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Effects of moderate hypobaric hypoxia on evoked categorical visuocognitive responses

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Abstract

Objective: Aim of the current study is to provide electrophysiological evidence about the effects of moderate hypobaric hypoxia on human visual cognition.
Methods: We investigate ERPs at occipitoparietal cortical areas in an ultra-rapid categorical discrimination task with psychomotor responses under the conditions of normoxia vs. moderate hypobaric hypoxia. Subjects had to produce motor response upon the categorization of target images containing animals, while suppress it for nontarget images containing only nonanimals.
Results: Statistical analysis on peak amplitudes and latencies of ERP components indicated significant: (i) attenuation of P1 and enhancement of N1–P3 amplitudes, (ii) delay of P2 latency for both stimuli whereas the delay of P3 latency only for nontargets, (iii) reduction in behavioral performance rates only for nontargets.
Conclusions: For both categorical stimuli, impairment of early visual sensory and compensation through late cognitive processes was noticed. For targets, compensatory discrimination–categorization processes (reflected on P3 amplitudes) were sufficient to override our mild transient hypoxic challenge. For nontargets, differential P3 latencies and behavioral performance manifested the early impeding effects of systemic hypoxaemia.
Significance: Evoked brain responses allow for early detection of subtle electrophysiological modulations coupled to cognitive-behavioral alterations, assessment of 'functional' hypobaric hypoxic sensitivity thresholds for 'altinauts' and reveal the susceptibilities of complex visuocognitive processes even to moderate hypoxic insults.

Keywords: Hypobaric hypoxia; $SatO_2$%; Categorical visuocognitive ERPs; ERP components peak amplitude and latency; *n*-ANOVA; Productive and inhibitory psychomotor responses

1. Introduction

Hypobaric hypoxia is the condition of hypoxia that results from exposure to low atmospheric pressure levels, with a consequent reduction of the pressure gradient down the respiratory system in the arterial blood partial pressure of oxygen-O_2 (PaO_2). Such conditions are present in high altitudes (mountains, airplanes, space shuttles) and stimulate complex human physiological respiratory (ventilation), circulatory (blood flow) and different tissue adaptation (metabolic) processes to withstand the hypoxic challenge (Guyton and Hall, 1996; Peacock, 1998). The single best collective index of sufficient atmospheric O_2 supply, respiratory uptake and systemic delivery to different tissues (O_2-carrying capacity of blood) is the oxygen saturation level of hemoglobin–Hgb ($SatO_2$%), (Fauci et al., 1998; Ganong, 2005). Normally the $SatO_2$% is $\geqslant$97%

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corresponding to a PaO_2 of >90 mmHg. The brain is certainly the most sensitive organ to hypoxic insults, whether systemic or local (i.e. cerebral ischemia) (Raichle, 1983; Lutz et al., 2003). The 75% $SatO_2$% level of hemoglobin (that is, on average 3 out of 4 heme groups binding O_2) marks the inflection point in the sigmoidal O_2–Hgb dissociation curve, below which rapid decline <40 mmHg in PaO_2 ensues, and exhausts the arterio-venous O_2 gradient (Pa-vO_2) so that the brain tissue relies merely upon local metabolic and vasoregulatory (blood flow) circulation mechanisms for extracting any further O_2 (Guyton and Hall, 1996; Fauci et al., 1998; Ganong, 2005). Severe systemic hypoxaemia ($SatO_2\% < 50\% \approx PaO_2 < 25$ mmHg) by means of acutely developing 'cerebral anoxia' may result in the loss of consciousness within 15–30 s and irreversible brain damage after 5–10 min (Adams et al., 1997; Fauci et al., 1998). Less severe systemic hypoxaemia ($SatO_2\% = 50–75\% \approx PaO_2 = 25–40$ mmHg) through excessive or prolonged cerebral hypoxia can cause variable deteriorative effects ranging from subtle cognitive and psychomotor disturbances (Denison et al., 1966; Green and Morgan, 1985; Fowler et al., 1993; Bartholomew et al., 1999; Li et al., 2000) to marked neuropsychological impairment affecting memory, visual spatial functions, cognition and personality (Regard et al., 1989, 1991; Caine and Watson, 2000; Virués-Ortega et al., 2004, 2006; Fayed et al., 2006). In this context, 'hypoxaemia' refers to blood hypoxia and constitutes our measurable quantitative experimental variable, whereas 'hypoxia' is more general qualitative term that refers either to causative ambient (hypobaric hypoxia) or consequent tissue (including brain tissue hypoxia) lower than normal O_2-tension levels. The level of hypoxia, how rapidly it develops, how long it is sustained, pre-conditioning and subject sensitivity, are all critical factors of the degree of functional compensation vs. impairment or reversibility of damage it may be inflicted (Adams et al., 1997; Lutz et al., 2003).

In general, during rapid ascent to high altitudes human cognitive performance deteriorates abruptly, although some people can still successfully perform cognitive tasks even under extreme hypobaric conditions (up to 6000 m) (Kida and Imai, 1993). The differences in responses may be attributed to different arousal levels, as can be reflected on EEG (Kraaier et al., 1988), and different short-to-long term adaptation capacities (Hornbein and Schoene, 2001). Although human EEG studies of systemic hypoxia under hypobaric (Papadelis et al., 2007) or normobaric (Schellart and Reits, 2001) conditions have demonstrated increased power across all spectral bands, they cannot capture electrophysiological alterations coupled to underlying visual cognitive-behavioral subprocesses. On the other hand, 'altinauts' (pilots, climbers, etc.) trained under simulated hypobaric hypoxic conditions to endure with execution of their demanding tasks, need to be assessed in terms of sensitivity thresholds of their brain functions to hypobaric hypoxia. Our approved conditions of moderate hypobaric hypoxia (around 4500 m for 15 min, subject mean $SatO_2\% > 75\%$) lie within a powerful and effective compensation range of human physiological and cognitive functions. They may not be sufficient to cause an overt decline in basic visuocognitive functions tested by our stimulus categorization paradigm (ceiling effect), but may still affect some psychometric parameters. The objective of this essay is to probe early alterations in visual categorical and psychomotor functions due to moderate hypobaric hypoxia as reflected on quite sensitive electrophysiological parameters: event-related potentials (ERPs) at occipitoparietal brain sites. The spatial distribution and temporal latency of the ERP deflections represent the resultant of spatiotemporally summated cortical neuronal responses. Visuocognitive ERPs indicate the time course of visual information processing, including early exogenous components (P1: 80–120 ms) sensitive to stimulus luminance, spatial frequency, contrast and low-level cues, as well as late endogenous components (N1: 120–180 ms and P3: 300–500 ms) modulated by the more complex processes of expectancy, attention, cognition search, categorical discrimination, identification, decision making, response choice and memorization (Licht and Homberg, 1990; Luck and Hillyard, 2000; Luck, 2005).

Ultra-rapid presentation of images merges onset and offset stimulus evoked cortical responses, prevents contamination of EEG data by image tracking exploratory eye movements, and imposes critical constraints that test the limits of visual cognition. The human brain demonstrates an astonishing capacity in superordinate level fast-track categorization of images, i.e. containing animal vs. nonanimal objects (Thorpe et al., 1996). The electrophysiological correlates (ERP components) of the underlying visuocognitive processes have only recently started to be elucidated (Tanaka et al., 1999; VanRullen and Thorpe, 2001a). An early process of coarse feature extraction (VanRullen and Thorpe, 2001b) and categorical discrimination (Tanaka and Curran, 2001; Grill-Spector and Kanwisher, 2005) has been linked to modulations in the N1 component for objects (Curran et al., 2002; Proverbio et al., 2007) or N170 component for faces and animate homomorphic entities (Allison et al., 1999; Itier and Margot, 2004), whereas a late process of fine grain identification (recognition), memory pattern matching (old/new effects) and context updating has been associated with modulations in the P3 component (Donchin and Coles, 1988; Curran, 2000). Although P3 is influenced by the probability of an event, for stimuli with a 50/50 probability, the P3 amplitude will be higher for go (targets) than no-go (nontargets) trials in a go/no-go paradigm (Pfefferbaum et al., 1985). So it has long been argued that stimulus categorization is more important than stimulus probability for the P3 amplitude (Nasman and Rosenfeld, 1990; Mecklinger and Ullsperger, 1993). In general, P3 amplitude is accepted to reflect the number of neurons allocated to the eliciting task (Wickens et al., 1983), while P3 latency time to reflect the duration of stimulus evaluation (Kutas et al., 1977), and can thus be prolonged by task difficulty. In conclusion, visual cognitive ERPs studied together with psychometric tests