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HUMAN RESPONSE TO HYPOXIA-MOTION SICKNESS STRESS AS A PREDICTOR OF THE SPACE SICKNESS SYNDROME

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Introduction

With the onset of manned space travel, allowing unconstrained mobility in a zero-gravity (zero-g) environment, man has developed a space sickness syndrome (Ref: 1,2). With the prospect of frequent space shuttle missions just on the horizon, a method of screening crews and developing a cure for this syndrome seems a paramount objective.

Presently it is thought that a sensory conflict between the visual, otholithic and proprioceptor inputs in zero-g creates an imbalance that the brain cannot resolve (Ref: 1,2). This imbalance is thought to cause space sickness. However, this theory fails to explain the variable and ambiguous motion sickness presentations seen in zero-g. It also has not yet offered a reliable way of predicting, in earth gravity, individual susceptibility to this syndrome.

Previous earthbound studies have shown a positive correlation between (Motion Sickness Susceptibility) MSS and states of hypoxia (Ref: 3,5,6). A review of the literature failed to reveal any studies on hyperoxia and motion sickness.

In zero-g, a relative hypoxia produced by various physiological alterations within the body may also contribute to the etiology of space sickness. Subjective reports and objective measurements of our Apollo and Skylab astronauts revealed facial edema, nasal congestion, jugular distension, fluid and electrolyte shifts, and a decreased lower limb volume. Based on these reports, a decreased cerebral blood flow-oxygenation theory is hypothesized.

Upon insertion into Earth orbit and the weightless environment, the human body experiences a change in intravascular and extravascular pressure dynamics resulting in: (1) a progressive cephalad fluid shift, (2) electrolyte alterations, and (3) fluid overload in the circulatory system. These alterations are hypothesized to decrease blood flow in the cranial circulation creating cellular hypoxia and leading to acid-base and metabolic disturbances. These hypoxia-metabolic alterations are theorized to abnormally sensitize the vestibular system and/or it's central modulating centers to any type of motion sickness stimuli.

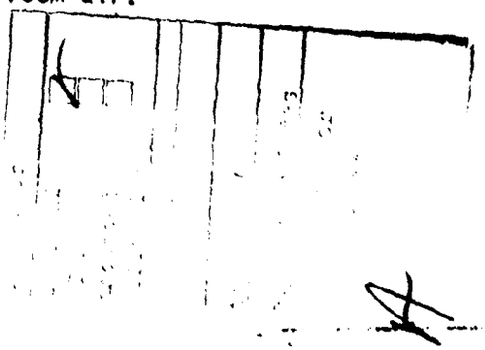
Running parallel to the hemodynamic changes is a breakdown in visual-otholithic processing resulting from the altered reference frame seen in zero-g. Accommodating to this environment imposes a significant learning task on the crewmember, increasing vestibular and central nervous system oxygen demand, which further accentuates the already hypothesized stagnant hypoxia. These factors all combine to give an inappropriate vestibular response. The autonomic discharge resulting from this abnormally increased vestibular output is characterized by nausea, vomiting, anorexia, and lethargy. From past experiences, all symptoms have usually abated in two to five days, which, not surprisingly, coincides with the time required for the body to stabilize with respect to fluid shifts, electrolyte balance, and total circulatory volume. This normalization of parameters, i.e., blood flow, hypoxia-acidosis, and vestibular processing, leads to a cessation of symptoms and a crewmember now resistant to future attacks during the mission. To date, return to earth-gravity has not resulted in motion sickness symptoms. The hemodynamic changes seen on return to one-g are not postulated to lower the vestibular threshold. However, the crewmembers do require an accommodation period of from seven to ten days to integrate their visual reference with a one-gravity loaded vestibular system.

A preliminary hypoxia-motion sickness study was conducted at the NASA-Johnson Space Center (JSC) Neurophysiology Laboratory, while the author was completing phase I of the USAF Aerospace Medicine Residency. Barbara T. Sauerland, M.D., supplied medical moitoring assistance during the test runs.

Materials and Methods

The human volunteers for this experiment consisted of five male and five female subjects from NASA-JSC. Subject selection criteria used were a demonstration of normal vestibular function, a recent Air Force Class III physical exam, and a negative smoking history. The vestibular function battery included a motion experience questionnaire, cupulogram, audiogram, and standard rail test. Following these measurements, all subjects were tested for baseline susceptibility with from three to five MSS trials until a repeatable endpoint of symptoms and CSSI score (Coriolis Sickness Susceptibility Index) was attained.

A skylab M-131 rotating chair was used for all MSS testing. Modification was necessary to allow installation of the compressed gas cylinders and various amplifiers. Variable head constraints were mounted to standardize the amount of head excursion during the test procedure. The level of motion sickness was scored using the method developed by Miller and Graybiel (Ref: 1,4). The level of motion sickness used as the endpoint in all baseline and test runs was M-IIA (Ref: 4). Following baseline measurements, subjects were randomly exposed, two to four runs each, to compressed air, nitrogen-enriched room air, and oxygen-enriched room air.



The test gases were supplied by a rotating chair mounted cylinder, through a bubble-jet humidifier and then to an adjustable venturi-type oxygen mask. This mask allowed adjustment of the oxygen-nitrogen ratio in response to individual variations in O_2 saturation. Compressed air was used as the normoxia control gas and was delivered by the same mask as the other gases.

A Hewlett-Packard eight wavelength ear oximeter was used to continuously monitor blood oxygen saturation during the thirty minute pre-breathing-stabilization period prior to all chair runs. The target blood oxygen saturation value for each hypoxia run was 80%, which is equivalent to a pressure altitude of 15,000 feet (429 torr) (assuming a constant blood pH of 7.40). A 50% oxygen mixture was supplied for the hyperoxia runs, giving O_2 saturations of 98-99%. The oximeter was used in a feedback loop to vary the oxygen-nitrogen ratio and so maintain the blood O_2 saturation at the target values.

Results

In all subjects, baseline measurements were taken until a stable pattern of motion sickness symptoms were elicited. The target range of head movements tolerated was between 40 and 100, to allow for a detectable change, given that the automatic rotating chair sequencer stops the run at 150 head movements regardless of the RPM chosen.

The mean CSSI score tolerated during the hypoxia runs decreased from the normoxia runs as mean = -2.91 with S.D. = $\pm$2.13 ($p<.01$). All but two of the subjects showed a decrease in mean CSSI score compared to their normoxia runs. The mean percent decrease in head movements tolerated ranged from 0% to 50%, with a population mean of 30% $\pm$ 19%.

The mean CSSI score tolerated during the hyperoxia runs increased from the normoxia condition as mean = 6.38 with S.D. = $\pm$2.13 ($p<.01$). All but two percent increase in head movements tolerated ranged from +12% to 233% with a mean of 60% $\pm$ 66%. All subjects showed an increase in CSSI score with the hyperoxic conditions.

Discussion

The change observed during the hypoxia runs supports the hypothesis that hypoxic states lower human tolerance to vestibular stimuli (Ref: 3,5,6). The frequency of occurrence of symptoms in this study is encouraging since little change in presentation was found between the normoxia and hypoxia runs. Large variations in the symptom presentations would have indicated that hypoxia symptoms were being misread as motion sickness symptoms, thereby skewing the results.

From these results it is obvious that a significant research effort is needed to futher delineate and verify the various aspects of this theory. Additional human investigations in one-g and zero-g are needed to verify that the proposed hemodynamic changes and oxygenation parameters potentiates

or causes space sickness. Animal studies will be needed to quantify the type and amount of circulatory disruption and hypoxia caused by zero-g exposure.

If hypoxia proves to be implicated in the space sickness syndrome, it may be possible to use this hypoxia-motion sickness stress response as an indicator (in earth gravity) of a person's susceptibility to zero-g space sickness.

Summary

1. It is expected that a number of Space Shuttle astronauts will experience motion sickness to a degree that mission productivity may be compromised.

2. It is known that present models are inadequate for prediciting a given person's susceptibility to space motion sickness.

3. It is known that various physiological changes take place in zero-g, i.e., fluid shifts and attendant cardiovascular changes, without knowing their effects on cerebral and vestibular circulation.

4. It is known that hypoxia in earth gravity can affect susceptibility to motion sickness.

5. It is hypothesized that circulatory changes in zero-g result in a type of stagnant hypoxia and that the vestibular apparatus and/or its central connections respond to it a functionally negative manner, i.e., space sickness.

6. It may be possible to use the hypoxia-motion sickness stress response in one-g to predict individual susceptibility to space sickness.

7. It may be possible to prevent or alleviate space motion sickness by altering tissue perfusion and/or oxygenation.

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