Letters to the Editor

Health Screening on Aconcagua

To the Editor:

In recent years National Park Authorities controlling access to Cerro Aconcagua (6962 m) have insisted that all mountaineers must first agree to participate in a health screening program before climbing the peak. On arrival at 2 altitudes—3395 m and 4365 m—individuals undergo a series of tests that include arterial oxygen saturation, heart rate, and blood pressure measurement. In some cases this has led to members of expeditions being diagnosed with hypertension and prevented from ascending further until their blood pressure has been treated. This approach raises a number of concerns:

1. On Aconcagua, a diagnosis of hypertension is usually made following 2 blood pressure measurements taken on successive days. However, in some instances treatment is begun after just a single measurement. Both approaches are contrary to a number of national guidelines, including the latest from the British Hypertension Society, which states, “An average of 2 readings at each of a number of visits should be used to guide the decision to treat” and which emphasizes the importance of avoiding treatment “on the basis of an isolated measurement.”

2. Even if an accurate recording is obtained, the authors of this letter are not aware of any peer-reviewed evidence identifying blood pressure as a reliable predictor of high-altitude illness or other diseases at altitude. Indeed, a rise in blood pressure tends to be a normal healthy response during the first few weeks at altitude.

In one study conducted on Aconcagua, diastolic blood pressure rose by 10 to 25 mm Hg in 14 healthy men during the course of 22 days spent at altitudes above 4270 m. There also appears to be a lack of evidence to support the treatment of newly diagnosed hypertension at altitude. Rather than reduce medical problems at altitude, the side effects of newly prescribed antihypertensives may trigger a host of side effects ranging from minor gastrointestinal disturbances to life-threatening cases of anaphylaxis.

Each year the world’s highest mountains attract many who are inexperienced and poorly equipped. This inevitably leads to a considerable amount of illness that ranges from simple soft tissue injuries to potentially fatal conditions such as high-altitude pulmonary edema and high-altitude cerebral edema. It is therefore vital that appropriate medical treatment can be easily accessed. However, the use of blood pressure measurement as a screening tool for high-altitude illnesses or other diseases clearly has its limitations.

For many years the Himalayan Rescue Association has supported a number of high-altitude medical posts throughout the Nepali Himalaya. These not only provide a high standard of affordable medical care, but they also focus upon educating all those who venture to altitude through a series of regular lectures and printed notices. This approach, supplemented by a number of free Web-based publications such as “Travel at High Altitude” (www.medex.org.uk), may be a more reliable way of making mountains such as Cerro Aconcagua safer places to visit.

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References


Letters to the Editor

Doctor as Environmental Steward

To the Editor:

In our society the doctor plays many roles: healer, community leader, scientist, advocate for patients, confidante, and role model. Another vital role for today’s physician is as environmental steward. The health of human beings relies on the health of our planet, and doctors can do more to protect both. Doctors need to begin fighting for global environmental issues. On a local level, the doctor is in a unique position to affect change. Furthermore, doctors can be healthier people and role models themselves when they are more personally connected to the environment around them.

As science propels forward at breakneck speed, scientists are becoming better at discovering and describing just how sick our planet is. From global warming to water pollution to smog, the Earth is in trouble. With a sick planet come sick inhabitants. It is estimated that 1.8 million people die each year from diarrheal diseases, with a majority of these cases attributed to unsafe drinking water. In February 2008, chemicals leaking into the Dongjing River in central China turned the water red and foamy, cutting the water supply to over 100,000 people. In Australia, as the ozone layer becomes more depleted, there has been a 41% increase in melanoma since 1993. Our cities are dirty and smog-filled, as an estimated one fourth of the world’s population lives exposed to unhealthy concentrations of air pollutants. As doctors, it is our duty to point out the connections between the health of the Earth and the health of the human race. We must perform the studies, raise the issues before government officials, start campaigns, strike up public awareness, and facilitate change.

Perhaps a more direct and practical way for many doctors to become stewards for the environment is by working on a local level. As more and more adolescents grow up obese, with their only interactions with the wilderness occurring through video games and movies, it is time for doctors to stand up and shout, “Get out and see the mountains!” Put trail maps and magazines like National Geographic next to the People Magazine in the waiting room. Start wilderness interest groups and recycling programs at the hospital. Advocate for city-wide recycling programs. While these activities may be unrelated to the daily practice of medicine, as a doctor your voice goes far. Question the quality of your town’s water and air and fight to improve access to safe parks. These quality improvement checks are the doctor’s responsibility—if doctors don’t take on these challenges, environmental problems with real solutions may slip through the cracks. When patients begin to take advantage of a healthier local environment, they become healthier patients.

As Paul Auerbach suggests, the field of medicine can become more connected with environmental issues by incorporating them into the medical school curriculum. My own recent experience as a student on a wilderness medicine rotation has impacted the way I want to live my life personally as a doctor. I want to live a healthy life and help others to do the same. I plan to trade treadmill for trail, to enjoy and participate in nature. When doctors have a personal connection with the environment, advocacy for environmental issues becomes more important. As role models, doctors can then share stories and thoughts about their relationships with the wilderness and inspire others to explore. Take a trip into the woods during your vacation. Explore the wild world while it still exists, and bring a friend with you who has not experienced the wonderful world away from roads and billboards and smokestacks.

Doctors are healers, and the Earth is a patient worth saving. On a global, local, and personal level, let us use our positions of influence to make a difference. Let doctors first do no harm and, second, try to help steward our planet to health.

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References

Ultrasound Identification of Patella Fracture

To the Editor:

The use of ultrasound as an adjunct technology is being increasingly employed for the diagnosis and treatment of musculoskeletal injury. Applications for its use include identification of radiolucent foreign bodies, fluid collections, and bony fractures. Advantages of sonography include portability, lack of ionizing radiation, and the ability to obtain multiple images in a relatively short period. We describe its use in the identification of a transverse fracture of the patella and compare it to a standard radiograph. This application of sonography has potential use in austere environments, in which access to traditional radiography is limited.

Case report

A 59-year-old male presented to the emergency department 1 day after a fall directly onto his right knee. He reported pain to the right knee with swelling and decreased range of motion. On exam, the patient was in mild discomfort, with tenderness to palpation of the right patella. There was a significant amount of soft tissue swelling, a moderate suprapatellar effusion, and decreased right leg extension. Given anticipated delays in obtaining plain radiography as a result of high patient volume in the emergency department, an ultrasound of the right patella was performed using a 10-5 MHz linear transducer (SonoSite MicroMaxx, Bothell, WA). Longitudinal images demonstrated a cortical disruption with associated hematoma (Figure 1). Radiographs confirmed a mid-patellar transverse fracture (Figure 2) with a contour identical to that obtained by bedside sonography.

Discussion

The patella is the largest sesamoid bone in the body.\(^1\) It serves as a protective barrier for the anterior portion of the knee and as a fulcrum for the extensor function of the quadriceps tendon on the lower extremity. Any disruption of the quadriceps tendon, patella, patellar tendon, or its insertion onto the tibial tuberosity limits full extension of the lower extremity. A bipartite patella occurs in approximately 2% of the general population.\(^2\) In this case, the patella develops from 2 separate ossification centers, and the 2 sections are connected by thick fibrous tissue. This should not be confused with an acute traumatic injury.

The use of ultrasound for the identification of fractures has been described previously.\(^3,4\) The exam is performed with a high-frequency linear transducer, as this provides maximal spatial resolution and is optimal for imaging superficial structures in great detail. The ultrasound exam may require the use of a water bath or stand-off pad (a gel- or water-filled cushion) for superficial osseous structures such as the phalanges and carpal...
bones. Normally the bone-soft-tissue interface is seen as a continuous, reflective, hyperechoic line. Acoustic shadowing is seen deep to the interface as a result of the high acoustic impedance of the bony cortex. Sonographic evidence of a fracture includes disruption of this hyperechoic line and may be associated with an anechoic or hypoechoic collection near the cortical break, suggestive of a hematoma. The diagnostic accuracy of ultrasound for fracture detection differs based on the location and severity of fracture. However, there is some evidence that ultrasound performs with similar sensitivity and specificity compared with plain radiography when used by experienced operators. Of note, while our patient suffered a substantial patellar fracture, the sonographic diagnosis of subcutaneous soft tissue fracture has also been described by Ditchfield et al., Klerx-Melis and Watt, and Grobbelaar and Bouffard.

In addition to fracture detection, additional knee pathology may be diagnosed using ultrasound. Specifically, ultrasound can be used to identify the quadriceps tendon (by sliding the probe cephalad from the superior pole of the patella) and the patellar tendon (by sliding the probe caudad from the inferior pole of the patella) and to assess for tendon disruption. In addition, knee effusions and meniscal injuries may be accurately detected using ultrasound.

To our knowledge, this is the first description of the use of bedside ultrasound by emergency physicians to diagnose a patellar fracture. The identification of bony injury using portable ultrasound units may be useful in circumstances in which radiographs are not possible or when the provider wishes to avoid the use of ionizing radiation. In fact, portable ultrasound units have already been used in mass casualty situations for identification and triage of trauma victims. The use of portable ultrasound for the detection of fractures could specifically affect the practice of wilderness medicine by determining whether splinting and/or medical evacuation are necessary following acute musculoskeletal trauma.

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Causes of Death From Avalanche

To the Editor:

In 2 recent studies McIntosh et al1 and Hohlrieder et al2 have focused on the causes of death from snow avalanches, putting the pathophysiology of avalanche burial in a new light. This has been acknowledged by Martin Radwin in the Editorial for a recent issue of Wilderness and Environmental Medicine.3 We would like to add a comment related to the techniques of postmortem examination of avalanche victims and the study methodologies used in the cited publications.

We endorse the opinion that autopsy, strictly defined as a full postmortem examination of both external and internal organs, is the gold standard in establishing the cause of death. If autopsy is not conducted, then a documented full external examination and the review of any premortem clinical records, including radiology and computer imaging technologies, should be the required
minimum for a reliable postmortem diagnosis. Otherwise, unreliable diagnoses are to be expected. Furthermore, the study sample should be representative and should minimize selection bias.

In this regard some of the often-cited studies should be critically reviewed, focusing particular attention on 1) the method of postmortem examination and 2) the reliability of the study sample. We have reviewed all studies dealing with the causes of avalanche deaths from 1970 to date.

The dissertation of Markwalder in 1970 comprised a review of 66 Swiss avalanche accidents occurring from 1961 to 1967. Two of the 43 fatalities described underwent full autopsy, while the method of postmortem examination remained unclear in the remaining 41 cases. All 7 traumatic deaths occurred in buildings or vehicles, in contrast to the results of other studies involving investigations of victims hit in open areas. The author did not exclude a selection bias, as the sample was only a small fraction of the 171 avalanche fatalities documented in Switzerland in this time period.

In 1972 Lugger and Unterdorfer published a selection of 20 autopsies of avalanche victims in Tyrol, Western Austria; deaths occurred between 1964 and 1970. However, similarly, the authors did not indicate whether their sample represented the total number of fatalities in that area in that time period.

The authors of 2 French studies, Eliakis in 1974 and Lapras in 1980, did not specify how the cause of death had been determined. Eliakis reported 24 avalanche fatalities, quoting a Swiss investigator without description of the circumstances or the time period, and, therefore, additionally, this sample may not be representative.

In 1989, Stalsberg et al reported 2 major snow avalanches with 18 fatalities in Norway in 1986 and 1987. Six victims underwent full external examination; 12 underwent full autopsy. Though all fatalities from these 2 investigated accidents were included, there was no reference to the total number of avalanche fatalities in this area, and since the sample only included 2 successive winters, the findings may not be representative.

In 1989 Grossman et al reviewed all avalanche fatalities in Northern Utah from 1982 to 1987. Four out of 12 fatalities were apparently not subject to external examination, while 7 were subject to clinical examination during resuscitation, and only 1 victim was subject to autopsy. The official causes of death differed from the clinical records. The latter incorporated combinations of asphyxia, multiple trauma, and hypothermia.

Tschirky al did not present original data in their article about avalanche rescue devices in 2001, but they quoted a dissertation of Weymann, published in 1999. This prospective study included all persons caught by avalanches in Switzerland from 1991 to 1996. In 91 of 99 fatalities the cause of death was determined using a standardized questionnaire. However, the author described only 4 autopsies and did not indicate how the remaining 87 causes of death were established.

In 1996, Locher and Walpolt retrospectively analyzed the causes of cardiac arrest in 19 of 32 hospitalized avalanche victims in Switzerland from 1980 to 1987. During this time period, however, a total of 245 avalanche fatalities were documented in Switzerland, and a selection bias therefore cannot be excluded.

In our opinion the above-mentioned studies might not meet the requirements for a valid determination of cause of death because either samples were not representative or the method of postmortem examination was not adequate or clearly documented. Therefore, caution should be taken in including these investigations when analyzing causes of death in avalanche victims.

Only 3 studies (the Table) complied with the requirement that 1) the research was based on a representative sample and 2) either full external examination or full autopsy was performed to determine the cause of death.

In 1993, Tough and Butt presented all fatal accidents associated with backcountry skiing in Alberta, Canada, from 1980 to 1991, including 15 avalanche deaths. External examination was performed in 11 and autopsy in 4 cases.

The study of McIntosh et al included all avalanche fatalities in Utah from 1989 to 2006. Of the 56 fatalities, 28 were subject to full autopsy, while 28 were subject to external examination alone.

Concurrently, Hohlrieder et al analyzed all avalanche accidents in Tyrol, Western Austria, between 1996 and 2005; their data included 36 fatalities. In 30 cases autopsy was performed, while in the remaining 6 cases cause of death was determined by extensive clinical investigation.

In these studies we found no significant difference in the proportions of causes of death determined by external examination compared to autopsy ($P = .622; \chi^2$ test) and between the countries in which the studies were conducted (ie, Tyrol, Austria; Alberta, Canada; and Utah, USA) ($P = .730; \chi^2$ test). It should be emphasized, however, that as a result of the limited number of available studies with an overall rather-small study population, results might not be generally valid.

The resulting percentages of lethal trauma (5.6% in total, with 95% CI [2.3%, 12.3%]; 4.4% CI [1%, 16%] with external examination and 6.5% CI [2%, 16.5%] with autopsy) are also consistent with the survival function calculated by Falk and Brugger and Brugger et al. The initial course of the survival curve showed a 91% survival probability within 18 minutes after burial.
Causes of death from avalanche in relation to method of postmortem examination

<table>
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<tr>
<th>Method</th>
<th>Autopsy (%)*</th>
<th>HT Subtotal</th>
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<td>26 (92.9)</td>
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<td>2 (4.4)</td>
<td>43 (95.6)</td>
<td>4 (6.5)</td>
<td>57 (93.5)</td>
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<td>1 (0.9)</td>
<td>107 (99.1)</td>
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*Trauma as sole cause of death.
§Including cases presenting traumatic injuries, not considered the primary cause of death. HT indicates hypothermia.

That would indicate a rate of trauma deaths immediately after burial of about 9% with CI (7.1%, 11.6%).

Traumatic injury leading to death within a few minutes needs substantial body impact during the passage of the avalanche. It is unlikely that snow masses alone could be strong enough to inflict lethal trauma to a human body. From the survival analyses, such lethal trauma from the snow alone is not supported. External factors (eg, impacts with rocks or trees during the avalanche or impacts with man-made structures when hit in vehicles or buildings) are the logical causes of trauma. We would like to point out, however, that a theoretical incidence of traumatic deaths of 9% within the first 18 minutes does not preclude an overall higher rate of trauma. Victims suffering potentially lethal trauma may die first from asphyxia if their breathing is impaired.

Though we found no variation between the countries represented in these 3 studies, we agree with Radwin that trauma mortality may differ in relation to the local geography, not just between countries, but even within a mountain chain.

In our review, hypothermia (0.9% of cases, 0% with external examination and 1.6% with autopsy; the Table) appears to play a negligible role as the sole cause of death from avalanche. It has been assumed, however, that avalanche-related hypothermia emerges from prolonged snow burial in the presence of an air pocket and free airways and is associated with hypoxia and hypercapnia, the so-called triple H syndrome. It is questionable whether this combination of slow asphyxia and hypothermia is detectable solely by external examination or autopsy without clinical data. Additional investigation is needed to clarify the significance of this recently designated pathology as a cause of death, paying attention to body temperature on extrication from the snow masses.

In conclusion, thanks to the studies of Hohlrieder et al and McIntosh et al on the European and American continents, we catch a new glimpse on the pathophysiology of avalanche burial. In these 2 well-performed studies asphyxia was overwhelmingly the most life-threatening factor, and this reflects its dominant role in avalanche burial, whereby hypothermia may act as a co-factor in some cases. Trauma remains an important but unresolved issue requiring further study, as stated by Radwin.

To avoid future misunderstanding, investigators should exercise caution with often-quoted older studies and seek those in which the cause of death is established by full external examination or the gold standard—full autopsy. Moreover, particular attention should be paid to the reliability of the sample by seeking a comprehensive series within a geographic area.
Skiing Injuries in Perspective

To the Editor:

Congratulations to Flores, Haileyesus, and Greenspan with regard to their excellent article on National Estimates of Outdoor Recreational Injuries Treated in Emergency Departments, United States, 2004–2005. The article calls attention to recreational injuries, an important component in the need for injury control. However, when we first looked at their table 6, we thought “Where is skiing?” The omission of skiing in the table might leave the casual reader with the impression that skiing is not an important source of outdoor recreational injuries. The authors apparently excluded skiing injuries, as they were “concentrated in a few hospitals.” It is not surprising that there are more skiing injuries in hospitals near ski areas and that the hospitals selected in the Consumer Product Safety Commission’s sample do not provide a reasonable estimate of skiing injuries.

A major problem in measuring the importance of recreational injuries is the following: What is to be used as a denominator? The authors have used rates per 100,000 population, which gives an estimate of the societal importance of injuries resulting from each activity. Equally important is measuring the relative danger of persons exposed to the various activities. For skiing, the denominator is particularly difficult. Is it the number of people skiing? The days of skiing? Hours spent skiing? “Double black diamonds” as compared to hours on “bunny slopes”? Although the number of “ski visits” to the northeast areas decreased from 14.7 million in 1986–1987 to 11.8 million in 2006–2007, the number of skiers still exceeds the number of snowboarders.2

Although China does not seem a likely place for skiing injuries, the problem of ski injuries in the United States may be a forewarning of greater problems in China. Surprisingly, the number of people in China who skied went from 300,000 in 2000 to 3 million in 2005. China has great opportunities for “extreme skiing,” which is growing in popularity and should be of interest to the readers of Wilderness and Environmental Medicine. In conclusion, we call attention to an article on skiing injuries by William Haddon, Jr, “The father of...
Injury, epidemiology and control," and colleagues, whose landmark 1962 paper described the frequency of specific injuries and variations with age, sex, and skiing experience.\(^5\)

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References


Persistent Elevation of VEGF and Prostacyclin Following Poor Cardiopulmonary Adaptation to High Altitude

To the Editor:

Hypobaric hypoxemia is a potent stimulus for the release of erythropoietin and for some markers of angiogenesis, such as vascular endothelial growth factor (VEGF).\(^1\) Vascular endothelial growth factor stimulates angiogenesis, and angiopoietin-2 acts with VEGF to stimulate angiogenesis in the myocardium.\(^2\) Similarly, prostacyclin (PGI\(_2\)) is a member of the prostaglandin family of lipid mediators, which have potent vasodilator and antithrombotic activities.\(^3\) Some limited data have been reported on the release of selected markers of angiogenesis, including VEGF in climbers exposed to high altitude with or without symptoms of acute mountain sickness (AMS). However, no study has explored the concomitant changes in VEGF, angiopoietin-2, and PGI\(_2\), as well as selected markers of inflammation and oxidation, concomitantly in relationship with good vs poor adaptation to high altitude.

We investigated the changes in selected markers of angiogenesis, prostaglandin, and some markers of subclinical inflammation and oxidative stress 24 hours prior to departure for the Bolivian Altiplano and within 24 hours of return to sea level. The objective of this expedition was to summit Mount Sajama, the highest peak in Bolivia, at an altitude of 6522 m. Participants were exposed to an altitude between 3600 and 6522 m for 19 days.

The study population consisted of 5 participants (4 males/1 female) aged 30 to 64 years living chronically at sea level. The diagnosis of AMS was performed using the Lake Louise\(^4\) and the Hackett\(^5\) scoring systems at the highest point reached during the expedition.

From the 5 climbers, 2 participants developed moderately severe symptoms of AMS. One climber, aged 64 years, experienced mild headache, recurrent difficulty sleeping, profound fatigue, and dyspnea on minimal exertion at an altitude of 4800 m (Hackett score = 4; Lake Louise score = 6). This climber stopped his climb at 5300 m. One participant developed some fatigue and severe intractable headache at 5680 m and returned to base camp at 4800 m (Hackett score = 3; Lake Louise score = 4). Data are presented in the Table.

Table. Changes in plasma markers prior to departure and within 24 hours upon return to sea level*  

<table>
<thead>
<tr>
<th>Marker</th>
<th>No AMS (n = 3)</th>
<th>AMS-CP (n = 1)</th>
<th>AMS-CNS (n = 1)</th>
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<tbody>
<tr>
<td></td>
<td>BSL</td>
<td>Post</td>
<td>BSL</td>
</tr>
<tr>
<td>VEGF (pg·mL(^{-1}))</td>
<td>204 ± 38</td>
<td>210 ± 43</td>
<td>222</td>
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<tr>
<td>Ang-2 (pg·mL(^{-1}))</td>
<td>1605 ± 601</td>
<td>1257 ± 280</td>
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</tr>
<tr>
<td>6-Keto-PGF(_{1α}) (pg·mL(^{-1}))</td>
<td>37.7 ± 36.1</td>
<td>49.5 ± 40.0</td>
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<tr>
<td>IL-6 (pg·mL(^{-1}))</td>
<td>12.7 ± 15.9</td>
<td>12.2 ± 8.1</td>
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<tr>
<td>TBars (pg·mL(^{-1}))</td>
<td>10.8 ± 2.6</td>
<td>9.42 ± 3.70</td>
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</table>

*AMS indicates acute mountain sickness; CP, cardiopulmonary; CNS, central nervous system; BSL, baseline; Post, postexpedition; VEGF, vascular endothelial growth factor; Ang-2, angiopoietin-2; 6-Keto-PGF\(_{1α}\), 6-keto-prostaglandin F\(_2\); IL-6, interleukin 6; and TBars, thiobarbituric acid-reactive substances.
This exploratory study reports for the first time the plasma levels of multiple mediators of angiogenesis and selected markers of inflammation and oxidative stress concomitantly in 5 healthy volunteers prior to departure and within 24 hours of returning from 19 days on the Bolivian Altiplano. We report different plasma levels of VEGF and PGI\textsubscript{2} in 2 climbers who experienced poor adaptation to high altitude. Here, we report a marked increase in VEGF and PGI\textsubscript{2} within 24 hours of returning from an altitude of more than 4000 m for 19 days in the older climber who experienced some delay in the acclimatization process but who also exhibited some cardiopulmonary features of poor adaptation to high altitude. These observations contrast with the findings from the climber who experienced AMS mostly with central nervous system symptoms.

Vascular endothelial growth factor concentrations before the expedition were consistent with levels reported in previous investigations. The change in VEGF in response to high altitude has been a matter of controversy. Reports have shown no increase or a significant increase within 20 hours to 7 days upon exposure to an altitude of 3600 m or higher.\textsuperscript{1,6} Vascular endothelial growth factor appears to increase in nearly all climbers in response to hypobaric hypoxemia. Tissot van Patot\textsuperscript{1} reported significantly higher levels of plasma VEGF and lower levels of VEGF soluble receptor in participants who had AMS within the first 24 hours upon exposure to 4300 m. In contrast, Dorward et al\textsuperscript{6} reported no evidence of an association between AMS and changes in VEGF at either 3650 or 5200 m. However, the clinical characteristics and the mode of presentation of AMS were incompletely reported in that specific study. In addition, none of these reports involved exposure to high altitude for more than a week. Here we also reported a concomitant increase in PGI\textsubscript{2} level in the climber suffering from poor cardiopulmonary adaptation to high altitude, indicating a significant increase in cardiovascular stress.

There are some significant limitations to these observations. Vascular endothelial growth factor levels were not measured at different periods of the acclimatization process nor later during the expedition. In addition, VEGF soluble receptor-fms-related tyrosine kinase 1 was not measured. The participant with poor cardiopulmonary adaptation to high altitude was much older than the other climbers. Also, the small sample size did not allow for adjustment for the age effect, nor did it allow for further analyses about the relationship between clinical characteristics and biomarkers.

In conclusion, our study demonstrates that there were no significant changes in selected markers of angiogenesis, inflammation, and oxidative stress in climbers who did not experience mountain sickness, whereas in the 2 participants who experienced mountain sickness, the plasma concentrations of these biomarkers were perturbed. Vascular endothelial growth factor and PGI\textsubscript{2} may be associated with poor cardiopulmonary maladaptation to high altitude. Whether these vascular biomarkers are truly related to altitude illness or inadequate acclimatization requires study in a larger population.

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References
A Cardiac and a Kidney Transplant Patient Above 6000 Meters in Bolivia

To the Editor:

Over the past 2 decades, survival and quality of life following cardiac transplantation have markedly improved, mainly as a result of advances in immunosuppressive drug regimens. However, the survival rate following heart transplantation remains quite limited and is now approaching 50% at 15 years. The decrease in life expectancy in this population is largely related to associated comorbidity, such as cardiac-allograft vasculopathy, accelerated atherosclerosis, renal failure, hypertension, and diabetes. As is the case for patients following cardiac transplantation, patients experiencing kidney transplantation are at high risk of cardiovascular events. The etiology for accelerated atherosclerosis remains largely unexplained, but subclinical inflammation may play a significant role. Hypobaric hypoxemia is a potent stimulus for the release of erythropoietin and for the increase in some markers of angiogenesis, such as vascular endothelial growth factor. Similarly, prostacyclin is a member of the prostaglandin family of lipid mediators, which have potent vasodilator and antithrombotic activities that increase in response to vascular stress.

Some mountains have been successfully summited by organ transplant recipients. Here we report the experience of 2 climbers, one a recipient of a cardiac transplant and the other of a kidney transplant, on their ascent to Mount Sajama, the highest summit in Bolivia (6522 m). We also explored the changes in selected biomarkers in these climbers before and after the expedition.

Two patients participated in this expedition. The cardiac transplant recipient was aged 35 years and the kidney transplant subject was 41 years. The cardiac transplant patient climbed to an altitude of 4800 m (CTx = 90%; KTx = 82%; Group = 80%). Similarly, saturations were similar at Sajama base camp at an altitude of 4800 m (CTx = 90%; KTx = 82%; Group = 80%). The cardiac transplant recipient reached an altitude of 6120 m on the foothills of Mount Sajama. He presented with overall exhaustion. His O2 saturation measured before turning back to base camp was 69%. The kidney transplant recipient reached the summit at an altitude of 6522 m. Saturations were not measured on summit.

Both cardiac and kidney transplant subjects exhibited no significant changes in any of the markers of angiogenesis upon return to sea level. Similarly, there were no changes in plasma interleukin-6 and thiobarbituric acid-reactive substances before or upon return from the expedition.

This expedition was significant in the field of organ transplantation as it highlights the fact that organ transplant is not a contraindication for high-altitude mountain climbing. This is evidenced by the fact that both patients achieved an altitude over 6000 m. Both transplant patients tolerated the high altitude very well and exhibited similar changes in hematology and biochemistry profiles.

Cardiac and other organ transplant recipients have successfully summited high mountains within the last few years. These very special patients exhibited no obvious limitations. Here we report similar biochemistry profiles and adaptations to high altitude compared with other climbers who adapted well to high altitude (3 of the 5 healthy climbers).

The cardiac transplant patient climbed to an altitude of 6120 m and stopped because of overall exhaustion. He presented with no significant cardiovascular or overall health problems in response to high altitude. The low saturation achieved at maximal altitude reached most likely represents a normal response to exercise at such a high altitude. In addition, neither of the organ transplant recipients presented with infection or any signs of right heart failure.

We found similar levels of selected markers of angiogenesis, subclinical inflammation, and oxidative stress in organ transplant recipients compared with the healthy climbers who summited Sajama. In addition, there were...
Letters to the Editor

Table. Laboratory parameters on admission in a 12-year-old boy bitten by *Vipera berus*. All values are within normal limits.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Unit</th>
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</thead>
<tbody>
<tr>
<td>Partial thromboplastin time</td>
<td>31</td>
<td>seconds</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>96</td>
<td>seconds</td>
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<td>Antithrombin 3</td>
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<td>%</td>
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<td>Hemoglobin</td>
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<td>Creatinine</td>
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<td>Aspartate aminotransferase (GOT)</td>
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<td>U·L$^{-1}$</td>
</tr>
<tr>
<td>Gamma-glutamyl transferase</td>
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</table>

Vipera berus Bite in a Child, With Severe Local Symptoms and Hypotension

To the Editor:

Snakebites are very rare incidents in central parts of Europe (Austria, Germany, and Switzerland), and mortality has been reported to be 0% for adults.\(^1\) The experience of medical personnel with treating snakebites may not be substantial, which at times may lead to an unnecessary time delay in treatment or difficulties in decision making, especially where children are concerned. Viper venoms contain more than 100 components and may cause local symptoms and signs, such as pain and swelling, but also systemic findings, such as gastrointestinal and circulatory disturbances and coagulation disorders. Children and elderly people are more likely to develop severe symptoms.\(^2\)

We would like to report a 12-year-old boy who was bitten on his right index finger by an adder (*Vipera berus*) in the outskirts of Innsbruck. Almost immediately after the bite, the patient developed signs of shock and complained of gastrointestinal pain and difficulty breathing. Upon arrival of the emergency medical system personnel 10 minutes after the bite, the patient was somnolent, hypotensive (blood pressure: 75/55 mm Hg), and tachycardic (heart rate: 145 beats/min). He had tachypnea with inspiratory stridor and a peripheral oxygen saturation (SpO$_2$) of 90%. The patient was immediately supported with oxygen and rapid infusion of 1000 mL of a 0.9% NaCl solution. He was given 0.1 mg epi-

References

nephrine, 4 mg dimethyprindene, and 125 mg prednisolone hemisuccinate intravenously. He appeared to stabilize and was transported to the clinic. Upon arrival at the clinic, 45 minutes after the bite, neither gastrointestinal symptoms nor breathing difficulties were present. Apart from local pain, the patient was free of symptoms. His blood pressure was 90/60 mm Hg.

The local swelling, already present upon arrival, was affecting the right index finger and part of the hand. Local symptoms turned out to be the major problem in this case, as the swelling increased massively during the next 3 days, with a maximum extension to the right thorax with development of a subcutaneous hematoma. The patient developed no evidence of compartment syndrome, however. The patient received symptomatic treatment under continuous monitoring of electrocardiography, SpO₂, and blood pressure. Laboratory findings at admission are noted in the Table. The patient’s coagulation studies remained normal.

On the third day after the bite, the patient complained of heart palpitations, and the electrocardiogram showed ventricular extrasystoles. These were monitored and resolved without treatment.

The patient was treated as an inpatient for 6 days, until the swelling and hematoma finally began to resolve. At that time, a superficial area of necrosis (5 mm²) at the bite site had formed. This healed well, leaving only a small scar.

Since the incidence of snakebites is far higher in Sweden than in many other European countries, this country has a long tradition and much experience in the treatment of snakebites. Dr Karlsson-Stibber and Dr Persson from the Swedish Poison Centre published the Stockholm criteria with indications for the use of antivenom after snakebites in Sweden. To date, however, these criteria have been recommended for adults only. Even though the incidence of adder bites in children seems to be higher than in adults, there are no specific recommendations for antivenom treatment after adder bites in children.

Allergic reactions are uncommon following snakebites, particularly if the patient has never before been envenomated. Such reactions can, however, be life threatening. Initial management of anaphylaxis includes the use of epinephrine, airway management, and administration of intravenous fluids, antihistamines, and steroids. We could not find any studies related to the effectiveness of such treatment for bites from Vipera berus. In our patient the decision not to give antivenom was made because of concern regarding allergic reactions after antivenom treatment. Such concerns could certainly lead to a late or insufficient treatment of patients with significant snakebites. Specific recommendations for antivenom use in children suffering from bites by European vipers could help reduce time delay in treatment or difficulties in decision making.

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Acknowledgments
The authors acknowledge the kind support of the Austrian Poisons Information Centre, the Natural History Museum in Vienna (Mag Johannes Hill), and especially the Swedish Poison Centre (Dr Christine Karlsson-Stiber).

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Death by Volcanic Laze

To the Editor:

Despite the potential risk to human life, active volcanoes have become popular attractions for tourists and others interested in adventure recreation pursuits. One such example is the ongoing eruption of Kilauea Volcano and
the Pu’u O’o Vent in Hawaii Volcanoes National Park, where tourists and other spectators regularly gather to hike to active lava flows and view lava flowing into the ocean. In November 2000, authorities at Hawaii Volcanoes National Park received a report of 2 dead bodies found near the ocean entry in an area of the park referred to as the Eruption Site. Both bodies (1 Caucasian male; 1 Caucasian female) were located approximately 91 m directly inland from the ocean entry and were located on the eastern side of active lava flows. The bodies were located approximately 12 m apart from each other. In addition, the male victim had a backpack that was found 6 m west of his body. An expert geologist at the scene with considerable experience in the park reported seeing no sign of volcanic spatter and no evidence of a recent explosion in the area.

The bodies were removed via a sling load attached to a Hawaii County rescue helicopter. Two days later autopsies of the victims were conducted by a medical examiner for the County of Honolulu. Dental records identified the victims as a 43-year-old male and a 42-year-old female. Examination of the female victim found no obvious burns on her clothing. However, her state of decomposition was extremely advanced for the estimated time of death (maximum 48 hours prior to body recovery) and in comparison to the male victim. According to the medical examiner, the female had perimortem first- and second-degree burns to her head, neck, shoulders, upper chest area, and to all limbs. She also had perimortem wounds to the head, face, and limbs that were superficial in nature. Examination of the male victim also found no obvious burns to his clothing. Moreover, abrasions and lacerations to his body were also perimortem and superficial in nature. However, there were very obvious perimortem first- and second-degree burns to his head, neck, limbs, and areas of his trunk.

During the autopsies, no evidence indicating that lightning or violence were factors in the deaths was found. The medical examiner did report, however, that the burns were consistent with those caused by a hot gas or vapor rather than contact with hot liquid, contact with a hot object, or radiant heat. This was based on the findings of undamaged clothing and the regions of the bodies that were burnt. For example, both victims sustained burns to areas that were unprotected or protected by a single layer of clothing. No burns were indicated or obvious where there were 3 layers of clothing. In areas where there were 2 layers of clothing there were some burns indicated and observed where the clothing may have been penetrated or tucked up. The final cause of death determined by the medical examiner was death as a result of pulmonary edema caused by inhalation of
volcanic laze, sustained when the victims were exposed to the plume near the ocean entry. This incident highlights a potential hazard when entering areas of volcanic activity. What makes this a case of interest, however, is that it was the first known incident of its nature in Hawaii and that it specifically highlights a potential global hazard present in locations where lava enters ocean waters. Conditions near the ocean entry typically involve exposure to volcanic laze, a dense hydrochloric acid (HCl) mist that is formed when hot lava enters the ocean.\(^1\)\(^-\)\(^3\) This laze is often mistakenly referred to as a steam plume. Heat from the lava entering the ocean rapidly boils and vaporizes seawater, producing a large white plume. This plume contains a mixture of HCl and concentrated seawater that is a brine with a salinity about 2.3 times that of seawater and a pH of 1.5 to 2.0.\(^4\) Moreover, dense laze plumes are known to contain as much as 10 to 15 ppm of HCl.\(^4\) The density of the plume decreases as it moves away from the ocean entry, but acid rain commonly precipitates on individuals and land near the plume’s proximity.\(^4\) Hence, following the inhalation of the laze, the bodies of the victims were exposed to extreme heat and acidic conditions during the maximum 48 hours they were at the ocean entry.

In addition to the loss of life, the final cost of this incident included $3025 for aircraft assistance and $9507 for personnel costs. Volcanic hazards at the Eruption Site and in the vicinity of the ocean entry are not always recognized, and access to the area is not restricted. However, warning signs and safety messages should be strongly heeded by all visitors.

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Frostbite Injury Related to Chlorethane Application

To the Editor:

Cryotherapy is commonly used in sports medicine to reduce the pain of acute injuries. Ice packs, cryomassage, ice bath immersion, contrast baths, and vapor coolant sprays are various methods of performing cryotherapy. Vapor coolant sprays used for cryotherapy usually contain propane, butane, or chlorethane [ethyl chloride, C\(_2\)H\(_5\)Cl\(\text{H}11034\)H\)). Chlorethane drops the skin temperature from 33°C to less than 10°C within 10 seconds after application.\(^1\) For this reason, cooling the skin area by spraying closely for a prolonged time can cause frostbite injury. Chlorethane is used rarely in health care facilities, given the availability of topical anesthetic gels. However, it is popular in sports medicine and prehospital areas and might be included in some wilderness first aid kits.

Cold injuries may be seen with the improper use of both cold packs and vapor coolant sprays. Nevertheless, complications such as sensations of pressure, burning, and pain; alteration of pigmentation; corneal damage; dermatitis and skin irritation (especially after chronic exposure); and frostbite and necrosis due to prolonged application may rarely occur.\(^2\)

Frostbite injury is considered a potential complication of improper topical application of chlorethane and has been described as a complication of other topical coolants/anesthetics.\(^3\)\(^-\)\(^4\) Nevertheless, no case reports describe frostbite injury due to chlorethane application. Here we describe a case of frostbite due to improper application of chlorethane vapor coolant spray. A 19-year-old male patient presented to our emergency department complaining of blistered lesions on his right calf. The previous day he experienced pain in his right calf while running, and he applied vapor coolant spray (Chloraethyl spray, Adroka AG, Allschwil, Switzerland) to the injured area for 20 to 30 seconds. The application of chlorethane was from approximately 15 to 20 cm above the skin. Twenty-four hours later he developed blisters on his calf at the site of application. He denied having exposed the area to any heat source. Physical examination revealed noninfected blisters accompanied by erythematous skin in an area 8 cm \(\times\) 15 cm on his
Letters to the Editor

Figure. Second-degree frostbite injury caused by chlorethane application spray on the right calf of the patient.

calf (Figure) felt to be consistent with superficial frostbite. Management included aspiration of the blisters and application of bacitracin antibiotic ointment. He followed a course of regular dressing changes, and the lesions healed without complication over 14 days. On telephone follow up 6 months later, the patient complained of black pigmentation at the injured area.

There are occasional case reports in the literature of toxic dermatologic effects of propane and butane. La
cour et al. reported a case of deep frostbite in an 8.5-year-old child after the improper use of a toilet air freshener containing propane and butane. The injury was so severe that a skin graft was ultimately required.

Management of blisters associated with frostbite is somewhat controversial. Current approaches include leaving blisters intact, simple aspiration, and debridement. Hemorrhagic blisters should not be debrided, because this often results in tissue desiccation and worsened outcomes. Aspiration for hemorrhagic blisters is also controversial. Topical antibiotics, such as bacitracin, are standard therapy for these injuries. However, systemic antibiotics should not be used unless there is evidence of superinfection of the injured tissue (eg, surrounding cellulitis). Although the evidence supporting its use is scant, aloe vera cream may be helpful in the treatment of frostbite blisters given its anti-inflammatory effects through inhibition of the arachidonic acid cascade.

This case illustrates the importance of using care in applying cryotherapy, including topical application of vapor coolant sprays, to areas of injury in order to avoid inducing a cold injury.

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Acknowledgment

This letter was supported by the Akdeniz University Foundation.

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