International Symposium on Aviation
Psychology - 2009

2009

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Gravity-induced loss of consciousness (GLOC) is a major problem facing fighter pilots. In the throes of GLOC, pilots can travel 12 miles without control of their aircraft. The GLOC problem has proven to be difficult to resolve. Solutions involving repeated exposures to GLOC, G-suit pressure manipulation, intense sensory stimulation, and exposure to –G following the GLOC event have been unsuccessful. It is evident that a different approach is needed. One possibility might be an adaptive automation system to warn pilots of the imminence of a GLOC event. A key issue in adaptive automation is the cue to be employed in triggering the onset of automation. Using a centrifuge to simulate gravitational forces together with tracking and math tasks to simulate flight control and navigation, this study assessed the utility of a of cerebral tissue oxygen saturation (rSO_2) measure as a possible triggering mechanism for an adaptive automation recovery system.

Gravity-induced loss of consciousness (GLOC) is a major threat to pilots of modern fighter aircraft. It is brought about by a sudden reduction in cerebral O_2 as a result of increased $+G_z$ force (Tripp, Chelette, Savul, & Widman, 1998). Originally it was thought to consist of 12 sec of complete unconsciousness (absolute incapacitation) coupled with 12 sec of confusion and disorientation (relative incapacitation) However, it is now clear that the course of the GLOC episode is much worse than originally believed. A recent study by Tripp et al. (2006), showed that pilots ceased performing flight tasks approximately 7 sec prior to the onset of the absolute incapacitation phase of the GLOC episode and performance efficiency does not return to baseline values for 55.5 sec following emergence from the relative incapacitation phase (Tripp et al. 2006). Thus, fighter pilots who encounter a GLOC episode can fly approximately 12 miles while not in control of their aircraft. From 1983-1990, GLOC was responsible for the loss of lives of 24 USAF pilots and many other non fatal mishaps (Albery & Van-Patten, 1991).

The GLOC problem is complex and to this point has proven to be difficult to resolve. Measures involving repeated exposure to GLOC, modifications in anti-G suit deflation schedules, intense sensory stimulation deigned to elicit startle responses, and exposure to negative G following the GLOC event have not been successful in attenuating the GLOC problem (Tripp et al, 2006; 2007). In light of these findings, it is evident that a different approach to countering the effects of GLOC is needed.

On a general level, one remedy for reductions in pilot efficiency is *adaptive automation* in which a machine function capable of carrying out duties normally performed by the pilot is activated when the pilot is unable to perform those duties (Parasuraman, Mouloua, Molloy & Hilburn, 1996; Scerbo, 2007; Satchell, 1993; Wickens et al., 1998). Along this line, Scerbo (2007) has argued that in hazardous situations in which pilots are vulnerable and lives are at stake such as GLOC, it is extremely important to have available the capability for an avionics-initiated invocation of automation. Such automation could warn pilots of the imminence of GLOC, thereby alerting them to the need for taking action to decrease the G-force and also assume control of the aircraft if GLOC sets in. As described by Parasuraman, Bahri, Deaton, Morrison, and Barnes (1992), a key issue in adaptive automation is the cue to be used in triggering the onset of automation. This can be achieved through methods based upon critical events, performance measurement, operator monitoring, and physiological assessment. Given the short interval between the onset of high-G and the occurrence of early performance failure and subsequent unconsciousness, it would appear that a physiological cue might be the most effective in the case of a GLOC event.

Although the brain represents only 2% of the human body's weight, it consumes 20% of the body's oxygen requirement (Raichle & Gusnard, 2002). Using near infra-red spectroscopy (NIRS), a non-invasive optical imaging technique for measuring cortical oxygen levels (Gratton & Fabianai, 2007), several studies have shown that noninvasive optical imaging reveals aspects of neuronal activity in the brain (Franceschini & Boas, 2004; Helton et al. 2007; Gratton & Fabiani, 2007; Steinbrink et al., 2000; Tse, Tien, & Penney, 2006). In addition, there are reductions in cortical tissue oxygen saturation during +Gz acceleration (McKinley, Tripp, Bolia, & Roark 2005; Tripp et al., 1998). Accordingly, one goal for the current study was to chart the changes in cerebral oxygen saturation that occur prior to a GLOC episode in order to identify those that could be used operationally to warn

pilots of impending loss of consciousness or to provide the trigger for an adaptive automation system that could take over the aircraft until the pilot was able to regain flight control.

Although reductions in cortical tissue oxygen saturation during Gz acceleration have been well documented (McKinley et al., 2005; Tripp et al., 1998), it is critical to note that no data are currently available on the rate of return of tissue oxygen saturation following acceleration offset. Accordingly, a second goal for the present study was to use the NIRS technique to provide the initial examination of the rate of oxygen recovery following Gz offset. Given the prolonged performance recovery time following a GLOC event, one might surmise that the rate of oxygen recovery would be sluggish. That hypothesis was tested in the current study.

Method

Participants   Six active duty members of the United States Air Force (three men and three women), participated in the study. They ranged in age from 19 to 34 years, with a mean of 25.5 years. All participants were members of the sustained acceleration stress panel at Wright-Patterson AFB, OH. Participants were required to meet Air Force Flying Class III medical standards prior to their participation.

Facility The study was conducted at the Air Force Research Laboratory's Dynamic Environment Simulator at Wright Patterson AFB.

Acceleration Profiles: A computer control system was utilized to generate a positive Gz acceleration profile. The acceleration profile consisted of a 3G/sec rapid onset to an endpoint of unconsciousness.

In agreement with the flight surgeon, the principal investigator aborted the acceleration profile immediately upon the onset of the GLOC episode. This was followed immediately by a 1.5 sec return to full stop at +1 Gz.

GLOC Criteria: The presence of GLOC was determined using the Whinnery, Burton, Boll and Eddy (1987) criteria that included the following signs: (1) dual eye closure, (2) slumping of the head and upper body, (3) jaw muscle relaxation evidenced by a gaping mouth. All three signs needed to be present in real time surveillance images of the participant in order to determine that the participant had entered GLOC. The principal investigator and the flight surgeon had to be *in total agreement* to make the call. Following the Whinnery et al. (1987) protocol, participants were considered to have regained consciousness when they reopened their eyes. Again, the principal investigator and the flight surgeon had to be in *complete agreement* using real-time observation of the participant.

Performance Tasks A compensatory tracking task used by Tripp et al. (2006) was employed to tap the motor skills required by a pilot to maneuver an aircraft in flight. In addition to the tracking task, participants were required to perform a computation task used previously by Tripp et al., (2006) to tap the higher order cognitive skills needed by fighter pilots to navigate their aircraft. The task involved a series of addition and subtraction problems.

Procedure

Upon arrival at the laboratory, participants were instrumented with a Somentics (Troy, MI) INVOS 4100 Cerebral Oximeter which was used to measure cerebral tissue oxygen saturation (rSO_2) in the right frontal lobe. The self-adhesive oxisensor was affixed to the participant's forehead underneath a flight helmet. Participants wore the standard issue air force flight suit and Gentex helmet with the helmet's visor removed to permit observation of the participant's eyes during GLOC. Oxygen saturation prior to, during, and after the GLOC episodes was measured in terms of percent baseline values. To secure these measures, each experimental session was preceded by a 10-sec resting phase. Following the baseline resting phase, participants remained at rest for another 20-sec prior to the onset of acceleration. Mean oxygen saturation during the initial 10-sec resting phase was the baseline platform from which subsequent oxygen changes in terms of percent baseline were derived. The post-baseline period in which participants remained at rest was necessary to establish the stability of the baseline measure. An unstable baseline platform would render any changes in cerebral oxygen levels associated with GLOC difficult to interpret. Following the O_2 baseline and prior to the acceleration phase, performance baselines for the two tasks were established during a 30 sec testing period in which the gondola was static

Participants were instructed to engage the performance tasks as long as they could before lapsing into unconsciousness, to re-engage the tasks as soon as possible following emergence from the relative incapacitation period, and to continue engagement for five min thereafter. Oxygen saturation was measured continuously from the onset of the pre-acceleration baseline period until the end of the five-min recovery period that succeeded the relative incapacitation phase.

Pre-GLOC Performance The issue of pre-GLOC deterioration in performance was addressed in this study in terms of whether participants *ceased to respond* to either the tracking or the math task prior to the onset of unconsciousness in a GLOC episode. Cessation of response rather than the relative quality of performance was used as the dependent variable because response cessation represents the maximum measure of when pilots are not in control of the aircraft.

One-tailed *t*-tests indicated that the means for the tracking (-3.76 sec) and math tasks (-5.69 sec) were both significantly below zero (or coincident with the onset of GLOC), indicating that in each instance, response cessation significantly preceded the onset of GLOC, $t_{tracking}(5) = 6.71$, $t_{math}(5) = 9.33$, $p_{(Bonferroni\ corrected)} < .05$ in each case. Cessation times for the math and tracking tasks in this study did not differ significantly from each other, $t_{math\ and\ tracking}(5) = 3.46$, $p_{(Bonferroni\ corrected)} > .05$.

Post-GLOC incapacitation performance. The moving window procedures for determining post-GLOC performance recovery times in the tracking and math tasks developed by Tripp et al. (2006) were utilized in this study. These procedures determine the temporal point at which a participant's performance returns to baseline level. A t-test indicated that there was no difference in recovery time between the two tasks. A one-tailed *t*-test indicated that the average recovery time across tasks, **49.45 sec**, differed significantly from zero or immediate recovery from the relative incapacity phase of the GLOC episode,. $t_{one-tail}(5) = 7.22$, $p_{(Bonferroni\ corrected)} < .05$. However, the average recovery time was not significantly shorter than the 55.50 sec value reported by Tripp and his associates (Tripp et al., 2006) in their initial discovery that participants' performance is degraded for a period of time following GLOC, $t_{two-tail}(5) = 0.883$, $p_{(Bonferroni\ corrected)} > .05$.

Cerebral Tissue Oxygen Saturation

The mean percent changes from baseline in cerebral oxygen saturation (rSO_2). Data are plotted as a function of successive 2-sec intervals is illustrated in Figure 1. The figure is divided into pre-GLOC, GLOC- incapacitation, and post-incapacitation recovery phases. The acceleration onset landmark reflects the onset of acceleration following the 20-secs of rest that initiated each experimental session. The remaining landmarks reflect the average values across participants for the appearance of math cessation, the beginning and ends of the absolute and relative incapacitation periods, and performance return to baseline.
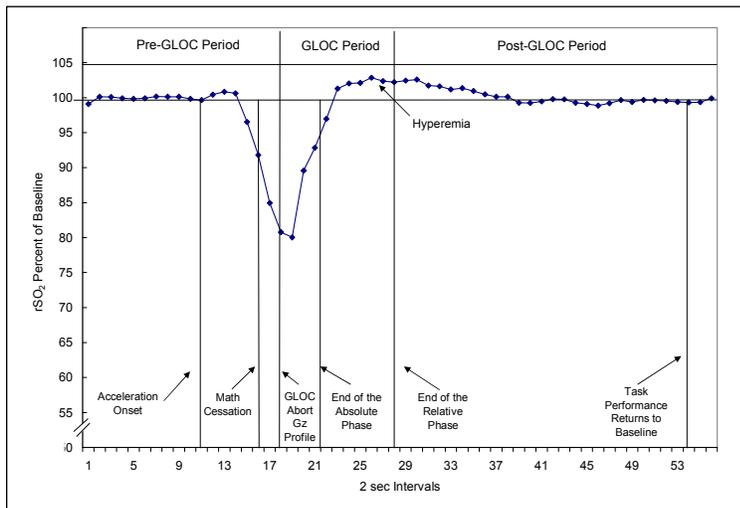

Figure 1. Changes in rSO_2 across time in a composite of the
three Gz offset conditions.

A one-way repeated measures ANOVA of the data during the pre-GLOC phase indicated that the rSO_2 scores differed significantly from the onset of the experimental session until the point of GLOC, $F(1.866, 9.330) = 36.715$. $p < .0001$.

Perusal of the figure reveals that rSO$_2$ remained stable for the 20-sec prior to the onset of acceleration and began a rapid decline at about 28 sec into the acceleration profile until GLOC occurred. Participants ceased performing the math task when the decline in rSO$_2$ reached approximately 95 percent of baseline and GLOC set in when the decline in rSO$_2$ reached approximately 80 percent of baseline. A similar ANOVA of the data during the time intervals from the onset of GLOC incapacitation to the point of performance recovery indicated significant differences in the rSO$_2$ scores across these intervals, F (2.628, 13.140) = 21.108, $p <$. 0001.

It is clear in the figure that the rSO$_2$ increased rapidly from the point of deceleration of the centrifuge which occurred at the onset of GLOC, tended to exceed baseline values throughout the relative incapacitation phase, and during the early portions of the recovery phase, and returned to baseline levels at approximately 18 sec into the recovery period, where it remained until performance returned to its baseline level. In all of these ANOVA's, the Box correction was used to compensate for violations of the sphericity assumption (Maxwell & Delaney, 2004).

Discussion

Cerebral Tissue Oxygen and Adaptive Automation

A one goal for this study was to chart the changes in cerebral oxygen saturation that occur prior to GLOC to identify those that could be used by an adaptive automation system (AAS) system in monitoring the pilot in flight in order to warn him/her of impending loss of consciousness and to assume control of the aircraft when the pilot was incapacitated. The results indicate that changes in rSO$_2$ offer the promise of being useful in this way. The oxygen saturation figure shows that rSO$_2$ levels tended to remain relatively stable when participants were at rest at +1 Gz but that they declined rapidly from baseline during an acceleration profile. Within that declining function, the figure reveals that participants were unable to process simple mathematical information once rSO$_2$ fell to a level that was *95 percent of baseline* and that there was an approximately six-sec window before rSO$_2$ levels fell to *80 percent of baseline* and GLOC set in. These rSO$_2$ values can be critical landmarks for alerting the pilot that that loss of consciousness is approaching and for triggering the adaptive automation system to assume flight control.

The ability to non-invasively characterize changes in neurophysiology in near real-time in a dynamic flight environment and to use that information to predict a pilot's physiological and cognitive state would be a powerful tool for the high performance aviation community. However, employment of that tool could have serious costs. Consequently, thought is required to determine the scenarios in which it might be utilized advantageously.

Currently, the aircraft cockpit is densely populated with displays that provide the pilot with information regarding variables pertaining to the state of the aircraft such as air-speed, altitude, flight attitude (pitch, roll, and yaw), and hydraulic and electrical system status, but the pilot is virtually blind to pilot-state variables. One remedy might be to provide the pilot with cerebral oxygen information in the form of a three-color light system i.e. green = stable normal rSO2, yellow = compromised cerebral rSO$_2$, and red = impending GLOC. Such information might serve to reduce the high and often fatal incidence of GLOC that occurs in pilot training (CHI Systems, 2000) by alerting the novice pilot about an impending loss of consciousness so that the pilot could change the energy variable of the aircraft to avoid GLOC, and in cases where GLOC in not avoided, to trigger an auto-recovery system that would return the aircraft to level flight while the pilot recuperates.

A system of this sort might be employed to achieve similar goals in a combat environment. However, there is the question of whether experienced combat pilots would accept such a system because it could be viewed as peripheralizing their roles (Satchell, 1993). Moreover, it may also have negative consequences in combat. The steep climbs and sharp turns that can lead to GLOC in combat are part of the maneuvers often employed by pilots to avoid airborne enemy threats or to gain an offensive advantage over those threats (Shaw, 1985). While taking control from pilots in such situations and returning the aircraft to level flight might avoid the incapacitation induced by GLOC, it could also counter the tactics employed by the pilot to avoid or destroy the enemy and thereby place the pilot's aircraft in harm's way. Hence, it may be more appropriate in the air-combat setting to provide pilots with rSO$_2$ information that enables them to extend the tactical envelope by allowing them to fly the aircraft to the edge of their physiological capabilities.

A second goal for this investigation was to use the NIRS technique to provide the initial examination of the rate of oxygen recovery following Gz offset and to test the hypothesis, based upon the extended time needed after GLOC incapacitation for performance efficiency to return to pre-GLOC levels, that the rate of oxygen recovery would be sluggish. As can be seen in the oxygen saturation figure, that hypothesis was not confirmed. The rSO$_2$ level began to increase almost immediately upon termination of the acceleration profile. It rose steeply to a level that

exceeded baseline during the relative incapacitation phase and the early portions of the recovery phase, a phenomenon termed *reactive hyperemia,* and settled back to baseline approximately 18 sec prior to the point at which performance efficiency returned to a pre-GLOC level. Two aspects of the time course of return in the level of rSO_2 are noteworthy. The presence of hyperemia is consistent with similar effects observed in medical situations when patients recover from hypoxia. It is due to the dilation of cerebral blood vessels brought about by a build- up of cellular metabolites (Gyton & Hall, 2005). The second key point is that the prolonged period of performance recovery cannot be attributed to delays in the rate of return of rSO_2. Thus, another mechanism must be responsible for the prolonged period of recovery after GLOC. As described by Dirnagl, Iadecola, and Moskowitz (1999), hypoxia causes a critical shortage in brain energy as neurons use glucose and oxygen faster than they are being supplied. At the cellular level, this energy depletion is accompanied by a failure of the Na^+ and K^+ pumps critical for depolarizing neuronal membranes which, in turn, causes conductivity to cease resulting in a significant loss of neural firing. The accumulation of metabolite by-products during ischemic hypoxia delays the recovery of normal neurological function following rSO_2 return. Therefore, it would appear that the need to clear away the metabolic residue of GLOC-induced hypoxia may be responsible for the prolonged performance recovery period even though rSO_2 levels are at or above baseline values. An explanation along these lines reinforces the view that shortening the duration of the GLOC event by fostering a return of blood to the brain would not be a viable alternative to combating the overall GLOC problem, since the performance deficit following GLOC may be more biochemical than hemodynamic in origin.

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