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U. S. NAVAL AIR DEVELOPMENT CENTER
JOHNSVILLE, PENNSYLVANIA

Aviation Medical Acceleration Laboratory

NADC-MA-6140

12 December 1961

Limitation of Ocular Motility and Pupillary
Dilatation in Humans Due to Positive Acceleration

Bureau of Medicine and Surgery
Subtask MR005.13-0005.8 Report No. 1



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SUMMARY

In a search for a reproducible and objective endpoint for measurement of reaction to positive acceleration, it was observed that volitional ocular motility disappeared at approximately the same magnitude of acceleration that produced a loss of peripheral vision. Further studies designed to elucidate the mechanism of this phenomenon were carried out on trained centrifuge subjects and it was disclosed that these subjects could, in fact, with greater volition, rotate their eyes after losing peripheral vision but in an ataxic manner. The optokinetic reflex seemed to disappear concomitantly with limitation of ocular motility (LOMA). The ability to follow a moving target was also lost after peripheral vision failed. Progressive pupillary dilatation was observed, beginning at the time that the peripheral vision was lost and became maximal after central vision failed. The above phenomena were also tested while 30-35 mm Hg negative pressure was applied over the eyes by use of a modified underwater swimmer's mask. With this technique, vision after blackout (loss of central and peripheral vision) during acceleration was restored almost to normal. The volitional eye movements likewise returned to normal. The optokinetic reflex was likewise restored as was the target following reflex. The pupils, however, remained dilated. On the basis of these findings, it is concluded that there is a cerebral cortical dysfunction which occurs during exposure to positive acceleration at levels which cause "blackout". This is in addition to the commonly acknowledged retinal failure.

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INTRODUCTION

The loss of vision or blackout which aviators experience during turns or pull out from a dive can also be produced in subjects exposed to acceleration on the human centrifuge. Since the maintenance of normal vision is of paramount importance to the aviator, its deterioration under this form of stress has been extensively studied. 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 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 Lambert (3) to change the threshold to blackout. These findings have been confirmed recently by Howard (4). They have been further amplified by Keighley, Clark and Drury (5) 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 - 3.2 G). However, with the application of negative pressure to the eyes and higher G levels (3.4 - 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 head was different and the latter accounted for changes in the FFF. In other words, reduced circulation in extra-ocular 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 (6) and that this retinal hypoxia does not primarily involve the pericipient layer (7).

In 1943, Rossen and his associates (8) 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

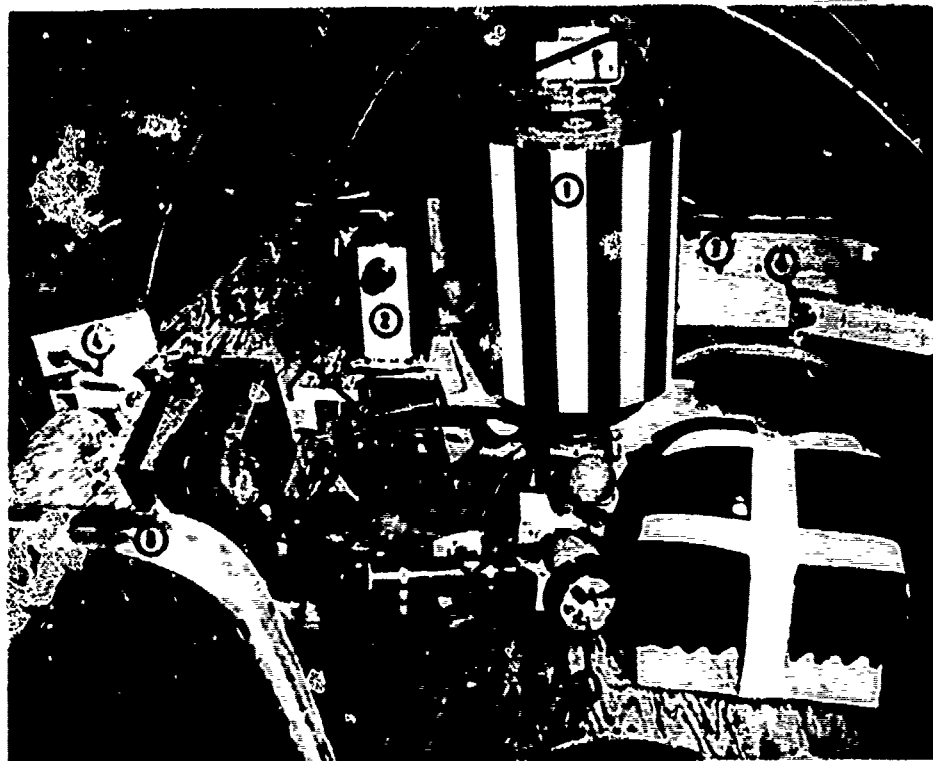
fixed suddenly in the midline and the subject was incapable of moving the eyes, although he was still conscious (loss of consciousness occurred 1/2 to 1 second after fixation of the eyes). The subject stated afterward that he tried to follow the examiner's finger and could see it moving but was unable 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 for the same person at different times. Besides the fixation of the eyes, many subjects experienced a rapid narrowing of the visual fields, 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 (9) of monkeys undergoing positive acceleration revealed that, in addition to the "end of blinking" observed by them, concomitantly a fixation of the eyes occurred.

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 (10), Walsh (11), Duke-Elder (12), Cogan (13), and Kestenbaum (14).

METHODS

Two different G-time profiles were used in this study since cardiovascular compensatory reflexes are not effective until 6 to 8 seconds after the onset of acceleration. In one, the accelerations were gradually increased at a rate of approximately 4 sec/1 G to allow the above mentioned reflexes 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.

A TV camera and a 16 mm cine camera were mounted in front of the subjects to permit observation and to record their reactions (Figure 1). Amber test lights were located 23° to either side of the mid-sagittal plane at the subject's eye level. Similar tests were conducted with the test light positions varied to 65°. These were alternately illuminated at random intervals varying from 0.5 to 1.5 seconds. The subject was instructed to fix on the illuminated light and when it was extinguished, he was to transfer his gaze immediately to the opposite light. The subject was to continue this procedure as long as possible. A negative pressure of 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. External nares were occluded by means of adhesive tape.



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|----------------------------|-------------------------------|
| 1. Optico-Kinetic Drum | 6. Intercommunications System |
| 2. Television Camera | Speaker/Microphone |
| 3. 16 mm Movie Camera | 7. Supports for Canvas |
| 4. Lateral Fixation Lights | Windscreen |
| 5. Electronic Flash Unit | 8. Subject's Headrest |

Figure 1. Measuring instruments mounted in carriage on centrifuge as viewed by subject.

The optokinetic reflex was studied by use 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 was rotated at 10 rpm. It was mounted on a vertical axis, approximately 56 cm in front of the subject (Figure 1). 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. Bitemporal skin electrodes were used on some subjects to record the changes in corneo-retinal potentials which indicated the cessation of tracking of the lights or drum. 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, C8 - by response to verbal commands. The pupillary responses (of C3) and the visual responses (C2) were investigated and will be described more fully below.

RESULTS

Sixty subjects, male and female, ranging from 18 to 55 years of age, participated in a total of over 400 centrifuge runs. Of this number 10 were trained centrifuge subjects of the Aviation Medical Acceleration Laboratory (AMAL) staff. The others were essentially inexperienced. 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 "grayout" 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 some 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 means of extra effort the eyelids could be opened and closed and the musculature of the face controlled. 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 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 3 subjects, miosis was produced unilaterally with locally applied pilocarpine. These pupils failed to

dilate under accelerations which produced blackout and maximal dilatation of the fellow (unmedicated) eye. In three other subjects, miosis was produced by parenterally administered morphine. In each of these, 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. Clearing of visual fields 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.
6. When one of us (TDD) viewed the retinal vessels of a subject ophthalmoscopically during accelerations which produced blackout, application of negative pressure to the face mask was observed to cause immediate filling of the emptied retinal vessels.

DISCUSSION

During positive acceleration, there is a diminished blood flow 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 because the standard tests of vision on the centrifuge have been performed with the eyes in the primary position.

The limitation of ocular motility which was observed in this experiment, during positive acceleration, closely parallels similar observations reported in other forms of anoxia. Rossen and his associates (8) observed this to be a constant end point in their studies with a cervical cuff as summarized in the review of literature above. 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 (15)

reported an experiment in which they stimulated the paramedian portion of the diencephalon in monkeys and cats with the Horsley-Clarke stereotaxic technique and found that a mid-positioning of the eyes and pupillary dilatation resulted. Stimulation of the brain stem, the frontal and occipital area of the cortex and in the cerebellum sometimes produced similar results. In a later paper, Bender (16) 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 physiological 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 although we have no evidence as to where such an "eye centering" area may be located.

Some subjects were able to overcome the tendency for 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 dyssynergia may be a type of increased scanning or bracketing which operates in any shift of gaze from one object of regard to another. Phylogenetically, the extra-ocular muscles served the function of maintaining the eyes in a fixed position with reference to the environment (17). 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 due to this increased load on the extra-ocular 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 (8) and Bender (16). 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 extra-ocular 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 (4, 7) 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 cerebral cortical dysfunction during LOMA.

The studies with the negative pressure mask verified the results of Lambert (3) 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 on the basis of a cerebral dysfunction (i.e., in the "eye centering" area, 16, 18) 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 to general anesthesia. "Cessation of eyeball activity marks the passage of anesthesia into the second plane of the third stage" (19). Concurrently, the normal pupil begins to dilate. Guedel (20) 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 physiological sleep, where 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 suggest that the dilatation may be due to cortical hypoxia.

A mechanism for production of LOMA and pupillary dilation 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.

The finding that the pupillary dilatation of positive G was not reversed when vision was restored by application of negative pressure over the eyes suggests that some mechanism in addition to that of primary visual perception is involved. The reversal of the morphine miosis, which has been considered to be of central origin (21), during positive G exposure likewise suggests that a central nervous system function has been disrupted. It may be therefore inferred that the production of LOMA and pupillary dilatation during exposure to positive acceleration involves not only the circulatory failure and resultant hypoxic dysfunction of the retina, but also a circulatory failure and hypoxic dysfunction of the cerebral hemispheres as well.

These findings have certain practical applications. LOMA has been utilized at AMAL as an endpoint in evaluations of the effectiveness of anti-blackout suits in protecting against the effects of acceleration in inexperienced subjects. Studies are continuing to compare the effectiveness of LOMA as an endpoint with other endpoints which are in common use in the measurement of tolerance to positive G ($+G_x$).

During the course of these investigations it was also observed that the degree of pupillary dilatation was the most reliable and critical objective indication of the response of a given subject to positive G. The degree of pupillary dilation could not be directly correlated between the subjects. However, the degree of pupillary dilatation for any individual subject was found to be a reliable and quantitative indication of that individual's response to positive G. This objective sign likewise may prove to be useful in future studies of tolerance to acceleration.

LOMA also has certain implications which are important to operational flying in which high G maneuvers are used. If these maneuvers are of such a magnitude as to produce visual symptoms of grayout in the pilot, it must be understood that LOMA with subsequent fixation of the eyes in the primary position may also be produced, even though considerable central vision remains. If LOMA should occur under these conditions, the pilot therefore would be incapable of changing his point of visual fixation to another part of the instrument panel or to look from the bombsight to his primary flight instruments in the cockpit. This visual fixation may be important in explaining some of the many accidents involving experienced pilots who failed to recover from a high G maneuver in time.

CONCLUSIONS

1. During positive acceleration in man, a stage is reached where there is a limitation of ocular motility.

2. These limitations can be overcome by voluntary effort but the superseding movements are ataxic.

3. The lower motor neurons to the extra-ocular muscles are not involved in LOMA.

4. The pupils dilate as the visual fields constrict during positive acceleration and reach a maximum with loss of central vision.

5. The optokinetic reflex does not continue during blackout. However, a form of horizontal nystagmoid movement of the globes may persist in eyes previously stimulated by the optokinetic drum.

6. Vertical nystagmus is observed during high rates of change of positive acceleration.

7. With a modified skin-diver mask, 30 mm Hg negative pressure was applied to the orbit and under these conditions:

- a. Vision was restored.
- b. Ataxic voluntary movements became coordinated.
- c. Optokinetic reflexes were restored to their original frequency.
- d. The pupils remained partially dilated.

8. The pupillary dilatation which accompanies blackout is prevented by the local application of pilocarpine and is unaffected by morphine.

9. The observation of LOMA is a useful objective endpoint for the evaluation of response to positive acceleration.

10. The observation of pupillary dilatation is a useful quantitative sign for evaluation of response to positive acceleration.

11. The direct light reflex was absent during blackout of positive G when tested with an electronic flash bulb with a flux of 1800 lumen.

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Limitation of Great Mobility and Postural Distortion in Human Response to Positive Acceleration by Ede, L. Berman, CAPT, MC,
USN, T.D. Quam, M.D., PH.D., and R.H. Cohen, LCDR,
NMC, USN, 11 pp., 11 December 1961

In a search for a reproducible and objective endpoint for measurement of reaction to positive acceleration, it was observed that the vestibular system, particularly the peripheral system, was the most sensitive to acceleration. Further studies designed to elucidate the mechanism of this phenomenon were initiated on trained test subjects and it was determined that these subjects could, in fact, maintain a steady gaze in the presence of positive acceleration with greater stability than when they were in a neutral position. The vestibular system, therefore, was found to be a more reliable indicator of reaction to positive acceleration than the visual system. The ability to maintain a steady gaze in the presence of positive acceleration was found to be a more reliable indicator of reaction to positive acceleration than the visual system. The ability to maintain a steady gaze in the presence of positive acceleration was found to be a more reliable indicator of reaction to positive acceleration than the visual system.

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