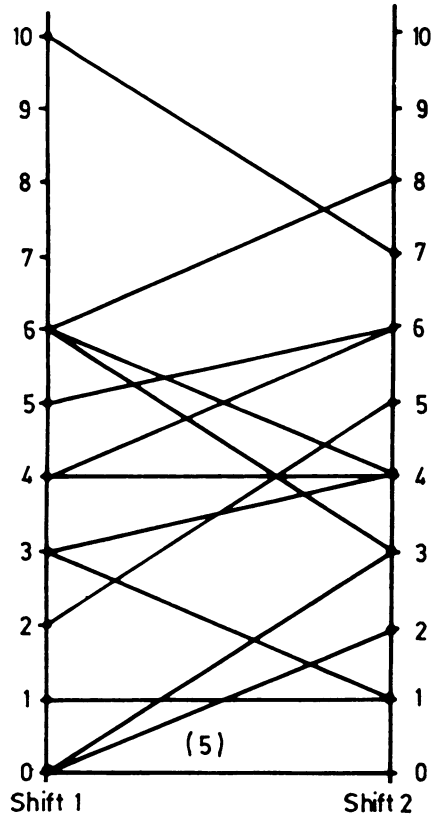
Reproducibility of individual response to exposure to high altitude

The response of people who live at sea level to the low oxygen concentrations at high altitude varies widely. Some people are relatively unaffected while others suffer severe acute mountain sickness.[1] Anecdotal reports and experiments in decompression chambers have indicated that responses to high altitude are reproducible,[2] but this has been difficult to substantiate under controlled field conditions. A two year study of the staff manning an astronomical observatory 4200 m above sea level permitted an examination of individual performance during repeated ascents.[3]

Methods and results

The 3·8 m United Kingdom infrared telescope is sited at the summit of Mauna Kea (4200 m) in Hawaii. Staff alternately work on the mountain for five days and rest at sea level for five days. During work shifts they sleep at 3000 m and work for 10 hours at the summit. The daily ascent from 3000 m to 4200 m takes 30 minutes by vehicle. Self assessment questionnaires listing the symptoms of acute mountain sickness and scoring their severity on a scale of 0 to 10 were completed by 18 men (aged 17-45) during two separate five day shifts at the telescope. Arterial capillary blood was drawn from the ear lobes of 12 men on days 1 and 5 of each shift. Oxygen and carbon dioxide tensions were measured with an Instrumentation laboratory IL213 analyser.



Comparison of scores for symptoms of altitude sickness recorded by 18 men on day 1 of two five day work shifts at 4200 m.

Five men were asymptomatic at the summit. The remaining 13 experienced characteristic symptoms of acute mountain sickness (headache, lethargy, insomnia, and anorexia). The figure shows that the order of the 18 men ranked according to severity of altitude sickness on the first day of the two shifts correlated significantly ($r=0·63$, $p<0·001$, Kendall's rank test). After five days on the mountain most workers were free of symptoms.

Arterial oxygen tension on day 1 of the two shifts ranged from 4·4 kPa (33 mm Hg) and 5·1 kPa (38 mm Hg) in one man to 7·0 kPa (53 mm Hg) and 7·6 kPa (57 mm Hg) in another; the mean for the group was 5·5 kPa (41 mm Hg). The two values for each man correlated well ($r=0·74$, $p<0·01$). The order of men ranked according to arterial oxygen tension on day 1 of the first shift correlated with that on the second shift ($p<0·05$). Individual workers' oxygen tensions were similar on day 5 of the two shifts ($r=0·67$; $p<0·02$), and in the two men mentioned above were 5·1 kPa (38 mm Hg) and 5·7 kPa (42 mm Hg) in one and 6·7 kPa (50 mm Hg) and 7·9 kPa (59 mm Hg) in the other.

Mean arterial carbon dioxide tension was higher on the first shift (3·9 (SD 0·52) kPa (29 (3·91) mm Hg)) than on the second (3·5 (0·54 kPa (26 (4·1) mm Hg)), but there was a significant correlation between the values on the two ascents ($p<0·01$). Carbon dioxide tension correlated inversely with oxygen tension ($r=-0·62$; $p<0·001$). Assuming a gas exchange ratio of 0·8 and an inspired oxygen tension of 12·5 kPa (94 mm Hg) at 4200 m, the mean (SD) alveolar to arterial oxygen tension gradient was 2·1 (0·64) on day 1 and 2·0 (0·62) on day 5. These values are comparable with a mean gradient of 2·2 (0·9) at sea level.

Comment

Some telescope operators suffered from acute mountain sickness on each ascent to 4200 m while others reported no symptoms. Lack of judgment and denial of symptoms are well recognised features of acute mountain sickness. Nevertheless, self assessment has been shown to be as reliable as peer review in assessing the severity of acute mountain sickness in supervised people.[4] Arterial oxygen and carbon dioxide tensions were similar for each man on the two ascents, indicating a reproducible degree of hypoxia and hyperventilation on exposure to high altitude.

The inherent characteristic that determines susceptibility to altitude sickness is unknown, but experimental evidence suggests that susceptibility may be inversely related to the ventilatory drive to hypoxia.[5] The oxygen tension gradient did not increase on ascent. This observation confirmed the clinical impression that pulmonary oedema was not a causal factor in the hypoxia of our subjects.

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Department of Pathology, Royal Liverpool Hospital, Liverpool L69 3BX
PETER FORSTER, MD, MRCP, medical research fellow

Correspondence to: Dr P Forster, Rheumatic Diseases Unit, Northern General Hospital, Edinburgh EH5 2DQ.

Sublingual glyceryl trinitrate compared with Nitrolingual spray using cardiac scintigraphy

Glyceryl trinitrate is administered sublingually to treat acute angina and for prophylaxis, but the tablets have poor stability and a short half life, and absorption may be delayed, especially in patients who have dry mouths or are edentulous. A new way of administration avoids these problems and may retain the advantages of rapid absorption via the oral mucosa. Studies of an aerosol solution (Nitrolingual spray, Pohl-Boskamp) have shown appreciable improvements in ejection fraction, pulmonary wedge pressure, and exercise tolerance.[1-3] We conducted a trial comparing haemodynamic changes induced by the spray and sublingual glyceryl trinitrate.

Patients, methods, and results

We studied 10 patients (eight men, two women) aged 49-75 (mean 63) with stable angina. Eight had had myocardial infarction but none had clinical or radiological evidence of heart failure. All were accustomed to using nitrates in some form but did not take long acting nitrates within 24 hours or sublingual nitrates within two hours of the tests. No patient was currently taking calcium channel blockers. All gave informed written consent.

We compared the haemodynamic effects of the two methods of administration by assessing changes in heart rate using an electrocardiogram, blood pressure using a sphygmomanometer, and left ventricular ejection fraction using a single crystal scintillation probe (Nuclear Stethoscope, Bios Incorporated, United States).[4] Patients rested for 40 minutes beforehand and