

ALTITUD y RIESGO NEUROLÓGICO

Alpinistas Europeos *versus* Sherpas del Himalaya

Eduardo Garrido Marín



Barcelona

1997

PUBLICACIONES ORIGINALES
(Science Citation Index)

Cortical atrophy and other brain magnetic resonance imaging (MRI) changes after extremely high-altitude climbs without oxygen.

Garrido E, Castelló A, Ventura JL, Capdevila A, Rodríguez FA.
International Journal of Sports Medicine 1993;14:232-4.

New evidence from magnetic resonance imaging of brain changes after climbs at extreme altitude.

Garrido E, Segura R, Capdevila A, Aldomá J, Rodríguez FA, Javierre C, Ventura JL.
European Journal of Applied Physiology 1995;70:477-81.

Are Himalayan Sherpas better protected against brain damage associated with extreme altitude climbs?

Garrido E, Segura R, Capdevila A, Pujol J, Javierre C, Ventura JL.
Clinical Science 1996;90:81-5.

Cardiorespiratory response to exercise in elite Sherpa climbers transferred to sea level.

Garrido E, Rodas G, Javierre C, Segura R, Estruch A, Ventura JL.
Medicine and Science in Sport and Exercise 1997;29: (en prensa).

Afasia motora transitoria a gran altitud.

Botella J, Garrido E, Catalá J.
Revista Clínica Española 1993;193:296-8.

Extreme altitude transient aphasia.

Garrido E, Javierre C.
British Journal of Sports Medicine 1996;30:364.

El mal de montaña.

Garrido E, Botella J.
Medicina Clínica 1997; (en prensa).

Cortical Atrophy and Other Brain Magnetic Resonance Imaging (MRI) Changes After Extremely High-Altitude Climbs Without Oxygen

E. Garrido¹, A. Castelló^{1†}, J. L. Ventura^{1,5}, A. Capdevila³, F. A. Rodríguez^{2,4}

¹ CEARE (Centre d'Estudis d'Alt Rendiment Esportiu)

² CAR (Centre d'Alt Rendiment), Sant Cugat del Vallés

³ CDP (Centre Diagnòstic Pedralbes)

⁴ INEFC (Institut Nacional d'Educació Física de Catalunya, Barcelona)

⁵ CS Bellvitge (Ciutat Sanitària Bellvitge), Barcelona, Spain

Abstract

E. Garrido, A. Castelló, J. L. Ventura, A. Capdevila and F. A. Rodríguez, Cortical atrophy and Other Brain Magnetic Resonance Imaging (MRI) Changes After Extremely High-Altitude Climbs Without Oxygen. *Int J Sports Med*, Vol 14, No 4, pp 232–234, 1993.

Accepted after revision: January 5, 1993

The purpose of the present study is to detect by means of MRI any structural changes in the brain and their correlation with the clinical history of climbers who have ascended to extremely high altitudes without supplementary oxygen. Clinical history, neurological examinations and brain MRI were obtained from a group of 26 climbers who ascended to over 7000 m without supplementary oxygen, and the results were compared with a control group (n = 21) of healthy subjects. All the MRI studies were carried out between 26 days and 36 months after return to sea level. Significant neuropsychological disorders were experienced by all climbers during the ascent with residual neurobehavioural impairment after returning to sea level in 58% of them. The neurological examination was normal in all subjects. Almost half of the climbers showed MRI abnormalities (46%). Characteristic signal patterns of cortical atrophy were detected in five subjects. Periventricular hyperintensity lesions in the T2-weighted images were observed in other five climbers. Both types of lesions were found in two subjects. These pathological findings did not correlate with age, sex, clinical symptoms, maximal altitude reached, or length of exposure to extreme altitude. The exact long-term pathological significance of these MRI abnormalities is as yet unknown.

Key words

Magnetic resonance imaging (MRI), hypoxia, brain, altitude, climbing

Introduction

Ever since the first ascent by R. Messner and P. Habeler to the summit of Mount Everest without any supplementary oxygen in 1978, other mountain climbers have made attempts to reach the highest peaks of the world without the use of oxygen. Whether permanent brain damage occurs under these extremely hypoxic conditions has been debated, and nobody knows if the neurological dysfunction is acute or subacute (1,5,8) and there is a common doubt as to whether more disorders will appear in the long term (9). The papers written previously show contradictory results. Some claim that motor disturbances can appear between 12 or 24 months after the expedition (8,9) and others cannot find any brain or mental dysfunction using neuropsychological tests (2). As far as we know there are only two papers which deal with the use of imaging techniques, but in each of them only one climber was examined. In one of them (4), they found cortical atrophy by means of Computed Tomography which was carried out six years after climbing. The other one performed by MRI (6), one year after climbing, showed bilateral pallidum damage.

This study has been designed to detect possible brain damage and correlate it with clinical neurological dysfunction. At present, it is wellknown that MRI allows us to obtain the best contrast resolution in brain lesions and we are also trying to demonstrate that MRI could be the best non-invasive technique to assess structural brain changes on account of its high contrast resolution, relative comfort and the possibility of repeating images, which have no ill-effects on the climber.

Material and Methods

Magnetic resonance imaging (MRI) brain scans, medical histories and clinical neurological examinations were obtained in 26 elite high-altitude climbers (22 men and 4 women) between 26 days and 36 months after returning to sea level (285±280) days after ascents to altitudes of over 7000 meters without oxygen (Everest 8848 m, K2 8611 m, Lhotse 8516 m, Yalung Kang 8505 m, Makalu 8463 m, Lhotse Shar 8383 m, Cho Oyo 8201 m, Dhaulagiri 8172 m, Broad Peak 8047 m, Muztaghata 7500 m). The average age of the climbers was 35±5 years (ranging from 25 to 42 years). The maximum altitude reached in the last expedition varied between 7300 and 8500 meters (7802±416 m). Twenty-two subjects had previously climbed one (12 subjects) or several (10 subjects) peaks

Supported by Sandoz-Sport Research

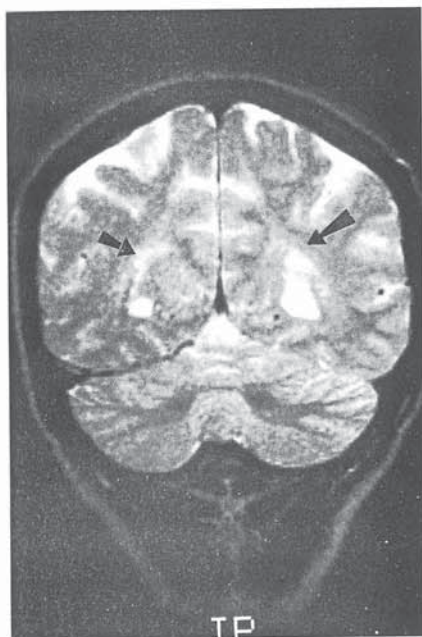


Fig. 1 Coronal T2 weighted image shows periventricular changes (arrows) and deep sulci.

Table 1 MRI findings in elite high-altitude climbers (n = 26).

MRI findings	Frequency	Percentage
Brain cortical atrophy	5	19
Parietal Posterior	4	15
Global	1	4
Areas of high signal	5	19
<i>Symmetrical Bilateral</i>	2	8
Centrum semiovale-posterior	1	4
Posterior horns	1	4
<i>Unilateral</i>	3	12
Right posterior horn	1	4
Left anterior horn	1	4
Right temporal horn	1	4
Cortical atrophy + high signal areas	2	8
Pariet-post atrophy + right post horn	1	4
Pariet-post atrophy + left post horn	1	4
Normal	14	54

over 7000 m without supplementary oxygen (14 climbers had previously reached an altitude higher than 8000 meters). The cumulative exposure to altitudes of 7000–8848 m ranged from 6 to 1440 hours (419 ± 429 hours). None of them had a neurological or psychiatric history. The medical history was specially designed and conducted by one doctor who is an expert in sports medicine, working closely with the climbers. The questionnaire required information concerning toxic habits, fitness level, head injuries, metabolic disorders and was specially aimed at detecting neuromotoric, biological, cognitive, emotional, perceptual, and motivational dysfunctions or abnormalities during acclimatization, at high altitude, and on return to sea level. The control group included 21 age and sex matched subjects. None of them had ever been exposed to extreme altitudes.

Clinical neurological examination was carried out at the time of the MRI study. The system used was a 1.5 T superconducting magnet (General Electric, Milwaukee, USA). The following cranial scans were obtained (Proton density, T1 and T2 weighted images): sagittal plane (SE TE : 20 TR : 575); axial and coronal plane (VE TE : 40–100 TR : 2000).

Two radiologists examined independently all the scans (blind study) reporting abnormal findings as a high signal or atrophy (+) or normal images (-), for the following anatomical zones: centrum semiovale, periventricular areas, brain stem, basal ganglia, internal capsule, thalamus, cerebellum, hemispheric brain sulci, and ventricles (Fig. 1).

Statistical results were based on frequencies and percentages for categories and on mean and standard deviations for continuous variables. The Kruskal-Wallis Test was used to compare continuous data and Fisher's Exact Test for frequency. Type I error was established at 5%.

Results

Medical history and neurological examination

None of them had either neurological or psychiatric history or toxic habits, head injuries such as contusions, alcohol or other drug addiction. All subjects experienced mental-neurological symptoms during acclimatization or at the high altitude, and 58% also after returning to sea level. Most of these post-ascent symptoms lasted more than one month and in three of them the symptoms lasted up to ten months later. One case of aphasia and one case of hemianopsia were also reported both at altitudes over 8000 m.

The most frequent mental-neurological disorders during high altitude exposure were: headache (77%), insomnia (58%), anorexia (42%), irritability (42%) and ataxia (42%). Other manifestations were visual dysfunctions (31%), apathy (31%), nightmares (27%), hypersomnia (23%), auditory dysfunction (15%), taste dysfunction (15%) and monothe-matic thoughts (15%).

After the expedition we found amnesia, emotional disorders and confusion. All neurologic clinical examinations were normal at the time of the MRI study.

Cranial MRI

While results were normal in all control subjects, MRI showed structural abnormalities in 12 (46%) of the 26 climbers ($p < 0.0001$). The MRI abnormalities observed are presented in Table 1.

Clinical symptoms and MRI findings

The pathological findings did not significantly correlate with age, sex, body size, maximum altitude reached,

length of exposure to extreme altitude, life style (fitness level and toxic habits), and type and number of symptoms reported during and after the expedition.

Discussion

Neurological impairment and mental dysfunction due to severe altitude hypoxia are extremely frequent and varied (5), and are perhaps related to general or local thrombotic and/or embolic ischaemic processes in the brain vessels (3,5,7), (although this, as yet, has not been medically demonstrated). Whether CNS impairment is transitory (2) or permanent is controversial (1,9).

The high number of MRI abnormalities (46% of the climbers), brain cortical atrophy and areas of parenchymal increased signal intensity being the most predominant, would seem to point to long-term brain injury. Unfortunately we do not have previous MRI, but in view of the changes observed, new studies should be performed.

The wide range used in exploration time was to obtain the biggest group possible, as we were well aware of the intrinsic difficulties to find an elite group of climbers. Although this is not a longitudinal study, the fact that no one in the control group showed MRI abnormalities strongly suggests that anomalies found in the climbers could be due to extremely high altitude conditions. Cortical atrophy which has been observed only in those subjects examined 6 to 36 months after their return seems to show that these changes may be permanent. The same has been suggested by other investigators (8,9). Fukushima et al. (4) reported a case of mild cortical atrophy on CT scan in a young climber six years after having experienced high altitude cerebral oedema. Shiota et al. (6) confirmed bilateral lesions of the globus pallidus by MRI in a middle-aged woman after one year of acute mountain sickness.

Our results strongly suggest that exposure to extreme high altitude could result in permanent brain damage. Neither the cause-effect relationship nor the physiopathogenic mechanism can be established. Based on our findings, we speculate that the possible causes of these anatomical brain changes may include genetic predisposition, fast climbs, different acclimatization capacity and/or other unknown mechanisms. The exact pathological significance of these MRI changes is also unknown and needs to be clarified. Nevertheless, if these results are confirmed, would this be a reasonable price to pay for reaching the highest summits of the world without supplementary oxygen?

Acknowledgements

We thank Gerald P. Fannon for his English corrections and all the group of climbers for their patience and collaboration in this work. We wish them great success in all their future goals.

References

- 1 Cavaletti G., Moroni R., Garavaglia P., Tredici G.: Brain damage after high altitude climbs without oxygen. *Lancet* 1: 101, 1987.
- 2 Clark C. F., Heaton R. K., Wiens A. N.: Neuropsychological functioning after prolonged high altitude exposure in mountaineering. *Aviat Space Environ Med* 54: 202-207, 1983.
- 3 Clarke C. R. A.: Cerebral infarction at extreme altitude. In: Sutton J. R., Huston C. S., Jones N. L. (eds), Hypoxia, exercise and altitude. Proceedings of the Third Banff International Hypoxia Symposium, 453-454, New York: Alan R. Liss Inc., 1983.
- 4 Fukushima M., Kobayashi T., Kubo K., Yoshimura K., Shibamoto T. A.: A case of high altitude pulmonary edema followed by brain computerized tomography and electroencephalogram. *Aviat Space Environ Med* 59: 1076-1079, 1988.
- 5 Ryn Z.: Psychopathology in mountaineering-mental disturbances under high-altitude stress. *Int J Sports Med* 9: 163-169, 1988.
- 6 Shiota J., Sugita K., Isono O., Araki S.: A case of acute mountain sickness with bilateral lesion of pallidum. *Clin Neurol* 30: 630-634, 1990.
- 7 Song S. Y., Asaji T., Tanizaki Y., Fujimaki T., Matsutani M., Okeda R.: Cerebral thrombosis at altitude: its pathogenesis and the problems of prevention and treatment. *Aviat Space Environ Med* 57: 71-76, 1986.
- 8 Townes B., Hornbeim T., Schoene R. B., Sarnquist F. H., Grant I.: Human cerebral function at extreme altitude. In: West J. B., Lahiri S. (eds), High altitude and man. Bethesda: American Physiological Society 1984, pp 32-36.
- 9 West J. B.: Do climbs to extreme altitude cause brain damage? *Lancet* II: 387-388, 1986.

E. Garrido

CEARE (Centre d'Estudis d'Alt Rendiment Esportiu)
Avda. Països Catalans, s/n.
08950 Esplugues de Llobregat (Barcelona)
Spain