

Transient High Altitude Neurological Dysfunction: An Origin in the Temporoparietal Cortex

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ABSTRACT

Firth, Paul G., Hayrunnisa Bolay. Transient high altitude neurological dysfunction: An origin in the temporoparietal cortex. *High Alt. Med. Biol.* 5:71–75, 2004.—This case report describes three separate episodes of isolated ataxia, hallucinations of being accompanied by another person, and bilateral dressing apraxia occurring in a single individual without prior warning signs. These symptoms are attributable to disruption of vestibular processing in the temporoparietal cortex or associated limbic structures. Neurological dysfunction at high altitude is usually ascribed to high altitude cerebral edema or acute mountain sickness. However, transient neurological symptoms occur abruptly at more extreme altitudes, often following vigorous exertion, without overt altitude-induced prodromes. These symptoms may be caused by intense neuronal discharge or neuronal synchronization as a feature of epileptic discharges or cortical spreading depression. Transient high altitude neurological dysfunction should be recognized as a separate complication of extreme altitude, distinct from high altitude cerebral edema.

Key Words: hallucination; high altitude; high altitude cerebral edema; high altitude pulmonary edema; hypoxia; migraine; seizure

INTRODUCTION

NEUROLOGICAL SIGNS and symptoms induced by high altitude are generally attributed to acute mountain sickness (AMS) or high altitude cerebral edema (HACE), two well-recognized syndromes thought to be closely related. Transient episodes of isolated brain dysfunction are less commonly reported. Clinical descriptions of less severe manifestations of altitude-induced syndromes may yield insight into the pathophysiological mechanisms not evident from the global dysfunction produced

by HACE. This report describes three separate episodes of brief altitude-induced neurological symptoms in a medically trained climber and discusses possible pathophysiological mechanisms.

CASE REPORT

A healthy, right-handed, 28-year-old physician ascended from sea level to 5400 m on Aconcagua, Argentina, over 7 d. He had no signs or symptoms of AMS. On day 8 he at-

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tempted to reach the 6962 m summit with a partner. Climbing with regular food and drink breaks, the pair reached 6800 m before turning back when his companion felt too fatigued to continue. At an altitude of 6750 m, the climber abruptly lost his sense of balance, experiencing the sensation that he needed to hold on to something to prevent his falling off the level ground of the path. He was able to walk only with support from his companion. There were no prodromal or associated symptoms of headache, visual disturbances, or leg weakness. He had no nausea, vertigo, earache, or tinnitus suggestive of an acute Ménière crisis. His instability resolved after approximately 15 min. He descended unassisted to 5400 m without further incident.

The following day they moved camp to 5800 m, and he summited solo on day 10. While resting alone at 6700 m during his descent, he discovered he had severe frostbite in five fingers. Shortly after this, he abruptly developed a powerful sensation of the presence of another person. This caused him to turn around repeatedly to search for the other individual. Descending the mountain, he felt as if the person were following him and conducted a mental conversation with his apparent companion. While walking, he felt as if his legs were moving of their own accord and his torso was elongated. He felt detached from his person, as if he were observing himself from a distance. He was aware of the hallucinatory nature of his experiences. These anomalies lasted approximately 10 min before disappearing spontaneously. He returned to camp, experiencing no further perceptual disturbances, and descended the following day. He noted no overt neurological sequelae.

Four years later, the physician ascended from sea level to 5250 m on Mount McKinley, Alaska, in nine d. He suffered a viral upper respiratory tract infection for 2 d prior to ascent and mild symptoms of AMS on day 5 that resolved with a day's rest at 3350 m. On day 10, he climbed to the 6194 m summit with three climbers. He was able to manually focus his camera on the summit and traverse the narrow summit ridge using his ice ax and rope. On completion of a snack and rest stop at 5950 m during the descent, he was unable to zip his

jacket closed or fasten his backpack straps. Although able to conceive the action sequence, he had acutely lost the fine-motor control required to complete the desired actions. He noted slight difficulty concentrating. There were no prior or associated symptoms of dysarthria, diplopia, nausea, dizziness, headache, paresthesia, weakness, dysidokinesia, or ataxia. He was able to walk in a straight line, and Romberg's sign was negative. His companions dressed him. He was able to firmly grasp and utilize his ice ax, which was placed in his hand. He descended without assistance. On testing 2 h later at 5550 m, he found that the difficulty with dressing had resolved. He experienced no further symptoms. He descended from 5250 to 2100 m the following day. His recall of events correlated closely with those of his companions. Cardiac ultrasound and magnetic resonance imaging (1.5 T) of the brain a year later for incidental purposes revealed no structural abnormality.

DISCUSSION

The hallucination of a "sense of a presence" is an example of a broad range of perceptual dysfunctions of personal space and self-position. Automatism and a feeling of depersonalization are commonly associated experiences. (Brugger et al., 1997). Disruptions to concepts of self-perception in climbers at extreme altitude are well documented. These symptoms include the hallucination of a phantom presence (Smythe, 1935; Shipton, 1947; Herzog, 1952; Clarke, 1976; Bonington, 1977; Messner, 1989; Buhl, 1998; Groom, 1999; Brugger et al., 1997; Garrido et al., 2000.), sensations of floating (Ravenhill, 1913; Brugger et al., 1997; Dietz and McKiel, 2000), automatism (Habeler, 1979; Dietz and McKiel, 2000), or viewing oneself from outside one's body (Habeler, 1979). These hallucinatory experiences are typically transient and not associated with other frank neurological deficit. The striking similarity of these multiple accounts suggests that dysfunction of a specific region of the brain, responsible for body-related processing experiences, is a reproducible complication of extreme altitude. Vestibular processing and integration of sensory input occur in the parietal and temporal

cortex and associated limbic structures (Penfield and Perot, 1963; Gloor et al., 1982; Sveinbjornsdottir and Duncan, 1993; Blanke et al., 2000; Blanke et al., 2002; Allison et al., 2003; Pelphrey et al., 2003). The right angular gyrus is a part of the parietal cortex, situated at the junction of the parietal and temporal lobes. Experimentally induced electrical disruption in the right amygdala (Penfield and Perot, 1963; Gloor et al., 1982) and inferior parietal cortex at or near the angular gyrus (Blanke et al., 2000; Blanke et al., 2002) causes reproducible vestibular disturbances. These include loss of balance and sensations of falling, paroxysmal illusions that somebody was nearby, out-of-body experiences, and sensations of floating and bodily distortions. Interestingly, the amplitude and duration of the stimulus may determine the nature of experience (Blanke et al., 2000; Blanke et al., 2002, Pelphrey et al., 2003). While low-intensity stimulation of the right angular gyrus produced vestibular responses such as "falling from height," a higher stimulus intensity induced out-of-body experiences (Blanke et al., 2002). Differing intensity of disturbance in identical or anatomically close areas may therefore produce differing symptoms. While loss of balance may arise from disruption to vestibular functioning at differing levels of neurological processing (Gordon, 1999), a common source of both the hallucination and the disrupted sense of balance described in the present report may therefore be dysfunction of the parietal or temporal cortex in the region of the right angular gyrus.

Pure dressing apraxia, the inability to dress despite preservation of hand and arm function to perform other tasks, is a type of bilateral ideomotor upper limb apraxia. Limb apraxias comprise a wide spectrum of higher-order disorders of sensorimotor integration, not attributable to an elementary comprehension or motor-sensory deficit, that affect the performance of skilled, learned movements (Pramstaller and Marsden, 1996; Lieguard and Marsden, 2000). Ideomotor apraxia has been suggested to arise from either the destruction of visuokinesthetic motor representations of learned movement, stored in the parietal lobe, or from a separation of these representations from motor and premotor areas (Liegard and Marsden, 2000).

Unilateral parietal lesions can produce bilateral symptoms, with the recognition disorder that produces dressing apraxia arising from dysfunction of the nondominant posterior parietal lobe (Hier et al., 1983). The use of additional visual or tactile-kinesthetic cues may bypass or minimize an isolated ideomotor deficit (Liegard and Marsden, 2000). The ability of the subject to use his ice ax, despite being unable to perform the actions required to dress, may be an example of this. An alternative model suggests that apraxia may arise from disruption to communication between left and right hemispheres (Geschwind and Kaplan, 1962; Watson and Heilman, 1983; Lieguard and Marsden, 2000), for example, callosal apraxia arising from lesions in the corpus callosum. This clinical picture has been suggested to be appropriate (Baumgartner, 1999) for the diffuse callosal edema detected in patients with symptoms of HACE (Hackett et al., 1998). However, callosal lesions typically produce unilateral apraxia, and are commonly associated with additional lesions and symptoms (Hier et al., 1983; Pramstaller and Marsden, 1996; Lieguard and Marsden, 2000). The pure dressing apraxia of the present case, the isolated deficit, the absence of prodromal symptoms of HACE, and the abrupt onset and resolution of the symptoms further argue against callosal edema as an etiology. The three apparently disparate symptom complexes of the present case, hallucinations of another presence, vestibular disruption, and dressing apraxia, may therefore be explained by neuronal dysfunction in identical or anatomically close areas of the right inferior parietal and superior temporal cortex.

Auditory and complex visual hallucinations are additional well-recognized symptoms of extreme altitude that can be induced by experimental electrical stimulation of the temporoparietal cortex and associated deeper structures (Gloor et al., 1982; Sveinbjornsdottir et al., 1993; Blanke et al., 2002). Dysfunction of these parts of the brain therefore can account for a wide spectrum of acute, transient high altitude pathology. The temporoparietal cortex in the region of the supramarginal gyrus receives much of its blood supply from the lateral occipitotemporal and angular arteries,

lower divisions of the middle cerebral artery. These cortical areas are in the vicinity of the watershed with the posterior cerebral artery and in the presence of vascular variations may actually lie within a watershed region. Under extreme hypoxic conditions, it is possible that the blood supply to watershed areas is inadequate. Alternatively, the complex function of sensorimotor integration may be a task that is extremely sensitive to hypoxic disruption and may simply be an early sign of dysfunction in the face of global cerebral hypoxia. These suggestions are however, speculative, and it is unclear why these regions or functions appear to be particularly sensitive to hypoxic conditions.

Hallucinations and acute motor symptoms tend to occur unexpectedly in mountaineers sufficiently healthy to climb to above 5000 or 6000 m (Smythe, 1935; Herzog, 1952; Clarke, 1976; Bonington, 1977; Habeler, 1979; Messner, 1989; Buhl, 1998; Groom, 1999; Brugger et al., 1997; Garrido et al., 2000; Dietz and McKiel, 2000), while HACE usually presents more gradually, progressing following AMS and/or pulmonary edema at altitudes of 2800 to 6000 m (Hultgren, 1997; Hackett et al., 1998). Intense neuronal discharges or neuronal synchronization in the temporoparietal association areas would explain the abrupt, unheralded onset and offset of symptoms in the present case. Spreading depression in the cortex and hippocampus has been implicated in the pathophysiology of migraine aura and transient global amnesia, respectively. Conceivably, a broad spectrum of acute, transient neurological dysfunction at high altitude (hallucinations, motor dysfunction, aphasia, cortical blindness, global amnesia) arise from electrophysiological disruption similar to the cortical spreading depression of migraine. Cortical spreading depression may cause blood-brain barrier disruption (Bolay et al., 2003) and may conceivably progress to HACE if descent from altitude does not occur. While electrophysiological disruption may proceed or accompany HACE, the epidemiology, pathophysiology, and clinical presentation of hallucinations and transient motor deficits at extreme altitude may therefore be different. We suggest that these symptoms of transient high altitude neurological dysfunction be recognized as an expression of high altitude pathology separate from the end-stage syndrome of HACE.

The absence of prodromal symptoms at extreme altitudes, compared to gradual onset of HACE or AMS at lower altitudes, suggests an acute trigger against a background of more extreme hypoxia. Transient neurological dysfunction usually occurs during or shortly after a prolonged period of vigorous exercise at very high altitudes. Strenuous exercise and hyperventilation in hypoxic environments produce acute hypoxemia and hypocapnia, respectively (Sutton et al., 1988), but published data are currently inadequate to accurately assess the relative effect of hypoxic vasodilatation and hypocapnic vasoconstriction on cerebral perfusion and oxygenation (Hornbein et al., 1989). The association noted in clinical accounts may simply reflect the fact that strenuous climbing is usually required to reach extreme altitudes or that much time at these altitudes is spent either climbing or resting from climbing. Alternatively, there may be a reporting bias in that climbers who have exerted themselves sufficiently to attain great altitudes are perhaps more likely to publish accounts of their successful exploits. A causal relationship to exercise and hyperventilation, while attractive in theory, therefore remains unproved. If cerebral hemodynamics are the triggering cause, however, the delay in onset of symptoms implies that some secondary process is involved. Alternatively, symptoms might arise directly from acute hypoxia. Possible acute but transient impediments to blood flow and oxygenation might include vasospasm, dehydration, polycythemia, or localized cerebral edema or extravascular fluid shifts. It remains unclear, however, which if any of these various possibilities play a role in triggering symptoms.

Hallucinations, gait disturbance, and loss of fine-motor control are well-known complications of extreme altitude. The present report, however, is unusual because of the abrupt onset and resolution of apparently disparate symptoms in a single individual on separate occasions. The unique circumstances of the current case suggest that sensorimotor integration in the temporoparietal association areas may be particularly vulnerable to disruption at extreme altitude. Transient high altitude neurological dysfunction could be due to focal-intense neuronal discharges or neuronal synchronization similar to epileptic discharges or

migraine aura. The pathophysiology may differ from that of HACE, although these neuronal disturbances may play a role in the subsequent development of HACE.

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