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BLOWOUT FRACTURE

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Abstract

This paper reviews 29 cases of blowout fracture seen over a three year period. It outlines the theories on the mechanism of blowout fracture and concludes that it is most likely caused by a combination of bone and global force transmission. The ocular motility restriction is no longer thought to be due to muscle entrapment, but rather traumatic disruption to the connective tissue septa and the orbital tension, either by entrapment or fibrosis. Ten patients were noted to have visual field damage and emphasis is placed on routine visual field testing at the initial examination. The poor results achieved in young patients with blowout fracture were consistent with other surveys.

Key words: *Blowout fracture, mechanism, connective tissue, optic nerve damage, childhood blowout.*

INTRODUCTION

This paper presents a clinical overview of twenty nine patients with blowout fractures seen over a three year period. It details the clinical findings and discusses current thoughts on the mechanism of the fracture and its management.

Blunt trauma to the periorbital region may result in fractures of the orbital bones and or soft tissue damage. 'Blowout' fracture is a term first introduced by Smith and Regan in 1957[1] after a cadaver experiment. It was initially used to describe a traumatic fracture of the orbital floor with the rim intact. The term is now used in a less specific sense and encompasses similar fractures of other orbital bones. It is important to differentiate the two types of blowout fracture:

1. Pure blowout — rim intact
2. Impure blowout — rim involved.

ANATOMY

It is relevant to consider the anatomy of the bony orbit and some of its important relations, as understanding of this sheds some light on the nature of blowout fracture and the clinical consequences.

Seven bones take part in the formation of the orbit (Wolff):[2]

ROOF — consists of two bones, the major portion being formed by the orbital plate of the frontal bone. It is extremely thin, translucent and fragile, except where it is formed by the lesser wing of the sphenoid bone.[2] Traumatic fracture of the roof is uncommon and complex. It occurs as a result of high velocity trauma and the thin orbital plate of the frontal bone tends to be displaced downwards and is termed a 'blow in' fracture. It is not included in this paper.

MEDIAL WALL — consists of four bones, the largest of which is the orbital plate of the ethmoid bone. This is by far the thinnest orbital wall. Isolated blowout fracture of the medial wall is uncommon. Supposedly the numerous bony

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septa of the ethmoid air cells give the lamina some support. The continuity of the orbital plate of the ethmoid with the thin unsupported portion of the orbital floor facilities blowout in this area.[3] The optic foramen is at the posterior limit of the orbital plate of the ethmoid and therefore severe fractures may result in optic nerve damage. The naso-lacrimal duct and its bony canal are vulnerable in medial wall fractures.

FLOOR — consists of three bones, the largest of which is the orbital plate of the maxilla. The floor is separated from the medial wall by a fine suture and this area is vulnerable to blowout fracture. The floor lies over the maxillary sinus and is therefore unsupported. The infra orbital canal runs in the floor of the orbit and opens at the infra orbital foramen. It transmits the infra orbital nerve and anaesthesia of this is a frequent sign of blowout.

LATERAL WALL — formed by two bones. This is the thickest of the orbital walls and is especially strong at the orbital margin. The most posterior portion is the thinnest, and therefore more vulnerable to blowout fracture.

ORBITAL MARGIN — formed by the frontal bone superiorly, and by the zygomatic bone and the maxilla inferiorly. The strong superior orbital rim protects the thin orbital roof. A fractured zygoma is frequently encountered in impure blowout.

MECHANISM OF INJURY

The mechanism of blowout is a controversial issue. There are two main theories, the hydraulic theory and the buckling theory, both proposed as a result of cadaver experiments.

1. HYDRAULIC THEORY: Proposed by Smith and Regan in 1957.[1] Basically the theory proposes that blunt trauma transmits a force to the globe. The resulting increase in the intra orbital pressure results in fractures of the weaker plates of the maxilla and ethmoid. The orbital contents are forced into the fracture site.
2. BUCKLING THEORY: Proposed by Fujino in 1974.[4]

   This theory proposes that the blunt trauma transmits a force to the bony orbital rim, resulting in a transient deformation, with transmission of the force to the weaker portions of the orbit. The entrapment of soft tissue is explained by the different rate of recovery of bone and soft tissue after impact,[5] the recovery of soft tissue being slower than bony structures.

In a recent summary of current theories of the mechanism, Kersten[6] proposed that an isolated hydraulic effect, which is probably the most widely accepted theory at the moment, is highly unlikely. He feels it is inconsistent with the clinical findings in three major areas.

1. The very nature of the trauma would impact globe and rim. Very few injuries isolate the globe on impact force.
2. The mechanism of hydraulic force would imply a considerable force on the globe itself. Fujino,[5,7] proposes hydraulic pressure requires a force up to ten times greater than the force required to produce buckling of the orbital floor.[8] Thus one would expect a high incidence of ocular injuries with hydraulic transmission, but this is not the case in most surveys.
3. The theory of hydraulic transmission would imply that the thinnest and weakest bones would be affected. The most common site for blowout is the posterior medial aspect of the floor.[9] There is a low incidence of isolated medial wall blowout, although this is by far the thinnest bone.

Blowout fracture can occur from a variety of mechanisms and a combination of the hydraulic and buckling theory would appear to be the most compatible with the clinical findings in most surveys.

CLINICAL TESTS

Careful clinical assessment and accurate recording of data are of great importance in the diagnosis and management of blowout fracture. The aetiology of blowout points to the potential for legal compensation claims and this aspect also necessitates a comprehensive initial examination.

Ten of the twenty nine patients in this series became compensation claims. Following orthoptic/ophthalmic assessment of corrected near and distance visual acuity, media and fundus examination, the following clinical diagnostic tests should be done routinely —

DIAGNOSTIC TESTS

1. Observation — infra orbital nerve anaesthesia
   — retraction
   — enophthalmos
2. Cover test — near and distance measurements.
3. Binocular single vision assessment (if possible) — as it may shed light on pre existing problems.
4. Diplopia — A. Hess
   — B. Field of binocular single vision.
5. Ocular motility.
6. Visual field assessment.

X-RAYS, COMPUTER TOMOGRAPHY, MRI

These tests are all methods of producing visualisation of anatomical changes in blowout fracture.

1. X-rays
   These will provide information on facial and rim fractures and are often ordered initially. However they are not reliable in pure blowout. Occasionally antral or ethmoidal opacification gives a clue to blowout.

2. CT scans
   Most studies point to CT scans as providing the most informative diagnostic pictures.[10] A good coronal CT scan can show fracture site, extent of the fracture and soft tissue information. Although some clinicians feel it is unnecessary, there is an argument for routine CT scanning of all suspected blowouts as it removes the speculation (Figure 1).

3. MRI
   This is generally thought to be complimentary to CT scans rather than a superior test. It gives soft tissue information while CT scans give higher bone resolution.

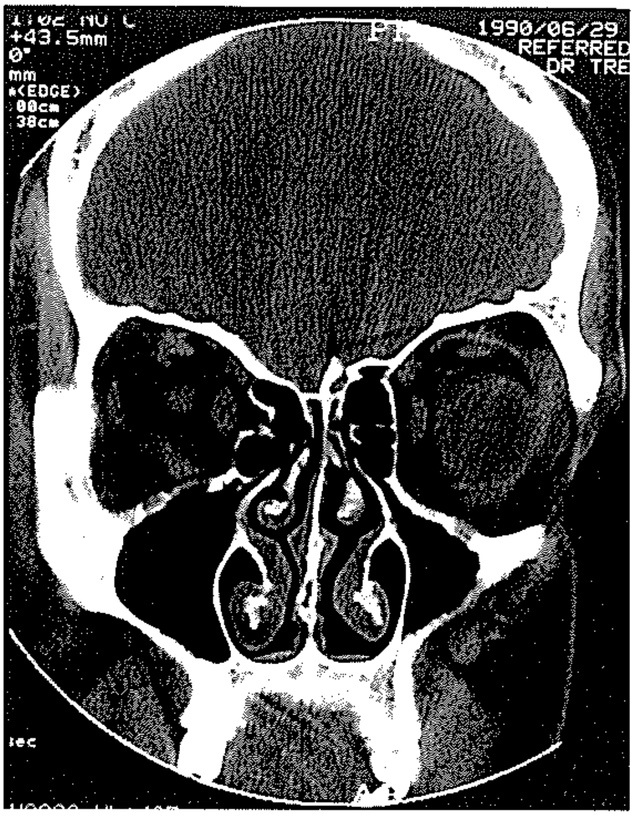

Figure 1. CAT scan of patient with left medial wall blowout. Fracture site and extra ocular muscles are clearly visible.

CLINICAL RESULTS

Twenty nine patients were examined the age range being seven years to fifty three years, with a mean age of twenty seven years. The results are analysed in the following Tables 1, 2 and 3.

TABLE 1
Distribution of Blowout Fracture

Unilateral	Bilateral	Right Eye	Left Eye	Male	Female
28	1	19	11	25	4

TABLE 2
Aetiology and Corresponding Type of Blowout Fracture

Aetiology		Pure Blowout	Impure Blowout
Motor vehicle accident	8	3	6
Assault	8	5	3
Football	5	3	2
Fall	5	4	1
Other	3	2	1

TABLE 3
Fracture Site and Incidence of Fractured Zygoma

Fracture Site		# Zygoma
Orbital floor	20	7
Floor and medial wall	6	4
Floor and lateral wall	3	2
Medial wall	1	

CLINICAL FINDINGS AND DISCUSSION

Classically the patient presents with a history of blunt trauma and may have local soft tissue signs such as periorbital haematoma, oedema, or subconjunctival haemorrhage. A subconjunctival haemorrhage with no posterior limit implies a fracture may be present as the blood may have tracked forward from the posterior fracture site.[11]

Patients with medial wall blowout fracture may present with subcutaneous emphysema or more seriously cerebro spinal fluid rhinorrhea. Trauma disrupts the meninges and cerebro spinal fluid leaks into the nose.

OPHTHALMIC SIGNS

Funduscopy revealed retinal damage in five of the twenty nine patients.

Retinal haemorrhage	1
Macula haemorrhage	1
Disc haemorrhage and chorioretinal tear	1
Retinal oedema	1
Macula oedema	1

Moderate to full traumatic dilatation of the pupil may occur with absent or diminished reactions to direct and consensual light reflexes. Five patients in this series showed a dilated pupil on initial examination.

VISUAL ACUITY

Traumatic impact on the globe may cause decreased visual acuity due to such complications as hyphema, lens dislocation, retinal tears and retinal or macula oedema.

In this series nine patients showed decreased acuity in the affected eye on initial examination although in five of these the acuity was reduced only to 6/9. The visual acuity loss was transient in seven of the cases. One patient had permanent visual acuity loss and one patient was lost to follow up.

DIPLOPIA

Diplopia was present on initial examination in all twenty nine patients. Diplopia is initially influenced by soft tissue swelling, and in some cases may be constant rather than confined to certain positions of gaze. It is typically vertical and may reverse when moving from elevation to depression. A horizontal component need not necessarily imply a medial or lateral wall blowout, as horizontal recti have connective tissue septa extending to the orbital floor (Koorneef).[12]

In this series the diplopia pattern was:

Purely vertical diplopia	22
Purely horizontal diplopia	1
Vertical and horizontal diplopia	6

It is important to remember diplopia may occur due to decompensation of a pre existing phoria or tropia, or traumatic damage to an ocular muscle or nerve (Table 4).

OCULAR MOTILITY

The concept of true muscle entrapment following blowout has been questioned. It is not consistent with diplopia patterns, with findings on high resolution CT scans or with surgical observations.

Koorneef,[12] in anatomical studies details a highly organised connective tissue septa around each eye muscle and between the eyeball and the

TABLE 4
Pre Existing and Acquired Palsies Associated with Blowout Fracture

Pre Existing Problems	No. of Cases	Acquired Palsies	No. of Cases
Congenital fourth nerve palsy	1	Seventh nerve palsy	2
Microtropia	2	Fourth nerve palsy	3
Intermittent divergent squint	1	Inferior rectus palsy	1

orbital walls. He feels it is disruption to this tissue septa and to the orbital tension that produces ocular motility imbalance. Some authors[13] state the connective tissue septa is so highly organised that it is possible to predict the fracture sight from the motility imbalance. In summary:

Reasons for Ocular Motility Imbalance:
1. Soft tissue oedema and haemorrhage.
2. Incarcerated tissue.
3. Herniation of orbital fat/connective tissue into the maxillary antrum. In rare cases it may be muscle.
4. Post traumatic oedema and scarring of tissue and fat causing disruption to orbital septa tension.
5. Traumatic muscle/nerve palsy.

In this series the ocular motility pattern was:

Restricted elevation	4
Restricted depression	4
Restricted adduction and abduction	1
Restricted elevation and depression	14
Restricted elevation, depression and horizontal movement	6

INFRA ORBITAL NERVE ANAESTHESIA

The infra orbital nerve is a branch of the Maxillary Division of the 5th cranial nerve. It runs forward from the inferior orbital fissure and travels in the floor of the orbit, first in a groove, then a canal and emerges on the face through the infra orbital foramen.[2] It is commonly affected in blowout directly from trauma to the floor, zygoma or maxillary region or from the surgery. Clinically it is important to test for anaesthesia in the areas it supplies.

The nerve supplies sensation to the skin and conjunctiva of the lower lid, the lateral aspect of the nose, the anterior cheek and upper lip. The anterior superior alveolar nerve descends from the infra orbital nerve into or along the maxillary bone and supplies sensation to the gums and anterior three teeth on the same side.[14] These may also be numb and indicate a fracture of the anterior plate of the maxillary bone or of the maxillary wall.

In this survey twenty patients had infra orbital nerve anaesthesia to some degree — one of these being a surgically induced trauma. Nine patients still had persistent anaesthesia on their final check.

ENOPHTHALMOS

Enophthalmos has been defined as a difference of greater than 2 mm in the position of the globes along the antero-posterior axis. Fells reported that a difference of greater than 3mm constitutes a cosmetic problem.[15] The cause is a discrepancy between the volume of the bony cavity and the volume of its contents.[16]

Reasons for Enophthalmos:
1. Enlargement of the bony cavity i.e. increased bony cavity volume.
2. Displacement of soft tissue out of the orbit — usually herniation of contents into the maxillary sinus i.e. decreased content volume.
3. Tethering of the globe posteriorly by trapped orbital fat or tissue.
4. Fat necrosis and decreased soft tissue volume.

Bite[16] performed three dimensional CT scans to measure cavity and soft tissue volumes in patients with established enophthalmos. Their results suggest that in the majority of cases post traumatic enophthalmos is caused by increased bony cavity volume rather than soft tissue loss. In the cases with no volume discrepancies, tethering and contracture of retrobulbar tissue were thought to be responsible. Only two patients in this series developed marked enophthalmos.

RETRACTION

Retraction of the globe can occur when the eye moves in a direction opposite to the trapped tissue or fibrosis. Absence of retraction provides a better prognosis for spontaneous recovery of ocular movements in pure blowout.[11] Blowout fracture of the medial wall may have retraction on abduction or more rarely adduction. This is sometimes termed 'Acquired Retraction Syndrome'. This occurred in one patient in this series (Figure 2).

VISUAL FIELD LOSS

In this series twenty seven patients were tested

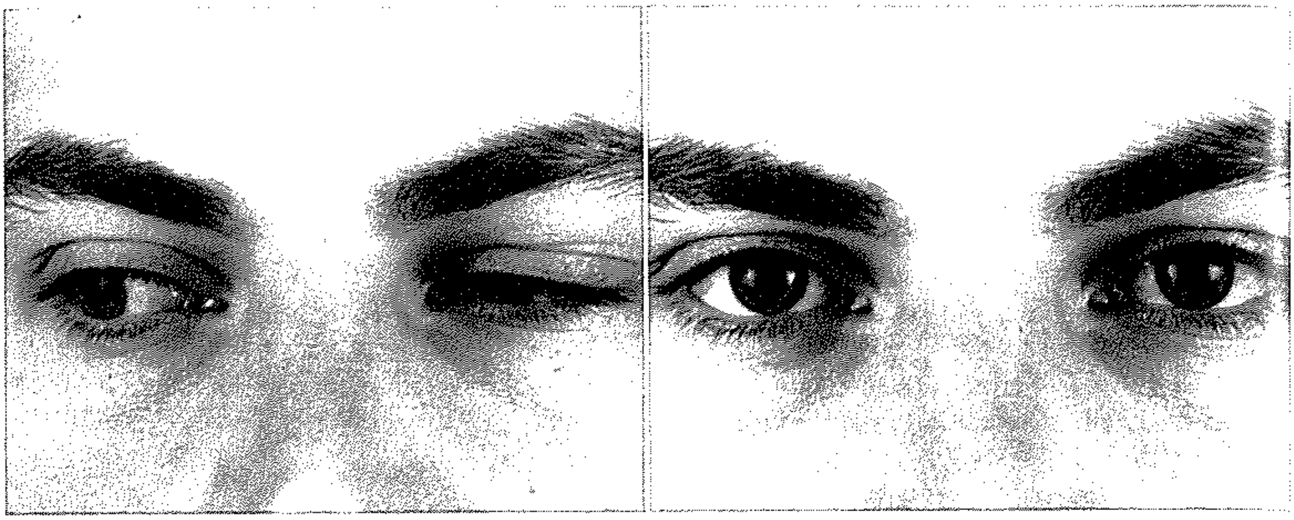

Figure 2. Patient with Left Acquired Retraction Syndrome in primary position and dextro gaze.

on the Goldmann perimeter. Ten patients showed varying degrees of visual field disturbance that related to optic nerve trauma. It was permanent in nine of them.

Optic nerve damage may occur due to:

1. Direct damage to the nerve from traumatic force.
2. Shearing injuries.
3. Compression of the nerve at the apex.
   A. Haemorrhage
   B. Bone fragments
   C. Increased orbital content volume e.g. post bone grafting.

Of the ten patients with field disturbance six were tested pre-operatively and the remaining four post operatively. This occurred as two were late referrals and two were tested early in the series, before the importance of routine testing became obvious.

It is not possible to pinpoint the exact aetiology in each case. However, four patients with known orbital apex trauma all sustained marked defects extending from the disc to the periphery. One patient with a normal field pre-operatively sustained optic nerve trauma in the post operative phase.

MANAGEMENT

In discussing management of blowout fractures, both pure and impure must be discussed as separate clinical entities. The management approach to each is quite different, although good ocular motility without enophthalmos is a common goal.

IMPURE BLOWOUT

Impure Blowout is essentially a surgical problem and is managed by ocular plastic surgeons. The rim is repaired, any accompanying depressed zygoma fracture is elevated and repaired and the orbital floor is explored and repaired at the same time.

Some aspects of the surgical management:

1. Lower Lid Incision — this is the common approach.
   (a) Lash margin
   (b) Orbital rim margin
   (c) Lid fold margin (between a & b)
2. Caldwell — Luc Approach — via the mouth above the canine and second molar teeth, to the maxillary antrum. This is a difficult approach and may cause damage to the orbital contents, alveolar nerve or in children the undescended molars.
3. Medial Canthal Incision — is commonly used to explore a medial wall. Sometimes a bicoronal flap is used to provide wide exposure of a medial wall fracture.

IMPLANTS USED

1. Silastic sheeting for smaller fractures.
2. Autogenous bone grafting for larger fractures.

BONE GRAFTING SITES

1. Calvarial bone grafts are usually from the parietal bone. The advantage of these grafts is that they are vascularised and do not resorb. The disadvantage is that they do not bend and are not easily contoured.
2. Split rib grafts are selected because the site is easily accessible, and has the ability to regenerate. Another advantage is that they are easily contoured and thus ideal for reconstructing curved surfaces such as the orbital floor.
3. Iliac crest or hip grafts are again easily accessible. Some consider they resorb more readily than calvarial grafts.

The twelve impure blowouts in this series all had surgery by plastic surgeons. Nine had implants and three rim repair only.

PURE BLOWOUTS

This was once considered an urgent surgical problem. However, recent studies on the anatomical structures involved and better understanding of the problem have led to a much more conservative approach. Reports by Putterman[17] and others suggest many pure blowouts recover satisfactorily without surgical intervention. The most sensible criteria for intervention advocated by two British surveys are:[18,19]

1. Diplopia not resolving within 14 days.
2. Fractures with large herniation.
3. Incarceration with global retraction.
4. Enophthalmos. 3 mm or more.

TIMING

The optimal time for surgery is within the first fourteen days and if left longer than two months the prognosis is poor.

In this series there were seventeen pure blowouts and eleven of these required surgery. Eight patients had surgery within the first week, one patient within two weeks and two patients within nine weeks.

While these findings indicate that a high percentage of patients required surgery, after studying the data, four of those operated on within the first week had serious blowout fracture which required further surgery.

MANAGEMENT RESULTS

IMPURE BLOWOUT	12 cases
Complete Recovery	3 cases
Good Recovery	3 cases (minimal residual problems)
Symptoms	3 cases
Failed to attend	3 cases

PURE BLOWOUT	17 cases
Complete Recovery	4 cases
Good Recovery	5 cases
Symptoms	6 cases
Failed to Attend	2 cases

BLOWOUT FRACTURE IN CHILDHOOD

Blowout fractures in childhood, as with facial fractures are relatively uncommon.[20] One reason for this may be that the facial bones in children are softer and more elastic than in adults. A second reason often given is that blowout fracture cannot occur until the sinuses have developed. Wolff[2] states that the sinuses are very

TABLE 5
Results of Children with Blowout Fracture

Age	Injury	Fracture Site	Treatment	Results
F 8	MVA	Medial floor	Silastic implant ten days later	Post op poor depression then lost to follow-up
M 9	Elbow and fall	Floor — apex	Two explorations. One (with implant) after one day and one six weeks later	1. Gross U/A elevation 2. U/A depression 3. Inferior field loss
M 9	Fall	Medial floor	Exploration after one day and tissue freed	1. Gross U/A elevation 2. Retraction

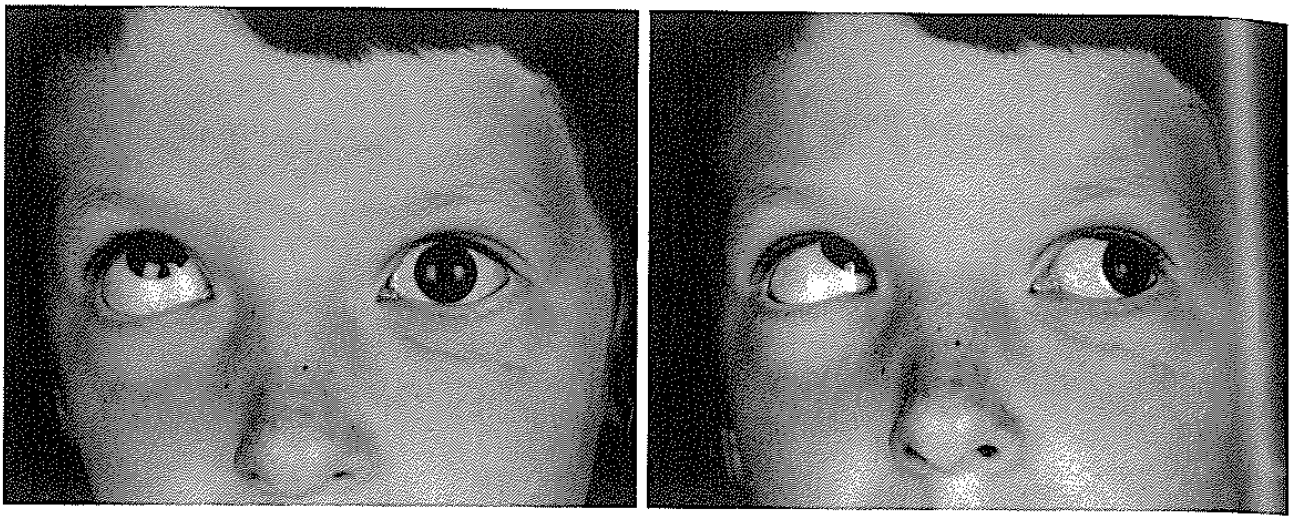

Figure 3. Restriction of left elevation in child following blowout fracture repair.

small in the young and start to increase in size around seven to eight years of age. However, children as young as three and certainly around seven to eight years can show clearly defined maxillary sinuses on CT scans.

When blowout does occur in the young child, the prognosis is often very poor. Studies by McCarry[21] and others have concluded that younger patients have a poorer prognosis particularly where there is limitation of ocular movement and retraction. Waddel[19] and others have proposed that scar tissue may develop more quickly in the young child.

In this series three children had blowout fracture. Of the two children who had a long term follow up the results were disappointing, showing marked ocular motility disturbances (Table 5) (Figure 3).

CONCLUSIONS

Experiments have shown that blowout fracture may be caused by a force to the globe, to the rim, or both. The data in this paper agrees with Kerstens[6] conclusion that isolated hydraulic pressure alone is unlikely to be the cause of most pure blowouts.

There was a high incidence of visual field damage especially with apex fractures. It can occur as a result of surgery, and it is important to test visual fields on initial examination if possible.

Children in this survey responded poorly to the treatment of their blowout fractures. This occurred in spite of early surgical intervention and the reasons for this are not as yet fully understood.

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