Evaluation of Intraocular and Orbital Pressure in the Management of Orbital Hemorrhage

An Experimental Model

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Objective: To evaluate orbital pressure (OP), intraocular pressure (IOP), and the effectiveness of canthotomy, cantholysis, and septolysis using an experimental orbital hemorrhage model.

Methods: Expired whole blood was injected into the retrobulbar space of 10 human cadaver orbits. At 1-mL increments, OP, IOP, and globe position were documented. The mean (SD) time interval between the injections was 84(36) seconds. Following injection of 22 mL, lateral canthotomy, cantholysis, and septolysis were performed. An additional 10 mL of blood was then injected.

Results: After injecting 22 mL of whole blood, mean (SD) OP and IOP were 68.4(32.2) and 71.5(19.1) mm Hg, respectively. The OP and IOP correlated closely throughout the experiment, with a mean (SD) difference of 11.4(4.9) mm Hg (Pearson coefficient, 0.97). Following canthotomy, cantholysis, and septolysis, there was a mean (SD) decrease of 48.0 (27.2) mm Hg (70%) and 50.0 (18.1) mm Hg (59%) in OP and IOP, respectively. With additional injection of 10 mL of blood, OP and IOP increased rapidly.

Conclusions: The IOP and OP rose in direct proportion to the volume of whole blood injected; OP lagged behind IOP by 11 mm Hg, and surgical release of the orbit reduced OP by 70%. This effect was short-lived in the setting of continued simulated hemorrhage.

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Orbital hemorrhage is seen in numerous settings, most commonly in association with periorbital fractures or contusion. It is also well described in association with blepharoplasty and may also follow retrobulbar or periorbital anesthetic injections.12 Anecdotal associations have been reported with blood disorders such as sickle cell disease, factor VII deficiency, platelet dysfunction, and vitamin C deficiency.6-8 Other less common causes, such as extension of subgaleal bleeds, have also been described.9

Given the diverse nature of the injuries, judging the likelihood or degree of visual loss is difficult. The incidence of orbital hemorrhage after blepharoplasty with subsequent partial or complete blindness is estimated to be between 6 and 40 cases per 100 000.2 This is presumptively owing to compressive occlusion of the vasculature supplying the optic nerve or retina. Animal studies have suggested that irreversible damage occurs within 90 to 120 minutes.

Thus, management has focused on identifying individuals with elevated orbital pressure (OP) and lowering it prior to infarction of compromised tissues.

Management consists largely of estimating OP with intraocular pressure (IOP) and, when elevated, performing a canthotomy and cantholysis. Canthotomy refers to a horizontal incision, dividing the lateral canthal tendon into its superior and inferior crus (Figure 1A). Cantholysis refers to cutting or disinserting the inferior (and less commonly the superior) crus (Figure 1B). Further decompression can be accomplished by opening the orbital septum, exposed at the base of the cantholysis incision, bluntly with a pair of surgical scissors (Figure 1C). This third step is referred to as septolysis. More definitive treatment includes orbital decompression or exploration with evacuation of any hematoma and ligation or cautery of problematic vessels. Unfortunately, treatment is largely based on anecdotal experience, with a relative lack of supporting evidence.

An experimental model of orbital hemorrhage was developed to gain a better understanding of the relationship between OP and IOP, primarily to determine how accurately OP can be estimated by IOP. Additionally, we evaluated the effectiveness of canthotomy, cantholysis, and septolysis.
METHODS

CADAVER PREPARATION

We simulated an orbital hemorrhage in 10 orbits of 5 unfixed, unfrozen white human cadavers (4 women; 1 man; aged 71 to 90 years). Dehydration significantly reduces IOP after death. To achieve the closest approximation to living-state IOP, specimen eyes were inflated with normal saline. A 30-gauge needle was passed in a beveled fashion from the corneal limbus into the anterior chamber. Saline was injected into both the anterior and the posterior chambers until an IOP of between 10 and 20 mm Hg was reached. Octyl-2-cyanoacrylate tissue glue was applied to the insertion site to ensure closure.

BLOOD INJECTION

Expired whole human blood was obtained from a hospital blood bank and injected into the retrobulbar space in 1-mL increments using a 1.5-inch, 18-gauge needle on a 10-mL syringe. The needle was inserted transcutaneously through the lateral aspect of the lower eyelid and advanced into the intracranial space. Needle position was based on clinical experience/judgment and was not confirmed. Initially, 22 mL of whole blood was injected. This volume was chosen based on trial runs in 2 cadavers (4 orbits) performed in the development phase of the experiment. This was the maximal volume that could be injected without expulsion of blood through the conjunctiva.

MEASUREMENTS

At each 1-mL increment, OP (using a Stryker Intracompartmental Pressure Monitor System; Stryker Instruments, Kalamazoo, Michigan), IOP (using a Tonopen; Mentor Ophthalmics Inc, Norwell, Massachusetts), and globe position (using a Hertel exophthalmometer; Richmond Products Inc, Albuquerque, New Mexico) were documented. Subsequent injections were performed immediately following completion of the series of measurements, with a mean (SD) interval of 83(31) seconds. The Stryker Intracompartmental Pressure Monitor System has an indwelling catheter system that was inserted adjacent to the 10-mL syringe of whole blood. For IOP estimation, the mean of 3 Tonopen measurements was used.

CANTHOTOMY, CANTHOLYSIS, AND SEPTOLYSIS

Following injection of 22 mL of whole blood, lateral canthotomy, inferior cantholysis, and septolysis were performed; measurements were repeated following each step. The superior crus of the lateral canthal tendon was not cut. To evaluate the effect of canthotomy, cantholysis, and septolysis in the setting of continued hemorrhage, an additional 10 mL was injected, with measurements again taken at 1-mL increments.

Differences in IOP and OP before and after canthotomy, cantholysis, and septolysis were assessed with the paired t test (1-tailed). The Pearson coefficient was used to evaluate correlations between IOP, OP, and exophthalmometry measurements.

RESULTS

CORRELATION BETWEEN IOP AND OP

Figure 2 summarizes the results. Mean (SD) resting OP and IOP were 4.1 (2.7) and 12.5 (3.2) mm Hg, respectively. After injecting 22 mL of whole blood intraorbitally, mean (SD) OP and IOP were 68.4 (32.2) and 71.5 (19.1) mm Hg, respectively. A close correlation between OP and IOP was observed, with a mean (SD) difference of 11.4 (4.9) mm Hg (Pearson coefficient, 0.97). Changes in exophthalmometry values less closely paralleled changes in OP and IOP (Pearson coefficients, 0.80 and 0.88, respectively). After injecting the initial 3 mL of whole blood, exophthalmometry measurements increased, with
little change in OP and IOP. As exophthalmometry values started to plateau, substantial rises in OP and IOP were observed.

**EFFECT OF CANTHOTOMY, CANTHOLYSIS, AND SEPTOLYSIS**

There was a significant reduction in mean (SD) OP after canthotomy from 68.4(32.2) to 36.5(18.3) mm Hg ($P < .001$) and cantholysis from 36.5(18.3) to 24.8 (10.1) mm Hg ($P < .005$). Septolysis further reduced the OP to 20.4(10.1) mm Hg ($P < .001$). There was a total reduction of 48.0(27.2) mm Hg (70% reduction) in OP following canthotomy, cantholysis, and septolysis. Parallel changes in IOP were observed, showing a total decrease of 50.0(10.1) mm Hg (58.7%).

With additional injection of blood, OP and IOP increased rapidly. After 10 mL, mean (SD) OP had risen from 20.4(10.1) mm Hg (postseptolysis pressure) to 65.7 (28.4) mm Hg. Similarly, IOP rose from 29.5(10.5) (post-septolysis IOP) to 78.4(11.8) mm Hg.

**COMMENT**

This is the first study to evaluate orbital hemorrhage in human cadavers using human blood. Several investigations have evaluated orbital pressure and associated visual loss. In patients with Graves disease with related optic neuropathies, orbital pressures were measured between 17 and 40 mm Hg (mean, 28.7 mm Hg). Hargaden et al\(^{13}\) simulated orbital hemorrhage by inflating balloon catheters within the orbits of 16 nonhuman primates for 3 or more hours. When inflated such that IOP was 50 mm Hg or greater, half of the animals suffered irreparable damage. Using a similar technique, Schabdach et al\(^{13}\) observed permanent injury in only 1 of 9 monkeys.

In contrast, Young et al\(^{14}\) simulated retrobulbar hemorrhage in nonhuman primates by autogenous blood injection. In their study, all animals regained vision after 120 minutes of ischemia. In nonhuman primates, Hayreh et al\(^{11}\) demonstrated that with clamping of the central retinal artery, permanent visual loss occurred after 105 minutes. In contrast, Katz et al\(^{7}\) described 2 patients with orbital hemorrhage who regained vision following more than 3 hours of no light perception.

Although the details may vary, it is clear that visual loss can occur with elevated OP and that this is dependent on the magnitude and duration of pressure elevation. Accordingly, treatment has been directed at detecting patients with elevated OP and reducing it. It has long been assumed that OP can be estimated by IOP, which is used as the primary measure in patients with orbital hemorrhage. Our study confirms this assumption. We found an extremely close correlation between IOP and OP ($r = 0.97$), with mean IOP remaining an average of 11 mm Hg greater than OP.

There was some variability in the difference between IOP and OP (SD, 5 mm Hg). The orbit is not a single, uniform, water-filled compartment. There are many subdivisions; for example, the orbit is divided by the periosteum, intermuscular septum, and fibrous septate between adipose lobules. Pressure originating in 1 compartment may not be equally distributed throughout the orbit. Other influences, such as the degree of posterior tension placed on a proptotic globe by the extraocular muscles and optic nerve, may vary. Conversely, varying aspects of eyelid anatomy such as laxity likely influence the degrees of anterior resistance and might alter the relationship between IOP and OP. One observation that was not quantified was that blood tracked into
the eyelids at different rates. Higher IOP was measured in orbits with more eyelid infiltration compared with ones where the blood remained posterior. This may be reflective of resistance to opening the eyelid causing fictitious IOP elevation; however, the clinical implications stand. Clinicians should be aware that some variability exists and not rely entirely on IOP as an indicator of OP.

Management options are orbital decompression or exploration with control of active bleeding. Prior to an orbitotomy, canthotomy and cantholysis are often employed as an initial attempt at normalizing orbital pressure. Yung et al. assessed the effect of canthotomy, cantholysis, and canthal tendon disinsertion on IOP in rabbits following retrobulbar saline injection. They documented a statistically significant decrease in IOP but did not monitor orbital pressure. Limited additional investigative data exists, and impressions of the effectiveness of canthotomy and cantholysis are based largely on anecdotal experiences.

Our data substantiates the effectiveness of canthotomy and cantholysis in human cadavers using human blood. With these maneuvers we observed significant reductions in OP (58.9%) and IOP (54.9%) after performing a canthotomy and cantholysis. Our study also assessed the effectiveness of septolysis. This additional step further reduced OP and IOP by 17.7% and 9.5%, respectively. Alarmingly, in the setting of simulated continued bleeding, the effect of canthotomy, cantholysis, and septolysis was very short-lived. This suggests that it will not have a lasting effect if performed prior to cessation and/or control of bleeding.

In our study, the location of the blood within the orbit during and after the conclusion of the experiment was not assessed. Our intention was to inject the blood into the retrobulbar space; however, the location of the needle was not confirmed. Additionally, we did not perform imaging studies or postexperimental dissection to evaluate the location of the blood within the orbit or adjacent tissues. Evaluating the extension of an orbital hemorrhage to adjacent tissue and/or cavities is a potential area for future investigation that might prove interesting. Comparison with a control orbit in which canthotomy, cantholysis, and septolysis are not performed might also provide useful information regarding the natural history of an orbital hemorrhage.

This study has several shortcomings. There are several limitations inherent in the use of cadavers. Tissue elasticity differs and may affect the rapidity at which pressure rises. We attempted to limit this effect by using fresh (< 48 hours after death), unfixed, and unfrozen specimens. Also, living eyes probably behave differently. With continued aqueous production, living eyes might exhibit comparatively higher IOP. Alternatively, the living eye has the ability to autoregulate aqueous production with changes in IOP. Our data may not be entirely reflective of hemorrhages of longer duration, which often contain accumulations of coagulated blood. Lastly, our results may have been different were alternate methods of decompression employed. For example, lysis of the entiure lateral canthal tendon (inferior and superior crus) might prove more effective. Although these limitations might have some measurable effect, it seems likely that our observed trends are valid.

In summary, our data strongly supports the belief that IOP is reflective of OP. The mean OP was 11.4 mm Hg less than IOP. Canthotomy, cantholysis, and septolysis significantly reduce orbital pressure; when performed together, a 70% drop in OP was observed. Unfortunately, this effect was short-lived in the setting of continued hemorrhage.

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REFERENCES