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PERFORMANCE RECOVERY IN THE BABOON FOLLOWING +G_Z-INDUCED LOSS OF CONSCIOUSNESS

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The animals involved in this study were procured, maintained, and used in accordance with the Animal Welfare Act and the "Guide for the Care and Use of Laboratory Animals" prepared by the Institute of Laboratory Animal Resources - National Research Council.

The Office of Public Affai·s has reviewed this report, and it is releasable to the National Technical Information Service, where it will be available to the general public, including foreign nationals.

This report has been reviewed and is approved for publication.

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13. ABSTRACT *(Maximum 200 words)*

Seven male baboons (avg. wt. 20.6 ± 1.6 kg) were trained in a simple shock-avoidance performance task on the Armstrong Laboratory human/animal centrifuge for subsequent $+G_z$-induced loss of consciousness (G-LOC) exposure. A red light was presented to the baboon at approximate 2-s intervals. The animal was allowed 1 s to turn off the light or receive a 1-s shock. The shock could be abbreviated by a late trigger pull (escape). Thus, the animal could avoid, escape, or accept the full shock. EEG was monitored from three transcranial stainless steel electrodes. Loss of consciousness (LOC) was induced by a rapid onset (4 or 6 G/s) exposure to 8 $+G_z$ (head-to-feet inertial load), and sustained until LOC was identified by a near isoelectric EEG signal. Performance recovery time was measured from the return of EEG activity to the time when the animal resumed the performance task. These data were compared with previously obtained human data and found to be very similar. Also, it was found that performance recovery time significantly ($p<.001$) increased with increased duration of unconsciousness. This study demonstrates the utility of the baboon as an animal model for G-LOC research.

14. SUBJECT TERMS

EEG; G-LOC; $+G_z$; loss of consciousness; performance recovery; baboon

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PERFORMANCE RECOVERY IN THE BABOON FOLLOWING $+G_z$-INDUCED LOSS OF CONSCIOUSNESS

Tactical maneuvering of high-performance aircraft can impose severe head-to-foot inertial stress ($+G_z$) on pilots, with the potential for $+G_z$-induced loss of consciousness (G-LOC) and possible loss of aircraft and life. The inertial stress associated with $+G_z$ results in displacement of blood away from the brain to vascular areas below the heart and decreases head-level blood pressure by 22-25 mmHg/G (20). Therefore, the rapid exposure of an upright seated, relaxed centrifuge subject, or of a pilot not performing an effective anti-G straining maneuver (AGSM) to 7-8 $+G_z$ will usually result in loss of consciousness (LOC) after 6-7 s.

Whinnery and coworkers have studied over 500 accidental and intentional human G-LOC episodes which have occurred on the Armstrong Laboratory centrifuge (27). They found that during the recovery period following a rapid onset $+G_z$ exposure to LOC, there are two distinct periods of incapacitation: (1) an unconscious period of absolute incapacitation, which lasts an average of 12 s, and (2) a conscious period of confusion, labeled relative incapacitation, which also lasts an average of 12 s. The sum of these two periods (24 s) is the total incapacitation time, and corresponds operationally to the time a pilot would not be in control of the aircraft following a G-LOC incident. Also, during the period of absolute incapacitation there is a convulsion-prone period, and toward the end of the convulsion period, a dream period.

The U.S. Air Force (USAF) is particularly interested in G-LOC because of previous and continuing loss of pilots and aircraft. Observations of repeated G-LOC in humans have indicated a possible training effect/benefit. It has been suggested that G-LOC training may be beneficial to aircrew (26). However, this hypothesis has not been validated with controlled experimentation. Moreover, it is not practical, and it may not be ethical to use human subjects repeatedly in G-LOC research; therefore, an animal model has been developed to better understand the process of G-LOC and recovery from G-LOC.

METHODS

Seven male baboons with an average weight of 20.6 ± 1.6 kg (range, 18.5-23.3 kg) were trained in a simple shock-avoidance performance task on the Armstrong Laboratory centrifuge. The animals were first trained unrestrained in a cage. After achieving task proficiency in the cage, the animals received restraint chair training in the laboratory, followed by centrifuge training. The transfer of task performance learning from cage, to restraint chair, to centrifuge was limited. A new performance task learning curve had to be established for each new environment. Centrifuge performance recovery data collection did not start until the animals were proficient at the performance task on the centrifuge following G-LOC. The amount of centrifuge training before data collection was variable; it ranged from 12 to 60 $+G_z$ exposures (avg = 27.7 ± 18.1 SD); therefore, data concerning initial response to $+G_z$, G-LOC and performance recovery were not available. The total number of exposures per animal ranged from 57 - 81 (avg = 68 ± 9.3 SD).

The performance task was generated and controlled by a computer. The task was initiated by the presentation of a white light that remained on throughout

the performance task. A red light was presented every 2-5 s. One second was allowed for a trigger pull response to turn off the red light. If the light was not turned off in 1 s, the animal received a 1 s 1-8 mv shock across the lower legs. The shock could be abbreviated by a late trigger pull (escape). Thus, the animal could avoid, escape, or accept the full shock. The white and red lights were mounted directly in front of the baboon, 45.7 cm from the head. The lights were separated by 30.5 cm; the white light was on the right and the red light on the left.

Three transcranial stainless steel EEG electrodes, providing contact with the dura mater, were surgically implanted; two over the parietal areas and one through the frontal sinus. EEG data was obtained from the two parietal electrodes referenced to the frontal sinus electrode using a battery operated microvolt differential amplifier. The EEG data was band-pass filtered between 1 Hz and 50 Hz.

LOC was induced by a rapid onset (4 or 6 G/s) exposure to 8 $+G_z$ (8 times normal gravity) and sustained until LOC was identified and then returned to 1 $+G_z$. LOC was identified by the dramatic decrease in EEG amplitude to near isoelectric (Fig. 1).

The first $+G_z$ exposure for a data collection sequence was a 4 $+G_z$/30 s exposure to refamiliarize the baboon to the centrifuge and the performance task. After a 10-min rest, the baboon was then exposed to 1-3 exposures of 8 $+G_z$ at 4 or 6 G/s for G-LOC data collection with a 10-min rest period between each exposure. The 8 $+G_z$ was maintained until LOC was identified, and then returned to 1 $+G_z$ at 4 or 6 G/s. To investigate the effect of extended LOC on EEG recovery, convulsion time, and performance recovery time, a number of exposures were extended by 10 or 20 s past the point where the isoelectric state was identified.

Analysis of human EEG data taken during LOC episodes resulting from hypoxic hypoxia or ischemic hypoxia, have demonstrated a shift toward increased delta wave activity just prior to LOC (7,16,19,23). To investigate the waveform characteristics of the baboon EEG before, during, and after G-LOC episodes, the EEG data was digitized at 256 Hz for power spectral analysis using fast Fourier transform (FFT) techniques. The EEG data were divided into 1-s windows. The power spectra for each window were calculated for 1 to 50 Hz, with 1-Hz resolution. The resulting spectra were divided into frequency bins corresponding to the component brain wave frequencies (delta, 1-3 Hz; theta, 4-7 Hz; alpha, 8-12 Hz; and beta, 13-30 Hz) for analysis and graphical representation.

The following measurements were made related to LOC and recovery: time to LOC; duration of LOC; EEG recovery time; G level at EEG recovery; convulsion time; and performance recovery time (Fig. 1). Time to LOC was measured from the time acceleration exceeded 1 $+G_z$ until LOC was identified; duration of LOC was measured from the time of loss of EEG activity until EEG activity resumed; EEG recovery time was measured from the offset of $+G_z$ until the return of EEG activity; convulsion time was measured from the return of EEG activity, which coincided with the beginning of video-observed convulsions, until the convulsions ceased; and performance recovery time was measured from the return of EEG activity to the time when the animal resumed the performance task.

RESULTS

Video observations: During the $+G_z$ profile the baboons usually exhibited horizontal, vertical, or a compound nystagmus (depending on the position of the head, which was free to move). Nystagmus increased in rate and decreased in excursion as LOC approached. Immediately prior to LOC the eyelids usually closed, nystagmus stopped, and facial skin relaxed. During LOC, especially during extended LOC, there was an occasional tonic extension of the head with a partial opening of the mouth, similar to an agonal gasp. No convulsions were observed during LOC. During deceleration the eyelids partially opened with a blank unfocused stare and nystagmus returned, followed immediately by return of EEG activity (Fig. 1) associated with convulsions, observed as rapid, uncoordinated head jerks and facial twitching. The television camera was focused on the head; therefore, it was not possible to determine if there was convulsive activity of the torso and limbs. Following the convulsion period, the animals sometimes looked around very rapidly for several seconds with wide eyes as though disoriented. This activity was followed some seconds later by performance task recovery (Fig. 1, Table 1).

The amplitude reduction of EEG toward isoelectric, which was used as an indicator of LOC, and the large amplitude EEG associated with convulsions during recovery from LOC, were consistent over all animals. Table 1 lists the average time relationships of the onset, duration, and recovery from G-LOC (excluding the 10 and 20 s extended LOC time). Figure 1 illustrates this same information superimposed over a strip-chart recording of a typical G-LOC episode.

Figure 2 shows the mean regression of performance recovery time versus the 10-s and 20-s extended time during unconsciousness in six animals. The regressions from the six animals showed positive correlations; three of the regressions were significant at $p<.05$. To match the data, a control G-LOC was taken from the same animal on the same day as the 10-s or 20-s extended LOC data were taken. An analysis of variance (ANOVA) showed a significant ($p<.001$) increase in performance recovery time at the 20-s extended time compared to control. Whereas, the 10-s extended time was not different from control, although there was a distinct trend toward an increase in performance recovery time at 10 s.

Performance recovery time was also compared with the number of G-LOC exposures (excluding the 10- and 20-s extended LOC time). One animal showed a strong negative correlation ($r= -.86$; $p<.0001$) suggesting that performance recovery time following LOC was shortened by multiple G-LOC exposures. The other 5 animals had either weak, nonsignificant negative correlations or a weak, positive correlation (one animal). Thus, no inferences can be made from the relationship between performance recovery time and the number of G-LOC episodes.

Figure 3 illustrates a representative three-dimensional serial power spectral plot of EEG from one animal during a G-LOC episode. The data start from control and progress through LOC (trough) and recovery. The trough coincides with the near isoelectric period shown in Figure 1. The serial median data of the four EEG component waveforms (delta, 1-3 Hz; theta, 4-7 Hz; alpha, 8-12 Hz; beta 1 and beta 2 13-30 Hz) throughout a G-LOC episode were averaged across all animals ($n = 7$) at 1-s intervals (Fig. 4). Control EEG data were taken during

rest; performance task data were collected before $+G_z$. Both control and performance task data before $+G_z$ were averaged over 10 s. The $+G_z$ data were taken during the 8 $+G_z$ profile and were time adjusted for LOC. The recovery data were also time adjusted from the beginning of EEG activity following G-LOC (Fig. 1). Median spectral data rather than mean data were used to reduce the effect of animal movement artifact.

Figure 5 illustrates an increase in resting heart rate (HR) as the number of exposure days were increased, suggesting an increase in apprehension, or anxiety. Five of the six animals involved in this plot showed significant ($p=.05$ or less) positive regressions, while one animal demonstrated a nonsignificant negative regression.

DISCUSSION

The relatively long latency period for G-LOC onset (mean = 11.5 s) following the onset of $+G_z$ supports the general consensus that G-LOC is not directly dependent upon the $+G_z$-related fall in head level blood pressure and blood flow, but rather is directly dependent upon the resultant cerebral hypoxia. This hypothesis is supported by the observation that abrupt interruption of cerebral blood flow (CBF) in humans by rapid cervical occlusion (19), or in animals by a variety of methods including occlusion of the cerebral blood supply (13), or rapid onset $+G_z$ in humans (25) or animals (12), requires a mean of 6.8 to 12.5 s for LOC. Normal CBF in mammals is 50-60 ml/100 gm/min (1). The CBF must be reduced by more than 50% before symptoms are observed (1), or below 45% of normal blood flow before the energy state of the brain is significantly affected (21). Thus, in the baboon, EEG alterations would begin at a CBF of about 20 ml/100 gm/min, and an isoelectric (flat) EEG would occur at 15-18 ml/100 gm/min (1,13), or a mean arterial pressure of 19 mmHg (11). If CBF is reduced and maintained at or below 6 ml/100 gm/min, irreversible cell damage is possible (1). Other studies have shown that brain concentrations of phosphocreatine, ATP, ADP, AMP, and lactate were maintained at constant levels as cerebral perfusion pressure was reduced, until perfusion pressure fell below 40 mmHg. At that point, there was a dramatic change in the concentration of these parameters (21) and graphically looked very similar to the amplitude reduction of the EEG toward the isoelectric state, as observed in this study (Figs. 1, 4).

Previous data have suggested that the usual animal EEG response to brain hypoxia, whether due to interruption of the cerebral circulation or through breathing hypoxic or anoxic gasses, is a reduction in amplitude and an increase in frequency, followed by a slowing of the EEG without delta activity, and then electrical silence (10,22). However, more recent data from rats that had been centrifuged to the point of G-LOC have shown increased delta activity throughout the $+G_z$ exposure when it is expressed as a percentage of total EEG power (8). Also, cerebral ischemia induced by embolism in rats produced almost identical results (14). Human exposure to hypoxia usually results in increased alpha wave activity which progresses to delta activity just before unconsciousness during ischemic hypoxia (3,16) or hypoxic hypoxia (7,17). Some of the delta and theta activity throughout the $+G_z$ exposure in this study (Fig. 4) could be the result of contamination by skeletal muscle activity and eye movement (2). It was very common for the animals to struggle as the centrifuge began to turn; nystagmus was persistent until the time of LOC. Moreover, the increased power of all four band

widths at the beginning of recovery was very closely associated with the convulsion period; and was most likely motor activity.

These data were compared with previous human G-LOC data (27). G-LOC induction time was somewhat longer in the baboon (12 s) than in man (7 s). There are several possible reasons for this difference: 1) The human centrifuge subjects were instructed to relax during the $+G_z$ exposure, whereas the baboons usually became excited and generally struggled during the $+G_z$ exposure, thus potentially raising their blood pressure and augmenting their $+G_z$ tolerance. 2) The baboon has a shorter eye-to-heart distance than the human, which provides an anatomic advantage. 3) There is the possibility that the end-point for baboon LOC (near isoelectric EEG) requires more time at $+G_z$ than the end-point for man (gaze fixation, eye rotation and head drop). The absolute incapacitation time was approximately the same for man (12 s) and baboon (11 s); it was measured from the time of $+G_z$ reduction to the time when convulsions stopped and the head was erect. Relative incapacitation time (performance recovery time minus convulsion time) was also approximately the same for the baboon (11 s) and man (12 s).

Convulsions during G-LOC in the baboon lasted nearly twice as long as that reported in the human (25), possibly because of the extended time at LOC in the baboon. The tonic spasms, or hyperextension, and the convulsive activity observed in humans or animals during LOC are considered to be the result of loss of control by the cerebrum over the reticular formation (18). The electrical activity of the reticular activation system (RAS) has been shown to increase during LOC when cerebral activity has stopped (6). When the reticular formation has been experimentally destroyed, anoxic spasms or seizures do not occur (24). In this study tonic spasms, such as hyperextension of the head, occurred during the early phase of LOC, whereas convulsions did not occur until cerebral blood flow was reestablished during and after offset of $+G_z$ while the animal was still unconscious. The same observation has been reported in humans after G-LOC (25), after neck occlusion (19) and after cardiac arrest (9).

We anticipated that multiple episodes of G-LOC would shorten performance recovery time following G-LOC due to acclimation or to a learning process, as suggested previously (4,28). This was the case in only one animal. Either the hypothesis is incorrect or other factors are influencing the results. For example, we noted that resting HR increased with increased number of $+G_z$ exposures over time (Fig. 5). Probably, the increase in resting HR is a manifestation of an increased anxiety toward the pending $+G_z$ exposure. This anxiety could possibly override the will to perform before and after the G-LOC episode.

There has been concern, especially in humans, that multiple exposures to LOC may result in brain damage. However, these animals remained alert, healthy, and active during the course of this study, similar to observations made in man (15,19,25,28).

The data clearly demonstrate the utility of the baboon as an animal model for G-LOC research. However, there are limitations to its usefulness: 1) Our use of an isoelectric EEG as an end-point was possibly a delayed determination compared to eye fixation or head drop in man. 2) Time to LOC was longer, as mentioned earlier, because of a shorter eye-to-heart hydrostatic column than man, and because the animal could not be trained to relax during G-onset. 3) Of

course, post recovery interviews regarding subjective and psychological (including dreams) observations and occurrences are not possible, nor are complex post-G tasks to measure the levels of performance recovery. Some of these data have been previously presented and published as part of a Panel on Deliberate G-LOC (5).

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TABLE 1. G-LOC DATA

	Mean	+/- SD	Range	n
Time to G-LOC	11.5 s	3.2	7.8-30.6 s	92
Duration of LOC	8.2 s	3.3	2.8-23.4 s	92
EEG recovery time	3.7 s	1.4	1.4-9.6 s	92
$+G_z$ level at EEG recovery	2.0 G	1.1	1.0-8.0 G	94
Convulsion time	6.9 s	2.5	3.6-19.6 s	87
Performance recovery time	17.8 s	.7	4.8-41.2 s	87
Resting HR before G-LOC*	150.2 b/m	21.9	76-210 b/m	327
HR during control performance task	173.4 b/m	23.4	110-233 b/m	264
Peak HR during G-LOC episode	226.2 b/m	18.5	137-261 b/m	280

G-LOC = $+G_z$-induced loss of consciousness

* Does not include resting HR before the 4 $+G_z$ familiarization exposure of each exposure day, which is presented in Figure 5.

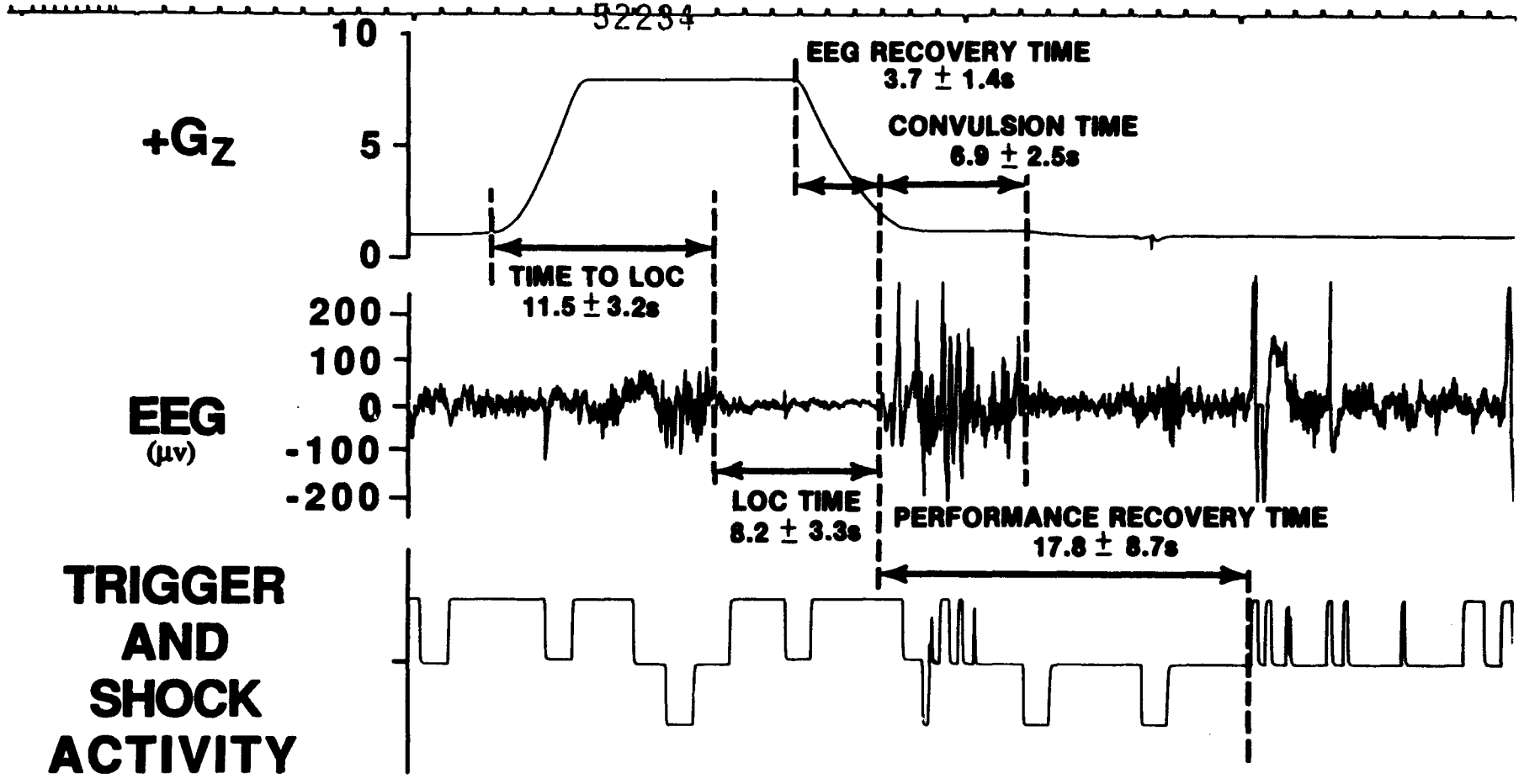

Figure 1. Average response of the baboon (n=7) to G-LOC. The animals were exposed to a rapid onset (4 or 6 G/s) exposure of 8 $+G_z$ until LOC was identified by a near-isoelectric EEG signal followed by a return to 1 $+G_z$. Performance recovery was measured from the return of EEG activity to the time of shock avoidance. Shock avoidance is indicated by the appearance of trigger pull activity above the baseline, whereas, a shock is indicated by the 1-s square wave pulses below the baseline. Time in 1-s intervals is illustrated at the top of the figure.

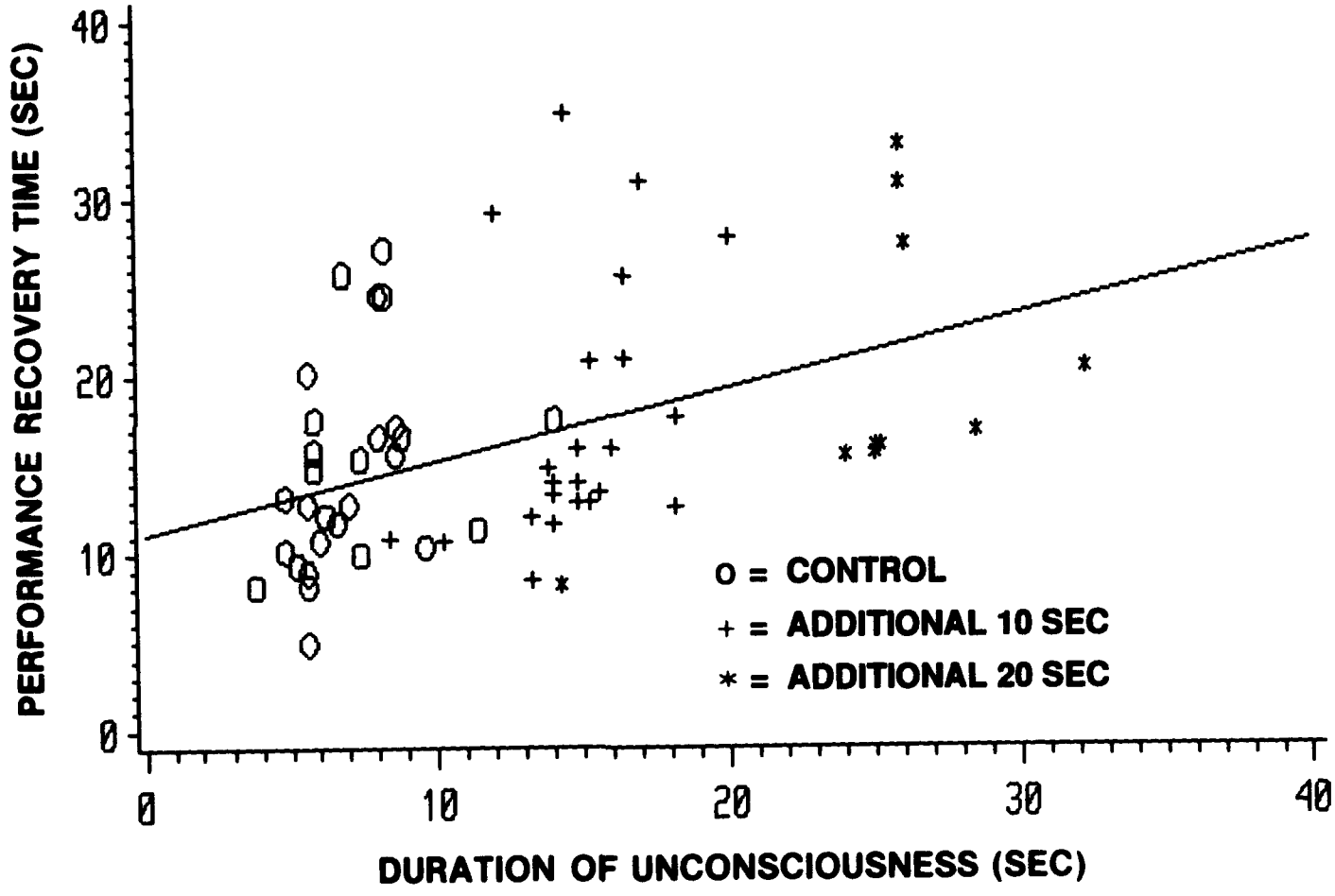

Figure 2. Comparison of performance recovery time vs. extended time at LOC. Data are matched samples of control and 10-s extended time or control and 20-s extended time taken from the same animal on the same day. ANOVA showed that the data at 20-s extended time are significantly different than control ($p<.001$). Mean performance recovery time for control, 10-s extended, time and 20-s extended time was 14.3 s, 17.4 s and 20.0 s, respectively.

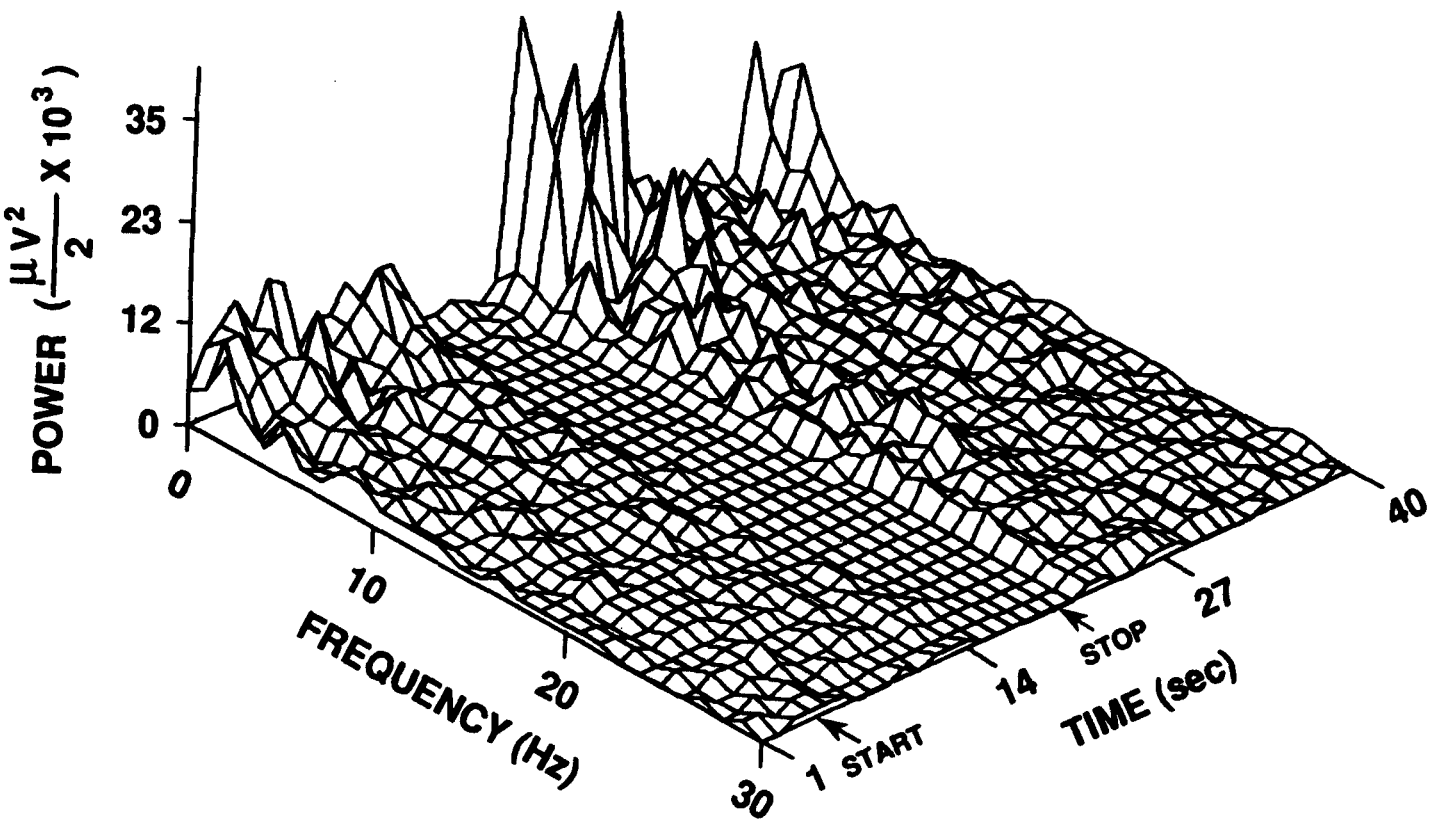

Figure 3. Serial power spectral analysis of EEG during a G-LOC episode from one animal. The occurrence of LOC is represented by the near-isoelectric trough at about 15 s. The start and stop arrows indicate the beginning and end of the 8 +G_z profile (see Fig. 1 for profile).

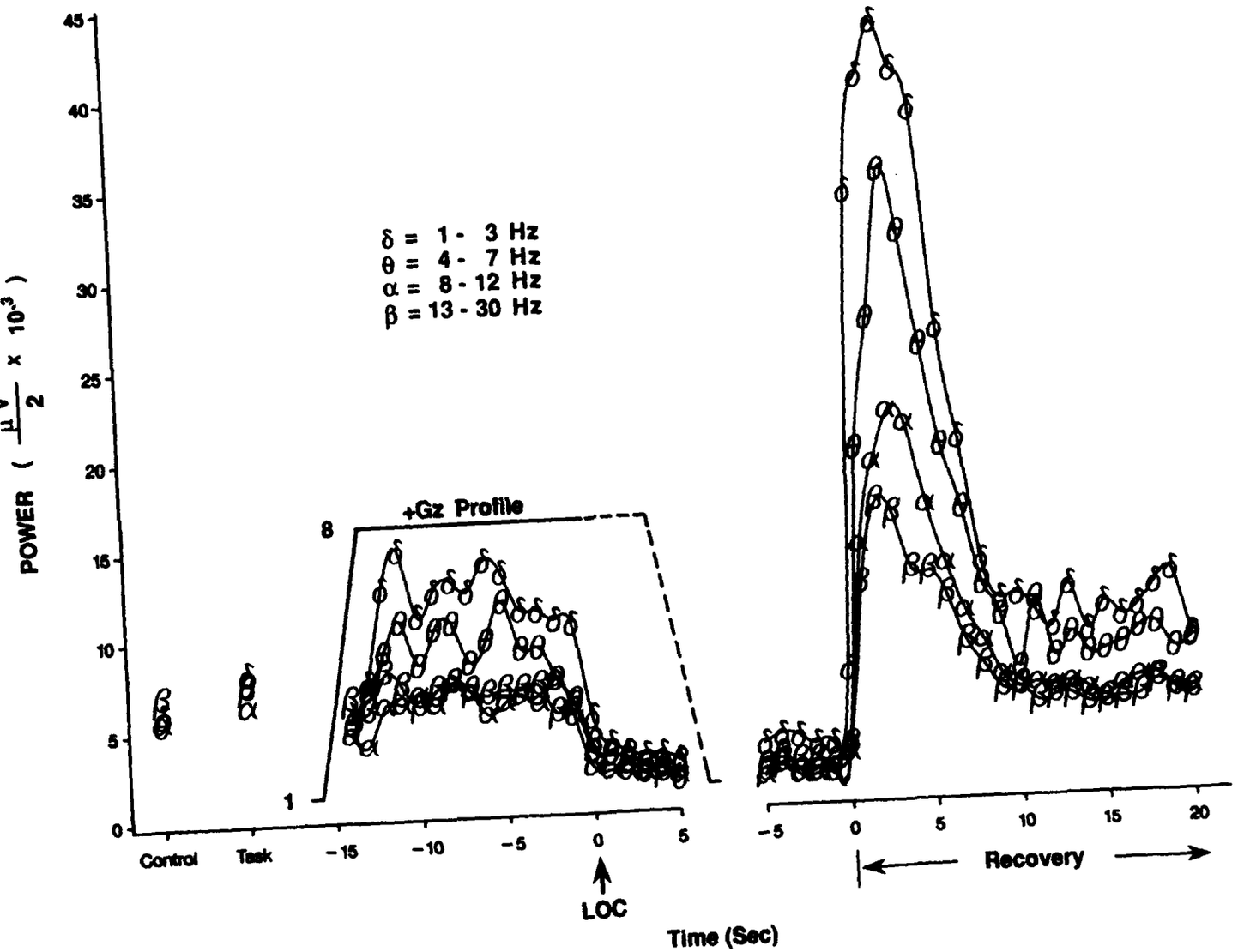

Figure 4. Serial median power spectral data of the four EEG component waveforms averaged across all animal (n=7) at 1-s intervals. Control data was taken before +G_z and before performance task was started. Task data was taken during performance of the task before +G_z. The data during the +G_z profile were time adjusted for LOC and the recovery data were time adjusted from the beginning of EEG activity following G-LOC.

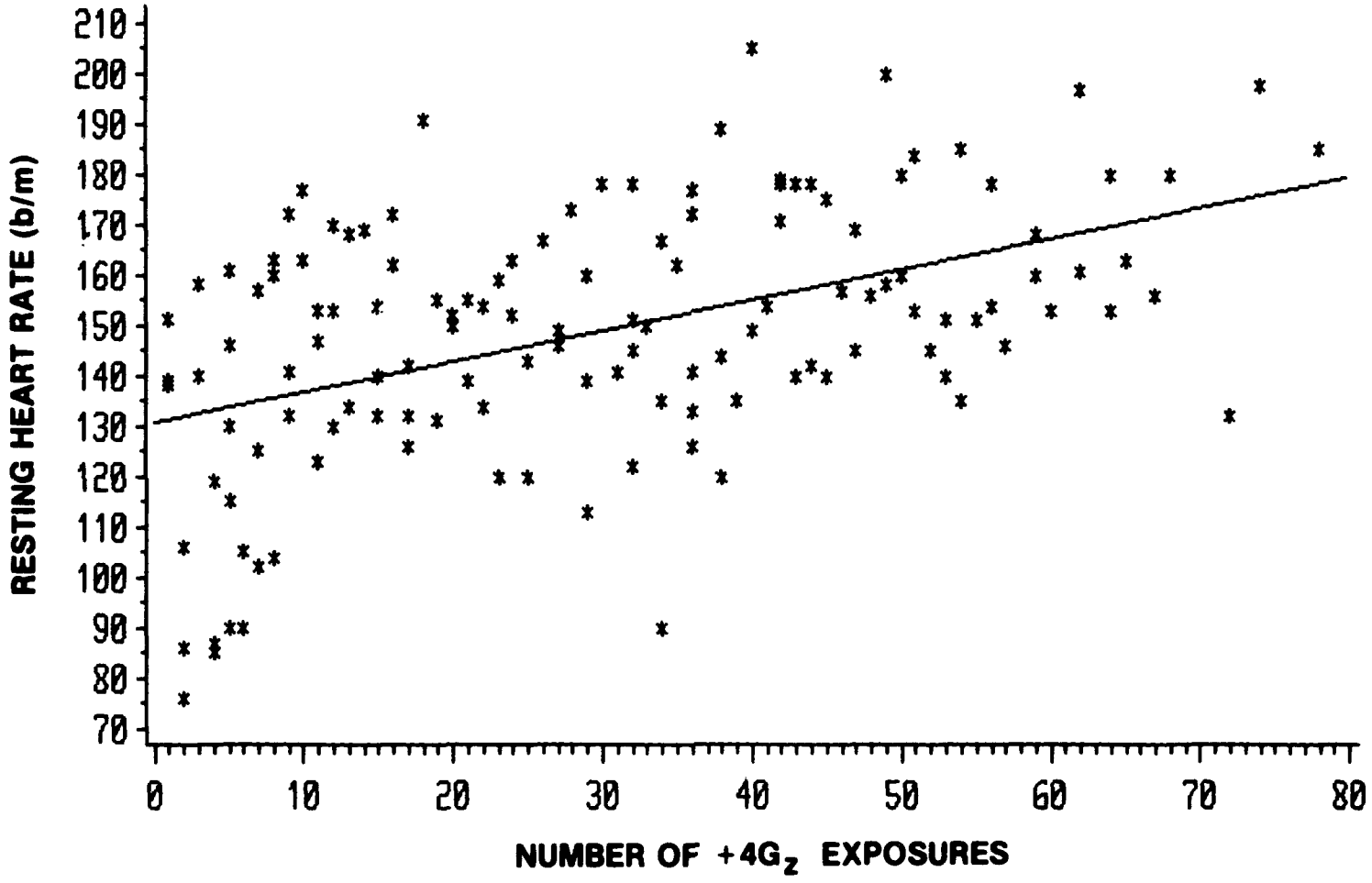

Figure 5. **Resting heart rate before the initial 4 +G_z exposure and before any performance task activity. Data includes multiple samples from six animals on different exposure days.**