Limitation of ocular motility and pupillary dilatation in human beings during positive acceleration

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During positive acceleration in man, a stage is reached at which there is a limitation of ocular motility. These limitations can be overcome by voluntary effort, but the superseding movements are ataxic. The lower motor neurons to the extraocular muscles are not involved in LOMA. The pupils dilate as the visual fields constrict during positive acceleration and reach a maximum with loss of central vision. The optokinetic reflex does not continue during black-out. However, a form of horizontal nystagmoid movement of the globes may persist in eyes previously stimulated by the optokinetic drum. Vertical nystagmus is observed during high rates of change of positive acceleration. With a modified skin diver's mask, 30 mm. Hg of negative pressure was applied to the orbit, and under these conditions vision was restored, ataxic voluntary movements became coordinated, optokinetic reflexes were restored to their original frequency, and the pupils remained partially dilated. The pupillary dilatation which accompanies blackout is prevented by the local application of pilocarpine and is unaffected by morphine. The observation of LOMA is a useful objective end point for the evaluation of response to positive acceleration. The observation of pupillary dilatation is a useful quantitative sign for evaluation of response to positive acceleration.

The loss of vision, or blackout, which aviators experience during turns or pull-out from a dive can also be produced in human subjects exposed to acceleration on the centrifuge. Since the maintenance of normal vision is of paramount importance to the aviator, its deterioration under this form of stress has been studied extensively. Most investigations in this area have been concerned with the sensory aspects, and little attention has been paid to oculomotor function and pupillary control. An objective test for the determination of the functional state of the central nervous system during positive acceleration was evolved on the basis of limitation of ocular motility, hereafter referred to as LOMA, which occurs in association with the loss of vision and pupillary dilatation. The mechanisms involved in LOMA and pupillary dilatation comprise the subject of this report.

Review of the literature

The general effects of acceleration have been described by Armstrong.1 That positive acceleration produces a decreased circulation to the head and eyes is amply documented by Lambert and Wood.2 Application of positive or negative pressure to the orbits has been shown by Lambert3 to
change the blackout threshold. These findings have been confirmed recently by Howard. They have been further amplified by Keighley, Clark, and Drury who studied FFF (flicker fusion frequency) with and without suction on the orbits. There was no change in the FFF in the unprotected eye when the G level was increased to the point of beginning visual impairment (2.5 G to 3.2 G). However, with the application of negative pressure to the eyes and higher G levels (3.4 G to 4.8 G) and the same state of retinal functioning, the FFF was slightly but significantly reduced and the authors concluded that under these circumstances the circulation in the eyes was the same (as at lower G levels, but unprotected), but the circulation in the head was different and the latter accounted for changes in the FFF. In other words, reduced circulation in extraocular parts of the visual pathway accounted for these findings. In addition, it has been established that decreased retinal circulation is the primary cause of the loss of vision or blackout which occurs during positive acceleration and that this retinal hypoxia does not primarily involve the pericentral layer.

In 1943, Rossen and his associates reported an experiment in which they acutely arrested the cerebral circulation in man with a cervical cuff and "the earliest most constant objective reaction to acute anoxia of the human brain was fixation of the eyes. This was tested by having the subject move his eyes rhythmically from side to side in the horizontal plane while they followed the moving finger of the examiner or a freely swinging pendulum. In the usual subject, after 5 or 6 seconds of cerebral anoxia, the eyes fixed suddenly in the midline and the subject was incapable of moving the eyes, although he was still conscious (loss of consciousness occurred ½ to 1 second after fixation of the eyes). The subject stated afterward that he tried to follow the examiner's finger and could see it moving but was unable to move his eyes." The greatest number of subjects showed fixation of the eyes 5 to 5.5 seconds after application of pressure to the cuff, and unconsciousness appeared in 6 to 6.5 seconds. There was considerable variation among the subjects with reference to the appearance of these signs, but the resistance to acute anoxia was fairly consistent in the same person at different times. Besides the fixation of the eyes, many subjects experienced a rapid narrowing of the visual fluids, blurring, and, finally, complete loss of vision. A number stated that they were unable to see but could still hear and were conscious. A review of motion picture records taken by Kydd and Stoll of monkeys undergoing positive acceleration revealed that, in addition to the "end of blinking" which they observed, fixation of the eyes occurred concomitantly.

Discussions of the voluntary and reflex pathways of ocular motility and pupillary mechanisms, as well as their importance to the understanding of the normal and diseased states, are presented in the textbooks of Adler, Walsh, Duke-Elder, Cogan, and Kestenbaum.

Methods

A television camera and a 16 mm. cine-camera were mounted in front of the subjects to permit observation and to record their reactions. Since cardiovascular compensatory reflexes are not effective until 6 to 8 seconds after the onset of acceleration, two different G-time profiles were used in this study. In one, the accelerations were gradually increased at a rate of approximately 4 seconds per 1 G to allow the reflexes previously mentioned to become effective before the desired G level was reached, and, in the other, the acceleration levels were attained in less than 10 seconds before these reflexes could operate. Durations of 15 to 60 seconds at peak G were employed in various phases of the experiment.

Amber test lights were located 23 degrees to either side of the midsagittal plane at the subject's eye level. Tests were likewise conducted with the test light positions
varied to 65 degrees. These were alternately illuminated at random intervals varying from 0.5 to 1.5 seconds. The subject was instructed to fix on the light that was turned on and, when it was extinguished, to transfer his gaze immediately to the opposite light. He was to continue this procedure as long as possible. A negative pressure 30 mm. Hg less than ambient was produced by placing a skin diver’s mask over the region of the orbits. The mask was connected to a vacuum pump. The external nares were occluded by means of adhesive tape.

The optokinetic reflex was studied by means of a drum, 30 cm. in height and 25 cm. in diameter. The width of stripes was varied from 1.8 cm. to 3.7 cm. The drum, which was rotated at 10 r.p.m., was mounted on a vertical axis approximately 56 cm. in front of the subject. The so-called following reflex was studied by random movements of the observer’s finger as the object of regard approximately 30 cm. in front of the subject.

The cranial nerves were tested as follows: C3, 4, and 6, by rotations of the globe in all cardinal directions; C5, motor (mandibular branch), by movements of the mouth, tongue, and cheek; C7, by facial grimaces; and C8, by response to verbal commands. The pupillary responses (C3) and the visual responses (C2) were investigated and will be described more fully.

Results

Fifty subjects, both male and female, ranging in age from 18 to 55 years, participated in a total of more than 350 centrifuge runs. Of this number, 8 were trained centrifuge subjects on the staff of the Aviation Medical Acceleration Laboratory (AMAL). Each subject demonstrated LOMA with his eyes coming to rest in the primary position. The occurrence of LOMA appeared to be related to the subjective sensations of gray-out and blackout and occurred at a G level between them. The magnitude of acceleration required to produce LOMA varied with individual subjects from 3.5 to 7.0 G. It was also observed that the blink rate decreased markedly or stopped altogether after LOMA occurred.

After the onset of LOMA, it was found that the trained centrifuge subjects were physically capable of voluntarily moving their eyes on all axes of motion if sufficient effort was made. However, the movements were ataxic in nature. By making an extra effort the subjects could open and close their eyelids and control their facial muscles. It would appear from this that the cranial nerves tested were functional. It must be stressed, however, that additional volition was required to produce these movements. The optokinetic reflex disappeared after LOMA, although irregular, horizontal, and intermittent movement persisted in some subjects. Vertical nystagmus was observed in several subjects just prior to loss of consciousness, as well as during the deceleration phase of the centrifuge run.

Concomitant with the subjective loss of peripheral vision, pupillary dilatation began. It reached a maximum at blackout, at which point direct and consensual pupillary reflexes were absent. In three subjects, miosis was produced unilaterally with locally applied pilocarpine. The pupils failed to dilate under accelerations which produced blackout and maximal dilatation of the fellow (unmedicated) eye. In three other subjects, miosis was produced with parenterally administered morphine. In each of these subjects, the pupil dilated widely at the level of acceleration where blackout occurred. Light reflexes were also absent.

Negative pressure applied to the orbits of blacked-out subjects produced the following changes: (1) Visual fields cleared with restoration of vision; (2) LOMA was reversed and eye movements ceased to be ataxic and became smooth and coordinated; (3) the optokinetic reflex was restored to the original pre-blackout frequency and continued as long as the subject kept his eyes on
the drum; (4) the following reflex which was lost during blackout was restored; (5) the pupillary diameter decreased somewhat.

Discussion

During positive acceleration, there is a diminished flow of blood to all regions above the heart. This is manifested by the commonly described blackout which has been extensively investigated. Little attention has been paid to ocular motility during acceleration on the centrifuge because the standard tests of vision have been performed with the eyes in the primary position.

In this experiment the limitation of ocular motility which was observed during positive acceleration closely parallels similar observations reported in other forms of anoxia. Rossen and his associates observed this to be a constant end point in their studies with a cervical cuff as summarized in the review of literature. Loss of ocular motility with fixation in the primary position has also been an accepted sign for evaluating the depth of surgical anesthesia. Bender and his associates reported an experiment in which they stimulated the paramedian portion of the diencephalon in monkeys and cats by the Horsley-Clarke stereotaxic technique and found that a midposition of the eyes and pupillary dilatation resulted. Stimulation of the brain stem, the frontal and occiptal areas of the cortex, and the cerebellum sometimes produced similar results. In a later paper, Bender speculated that there must be an eye-centering area in the central nervous system. He pointed out that, although much attention has been given to the physiologic and clinical significance of the slow component in various types of nystagmus, little attention has been directed to the quick component which returns the eyes toward the primary position. Yet it must operate or the eyes would remain deviated as they do in some types of cerebral disease. Our findings lend credence to this concept, though we have no evidence to indicate where such an eye-centering area may be located.

Some subjects were able to overcome the tendency toward ocular fixation, but the resulting voluntary movements were ataxic. The most probable explanation for this ataxia is that a loss of sensory feedback from the retina occurs. The resulting dys-synergia may be a type of increased scanning or bracketing which operates in any shift of gaze from one object of regard to another. Physiologically, the extraocular muscles served the function of maintaining the eyes in a fixed position with reference to the environment. Although the relative weight of the eyes was increased in proportion to the level of acceleration, there was no evidence to suggest that the ataxia was a result of this increased load on the extraocular muscles. This was substantiated by the fact that the ocular ataxia disappeared during the application of negative pressure while the relative weight of the eyes remained constant at a given G level.

Except for the optic pathways, there was no evidence of cranial nerve dysfunction accompanying LOMA. Hence, the site of malfunction is not likely to be found in the brain stem and areas caudal to it. The constancy of pupillary dilatation concomitant with LOMA is in keeping with the findings of Rossen and Bender. This suggests that the interruption of the cerebral pupillary dilatation pathways likewise may be responsible for LOMA. Dilatation of the pupils sometimes accompanies paralysis of the extraocular muscles as in Parinaud's syndrome. The direct and consensual pupillary reflexes could be elicited after LOMA occurred, but not after complete blackout occurred. This observation is not in agreement with the findings of others and warrants further investigation. Since the pupils remained dilated after negative pressure was applied to the globes, the subsequent return of vision serves to mitigate against the concept that the "blacked-out" condition of the retina was entirely responsible for the pupillary change.

Tests utilizing the optokinetic drum revealed that a nystagmoid movement in some cases persisted after blackout. This
phenomenon may be a continuation of the motor activity in which the eyes were engaged just prior to blackout, or it may be a form of modified optokinetic reflex.

The vertical nystagmus is considered to be a separate entity. It was commonly observed during periods of increasing or decreasing acceleration, and, subjectively, “head-over-heels” vertigo was experienced at the same time. A paroxysmal twitching of the lips was noted repeatedly in one subject following LOMA. This was of a nature commonly associated with a Jacksonian seizure and lends support to the concept of cerebrocortical dysfunction during LOMA.

The studies with the negative-pressure mask verified the results reported by Lambert3 and Howard.4 It has been demonstrated that the restoration of central and peripheral vision after blackout by the application of negative pressure during acceleration is accompanied by a restoration of circulation through the retina. At this time voluntary movements of the eyes became coordinated, and this is attributed to increased sensory feedback within the visual apparatus. The same mechanism would account for the return of the optokinetic reflex. Since the pupils remain partially dilated, despite the negative pressure, it may be presumed that this phenomenon is based upon a cerebral dysfunction (i.e., in the eye-centering area15,16) which would not be influenced by local changes in the retina.

LOMA and pupillary dilatation, in many ways, parallel the course of events during induction of general anesthesia. As Lundy19 has pointed out, “Cessation of eyeball activity marks the passage of anesthesia into the second plane of the third stage.” Concurrently, the normal pupil begins to dilate. Guedel20 states, “We must conclude that paralytic dilatation of the pupil is a measure of the degree of hypoxia incidental to the anesthesia; not a measure of the depth of anesthesia from the anesthetic agent per se and that its value as a ‘sign’ of anesthesia is limited to such an interpretation.” During physiologic sleep, when vision is absent but oxygenation of the cerebral cortex is normal, the pupils remain small. The observations in this experiment that the pupils remain dilated with or without vision suggests that the dilatation may be a result of cortical hypoxia.

A mechanism for LOMA may therefore be postulated. The cerebral hypoxia produced by positive acceleration was observed to produce pupillary dilatation and ocular fixation. The stimulations in the diencephalon, reported by Bender,15,16 also produced pupillary dilatation and eye centering. Therefore, an explanation of LOMA may be that the cerebral hypoxia produces a dysfunction in the normal oculomotor and pupillary control systems.

REFERENCES
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