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## THE EFFECTS OF SIMULATED ALTITUDE ON PENETRATING EYE INJURIES

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Trapped intraocular air expansion can cause extrusion of the lens or vitreous through ocular penetrations. Reduction in barometric pressure with a predictable relative gas expansion (RGE) occurs under conditions of altitude. Therefore, air evacuation presents a potential hazard to the patient having intraocular trapped air resulting from an ocular penetration. Information obtainable from a knowledge of the flight conditions and of the clinical eye examination can be related to the hypothetical formula  $V_{sa} = (RGE \times V_{ia} \times K) - V_{ra}$ . This formula should be regarded as a basis for understanding the mechanics involved, and it should serve to approximately define the extent of the hazard for individual patients. The precise prediction of  $V_{sa}$  (volume of secondary intraocular content loss) is not possible. Therefore, the best mode of transportation for individual ocular injury patients will rest heavily on clinical judgment.

Spontaneous loss of intraocular contents may occur for reasons other than expansion of trapped gas under conditions of reduced barometric pressure. This loss is usually attributable to extrinsic mechanical pressure, such as from muscle spasm. This fact suggests that great care should be used in handling the patient; also, it points to a logical use for sedation and analgesia.

Penetrating eye injuries demand prompt, highly specialized care by trained personnel. The victim whether civilian or military, is often living in a remote area hundreds of miles from the nearest ophthalmic surgeon. Not infrequently, an unpressurized aircraft is available as an alternate means, or as the only means, of transporting the patient to the treatment center. Little is known of the effects of altitude upon these injuries, particularly when bubbles of air are noted in the vitreous along the tract of penetration. The effects of the rate of ascent and of rapid decompression are also unknown.

Despite the protection afforded the eye by its location within a strong protective bony structure, serious injuries of the eye are more frequent in both wartime and peace time than is often realized. During wartime eye injuries constitute approximately 5 per cent of the hospital admissions for battle injuries and 10 per cent of the accidental non-combat-incurred injuries (4). Byrnes (2) reports that 7 per cent of the serious nonfatal casualties incurred by the British during World War II air raids were severe eye injuries. The incidence rose to 11.7 per cent with the use of more powerful bombs.

\*Performed at the USAF School of Aerospace Medicine, Brooks AFB, Texas, while the senior author was attached there in the capacity of a Federal Aviation Agency Flight Surgeon. Grateful acknowledgment is made to the Air Force for making available the facilities utilized in this study.

This same trend toward an increased incidence of eye injuries is expected to continue with the use of nuclear weapons; Byrnes estimates that 10 per cent of the total injuries will be traumatic eye injuries other than burns.

The incidence of serious eye injuries in peace time disasters is illustrated by the Texas City disaster of April 1947, where 123 eye injuries were reported (2). Thirty-eight of these were serious enough to require hospitalization. These 38 cases constituted 4.75 per cent of the total hospital admissions from this disaster.

Early repair of penetrating ocular injuries has been shown to be imperative if optimum results are to be obtained (5, 8, 9, 12, 15). Therefore, air evacuation of eye casualties to treatment facilities where eye surgery may be performed affords the most prompt mode of transportation.

Despite the extensive employment of air transportation for evacuation, little has been written regarding the effects of reduced barometric pressure on penetrated eyes. The problem here is basically one of trapped air within the injured eye. Bubbles of air may frequently be seen within an eye along the tract of penetration (5, 7). As the ambient pressure is reduced, the trapped air would be expected to expand and extrude the intraocular contents through the laceration or opening in the eye. Jacobi, in 1868, first described small, sharply defined spheres with bright centers and dark rims in the vitreous of patients with intraocular foreign bodies (7). He interpreted this as being blood. Morton, in 1876, properly interpreted this as being air—an observation subsequently confirmed by many other observers (7). Levy, in 1900, reported a case where a bubble of air was found in the lens associated with a foreign body.

Intraocular air injections may be used in several diagnostic and surgical procedures of the eye. Air has been injected into the vitreous as part of the treatment of retinal separation (3, 10, 13). Air may be injected into the anterior chamber in numerous surgical procedures. Since many patients in medical centers return to their homes by airplane, the persistence of any air within the eye, the attempted expansion

of this air at altitude, and the tensile strength of the ocular surgical wound are factors to be considered by the attending ophthalmologist in determining the date of discharge for a patient who plans to fly home.

Thalabard (14) instituted air evacuation of patients with eye injuries in North Africa, in July 1956, and reports noting better functional results. The delays and vibration of ground transportation were definitely felt to be detrimental. The flights were made in helicopters and light aircraft at altitudes of 900 to 4,500 feet, depending upon the terrain. Wide fluctuations of altitude were noted during most of the flights, the most abrupt of which occurred in the helicopters.

The report on medical criteria for passenger flying by the Committee on Medical Criteria of the Aerospace Medical Association (11) recommends that patients whose eyes contain any air remain below 4000 to 5000 ft. equivalent altitude, if possible.

For penetrating injuries of the cornea or of the sclera, the following hypothetical equation is postulated:

$$V_m = (RGE \times V_{in} \times K) - V_p$$

where  $V_m$  = volume secondary loss of intraocular contents from air expansion.

$RGE$  = the relative gas expansion of the air at the reduced barometric pressure to which the eye is exposed (treated as a trapped gas).

$V_{in}$  = the volume of air introduced into the eye.

$K$  = the reciprocal of fluid resistance. (In the relatively static conditions of this test,  $K$  is approximately equal to 1).

$V_p$  = volume primary loss of intraocular contents following initial injury.

The purpose of this study was to 1) investigate the effects of both rapid and slow reduction of barometric pressure upon these injuries, 2) test the hypothesis put forth in the above equation, and 3) attempt to establish criteria which might be used in the clinical appraisal of the suitability of air transportation for a patient with a penetrating eye injury, with or without associated traumatically introduced air.

## PROCEDURE

A preliminary study was conducted to develop techniques and determine volumes for the rabbit eye. Using a lubricated tuberculin syringe with a 30 gage needle, the following observations were made: approximately 0.4 to 0.5 cc. of air could be injected into the vitreous before the lens or vitreous were lost through corneal injuries. Further, it was found that for the rabbits that were used, the aqueous volume equaled approximately 0.4 cc. Thus, when a volume of trapped air equaling the volume of aqueous loss had been injected, extrusion of intraocular content through the corneal laceration occurred. Conversely, vitreous loss occurred immediately when air was injected into an eye with a scleral perforation where there had been no primary vitreous loss from the penetration. The significance of these and other findings are elucidated further under the Discussion.

For the experimental procedure, 25 six to seven pound New Zealand white rabbits were used. To facilitate both the observation of the placement of the air and the detection of any vitreous hemorrhage, mydriasis was accomplished prior to the experimental procedure with a one percent solution of cyclopentolate hydrochloride. Approximately one hour prior to anesthesia the rabbits were given an intravenous injection of 3 mg. of promazine hydrochloride per pound of body weight. Deep anesthesia was accomplished with 90 to 120 mg. of intravenous pentobarbital sodium. One tenth of a cubic centimeter of air was then injected into the posterior vitreous of each left eye under

observation with a Schopens binocular indirect ophthalmoscope. Penetration of the sclera with the needle was at 6:00 o'clock, 0.5 cm. from the limbus. A 6 to 7 mm. transverse incision through the center of the cornea was then inflicted upon both eyes with a Lawton No. 2 keratome. Thus, the right eye of each rabbit served as the control for the left eye of the same rabbit.

Within 30 minutes of the time that the wounds were inflicted the rabbits were placed into an altitude chamber and subjected to a simulated flight profile.

The flight profile consisted of: (1) a simulated ascent from ground level to 10,000 ft. at a 1000 ft per minute rate of climb; (2) a simulated ascent from ground level to 10,000 ft. in 10 seconds; and (3) a simulated ascent from 10,000 to 43,000 ft. at 1000 ft. per second, with the chamber leveled for observation at 25,000 and 36,000 ft. equivalents.

A standard Air Force aneroid airplane altimeter was placed in the chamber atop the rabbit boxes to facilitate recording by the observer of the simulated altitude at which ocular change occurred.

## RESULTS

Of the 25 injured eyes containing no injected air, loss of the lens and vitreous occurred in one during the entire flight profile to 43,000 ft. During the observation at 10,000 ft. following the rapid ascent from ground level, one of the rabbits with light anesthesia and without upper lid retraction with sutures lost lens substance simultaneously from both eyes during rather marked bilateral spasm of the orbicularis oculi muscles.

The simulated altitudes at which losses occurred in the eyes containing 0.1 cc. of air can best be demonstrated by the accompanying graph (fig 1). No loss occurred in six of the eyes. No losses occurred under 10,000 ft. equivalent altitude during either the slow or the rapid ascent.

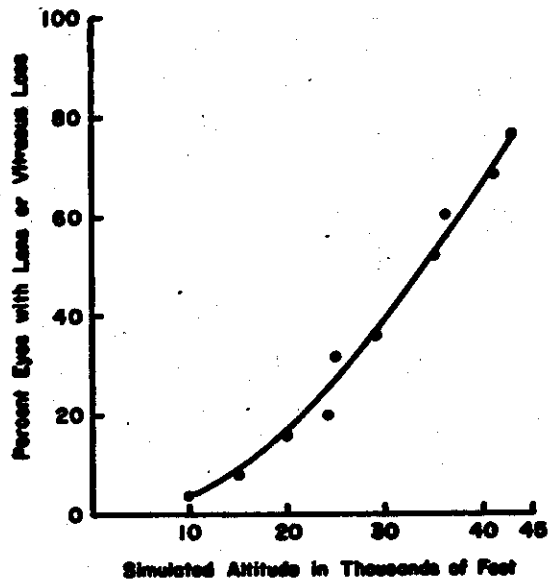


FIGURE 1. The cumulative incidence of loss of the lens or vitreous during simulated ascent from eyes with corneal lacerations and with 0.1 cc. of air in the vitreous.

## DISCUSSION

The eye is a system which combines non-compressible fluids and a reasonably rigid sclera. In such a fluid-filled system, expansion of a contaminating gas will be related to a relative increase in pressure inside the system. If the eye is intact, a rise in intraocular pressure could be predicted; however, in the case of a penetrated eye the pressure difference will become equalized by the extrusion of intraocular contents. If the gas is not trapped by the vitreous, the gas will escape; if the gas is trapped by the vitreous (the usual situation), extrusion of intraocular contents must occur. Under these conditions a mathematical relationship between the volume of trapped gas, which increases with an increase in altitude, and the amount of fluid displaced can be established. This relationship,  $V_{tr} = (RGE \times V_{ab} \times K) - V_{tr}$ , can be applied to both corneal and scleral penetrating ocular injuries.

In the case of penetrating corneal injuries the total aqueous volume is generally lost because of the low viscosity of aqueous. For the large rabbits used, this volume can be estimated to be 0.4 cc. and for man, 0.125 cc. Frequently, scleral injuries however do not result in vitreous loss because of the formed nature of the vitreous body. Therefore,  $V_{tr}$  for vitreous may vary considerably from 0.0 cc. to a few cubic centimeters loss, depending on the severity of the injury and the amount of orbicularis spasm.

In the preliminary study it was noted that in a scleral laceration vitreous loss occurred as soon as air was injected. In the corneal injury, however, the lens and vitreous loss occurred only after a volume of air had been injected that was equal to the volume of ocular contents lost during the injury; i.e., the 0.4 cc. of aqueous. These findings are consistent with the equation proposed.

From the proposed equation and from the preliminary study determination that vitreous loss occurred after 0.4 to 0.5 cc. of air had been injected into the eyes with corneal lacerations, it was felt that 0.1 cc. of air should give the same effect by relative gas expansion when the eye was subjected to one-fourth an atmosphere of barometric pressure ( $RGE = 4$ ) equivalent to an altitude of about 34,000 ft. This volume of air is probably equal to, or slightly less than, the volume which is usually injected with penetrating eye injuries. The mode and median for the experimental data are both 35,000 ft. Despite the considerable variation in the results, this figure compares quite favorably with the calculated value and supports our hypothesis.

Several factors were found to contribute to the variations in the results which were obtained. These include: (1) the length of the incision, (2) the clotting of rabbit aqueous, and (3) the depth of anesthesia. However, despite the many variables, conditions were held reasonably constant.

The failure to find any effects of simulated altitude on the eyes containing no air is not surprising since there is no basis upon which such a loss could be expected to occur.

It was felt that observed orbicularis muscle spasm accounted for the loss of the lens from both eyes of one rabbit at a simulated altitude of 10,000 ft. This observation would tend to amplify the importance of the use of sedatives, analgesics, and perhaps 7th nerve blocks in the early treatment of penetrating eye injuries regardless of the mode of transportation to be used.

The attempt to determine the effects of various rates of ascent was limited to two widely variant conditions: a simulated realistic 1000 ft. per minute climb and an extreme 1000 ft. per second ascent. It was also limited to realistic altitudes (under 10,000 ft.) that would be achieved either by actual altitude in an unpressurized aircraft or by cabin pressurization in an aircraft flying at higher altitudes.

To test our hypothesis and to establish a threshold for a detrimental effect of reduced barometric pressure upon rabbit eyes with a common type of injury and a known amount of air, the ascent was continued until loss of lens or vitreous occurred. For this determination a rate of ascent was chosen which would closely simulate the decompression of a Boeing 707 series aircraft following window failure while flying at 42,000 ft. (1, 6). In these studies the cabin altitude did not exceed 36,000 ft. Since several eyes had not reacted at a simulated 36,000 ft., the chamber was carried on to 43,000 ft. at the same rate of ascent.

That cabin pressurization failures do occur in high altitude passenger aircraft is pointed out by Bryan and Leach, (1) who report on a personal communication with Lucking concerning his study of 184 reported cases of cabin pressurization failure in commercial aircraft between June 1952 and February 1958.

It is not meant to imply that patients with penetrating eye injuries will be transported in commercial jet aircraft, but it does offer some

practical comparison for the threshold determination part of the experiment for the sake of interest and possibly for a rare application.

The formula proposed here should serve as a guide to understanding the clinical mechanism of secondary loss of intraocular contents in the penetrated eye following the relative expansion of trapped air. In most clinical instances, however, it would not be possible to apply the formula exactly.

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