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THE EFFECTS OF EXTERNAL OCULAR IRRITATION ON INTRAOCULAR PRESSURE

Roy H. Rengstorff

Edgewood Arsenal
Aberdeen Proving Ground, Maryland

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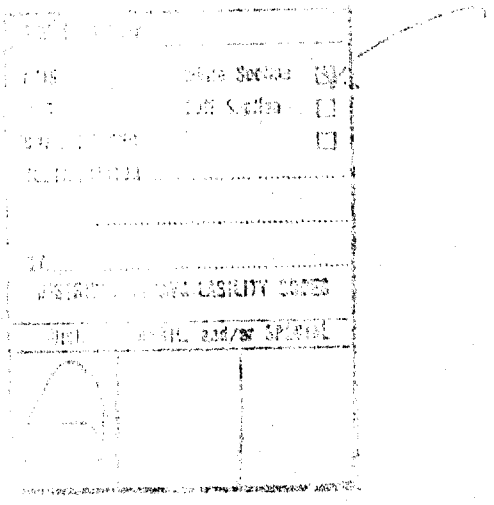

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Riot control agents (tear gases) cause intense eye irritation and reflex squeezing of the eyes. The intraocular pressure under these circumstances is unknown, but animal and human studies of ocular irritation and forces acting upon the eye indicate a transient increase in intraocular pressure which is not necessarily detrimental to vision.

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PREFACE

The work described in this report was authorized under Project 1W662606AD22, Medical Effects of Riot Control Agents. The work was started in February 1972 and completed in March 1972.

In conducting the research described in this report, the investigators adhered to the "Guide for Laboratory Animal Facilities and Care," as promulgated by the Committee on the Guide for Laboratory Animal Resources, National Academy of Sciences – National Research Council.

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CONTENTS

THE EFFECTS OF EXTERNAL OCULAR IRRITATION ON INTRAOCULAR PRESSURE

I. INTRODUCTION.

Do chemical irritants used in riot control agents (also known as tear gases)* cause increased intraocular pressure? If the answer is yes, then is there a risk of visual loss from such an increase in intraocular pressure? The answers to these questions are important because the eyes are one of the major sites of actions of the chemical irritants that are being used increasingly for controlling mobs.[1] In addition to thermally-generated aerosols that cover a large area, sprays of these irritants mixed in a liquid and pressurized in a container are sometimes directed at the eyes of individuals.

No human or animal experiments have been reported where the intraocular pressure was measured during exposure to the chemical irritants in riot control agents, nor will I present any such new data in this report. Instead, the evaluation of changes in intraocular pressure caused by these irritants will be based on other studies which describe the effects of external eye irritation on intraocular pressure.

Other eye and vision effects of riot control agents on humans have been studied, and one of the behavioral effects is the reflex squeezing of the eyes following ocular contact with the irritant.[2-4] For this reason, the effect of external forces acting on the eyeball is another consideration in evaluating changes in intraocular pressure, and I have included a review of pertinent studies.

II. EXTERNAL OCULAR IRRITATION.

A. The Fifth Cranial Nerve and Neurohumoral Mechanisms.

Perkins[5] revealed that electrical stimulation of the ophthalmic division of the trigeminal nerve in rabbits had relatively little effect on intraocular pressure, but mechanical stimulation of the nerve was highly effective. Duke-Elder[6] believes that the temporary rise in pressure is caused by the increased content of the eye brought about by accelerated aqueous formation and breakdown of the blood aqueous barrier which allows more proteins to obstruct the aqueous outflow. Furthermore, he likened the reaction to the axon reflex mediated by the peripheral branches of the nerve following sensory or noxious stimuli to the eye where some active histamine like substance is liberated.

Thomas[7] produced an acute transient rise in intraocular pressure in anesthetized rabbits and dogs by various methods: intracranial mechanical stimulation of the fifth cranial nerve, interruptions of the anterior ciliary vessels and aqueous and vortex veins, injecting air and methylcellulose into the anterior chamber, paracentesis of the anterior chamber with and without mechanical irritation of the iris, and topical application of diisopropyl fluorophosphate (DFP).

* Some examples are chloroacetophenone, also known as CN, and chlorobenzylidene malononitrile, also known as CS.

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External ocular irritation from chemicals was also studied. Injecting small amounts of chloroform beneath the conjunctiva in rabbits consistently resulted in a 10- to 20-mm Hg rise in ocular pressure. Similarly, topical application of nitrogen mustard produced a 5- to 15-mm Hg rise in ocular pressure. The effects of trauma, induced by repeated blows to the eye of anesthetized rabbits, also produced rises (10 to 20 mm Hg) in intraocular pressure. The author proposed that these methods increased the intraocular pressure by a neurohumoral mechanism; i.e., by release of a lipid extracted from the iris by Ambache,[8] irin. Since then irin has been classed as a prostaglandin, and these substances have produced similar ocular effects.[9]

Chiang[10] studied the intraocular pressure response in anesthetized rabbits after topical application of hydrochloric acid, sodium hydroxide, and ammonium hydroxide. The acid caused a gradual rise in intraocular pressure which was maximum within 10 to 30 minutes. The alkali solution caused a biphasic response: a rapid initial rise followed by a gradual secondary rise in intraocular pressure. Again, it was concluded that the intraocular pressure response was due to a common neurohumoral mechanism.

B. General Arousal Response.

Collins[11] has shown that other more subtle sensory stimuli, of low intensity, have evoked an increase in intraocular pressure. His study demonstrated how evoked intraocular pressure could be produced by low intensity sound, light, movement, temperature changes, and odor. The amplitude of the pressure increases was as great as 10 mm Hg in the eyes of unanesthetized rabbits. Continuous monitoring of the intraocular pressure was accomplished by a passive radio transensor (2 mm thick and 6 mm in diameter) which was implanted in the eye. The results showed that the latency of onset of the rise in pressure was too short (0.4 second) for humoral regulation, and the rate of rise was too fast (5 mm Hg per second) to be produced by aqueous secretion or outflow resistance. The author, therefore, tested some vascular and muscular phenomena as possible mechanisms and found that the intraocular pressure response was not due to an active vascular mechanism or any passive reflection of changes in blood pressure, nor was there any activity by the retractor bulbi muscles that might account for the changes. Further tests revealed the mechanism for these responses to be the contraction of the smooth muscle of Mueller. This muscle contraction seems to be α-adrenergically mediated through the sympathetic nervous system; the author[12] concluded that it was activated during a general arousal response to a sensory stimulus.

III. EXTERNAL FORCES ACTING UPON THE EYEBALL.

A. Contraction of the Extraocular Muscles.

Many early animal studies have shown that stimulation of the third cranial nerve causes a rise in intraocular pressure.[13-16] Stimulation of the rectus muscle of a dog with acetylcholine causes a rise in intraocular pressure of 30 mm Hg.[17] These effects are eliminated by sectioning the nerve or using drugs to paralyze muscle contraction.[18] Wessely[19] stimulated the motor nerves at the base of the brain and found that the abducens was half as effective as the oculomotor, and the trochlear was only one-tenth as effective in producing an increase in intraocular pressure. Two recent experiments on the extraocular muscles confirm their influence

on intraocular pressure: (1) an electrical current across electrodes on the skulls of dogs showed an increased intraocular pressure and a simultaneous contraction of the extraocular muscles,[20] and (2) injection of succinylcholine in cats and rabbits caused the contraction of the extraocular muscles with a corresponding increase in the intraocular pressure.[21]

In one of the early human studies, Hine[22] performed tonometry on 26 children as they converged their eyes. His results showed an increase in intraocular pressure from 2 to 10.5 mm Hg, average 4.9, which he attributed to the effect of mechanical pressure from the extraocular muscles. Glaser[23] administered edrophonium chloride (Tensilon) to 15 patients with myasthenia gravis and produced a rise in intraocular pressure; the same treatment caused no change in the intraocular pressure of normal subjects.

B. Blinking and Squeezing the Eyes.

A recurrent problem in measuring intraocular pressure in humans is the anxious patient who, unintentionally, causes an increased lid tension against the eye. Levene[24] observed two patients with uniocular paresis of the orbicularis muscle and found that intraocular pressure on the affected side was lowered by 2 to 3 mm Hg. The average blink of the eyelids in dogs was found to increase the intraocular pressure 5 mm Hg.[25] Comberg's[26] human studies showed that ocular pressure increased from 18 to 70 mm Hg during a hard lid squeeze.

Miller[27] studied the lid pressure against the eye in 10 normal subjects using a molded scleral contact lens with an inner rubber balloon connected to a pressure transducer. He found that the average reflex blink of the eyelids produced a 10-mm Hg pressure against the eye, and a hard lid squeeze resulted in pressures as high as 51 mm Hg.

Garner's[28] tonographic studies showed increased intraocular pressure from digital pressure against the eye, squeezing the eyes, and sneezing. Levene and Hyman[29] monitored the intraocular pressure as digital scleral compression through the upper lid was being produced. As a result, ocular pressure was elevated up to 60 mm Hg.

Recently,[30] direct manometric measurements of intraocular pressure in an unanesthetized man were made before the eye was removed because of ocular tumor. During accommodation the pressure increased about 4 mm Hg; turning the eyes or blinking caused increases of 10 mm Hg; and squeezing the lids shut increased the pressure to over 100 mm Hg.

IV. CONCLUSIONS.

Two questions were asked in the introduction of this report. In reply to the first question "Do chemical irritants used in riot control agents cause increased intraocular pressure?" The evidence from related studies indicates that the answer probably is yes. The answer to the second question, "Is there a risk of visual loss from such an increase in intraocular pressure?" is a qualified no. The qualification is that there is always the remote possibility that an acute transient rise in intraocular pressure will provoke a glaucomatous attack in the eyes of individuals predisposed to glaucoma. The overwhelming evidence, however, is that normal eyes undergo

considerable transient rises in intraocular pressure every day without dangerous consequences. Finally, in evaluating the effects of an irritant, Duke-Elder[6] made an important distinction: "In general, the intraocular pressure can undergo changes of two different types: (1) changes in the equilibrium between the factors responsible for the entry of the aqueous and those governing its escape and, (2) transient changes of intraocular pressure resulting from alterations in external forces acting upon the eyeball or from volumetric changes within it. The recognition of the differentiation between the two types of variation in the intraocular pressure is of the utmost importance for too frequently in the past the causation of long-term elevations of the intraocular pressure (as in glaucoma) has been erroneously inferred from experiments which merely demonstrate transient alterations."

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