Intraocular Pressure Changes Secondary to Reduction of Orbito-Zygomatic Complex Fractures

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Purpose: To investigate intraocular pressure (IOP) changes in the involved eye during orbito-zygomatic complex fracture reduction, compared with the IOP of the eye on the uninjured side.

Patients and Methods: Twenty patients who presented with displaced, isolated unilateral orbito-zygomatic complex fractures were enrolled. IOPs were measured using a Tono-Pen XL (Medtronic Solan, Jacksonville, FL) in the involved eye and in the non-involved eye. Pressure was recorded at 5 time points (before general anesthetic induction, after induction, immediately after fracture reduction, post operation while still under general anesthetic, and 15 minutes post general anesthetic).

Results: There were no significant differences between the injured and control data at any time point. Average IOPs for the involved and control eye decreased after anesthetic induction; however, IOP increased in both eyes after fracture reduction.

Conclusion: There is no statistical evidence of a difference between the IOP of the control and injured eyes. Routine IOP testing is not indicated during orbito-zygomatic fracture reduction.

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Loss of vision or decrease in visual acuity is a rare but devastating complication of surgery involving the orbital skeleton.[1-6] Factors responsible may include direct insult to the optic nerve from bony impingement, surgical injury to the nerve, or retinal artery occlusion secondary to increased orbital compartment pressure arising from edema or retrobulbar hemorrhage.[1-6]

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An increase in the compartment pressure of the orbit has been described by several authors as being responsible for changes in visual acuity following repair of orbital fractures or orthognathic surgery.[7-10] The underlying assumption of these reports is that the orbit, because of its rigid bony skeleton and firm anterior soft tissue border, provides an environment conductive to a compartment pressure syndrome caused by retrobulbar hemorrhage or traumatic edema. Visual loss is thought to result from this increased orbital pressure compromising the vascular supply of the optic nerve, choroids, and retina.[11]

It is postulated that the increased orbital compartment pressures can secondarily raise the intraocular pressure (IOP), which in turn can compromise the ocular blood supply.[12] Previous studies have shown a decrease in retinal perfusion in patients with glaucoma once the IOP reaches 80 mmHg.[13] However, it is not clear what the critical time limit for retinal ischemia is before irreversible damage occurs. Ischemic retinal cells have been documented to have a survival time of 3 to 5 minutes in rats and up to 97 minutes in elderly rhesus monkeys.[14]

Forrest et al[15] showed a statistically significant rise in IOP following the placement of bone grafts to

repair orbital floor fractures. The IOP changes returned to baseline within 30 minutes and there were no reported changes in visual acuity.

The aim of this study was to investigate the IOP changes of the involved eye following reduction of orbito-zygomatic complex fractures compared with the IOP of the eye on the uninjured side.

Patients and Methods

Twenty consecutive patients who presented to the Oral and Maxillofacial Surgery Unit at the Royal Brisbane Hospital (Brisbane, Australia) with displaced, isolated unilateral orbito-zygomatic complex fractures requiring reduction to prevent esthetic and functional deficits were enrolled in the study. The hospital ethics committee approved the research protocol and written consent was obtained from all participants. Patients were excluded from the study if they had previous orbital surgery on the affected side, or if they had sustained a globe injury. Each patient was prepared as per our routine practice; axial and coronal computed tomography scans, examination of visual acuity, pupillary reflexes, extraocular eye movements, assessment of enophthalmos or exophthalmos, assessment of infraorbital nerve paraesthesia, and an examination of the globe by an ophthalmologist.

IOPs were measured in mmHg using applanation tonometry with a Tono-Pen XL (Medtronic Solan, Jacksonville, FL) in the involved eye and non-involved eye. Pressure was recorded at 5 time points:

1. Before general anesthetic induction.
2. After induction.
3. Immediately after fracture reduction.
4. Post operation (while still under general anesthetic).
5. Fifteen minutes post general anesthetic.

Topical corneal anesthesia was achieved with 1 drop of oxybuprocaine hydrochloride (0.4%) (Chauvin Pharmaceuticals Ltd, Surrey, UK) placed in each eye before the first measurements and at the end of the operation for the final pressure readings recorded in the recovery room. Routine postoperative eye observations (pupillary response, visual acuity measurements) were carried out postoperatively on all patients as per usual practice.

STATISTICAL ANALYSIS

Paired t tests were used to test the hypothesis that there was a difference in the IOPs of the eyes on the injured and uninjured sides of the face. Nonparametric statistics were used to account for the non-normality of the IOP data. The Wilcoxon rank-sum test was used to test for differences at each time point. For results to be considered statistically significant, *P* values less than .05 were required.

Results

Of the 20 patients, 19 were male and 1 was female with the average age of 33 years (range, 16 to 54 years). Time from injury until presentation averaged 4 days (range, 0 to 20 days), the interval from presentation to operation averaged 6 days (range, 0 to 12 days). At the time of initial presentation 15 patients had inferior orbital nerve paraesthesia, 12 had subconjunctival hemorrhage, 2 had diplopia, 1 had exophthalmos, none had enophthalmos, and 18 had clinically obvious malar depression.

Five of the orbito-zygomatic complex fractures were repaired with a Gillies approach and lift[16] with no internal fixation deemed necessary. The other 15 patients underwent open reduction and internal fixation of the zygomaticomaxillary buttress and/or the fronto-zygomatic buttress and/or the inferior orbital rim. Two cases required orbital floor repair with outer table calvarial bone. For these 2 cases, at time point 3, IOP readings were carried out immediately after orbito-zygomatic fracture reduction but before orbital floor exploration and calvarial bone grafting. Three surgeons performed all of the operations. There were no intraoperative complications.

IOP readings for the 5 time points for both the control and injured eye were examined. Paired t tests were used to test the hypothesis that there was a difference in the IOPs of the eyes on the injured and uninjured sides of the face (Table 1). Initially, each time point was considered separately. There were no significant differences between the injured and control data at any time point. The data differences between control and injured sides over time indicated a quadratic relationship. Average IOP was greater in the injured eye than in the control at all times except for time point 3 (immediately after fracture reduction). Average IOPs for the control and injured eye decreased from time 1 to time 2. Interestingly, IOP increased in both eyes during the operation but more so in the control eye than the injured eye. However, the medians within the groups at each time point indicated that these changes are probably due to a few patients having large increases during the operation (Fig 1). Nonparametric statistics were used to account for the non-normality of the orbital pressure data. The Wilcoxon rank-sum test was used to test for differences at each time point. The conclusions from these tests were the same as those drawn from the t tests. There was no statistically significant evidence of

Table 1. MATCHED PAIR TESTS FOR IOP DIFFERENCES BETWEEN CONTROL AND INJURED EYES

Time	Mean IOP (mmHg) (C)ontrol	Mean IOP (mmHg) (I)njured	Difference in Means Δ = C − I	95% CI(Δ) Lower	95% CI(Δ) Upper	Pairwise t Test (*P* Value)	Median Control	Median Injured	Sign-Rank test (*P* Value)
1	16.7	17.2	−.45	−3.79	4.68	.826	16	14.5	0.970
2	16.1	16.3	−.25	−2.35	2.85	.842	12.5	11.5	0.694
3	21.1	20.8	.3	−6.78	6.18	.924	15	15.5	0.708
4	16.4	19.7	−3.3	−3.49	10.09	.322	15	16.5	0.599
5	18.2	18.8	−.6	−5.27	6.47	.833	14	17	0.276

Paton et al. Intraocular Pressure Changes. J Oral Maxillofac Surg 2006.

a difference between the IOPs of the injured eye compared with the control eye.

There is evidence of an increased IOP in both eyes from time point 2 to time point 3. Both means and medians appeared to increase by similar amounts. A matched pairs t test of between these time points suggested there was no evidence of a change in IOP differences between the eyes (Table 2).

Discussion

This study evaluated IOP changes during elevation of orbito-zygomatic complex fractures, using the uninjured contralateral eye as a control. A previous study by Forrest et al[15] reported a statistically significant rise in IOP immediately after bone grafting orbital floor defects in 19 patients, which returned to normal within 30 minutes. All 19 patients had orbito-zygomatic complex fractures that required elevation and 3 point miniplate fixation before orbital floor reconstruction with split calvarial bone. Control measurements were obtained from the opposite, non-operated orbit and from a comparison group consisting of 16 patients who had orbito-zygomatic complex fractures that also required elevation and 3 point miniplate fixation, but no orbital floor grafting. The same study also reported statistically significant lower preoperative IOP readings in orbito-zygomatic complex fractures requiring orbital floor repairs compared with orbito-zygomatic complex fractures not requiring orbital floor repair. It was postulated that this was because larger defects of the orbital floor allow decompression of the periorbital tissues. The assumption being that a lower intraorbital pressure translates to a lower IOP. The 2 subjects in the current study that required orbital floor repairs had injured eye IOP readings at time point 1 of 9 and 13 mmHg compared with the mean of 17.5 mmHg.

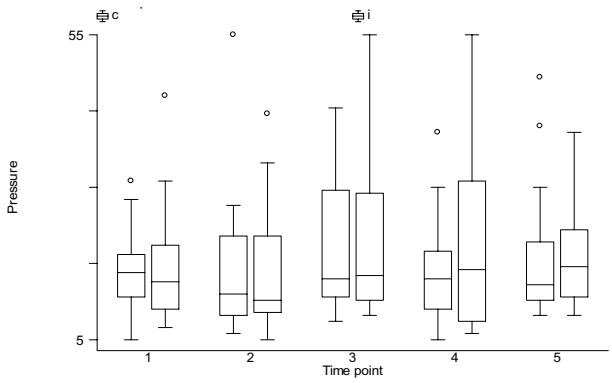

FIGURE 1. Distribution of IOP (mmHg) in control (*left plot*) and injured (*right plot*) eyes over 5 time points.

Paton et al. Intraocular Pressure Changes. J Oral Maxillofac Surg 2006.

Pressure changes within the orbital compartment, in the context of facial trauma, and its subsequent effect on the IOP and retinal microcirculation have not been fully elucidated. A distinction must be made between IOP and intraorbital pressure. IOP, or pressure within the globe, is normally within the range of 10 to 20 mmHg.[17] Intraorbital pressure is a measure of periorbital tissue pressure within the orbital com-

Table 2. MATCHED PAIR TESTS FOR IOP DIFFERENCES BETWEEN TIME POINTS 2 AND 3 IN CONTROL AND INJURED EYES

Eye	Mean IOP (mmHg) Time 2	Mean IOP (mmHg) Time 3	Difference in Means Δ = t3 − t2	95% CI(Δ) Lower	95% CI(Δ) Upper	Pairwise t Test (*P* Value)	Median Control	Median Injured	Sign-Rank Test (*P* Value)
C	16.1	21.1	5.05	−0.31	10.41	0.06	12.5	15	0.100
I	16.3	20.8	4.50	−1.99	10.99	0.16	11.5	15.5	0.231

Paton et al. Intraocular Pressure Changes. J Oral Maxillofac Surg 2006.

partment. Reimann et al[18] introduced an orbital manometer into the orbital compartment of healthy patients to determine pressure before and after a retrobulbar injection of 5 mL of anesthetic. The data collected showed a resting orbital compartment pressure of 4.0 ± 1.5 mmHg, which increased to 11.6 ± 2.6 mmHg, 5 minutes after the injection of 5 mL of anesthetic. An earlier study using a "split-catheter" technique also showed similar resting orbital compartment pressures, with a range of 3.0 to 6.0 mmHg.[19]

Forrest et al[15] also conducted a laboratory study where intraocular and intraorbital compartment pressures were measured during sequential orbital volume reduction in 3 groups of New Zealand white rabbits with intact orbits, acute orbital wall defects, and chronic orbital wall defects. It is interesting to note that IOP was significantly elevated in all 3 groups compared with non-operated control orbits; however, intraorbital pressure values did not change significantly from control levels throughout the grafting sequence. This result also raises questions regarding the relationship between orbital compartment pressures and IOPs.

In the present study, average IOP decreased from time point 1 to time point 2, indicating IOP changes caused by both the effect of the general anesthetic and postural changes. Mean IOP then rose in both the injured (16.3 mmHg to 20.8 mmHg) and non-injured (16.1 mmHg to 21.1 mmHg) orbits immediately after orbito-zygomatic fracture reduction. This result suggests that a neural component or other physiologic parameters may be responsible for IOP changes independent of orbital compartment pressure changes. This finding is at odds with that of Forrest et al,[15] who found that during orbital floor grafting the uninvolved eye IOP did not rise with the operated eye IOP (10.2 ± 1.2 mmHg vs 14.9± 1.2 mmHg).

In summary, there is no statistical evidence of a difference between the IOPs of the involved and control eyes. Parametric and nonparametric matched pair tests gave the same conclusion. There was no statistically significant evidence of a change in injured eye IOP between time point 2 and time point 3. Routine IOP testing is not indicated during orbito-zygomatic fracture reduction. Changes in orbital compartment pressure may not translate to equivalent changes in IOP. Further research measuring both the orbital compartment pressure and IOP, before and after elevation of orbito-zygomatic complex fractures is needed.

References

1. Ord RA: Post-operative retrobulbar hemorrhage and blindness complicating trauma surgery. Br J Oral Surg 119:202, 1981
2. Nicholson DH, Guzak SW: Visual loss complicating repair orbital floor fractures. Arch J Ophthalmol 86:369, 1971
3. Emery JM, Huff JD, Justice J Jr: Central retinal artery occlusion after blow-out fracture repair. Am J Opthalmol 78:538, 1974
4. Lederman IR: Loss of vision associated with surgical treatment of zygomatic-orbital floor fracture. Plast Reconstr Surg 68:94, 1981
5. Gordon S, Macrae H: Monocular blindness as a complication of the treatment of a malar fracture. Plast Reconstr Surg 6:228, 1950
6. Liu D: Blindness after blow-out fracture repair. Ophthalmic Plast Reconst Surg 10:206, 1994
7. Li KK, Meara JG, Joseph MP: Reversal of blindness after facial fracture repair by prompt optic nerve decompression. J Oral Maxillofac Surg 55:648, 1997
8. Smith B, Lisman RD, Simonton J, et al: Volkmann's contracture of the extraocular muscle following blowout fracture. Plast Reconstr Surg 74:200, 1984
9. Huang TT, Horwitz B, Lewis SR: Retrobulbar hemorrhage. Plast Reconstr Surg 59:39, 1977
10. Li KK, Meara JG, Rubin PAD: Orbital compartment syndrome following orthognathic surgery. J Oral Maxillofacial Surg 53: 964, 1995
11. Katz B, Herschler J, Brick DC: Orbital haemorrhage and prolonged blindness: A treatable posterior optic neuropathy. Br J Ophthalmol 67:549, 1983
12. Miloro M, Ghali GE, Larsen PE, et al: Peterson's Principles of Oral and Maxillofacial Surgery (ed 2). Hamilton, BC Decker, 2004, pp 472
13. Ffytche TJ, Bulpit CJ, Kohner EM, et al: Effect of changes in intra-ocular pressure on the retinal microcirculation. Br J Opthalmol 58:514, 1974
14. Hayreh SS, Zimmerman MB, Kimura A, et al: Central retinal artery occlusion. Retinal survival time. Exp Eye Res 78:723, 2004
15. Forrest CR, Khairallah E, Kuzon WM: Intraocular and intraorbital pressure compartment changes following orbital bone grafting: A clinical and laboratory study. Plast Reconstr Surg 104:48, 1999
16. Gillies HD, Killer TAP, Stone D: Fractures of the malar-zygomatic compound: With a description of a new x-ray position. Br J Surgery 14:651, 1927
17. Kaufman PL, Alm A, Alder FH: Adler's Physiology of the Eye: Clinical Application (ed 10). St Louis, MO, Mosby, 2003
18. Reimann CD, Foster JA, Kosmorsky GS: Direct orbital manometry in healthy patients. Ophthal Plast Reconstr Surg 15:121, 1999
19. Kratky V, Hurwitz JJ, Avram DR: Orbital compartment syndrome. Direct measurement of orbital tissue pressure: 1. Technique. Can J Ophthalmol 25:293, 1990