

TECHNICAL NOTE

Perfusion of the Visual Cortex During Pressure Breathing at Different High-G Stress Profiles

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The effects of pressure breathing for G protection (PBG) on perfusion of the visual cortex were studied in a subject during various high-G stress profiles. Blood flow velocity was measured in the posterior cerebral artery using a transcranial Doppler (TCD) ultrasound instrument. The G profiles examined included gradual and rapid onset rates. Mean cerebral blood flow velocity (MCBFV) declined with increasing $+G_z$ with G-suit protection alone. The MCBFV increased in direct proportion with increase in $+G_z$ acceleration with PBG. The mediating mechanisms for the effects of PBG may include improved gaseous exchange, the diminished sympathoadrenal discharges, and cardiopulmonary reflexes. A role for TCD in further research is indicated.

AEROSPACE MEDICINE endeavors to counter the effects of $+G_z$ acceleration by suggesting maneuvers and G-protection systems that function to maintain adequate perfusion of the brain. Recently, the U.S. Air Force initiated the Combined Advanced Technology Enhanced Design G Ensemble (COMBAT EDGE) Program for full scale development under the Tactical Life Support System Program. COMBAT EDGE is a G-protection system that provides the pilot with increasing levels of breathing gas pressure as gravitational force increases [15]. It does this by using the G-valve outlet pressure to drive the breathing regulator which in turn

provides the pilot with positive pressure breathing for G protection (PBG). The assessment of the benefits of PBG is constrained by lack of parametric measures of the effects of G forces on human physiology. Currently, it is customary to assess acceleration stress tolerance by recording the time of endurance after reaching peak $+G_z$ in a given G profile. However, such measurements do not necessarily reflect the state of cerebral perfusion and yet the adverse effects of high G on vision (gravitational loss of vision, G-LOV) and level of consciousness (gravitational loss of consciousness, G-LOC) both result from fall in cerebral perfusion (4-6). Indeed, there is a direct relationship between the applied acceleration and fall in cerebral perfusion (5). It appears that direct assessments of the state of cerebral perfusion during high G stress are needed. The present case study employed a Doppler ultrasound apparatus to monitor perfusion of the visual cortex in an experienced centrifuge subject during exposure to PBG and various G profiles. The purpose was to look for possible correlations between changes in brain blood flow, $+G_z$ -stress, and anti-G measures.

METHODS

The $+G_z$ exposures were carried out in a human centrifuge at the Center for Research in Special Environments, State University of New York, at Buffalo. The subject was a 31-year-old male F-16 pilot. He was healthy and had recently successfully completed physical and laboratory evaluations, which included routine electrocardiography, chest X-ray, blood sample and electrolyte analysis, ophthalmologic, ear, nose and throat examinations. There was no history of cardiopulmonary or neurological diseases. After informed consent was obtained, the following protocol was carried out in accordance with institutional guidelines.

Cerebral blood flow velocity measurements: Blood flow velocity in the right posterior cerebral artery

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(RPCA) which supplies the right visual cortex was monitored at a depth of 70 mm from the skin surface using a TCD instrument (TC2-64, Eden Medical Electronics, Edenbergen, Germany). The subject was fitted with a 2-MHz TCD transducer, with the probe housing modified for use in a centrifuge. The probe was first fastened in the subject's head with an elastic head band. The probe housing was anchored to the inner lining of the pilot's helmet. The RPCA was identified by depth, directionality of flow, audiovisual spectral output, anatomic relationship to the basilar and carotid bifurcation Doppler velocity signals, response to light stimulus (eyes closed and open) (1,12), and visual attention to colored objects (12). Head movements and pressure on the helmet were used during preparation to simulate the effects of G stress on the helmet which may affect the integrity of the Doppler signals. A satisfactory placement was accomplished when these simulations did not cause any deterioration of signal quality. This was then confirmed during initial test runs and, if necessary, adjustments were made to optimize signal quality in the 5-min rest period before each data run. All tests were performed with the subject seated at 13° backangle. The parameters monitored included the mean cerebral blood flow velocity (MCBFV) and the pulsatility index (PI) (1) calculated as: $PI = (\text{peak systolic velocity} - \text{end-diastolic velocity}) / \text{MCBFV}$.

Audiovisual communication with the subject was maintained at all times, and electrocardiography was monitored during the runs.

Centrifuge protocol: Centrifuge data were acquired in one session with rest periods of 5 min between successive runs. The +G_z exposure profiles included gradual onset rate (GOR) of 0.1 G/s to 10 G_z for 10 s, Profile 1. Profile 1 was performed without G-suit to determine relaxed tolerance and proceeded until G-LOV, after which time the subject began anti-G straining maneuvers. The rest of the runs were performed with subject wearing a fully operational anti-G suit equipped with PBG. The schedule of PBG was 12 mm Hg/G starting at +4 G_z and increased to a maximum pressure of 60 mm Hg at +9 G_z. Profile 2, consisted of a GOR of 0.1 G/s to 10 G_z for 10 s. Profile 3 was a ROR of 0.5 G/s to +8 G_z, sustained for a maximum of 3 min or to end-points given below. Profile 4 was a ROR Simulated Aerial Combat Maneuver (SACM), with 1.04 G/s transitions, between +5 and 9 G_z and with 10-s plateaus. The +G_z level, the positive pressure readings and the cerebral blood flow velocity waveforms were all simultaneously displayed on a monitor.

The end-point for all profiles included successful profile completion, fatigue, failure to respond to buzzer signal, cardiac rhythm abnormalities, or visual light loss. The cardiac rhythm abnormalities which constituted an end point were: ventricular tachycardia (3 or more successive ventricular ectopic beats at a rate of 100 bpm or more), supraventricular tachycardia (3 or more successive supraventricular ectopic beats at a rate of 100 bpm or more), and bradyarrhythmias (17). The visual changes during +G_z were determined by using a curvilinear metal bar in a 120° arc placed in front of the subject at eye level. On the bar were mounted a central white light for fixation and testing of central vision and

two red lights sustaining a visual angle of 48° for testing peripheral vision. The visual endpoints were loss of peripheral vision (grey-out) or peripheral light loss and loss of all vision (black-out) or central light loss. The grey-out was defined as a temporary, complete loss of red lights accompanied by dimming or blurring of vision of the white light. The black-out was associated with temporary, complete loss of central white light. The subject was instructed to press the trigger when he saw the red light as clearly as at 1 G, but release the trigger if he did not. Once this end-point was attained the subject was advised to strain as necessary to prevent G-LOC.

Data analysis: Correlation regression analyses were performed between +G_z level (as the independent variable) and percentage changes in MCBFV (%dMCBFV) as the dependent variable. $\%dMCBFV = ([\text{MCBFV data run} - \text{MCBFV baseline}] / \text{MCBFV baseline}) * 100$. The test of significance level was set at $p < 0.05$.

RESULTS

The MCBFV in the RPCA was 28 ± 3 cm/s and the PI was 116 ± 24 in the relaxed baseline state. In the initial GOR run without G-suit protection MCBFV declined in direct proportion to the increase in G-stress. This relationship was linear: $\%dMCBFV = -10 G_z + 7.8$, with $r^2 = 0.6$, $p = 0.0001$, the 95% confidence lower and upper limits on the slope were -12 and -8, respectively. The GOR end-point occurred at +6.5 G_z, with 50% reduction in MCBFV.

With G-suit protection during GOR, the MCBFV declined until +5 G_z were reached (Fig. 1). The relationship was given by: $\%dMCBFV = -6.4 G_z + 1$, $r^2 = 0.3$, $p = 0.002$, and 95% confidence upper and lower limits were -10 and -3, respectively. The negative slope still persisted, though lesser than in the initial run without G-suit operation. As the PBG became operational its effects changed the relationship between %dMCBFV and +G_z, such that as +G_z increased (above +5 G_z) the MCBFV increased in direct proportion. This is shown in the equation: $\%dMCBFV = 6.73 G_z - 59$, $r^2 = 0.7$, $p = 0.0001$, and 95% confidence lower and upper limits were 6 and 7, respectively. The changes in PI were directly opposite to that for MCBFV (Fig. 1). As expected, with increasing +G_z there was an increase in PI and, consequently, a decrease in blood flow velocity (below +5 G_z level). However, above +5 G_z level with PBG, MCBFV increased with +G_z as PI dropped, probably due to vasodilation.

With ROR profile, there was an initial drop in MCBFV typically at the onset ramp before the G plateau. Flow velocity in the superficial temporal artery correlates with eye level arterial pressure and has been used to predict an impending loss of central vision during rapid onset runs (14). In this subject, G-LOV, which indicates a considerable level of ischemic/hypoxic insult to the central nervous system, occurred as MCBFV reached -50% of baseline perfusion. The MCBFV declined over time, while PI increased initially, but oscillated towards the end of the run at sustained +8 G_z (Fig. 2). During the SACM the MCBFV fluctuations followed the pattern of changes in +G_z (Fig. 3). Interestingly enough, the values of MCBFV were higher at

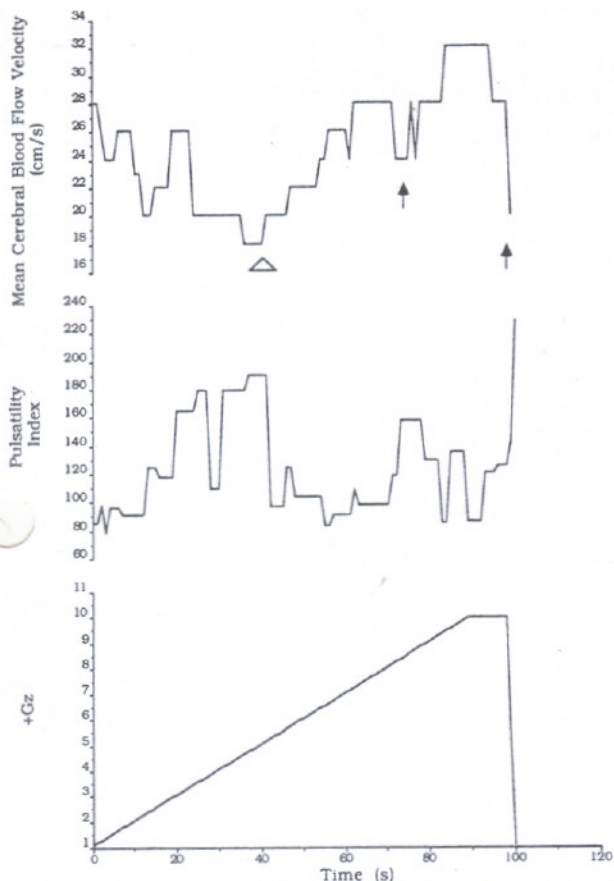


Fig. 1. The fluctuations of mean cerebral blood flow velocity (top panel) and pulsatility index (middle panel) during gradual onset rate profile of 0.1 G/s to 10 G_z for 10 s (bottom panel). The PBG initiated at +4 G_z (open arrow head) led to increased blood flow velocity. The light strain (black arrow) initiated subjectively by the pilot had also a positive effect on blood flow velocity.

G_z than at +5 G_z, which is probably related to the effects of PBG. This profile was terminated after a 20-beat ventricular tachycardia occurred in the test subject, at the 120th s. This was preceded at the 108th s by cerebral hypoperfusion of -80%, at +5 G_z.

DISCUSSION

Two important results were obtained in this case study. First, prior to PBG (Fig. 1), as +G_z increased, MCBFV in the posterior cerebral artery decreased. For each +1 G_z increase in acceleration there was a 10% decline in MCBFV without operational G-suit, but a 6% drop with functional G-suit. This relationship was reversed with initiation of PBG, such that for each +1 G_z acceleration increase, above +5 G_z, MCBFV increased by 7%. The elevated intrapleural pressure provided during PBG is applied to the heart and intrathoracic great vessels causing a rise in perfusion pressure and increasing the pressure required to fill the cerebral vessels. There are a few other additional mechanisms that are worth considering. One is the increase in the stimulation threshold of cardiopulmonary receptors which have

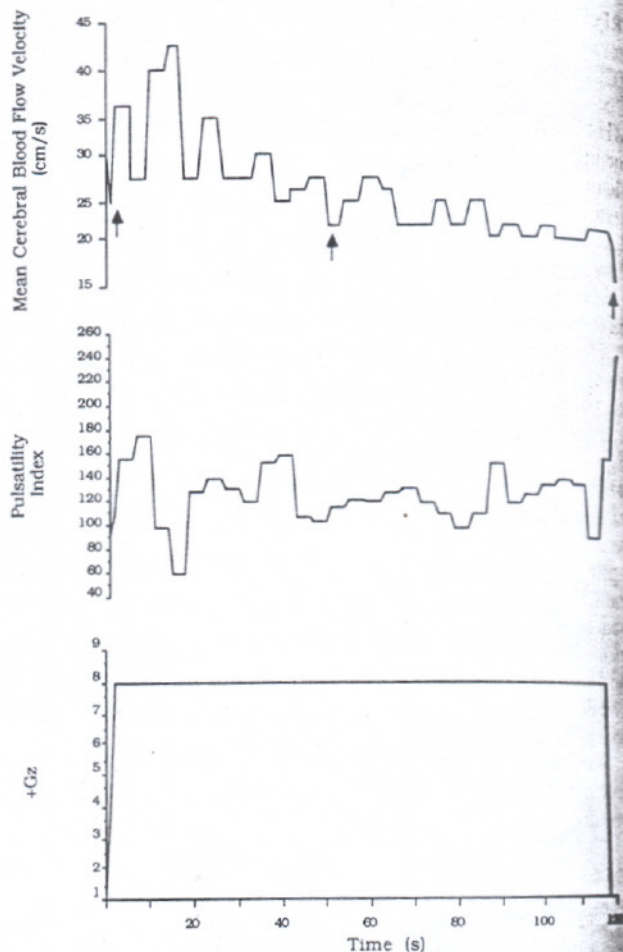


Fig. 2. The fluctuations of mean cerebral blood flow velocity (top panel) and pulsatility (middle panel) during rapid onset rate profile at sustained +8 G_z for 2 min (bottom panel). The light strain (black arrow) initiated subjectively by the pilot had a positive effect on blood flow velocity. However, the blood flow velocity showed a decremental trend as time at sustained +8 G_z elapsed.

been more or less implicated in vasodepressor syncope (8,10). Another, involving expansion of lung units, results in improved functional residual capacity, oxygenation, compliance and decreased intrapulmonary shunt (2). The vasodilation effect of CO₂ on cerebral small vessels at higher end-tidal partial pressures may play a role. It is also possible that, with classic stress response diminished, the effects of sympathetic hyperactivity on vascular tone which primarily manifests in vasoconstriction is eliminated or reduced to a minimum (10). It has been established that the effects of sympathetic renal discharge accompanying reduction in circulating blood volume are detrimental to, rather than responsible for, cerebral autoregulation (3). The effects of sympathetic activity have been suggested to be the mediating mechanism in "cerebral syncope" in the absence of cardiopulmonary reflexes (10).

Second, it is essential to note that there seems to be a critical lower limit of cerebral blood flow below which G-LOV was precipitated in this subject. This limit seems to lie at about -50% of baseline flow values. Some

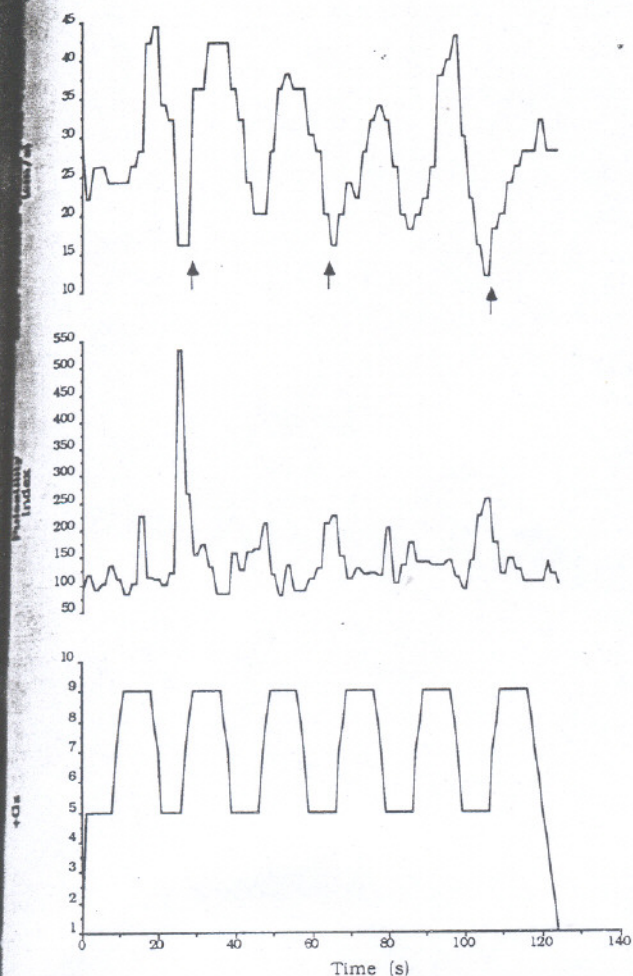


Fig. 3. The fluctuations of mean cerebral blood flow velocity (top panel) and pulsatility index (middle panel) during rapid onset rate profile of +5 to +9 Gz Simulated Aerial Combat Maneuver. The light strain (black arrow) initiated subjectively by the pilot had a positive effect on blood flow velocity. The slope of blood flow velocity rose synchronously with the initial G-profiles (first three peaks), but showed a stepwise rise with a few seconds lag thereafter (last three peaks). The overall trend for blood flow velocity was decremental.

observations have been made at +1 Gz (9,10,13). The latter refers to the possibility of determining "critical cerebral perfusion level," and hence supports the notion of "functional cerebral reserve." The latter may show individual variability, depending on training, experience and task performance. The critical level of perfusion may help estimate the tolerance ability to sustained acceleration. The fact that PBG can extend the limit of tolerance, therefore, suggests its influence on "the functional cerebral reserve" capability. In physiological terms, this can only be related to the level of oxygenation of brain tissue. It seems that with PBG, the latter is accomplished satisfactorily, but it remains to be seen to what extent the intrinsic neurogenic, myogenic and humoral mechanisms regulating cerebral perfusion are maintained intact in the +Gz environment with PBG.

It was also observed that changes in PI were in most part opposite to the fluctuations in MCBFV. It is established that changes in pulsatility reflect physiological

changes in the arterial peripheral resistance in large compliant chambers (7). This includes the conducting vessels of the circle of Willis and the distal resistance vessels. However, it must be noted that cardiac and systemic vascular factors affecting viscoelastic properties may modify pulsatility.

Another interesting observation is the occurrence of ventricular tachyarrhythmia preceded by a critical drop in cerebral perfusion, which occurred 12 s earlier. It is plausible that this temporal association between +Gz-induced transient cerebral ischemia and dysrhythmia in this subject represents a cause and effect relationship. Clinical evidence that cerebrogenic dysrhythmias may result from acute cerebral hypoperfusion in humans has been obtained during upright tilt-induced syncope [Njemanze, unpublished]. Since cardiac arrhythmias are not uncommon during +Gz stress tests (17), their association with cerebral hypoperfusion requires a more in-depth investigation, to establish interacting mechanisms of both systems.

This case study indicates that TCD monitoring may be useful in G-stress testing. Some difficulty is usually encountered in the stabilization of the TCD probe to ensure good quality recordings. However, this factor is to a large extent operator dependent, and also depends on the prominence of the subject's "acoustic windows" on the temples. The ability to measure changes that precede G-LOV much earlier than G-LOC (16) offers an application for design of G-protection systems. The capability of TCD to detect human cognitive activity (11,12), in addition to the possibility to predict G-LOC, offers a unique potential application of TCD (U.S. Patent No. 5121744) in aerospace medicine.

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