POINTING MOVEMENT IN SHORT AND LONG TERM EXPOSURE TO HYPOXIA

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INTRODUCTION

Movement control in hypoxia might be affected by several factors such as depression of propioceptive and vestibular afferents and changes in physical properties of ambient air at altitude (2,3). These factors may cause disturbance in coordination of aimed voluntary movements with a reduction of accuracy and altered kinematics variables. These changes may occur particularly in conditions as acute or chronic mountain sickness, where symptoms such as headache, dizziness, nausea, dimness of vision and generalized weakness are present.

Since movements are usually performed under high accuracy constraints, during hypoxic stress we expect an increase of movement duration. This could be explained as a re-orientation of the motor control system to the visual one. Under these circumstances, movements performed in the visual tracking may tend to have more than one deceleration, which results in prolongation of the deceleration phase. Studies on movement organization (1) showed that changes of motor control strategy lead to the deformation of the bell-shaped velocity profile, which become asymmetric with prolonged deceleration phase. This is also true for visually controlled movements. Therefore the symmetry of the velocity profile seems an important parameter, providing information about the role of the visual system in movement organization.

As already postulated, movements should become slower during exposure to a hypoxic environment although the final position of the limb at the end of movement should be preserved. Single-joint movement kinematics

are proportional to force production at normal air density. We undertook the present study to elucidate by kinematics analysis the mechanism involved in the reorganization of motor control during adaptation to breathing low O_2 mixtures.

MATERIALS AND METHODS

We measured the performance of 5 male subjects who were informed of the purpose of the experiment and gave their written consent. Mean age of our subject was 27 y. We performed two experiments. At first our subject participated in a short-term exposure (30 min) to the inhalation of a mixture of 13.5 O_2 in N_2 in a sealed chamber at a barometric pressure of 730 mmHg. The same subjects were, after about 1 month, exposed for 9 hours per day for 10 days to a barometric pressure of 490 mmHg. The subjects were tested in a sitting position. They were instructed to perform pointing arm movement toward a flashing LED as fast and as accurate as possible and to begin the movement as soon as the next flashing led appeared. The sequence of flashing LEDs was randomized in time and direction. Target appeared at 4 and 16 angular degrees from the center of the visual field. In this study only movement at 16 angular degrees were analyzed. For data collection, we used a matrix of LED for signal presentation and an opto-electronic system for detection of two IR-LED, positioned on the forearm and shoulder, by an IR-videocamera recording at a 25 Hz sampling rate. Analysis was performed with software produced in our laboratory. Three different time profiles were analyzed. First, position-time profile: movement amplitude (maximal angular displacement) and movement duration. Second, velocity-time profile: acceleration phase (time from the movement onset to the peak velocity), deceleration phase (time from the peak velocity to the end of movement). Third, acceleration-time profile: acceleration time (the time from the movement onset to the peak acceleration), "switch" time (the time from the peak acceleration to the peak deceleration) and deceleration time (the time from the peak deceleration to the end of movement). Mean value and standard deviations were calculated for each variable.

RESULTS

As illustrated in figure 1, high accuracy of angular displacement (for the movements of 16 angular degrees) is constantly observed in all test sessions at hypoxia independently from length of exposure. Mean deviation from the target did not exceed 5% of the movement amplitude. On the other hand, movement duration increased in both acute and long-term exposure to hypoxia up to about 200 and 300 ms longer, respectively.

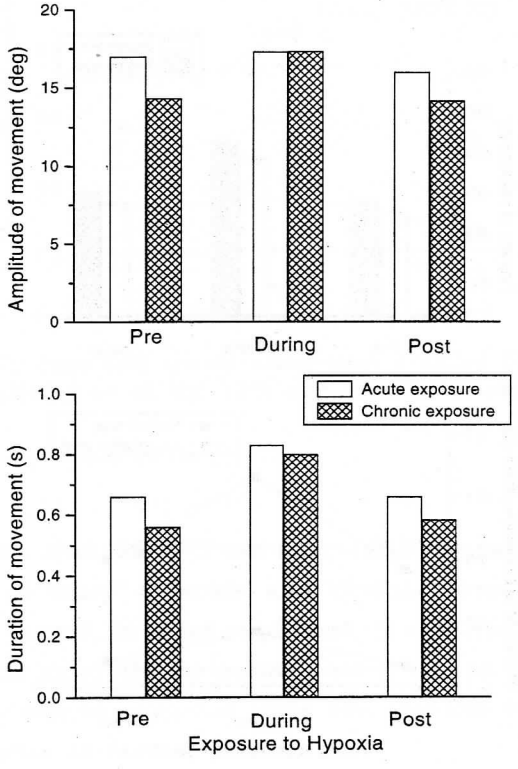

Fig. 1 - Angular displacement and duration of pointing arm movement before, at the end of a 30 min period or at the 10th day, and after acute and chronic exposure to hypoxia.

In figures 2 and 3, a significant decrease of peak and mean velocity is observed in both experimental conditions. Furthermore analysis of the acceleration-time profile shows that for acute hypoxia the acceleration and the deceleration times are almost equal in all sessions. For chronic hypoxia, the deceleration time was up to 100 ms longer than deceleration time. The "switch" time for both experimental conditions constituted about 50% of the movement duration. The increase of movement duration in acute and chronic hypoxia results from lengthening of all three phases of the acceleration-time profile. However, the absolute contribute of the "switch" time is considerably higher.

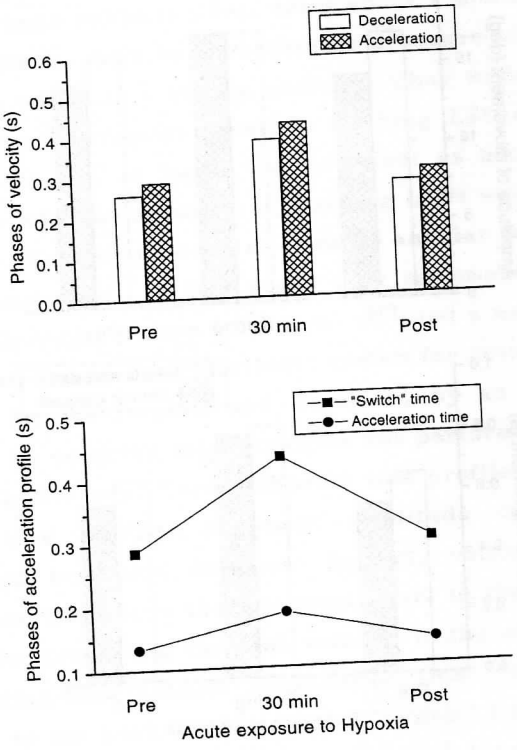

Fig. 2 - Velocity-time and acceleration-time profiles before, at the end of a 30 minute period or at the 10th day, and after acute exposure to hypoxia.

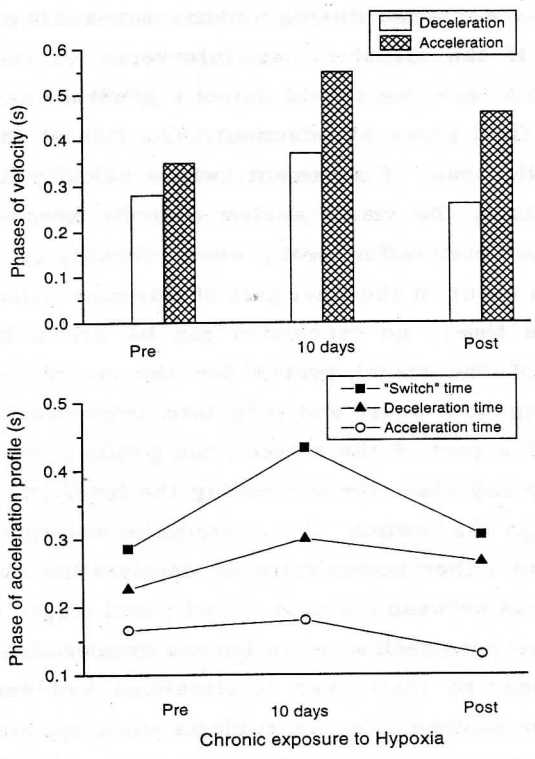

Fig. 3 - Velocity-time and acceleration-time profiles before, at the end of a 30 minute period or at the 10th day, and after chronic exposure to hypoxia.

DISCUSSION

Our date suggest that the CNS can successfully organize movement and maintain high accuracy in acute and chronic exposure to a low O2 environment. However, in these conditions, it requires an additional time for the organization of the movement, which results in prolongation of movement. This can be expected since our subjects were instructed to perform movements as fast as possible.

Pointing to visual target is controlled by proprioceptive and visual input. Movements are probably ballistic, as they seem not controlled during execution of the movement but only by terminal visual feedback.

The question arises whether during hypoxia movements are also executed ballistically or if the visual system intervenes on the late phase of movement. In this case, we should detect a general undershooting and/ or a prolonged final phase of movement. The role of the visual system during the middle phase of movement, where velocity is very large, is not clear. Possibly, the visual system controls movement only in the phases after the deceleration peak, where velocity is small. Since no specific changes occur in the final part of movement (deceleration phase and deceleration time), no conclusion can be drawn in regard to an increased role of the visual system for the control of this type of movements during both short and long term exposure to hypoxia.

The most sensitive part of the acceleration profile, the "switch" time, seems to be very important for controlling the amplitude of movement in hypoxia. Generally the "switch" time contributes more to the prolongation of movement than either acceleration or deceleration times. There are strong correlations between the peak velocity and length of the "switch" time, as well as between peak acceleration and deceleration "switch" time.

In brief, we observed that lower accelerations and declarations were characteristic for movement in our subjects when exposed to a hypoxic environment. At least two explanations can be sought for this effect. First, an alteration of proprioceptive loops so that more time is needed for coordination between agonist and antagonist activity (4). This explains the calculated increase of the time between peak acceleration and peak deceleration (data not shown) that corresponds to the "switch" times in figure 2 and figure 3. Second, because of hypoxia the CNS elaborates a general strategy of careful and slow movements in order to avoid errors and destabilization of the body during the movement. This may explain the decrease of peak values of accelerations and decelerations.

Kinematics variables of pointing movements where assessed in five adult subjects exposed acutely (30 min) and chronically (10 days) to a low O_2 mixture (13.5% O_2 in N_2). Amplitude of displacement did not vary in both experimental conditions but movement duration markedly increased compared to pre and post exposure conditions.

While in acute hypoxia the times of acceleration and deceleration are almost equal, in chronic hypoxia deceleration time exceeded of 100 ms the time of acceleration. The time from the peak acceleration to the peak of deceleration ("switch" time) increased in both experimental conditions and was about 50% of the movement duration. This time lengthening at hypoxia may be explained either by alteration of propioceptive loops or by a different strategy elaborated by the CNS to generally slow accurate movements.

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