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CEREBRAL OXYGENATION AND PUSH-PULL EFFECT

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1. INTRODUCTION

Pilots of high performance aircraft are exposed to $+G_z$ accelerations (gravity in the foot-to-head direction expressed as Earth gravity equal to 1 G), which may induce loss of consciousness, caused by a failure of cerebral blood flow. Indeed, $+G_z$ accelerations enhance the cardiovascular responses observed during moving abruptly from supine to standing. Over the heart level, the arterial blood pressure decreases inducing an increase in heart rate and in vascular resistance. Arterial blood pressure is maintained with a sympathetically mediated increase in vascular resistance.[1] These hemodynamic changes are responsible for the decrease in cerebral perfusion pressure and in oxygenation. Such a decrease is kept limited via cerebral autoregulatory mechanisms inducing a cerebral vasodilatation.[1] Under $-G_z$ acceleration, the arterial blood pressure is increased over the heart level, and a cerebral vasoconstriction occurs in response to prevent brain overperfusion.

The push-pull effect occurring in flight is known to be involved in many aircraft accidents, when the levels of $+G_z$ accelerations are less than those involved in loss of consciousness. The push-pull effect is defined as a decreased tolerance to $+G_z$ acceleration subsequent to a less than +1 G_z exposure.[2] Objective physiological tolerance to $+G_z$ acceleration is usually evaluated by change in blood pressure.[2] As a decreased blood pressure may lead to a decreased cerebral perfusion pressure, we wondered if the push-pull effect could induce a greater decrease in cerebral oxygenation. Furthermore, the delay in sympathetic drive during subsequent $+G_z$ exposure may be one of the mechanisms of the push-pull effect.[3] However, by using a vertical rotating-table to simulate the push-pull manoeuvre,[4] it has been demonstrated that cerebral vasoconstriction occurred to prevent brain overperfusion during head-down tilt. During subsequent head-up tilt, the elevated resistance of the cerebral vessel remained at the higher level for about 20 s, and may have worsened the cerebral perfusion from exposure to $+G_z$ acceleration.

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One goal of the present work was to use a human centrifuge to evaluate the implication of the cerebral vasoconstriction occurring under $-G_z$ acceleration in the mechanisms of the push-pull effect. Transcranial Doppler (TCD) velocimetry was used for monitoring cerebral blood flow velocity (CBFV) and cerebral vascular resistance. Near infrared spectroscopy (NIRS) method was used for monitoring cerebral oxygenation and cerebral blood volume changes. Another goal was then to evaluate the accuracy of the NIRS method in the assessment of changes in cerebral vasomotion, by comparison between TCD index and NIRS determined-cerebral blood volume changes. We hypothesized that brief exposure to $-G_z$ acceleration may reduce cerebral oxygenation during subsequent $+G_z$ acceleration.

2. METHODS

2.1. Subjects

Four healthy male non-pilot volunteers participated in this preliminary study. The subjects averaged 35 yr in age (range = 32-42 yr), 79.5 ± 10.1 kg in weight, and 180 ± 3 cm in height. They were informed of the purpose of the experiment, and the study was carried out with the approval of the national Ethics Committee.

2.2. Acceleration Setup

Acceleration profiles were generated using the 8-m-radius human centrifuge of the Laboratoire de médecine aérospatiale (LAMAS), Brétigny-sur-Orge, France. Each subject was seated and secured inside the centrifuge gondola in a 15° reclined seat.

2.3. Physiological Measurements

2.3.1. Cerebral oxygenation

Cerebral oxygenation was measured with NIRS, which allows to measure continuously and non-invasively changes in brain oxygenation concentration and in cerebral hemodynamics.[5,6] This method was also validated under +Gz acceleration,[7,8,9,10] particularly with the NIRO-300G,[8,9] a modified devised instrument (Hamamatsu Photonics, Hamamatsu, Japan). Our study was performed using the NIRO-300 monitor which produces four wavelengths of near-infrared light (775, 810, 850, and 910 nm) allowing monitoring changes in oxygenated (oxy-Hb) and deoxygenated (deoxy-Hb) hemoglobin concentrations, and cerebral blood volume as changes in total hemoglobin (total-Hb = oxy-Hb + deoxy-Hb) with a modified Beer-Lambert equation.[8] Furthermore, the NIRO-300 measures TOI (tissue oxygenation index = oxy-Hb/total-Hb) by using the spatially resolved technique.[8] By using a self-adhesive pad, the optodes (light source and light detector) were placed 4.0 cm apart on the right forehead, avoiding the temporal muscle regions. The differential path length factor was set at the level recommended for the adult head[11] to quantify the NIRS data in μM, and the sample frequency was set at 2 Hz.