Upper limb motor function at 5000 metres: determinants of performance and residual sequelae

Eli Silber

Abstract
Little is known about the effects of age and symptoms of acute mountain sickness on fine motor performance at altitude. There is uncertainty about whether time spent at altitude results in permanent neurological sequelae. Nine hole pegboard tests were performed on a group of trekkers at sea level (n=61), after ascending to Kanchenjunga base camp (5100 m; n=46), and 20 weeks after return to sea level (n=43). Comparison of baseline and altitude times showed a mean slowing from 36.2 to 39.0 seconds, a 7.8% deterioration in performance (p<0.0001), which was greatest in subjects aged 50 years or older (5.04 v 1.93 seconds, p=0.017), those tested within 24 hours of arrival at 5100 m (4.75 seconds, 13.3% v 0.48 seconds, 1.3% p<0.001), and persons experiencing symptoms of acute mountain sickness (p=0.012), each of which were independent determinants of deterioration. Repeat pegboard testing at sea level after 20 weeks showed no significant change compared with baseline (p=0.68). This confirms the deleterious effects of altitude on fine motor function, emphasises the benefit of acclimatisation, and suggests that older persons and those with symptoms of acute mountain sickness are particularly susceptible. The risk of long term motor dysfunction after exposure to these relatively moderate altitudes seems to be small.

Keywords: psychomotor testing; altitude; hypoxia

Despite the brain’s susceptibility to hypoxia, the ability to survive and function at high altitudes shows the potential to adapt to physiological extremes. Breakdown of adaptive responses results in acute mountain sickness and the potentially fatal high altitude cerebral oedema. There is also evidence of subtle neurological dysfunction at altitude, in particular a reduction in both fine motor and cognitive function,1,2 which may be of practical importance to people engaging in activities at these altitudes. Motor dysfunction is likely to be due to a combination of disturbances in visual, motor, and cerebellar processing. However, little is known about the determinants of psychomotor functioning at altitude, in particular age and symptoms of acute mountain sickness. Previous studies have investigated predominantly younger persons, but this may not be appropriate in the light of the growing number of older people who spend time at altitude. Few studies have assessed the potential for short term adaptation of neurological function at altitude. There are conflicting reports as to whether exposure to altitude results in long term neurological sequelae.1

This study was therefore conducted to investigate the effect of age, sex, symptoms of acute mountain sickness, and hypoxia on psychomotor function in a group of trekkers at 5100 m. It also assessed the potential effects of short term acclimatisation on motor function at altitude and whether exposure to altitude resulted in residual deleterious effects on psychomotor function.

Methods
This study investigated members of an expedition to Kanchenjunga base camp (5100 m) in eastern Nepal in 1998. The objectives of the expedition included medical research, with subjects giving consent to participate in this project. Some team members attempted to climb Kanchenjunga (reaching over 7000 m) and other peaks over 6000 m in the area. Expedition members followed recommendations by not ascending by more than 300 m/day and took rest days to aid acclimatisation.

Pegboard tests assess a composite of upper limb psychomotor function. The nine hole pegboard was chosen as it is a standardised test, convenient to carry and perform. It is sensitive3 and is the recommended test for assessing upper limb function in multiple sclerosis.4 Testing was performed while seated on a stable surface, with adequate lighting and in moderate weather conditions in subjects who were adequately fed and hydrated. A combined score of the mean of two attempts with the dominant and non-dominant hand is reported.
Baseline data were obtained on all potential test subjects at sea level 3 months before the expedition. During the trip, subjects recorded oxygen saturation (SaO₂) at rest with a portable pulse oximeter and maximal and tidal breath holding times, twice daily. Symptoms of acute mountain sickness were recorded using a questionnaire based on the Lake Louise score. All available subjects were assessed on reaching 5100 m before any significant further ascents; there was no difference in mean ages in the subjects tested (n=46) and those not retested at altitude are summarised in the table. Persons tested within 24 hours had a significantly greater mean deterioration compared with those assessed later (p=0.001). There were no differences in mean baseline performance between these two groups.

Pegboard times were influenced by symptoms of acute mountain sickness on the day of testing. Those with moderate (n=7; score 1–3) and more severe (n=6; score 4+) symptoms had a significant deterioration in performance (4.25 seconds, 12.3%, and 6.26 seconds, 18.4% respectively) compared with the 1.75 seconds (4.25 seconds, 12.3%, and 6.26 seconds, 18.4% respectively) among those assessed later (p=0.001). There was no significant difference in mean baseline performance between these groups (36.99, 35.49, and 33.98 seconds; p=0.11) and there was no difference in mean ages in the groups with different acute mountain sickness scores (42.7, 38.1, and 41.1 years; p=0.71).

As expected, there were significant differences in pegboard times between the dominant and non-dominant hand (17.59 v 18.59 seconds; p<0.0001) which persisted at altitude (18.93 v 20.06 seconds; p<0.0001). There was, however, no significant difference in the change in performance at altitude compared with baseline between the dominant and non-dominant hand (1.33 seconds, 7.9% v 1.47 seconds, 8.0% deterioration; p=0.64).

Performance at altitude did not show a significant correlation with the person’s SaO₂ on the day of testing (mean 82.1%, r=-0.23, p=0.15, n=40) or the percentage difference in SaO₂ between baseline and on day of testing (mean deterioration of 14.0%, r=-0.12, p=0.51, n=33). Analysis of only the subgroups

<table>
<thead>
<tr>
<th>Age</th>
<th>n</th>
<th>Baseline</th>
<th>Altitude</th>
<th>Time difference (s)</th>
<th>Deterioration (%)</th>
<th>p Value</th>
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<tr>
<td>&lt;50 y</td>
<td>33</td>
<td>34.85</td>
<td>36.80</td>
<td>1.93</td>
<td>5.8 (2.0–9.5)</td>
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<tr>
<td>≥50 y</td>
<td>13</td>
<td>39.43</td>
<td>44.48</td>
<td>5.04</td>
<td>13.0 (6.0–20)</td>
<td>0.002</td>
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<td></td>
<td>&lt;0.0001</td>
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<tr>
<td>Period spent at 5100 m before testing:</td>
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<td></td>
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<tr>
<td>&lt;24 h</td>
<td>25</td>
<td>36.14</td>
<td>40.89</td>
<td>4.75</td>
<td>13.3 (9.0–17.6)</td>
<td>&lt;0.0001</td>
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<tr>
<td>1–4 days</td>
<td>21</td>
<td>36.23</td>
<td>36.71</td>
<td>0.48</td>
<td>1.3 (–2.6–5.3)</td>
<td>0.5</td>
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<td></td>
<td>&lt;0.0001</td>
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<tr>
<td>Total‡</td>
<td>46</td>
<td>36.18 (35.21–37.16)</td>
<td>38.99 (37.44–40.53)</td>
<td>2.81 (1.61–4.00)</td>
<td>7.8 (4.5–11.2)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*p Altitude-baseline (positive number denotes deterioration).
†Mean (95% CI) time difference/baseline=100.
‡Mean (95% CI).

Results

Sixty one potential subjects were assessed at baseline; of these, 46 were available for retesting at 5100 m and 43 after return. The mean age at baseline was 40.4 years (range 18.4–67.9 years) with 13 aged 50 years and over. There was no difference in mean age between subjects tested (n=46) and those not tested (n=15) at altitude (40.3 v 40.4 years, p=0.99) or those tested (n=43) or not tested (n=18) on return (40.7 v 39.5 years, p=0.73). However, all of the subjects not available for retesting at altitude and 17/18 of those not retested after 20 weeks were men.

At baseline, the mean pegboard time of subjects aged 50 years or more had a significantly greater mean deterioration compared with those assessed later (p=0.017). There were no differences in mean baseline performance between these two groups.

As expected, there were significant differences in pegboard times between the dominant and non-dominant hand (17.59 v 18.59 seconds; p<0.0001) which persisted at altitude (18.93 v 20.06 seconds; p<0.0001). There was, however, no significant difference in the change in performance at altitude compared with baseline between the dominant and non-dominant hand (1.33 seconds, 7.9% v 1.47 seconds, 8.0% deterioration; p=0.64).

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with moderate or severe symptoms of acute mountain sickness (n=13) again showed no correlation between pegboard performance and \( \text{SaO}_2 \) (\( r=-0.52, \ p=0.05 \)). Similarly, there was no significant correlation between acclimatised subjects' maximum breath holding times on the day of testing (mean 36 seconds, \( r=-0.30, \ p=0.22, \ n=19 \)) or changes when compared with sea level (mean deterioration 47 seconds, 53.3%) and pegboard performance (\( r=-0.06, \ p=0.80, \ n=19 \)).

Multiple linear regression showed that age 50 years or older (\( p=0.034 \)), testing within 24 hours of arrival at 5100 m (\( p=0.008 \)), and symptoms of acute mountain sickness (\( p=0.007 \) for trend) were independent determinants of a deterioration in pegboard performance at altitude. The 41 subjects retested after return showed no deterioration compared with their baseline times (36.42 \( v \) 36.24 seconds; \( p=0.68 \)). There was no difference in long term performance between members of climbing teams (all of whom had spent time at \( \geq 6000 \) m) and the rest of the group (mean deterioration 0.73 \( v \) 0.03 seconds, \( p=0.78 \)) and no correlation between the difference in times between altitude and baseline and age (\( r=-0.084, \ p=0.6 \)).

**Discussion**

These findings confirm a deterioration in fine motor function, and add to our knowledge of the effects of age, acclimatisation, and clinical symptoms, on performance at altitude. The deterioration in performance occurred at relatively moderate altitudes, despite slow ascents and may have practical implications for persons engaging in activities relying on high levels of dexterity. Studies at similar altitudes have shown a slowing in motor speed and an increase in inaccuracy.1 Although older trekkers have a lower incidence of symptoms of acute mountain sickness,4 these data suggest that they are more susceptible to the psychomotor effects of hypoxia at altitude.

There is uncertainty about the extent to which acclimatisation restores neurological function and the delay before this is evident. Early reports describing a prolonged delay to recovery of function; upper limb coordination deteriorated over 10 months in soldiers at 4000 m after which it gradually improved.7 On the other hand, motor function tested in the first day after workers at an observatory had driven up to 4200 m deteriorated, but normalised on retesting after five days.9 This study shows the potential for rapid adaptation at these altitudes and suggests that ascent is associated with a continual process of neurological adaptation.

Identifying predictors of neurological dysfunction at altitude may provide clues to the pathogenesis of this form of hypoxic brain injury and may enable climbers to avoid complications. It has been suggested that climbers with a higher ventilatory response to hypoxia have greater residual neurobehavioural impairment.7 In this group, however, there was no correlation between fine motor performance and respiratory function measured on the day of testing. These results show a correlation between AMS symptoms and psychomotor function, similar to that which has been described for performance in some tests of cognitive function.10 This relation suggests possible similarities in pathogenesis of acute mountain sickness and psychomotor dysfunction at altitude and stress that particular caution be paid to subjects experiencing even mild symptoms.

Whether exposure to altitude has long term effects on nervous system function remains uncertain. A deterioration in short term verbal recall and motor performance (measured by a finger tapping test) was detected in members of an expedition to Everest immediately after return, which persisted at 1 year.7 Similarly, motor performance was poorer than that of matched controls in two of eight climbers who had reached summits of greater than 8500 m without supplemental oxygen11 and reaction times were prolonged in 11 persons tested 75 days after return from an altitude climb.12 By contrast, testing of 22 subjects after ascents of between 5334 and 8848 m,1 three subjects after an attempt on Everest,13 and six members of a simulated Everest ascent in a hypobaric chamber14 could show no significant deterioration in motor function. The present study suggests that people are unlikely to be left with marked residual neurological deficit after exposure to relatively moderate altitudes.

These findings have important practical implications for those spending time at altitude. The improvement in function after a day at a particular altitude emphasises the benefits of adequate acclimatisation. Particular attention should be paid to older persons and those experiencing symptoms of altitude sickness. It is reassuring that the risks of long term upper limb psychomotor dysfunction, at least after exposure to moderate altitude, seem to be small.

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