to no collapse. The degree of IVC collapse can be directly imaged with US and measured. A collapse of the IVC by 50% or more is suggestive of a low intravascular volume status and a fluid responsive state (1–10% notes minimal collapse; 100% notes complete collapse [marked hypovolemia]).[18,20,21]

The LVOT VTI technique provides information regarding left ventricular ejection velocity. As a pulsatile system, blood flow is not constant, but rather undergoes rapid acceleration and deceleration during ventricular contraction. By using a Doppler-enabled US, one can measure these changes in velocity and generate an ejection velocity waveform. By performing an integral calculation of the ejection velocity waveform as measured during a single cardiac cycle, the calculated number is expressed as the LVOT VTI. From this information, further physiologic parameters can then be derived as well. LVOT VTI multiplied by the LVOT area can assess beat-to-beat stroke volume, and, when multiplied by heart rate, cardiac output. LVOT VTI, when taken alone, is highly variable among individuals and is difficult to compare. However, dynamic changes can be measured and standardized. Performing a PLR involves transitioning subject from a semirecumbent position (with legs on bed in front of subject) to a supine position with legs raised to 45°. Raising the legs results in lower extremity pooled venous blood to be rapidly returned to the central venous system, which is mechanistically analogous to an intravascular fluid bolus (Figure 1).[15,23–25] Based on the Frank-Starling law of the heart, as venous return improves, ejection velocity, stroke volume, and cardiac output also improve. The largest gains in ejection velocity and stroke volume occur when preload is low (hypovolemia) and volume is added to the system. However, as preload is increased, output gains are reduced, and ultimately reversed as the ventricles are stretched beyond limits of maximal contractility. Previously reported literature in both emergency department and intensive care unit settings have reported changes in LVOT VTI before and after PLR based on volume status. Individuals who improve LVOT VTI by 12% or more after a PLR maneuver have been shown prospectively to improve cardiac output and end-organ perfusion after an intravenous fluid bolus, and were thus hypovolemic.[16,23,24,26] Although the images are technically more difficult than IVC CI to obtain, Royse et al[27] demonstrated that after only 20 studies, even novice US users obtain accurate and reproducible data.

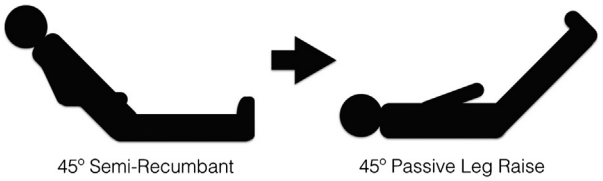

Figure 1. Left ventricular outflow tract velocity–time integral measurements are first taken while subject is in a 45° semirecumbent position. The subject is then moved into a supine position and legs are passively elevated to 45°, rapidly returning pooled venous blood to the central venous circulation and subsequently increasing left ventricular ejection velocity and stroke volume. A >12% increase in the left ventricular outflow tract velocity–time integral with passive leg raise gives evidence of a hypovolemic volume state. These individuals are "fluid responsive."

The aims of this study are to determine whether US assessment of subject volume status using IVC CI and LVOT VTI before and after a PLR maneuver is feasible in a wilderness setting and to demonstrate whether these techniques are capable of detecting intravascular volume differences between an AMS cohort and a healthy control group.

Methods

SUBJECT SELECTION

Subject recruitment took place through the Manang Branch of the Himalayan Rescue Association at an altitude of 3519 m (11545 feet). All of the subjects began the Annapurna Circuit trek in Besisahar at 760 m (2490 feet) and after 5 to 8 days of trekking, reached Manang, an altitude gain of 2759 m (9055 feet). Recruitment posters were placed at mandatory checkpoints and popular tea houses throughout the trek. Subjects, both with and without symptoms of AMS, volunteered either spontaneously or after attending our daily altitude sickness lecture, or were recruited through our clinic. Enrollment criteria included 1) ascent to Manang, Nepal, recruitment center 3519 m (11545 feet) between September 24 and December 2, 2013, 2)