THE OPHTHALMOLOGICAL CONSEQUENCES

OF MIDFACIAL TRAUMA

VOLUME ONE

BY
Isam A Al-Qurainy
MB ChB MCOphth DO

A Thesis Submitted in Two Volumes for the Degree of PhD in Ophthalmology

To

The University of Glasgow

On the Basis of Research Conducted at the Tennent Institute of Ophthalmology and the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital, Glasgow.

Submitted October, 1992

Copyright (c) 1992, By I A Al-Qurainy

ProQuest Number: 11007684

All rights reserved

INFORMATION TO ALL USERS
The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript
and there are missing pages, these will be noted. Also, if material had to be removed,
a note will indicate the deletion.

ProQuest 11007684

Published by ProQuest LLC (2018). Copyright of the Dissertation is held by the Author.

All rights reserved.
This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Thesis
9738
Copy 1
Vol 1

GLASGOW
UNIVERSITY
LIBRARY

C O N T E N T S

PART ONE

OCULAR ABNORMALITIES AND MOTILITY DISORDERS FOLLOWING MIDFACIAL TRAUMA

PART TWO

THE ROLE OF QUANTITATIVE TESTS IN THE DIAGNOSIS OF MOTILITY DISORDERS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGMENTS

It has been a pleasure to work with distinguished and understanding colleagues who made the production of this thesis possible.

I shall first express my gratitude to Professor W S Foulds for offering me the opportunity to conduct this study and supporting me over the years.

I am indebted to Dr G N Dutton whose generous support as a supervisor and a friend is unrivalled. His constructive criticism, encouragement and practical advice are always at hand and I shall ever be grateful to him.

My sincere thanks and appreciation go to Mr K F Moos and Mr A El-Attar, Consultant Oral and Maxillofacial Surgeons, for allowing me to examine their patients at Canniesburn Hospital and for lending me their continuous support. I am also grateful to my friends and colleagues, members of the medical/dental staff, whose collaboration in referring their patients to me provided me with the material of my study.

The forced duction equipment described in this thesis has been developed in collaboration with Mr M F Al-Hinnawi, my fellow researcher at the Department of Bioengineering at Strathclyde University. His contribution and enthusiasm were indispensable for the completion of this work. Professor J C Barbenel of the same department has given us much encouragement and support, and I am grateful to him.

I must thank Mr G Heron (Senior Lecturer, Department of Ophthalmic Optics, Glasgow Polytechnic) for his help in allowing the manufacture of the contact lenses in his department's workshop.

I am grateful to Professor M Titterington for his generous assistance with the statistical analysis in relation to the construction of the scoring system.

My thanks extend to Dr Donald Allan, Senior Physicist, Eye Department, Western Infirmary, for his help and guidance with regard to sorting out the clinical data on the computer.

Finally, I wish to thank all my friends and fellow researchers/postgraduate students who acted as my normal control subjects for the forced duction/force generation experiments. Their cooperation is greatly appreciated.

DECLARATION

The work described in this thesis is based upon the results of research conducted at the Tennent Institute of Ophthalmology, the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital, and the Bioengineering Department at Strathclyde University, Glasgow, between June, 1986 and September, 1990.

The protocol of the study was devised by the author who carried out all the examinations, clinical experiments and analyses of data apart from those which are appropriately acknowledged overleaf or in the text.

Supervision was provided by Dr G N Dutton.

The text of this thesis was written solely by the author.

Isam A Al-Qurainy
October, 1992

PRESENTATIONS TO LEARNED SOCIETIES AND PUBLICATIONS

Presentations:

1986, October	The Association of Ocular Injuries with Maxillo-facial Trauma: A Preliminary Report. The Scottish meeting of Oral and Maxillofacial Surgeons, Glasgow.
1986, November	The Association of Ocular Injuries with Maxillo-facial Trauma: A Preliminary Report. The Dutch meeting of Oral and Maxillofacial surgeons, Rotterdam.
1989, October	Symptomatic Diplopia Following Midfacial Trauma. The Autumn meeting of the British Association of Oral and Maxillo-facial Surgeons, London.
1989, October	The Association of Ocular Injuries with Midfacial Trauma. The Autumn meeting of the British Association of Oral and Maxillofacial Surgeons, London.
1989, November	Symptomatic Diplopia Following Midfacial Trauma. The Scottish-Irish meeting of Oral and Maxillofacial Surgeons, Glasgow.
1990, March	New Equipment for the Forced Duction and Force Generation Tests. Glasgow Ophthalmic Research Forum. Tennent Institute of Ophthalmology, Glasgow.
1991, May	The Fractured Orbit. Who should be referred for an Eye Opinion? The Annual Congress of the College of Ophthalmologists, Glasgow.
1991, May	Al-Hinnawi, M F, Al-Qurainy, I A & Barbenel, J C (1991). New Design for Carrying Out Forced Duction and Force Generation Tests. Poster. The Annual Congress of the College of Ophthalmologists. Glasgow.

Publications:

Al-Qurainy, I A, Dutton, G N, Moos, K F, Reynolds, S T & McMillan, N (1988). Orbital injury complicated by entrapment of the superior oblique tendon: a case report. **British Journal of Oral and Maxillofacial Surgery, 26,** 336-340.

Ilankovan, V, Al-Qurainy, I A, Moos, K F & Dutton, G N (1990). Acquired Brown's syndrome: Iatrogenic causes. **Journal of Oral and Maxillofacial Surgery, 48,** 420-424.

Dutton, G N & Al-Qurainy, I A (1991). Ophthalmic Consequences of Maxillofacial Injuries. In: Oral and Maxillofacial Trauma, ed. Fonseca, R J & Walker, R V. Ch 20, pp 543-72. Philadelphia: W B Saunders.

Al-Qurainy, I A, Stassen, L F, Dutton, G N, Moos, K F & El- Attar, A (1991). The association of ocular injuries with midface fractures. A prospective study. **British Journal of Oral and Maxillofacial Surgery, 29,** 291-301.

Al-Qurainy, I A, Stassen, L F, Dutton, G N, Moos, K F & El-Attar, A (1991). Diplopia following midfacial trauma. **British Journal of Oral and Maxillofacial Surgery, 29,** 302-307.

Al-Qurainy, I A, Titterington, D M, Dutton, G N, Stassen, L F, Moos, K F & El-Attar, A (1991). Midfacial fractures and the eye - The development of a system for detecting patients at risk of eye injury. **British Journal of Oral and Maxillofacial Surgery, 29,** 363-367.

Al-Qurainy, I A, Dutton, G N, Ilankovan, V, Titterington, D M, Moos, K F & El-Attar, A (1991). Midfacial fractures and the eye - The development of a system for detecting patients at risk of eye injury. A prospective evaluation. **British Journal of Oral and Maxillofacial Surgery, 29,** 368-369.

Al-Hinnawi, M F, Al-Qurainy, I A, Barbenel, J C & Dutton, G N. (1991). Quantitative mechanical assessment of the extraocular muscles. **Proceedings Part H, Journal of Engineering in Medicine, 205,** 101-108

Dutton, G N, Al-Qurainy, I, Stassen, L F A, Titterington, D M, Moos, K F & El-Attar, A (1992). Ophthalmic consequences of midfacial trauma. **Eye, 6,** 86-89.

SUMMARY

Ocular injuries commonly occur in patients with facial fractures.

A study was carried out of 363 patients presenting during a 2-year period with midfacial fractures. Patients underwent ophthalmological examination within one week of injury. The characteristics of the eye injuries sustained were related to the aetiology and type of fracture, and the sex and age of each patient.

Ninety-one percent of patients sustained ocular injuries of various severities. Sixty-three percent sustained minor injuries, 16% suffered moderately severe injury and 12% experienced severe injuries.

Road traffic accident was associated with the highest incidence of severe ocular disorder at 20% (9/45) followed by assaults at 11% (20/181). One third of all patients with comminuted malar fracture suffered a severe ocular disorder (9/27) whilst blow-out fracture came second at 16.7% (6/36).

Fifty-six patients (15.4%) had a decrease in visual acuity and 9 (2.5%) sustained significant traumatic optic neuro-

pathy. Decrease in visual acuity accompanied the majority of significant eye injuries. Road traffic accidents and assaults associated with alcohol abuse showed the highest incidence of major ocular dysfunction.

Motility disorder was also common. Seventy-two patients (19.8%) developed diplopia which was most common following road traffic accidents (31%) and least common with simple falls (10%). Blow-out fractures of the orbit (n=36) led to double vision in 58% of cases (n=21). Eighty-two per-cent of patients recovered from diplopia within six months of injury, 11% recovered within 6-12 months and 7% of patients took more than one year to recover including one patient who required squint surgery for double vision. The principal risk factors for diplopia comprise road traffic accidents, blow-out fractures and comminuted malar fractures. Early surgical reconstruction with conservative management of ocular motility disorders has, in this series, resulted in very few patients having diplopia in the long term.

Ocular injuries following facial trauma may be difficult to detect by the maxillofacial surgeon and may therefore be missed. Fifty-four parameters comprising maxillofacial, radiological and ophthalmic data were coded and recorded for each patient. These data were divided into predictors (the data potentially available to the maxillofacial surgeon) and outcome (the data potentially available to the ophthalmologist). Statistical methods of regression, and analysis of contingency tables, led to the identification of the principal predictors indicative of ophthalmic injury and thence to a scoring system which predicts the severity of such injuries. Impaired visual acuity was the principal predictor and when employed alone gave a sensitivity value of 80%. Pure blow-out fracture or comminuted facial fracture, double vision and amnesia, emerged as additional factors which yielded an efficient scoring system with a sensitivity of 89% and specificity of 90% for the population upon which it was based.

This scoring system was tested in a pilot study upon a new population of 100 similar individuals. The sensitivity value was 94.4% and the specificity value was 89%. Only one patient, warranting ophthalmic referral, was missed by the system whilst nine were incorrectly classified as warranting referral.

Defects of convergence and accommodation are common sequels of head injuries, the two functions failing usually to a commensurate degree although either may occasionally be separately deficient. A prospective evaluation of patients who have sustained midfacial trauma was carried out in order to determine the prevalence of impaired convergence and accommodation and to establish the risk factors for such defects.

Of 52 patients in this study, 11 suffered accommodation and/or convergence disturbances. They included 6 males (17% of the male population) and 5 females (29% of the female population) whose ages ranged from 13 to 72 years with a mean age value of 30.2 years. Nine of these 11 patients sustained their injuries due to alleged assaults (24.3% of all assaulted patients) and 2, following simple falls (25% of all fall victims).

Five patients complained of diplopia at near (5/11=45.5%) and another 4 of blurred vision and/or difficulty with reading (4/11=36.4%). The remaining 2 patients were asymptomatic.

Six patients were randomly selected to receive orthoptic exercises/treatment while the other five were monitored for signs of spontaneous recovery. Within six months of injury/surgery, 83% of the treated patients (n=5) and 80% of the non-treated patients (n=4) recovered to within the normal values of accommodation and convergence.

No significant statistical relationship was found between the incidence of accommodation and/or convergence failure, and the cause or the type of fracture sustained. It may, however, be related to the severity of impact and the associated closed head trauma.

A retrospective study of all patients treated for mid-facial fractures in the preceding 2 years (n=555) was performed. The majority of patients in this group were managed by the maxillofacial team only. Ophthalmic consultation and formal pre-operative ophthalmic assessment had been carried out in only 34 cases of midfacial fracture (6%).

Subconjunctival haemorrhage was the most frequently detected eye injury which affected 275 patients (49.5%). Pupils were tested for reaction to light and found to be either slow or non-reactive in 2% of all patients (n=12). Seventy patients (12.6%) were reported to have complained of diplopia while another 20 patients (3.6%) suffered of enophthalmos. Unspecified injuries to the globe were reported in 3 additional cases (0.5% of all patients). No other ocular or motility disorders were described.

Examination of data from the prospective and the retrospective series confirms that the accuracy of detection of ophthalmological disorder is higher when an ophthalmologist is involved in the clinical assessment of patients.

The active and passive behaviour of the extraocular muscles can be investigated by the forced duction and force generation tests. Review of previous work has highlighted the limitations of designs and methods used. A quantitative apparatus for carrying out both tests has been developed. Stages of development and test procedures are described. The test system described provides a reliable and acceptable means for carrying out both tests.

Test results from normal eyes fell within a relatively narrow range while patients' tests showed that specific ocular muscle deficits produced different patterns of results falling outside the normal range, and these were specific to the underlying pathology.

ABBREVIATIONS

ADC	Analogue to Digital Convertor.
ADC_{input}	Input from the analogue to digital convertor.
ADC_{output}	Output from the analogue to digital convertor.
CRP	Central Resting Position.
CT	Computed Tomography.
FDT	Forced Duction Test.
FGT	Force Generation Test.
F-Z suture	Fronto-Zygomatic suture.
kHz	kilohertz
kPa	kilopascal.
mN	milli-Newton = 10^{-3} Newton.
MRI	Magnetic Resonance Imaging.
mV	milli-Volt = 10^{-3} Volt.
NOE	Naso-Orbito-Ethmoidal.
PVD	Posterior Vitreous Detachment.
RPE	Retinal Pigment Epithelium.
rps	Revolution per second.
Type I #	Undisplaced malar fracture (any site).
Type II #	Zygomatic arch fracture only.
Type III #	Tripod fracture with undistracted fronto-zygomatic suture.
Type IV #	Tripod fracture with a distracted fronto-zygomatic suture.
Type V #	Pure blow-out fracture of the orbit.
Type VI #	Orbital rim fracture only.
Type VII #	Comminuted malar fracture.

CHAPTER 1

THE ASSOCIATION OF OPHTHALMIC INJURIES WITH MAXILLOFACIAL TRAUMA

1.1 INTRODUCTION

Facial fractures are frequently complicated by injury to the eye or its adnexa (Holt & Holt, 1983; Culbertson, 1983; Berine et al, 1981; Petro, 1979; Dastoor, 1975). If they are severe enough, such injuries may be detected with ease by any medical practitioner. Other injuries may either appear to be minor, or even be missed without specialised equipment and appropriate expertise. These may result in potentially preventable severe dysfunction of the visual apparatus if not diagnosed shortly after injury (Holt & Holt, 1983; Binder, 1978). Proper and timely simple diagnostic procedures followed by appropriate therapy may mean the difference between permanent loss of sight or recovery of useful vision (Records, 1979).

Estimates of the incidence of ocular disorder vary between 1% (McCoy et al, 1962) and 67% (Holt & Holt, 1983) and are dependent upon whether the evaluation is prospective or retrospective (Jabaley et al, 1975). Furthermore, the criteria employed to assess the severity of ocular disorder (Holt & Holt, 1983), and whether ophthalmologists or maxillofacial surgeons are responsible for carrying out the evaluation, significantly influence the interpretation of the results of such trials.

1.2 HISTORY

Ocular injuries have long been found in association with fractures of the facial skeleton especially those of the orbital floor (Fradkin, 1971).

Lang (1889) was the first to suggest that a fracture of the orbit may be responsible for the condition he called 'Traumatic Enophthalmos'. This was emphasized and further described by Pfiffer in 1943 and Converse and Smith in 1956, who later coined the term 'Blow-out Fracture'. King and Samuel (1944) reported a case of blow-out fracture caused by a sudden increase in the intra-orbital pressure which had resulted from a traumatic force to the soft tissue of the orbit. In 1957, Smith and Regan suggested that blunt trauma to the orbit resulted in increased pressure of the intra-orbital contents thereby causing a downward fracture of the thin walled orbital floor. Swelling and the effect of gravity combine to produce an expansion of the orbital contents into the maxillary sinus.

In recent years, a growing interest has developed in the relationship between facial trauma and the resulting ophthalmic disorders. However, a review of literature shows wide reported variations in the incidence of such injuries (Petro et al, 1979). Most authors mention only briefly or fail to characterize specific ocular complications (Gwyn et al, 1971). On the other hand ophthalmologists report high incidences of ocular injuries because of the nature of referral to their practices (Petro et al, 1979). In one published series, McCoy (1962), a plastic surgeon, noted only a 1% incidence of ocular injury in 855 patients with facial trauma. In contrast, Miller and Tenzel (1967) and Milauskas and Fueger (1966), all ophthalmologists, reported incidences of 17% and 14% in 30 and 84 patients respectively.

It is probable that the specialty of the authors involved (which may determine both the types of injury referred and the expertise in ophthalmic examination), is an important factor in the detection of ocular injury.

1.3 ANATOMIC CONSIDERATIONS

The anatomy of the face has been reviewed in detail by Schultz (1988), Haskell (1985) and Last (1984). The following description reviews the salient anatomical features of the eye and face with reference to the potential involvement of such structures in eye injury.

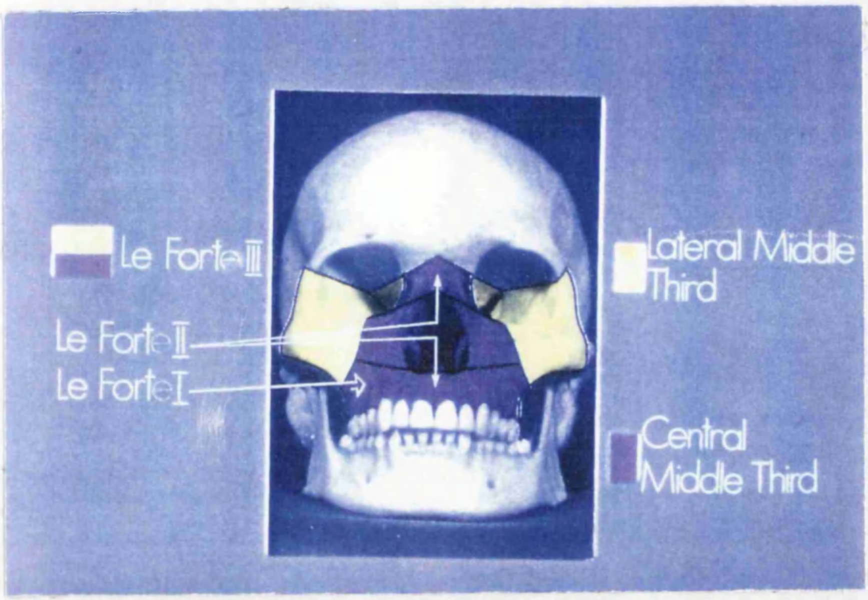
1.3.1 Facial Bones

From the stand point of function and repair, facial bones are best considered by dividing the face into thirds (Jackson et al, 1987) (Fig 1.1).

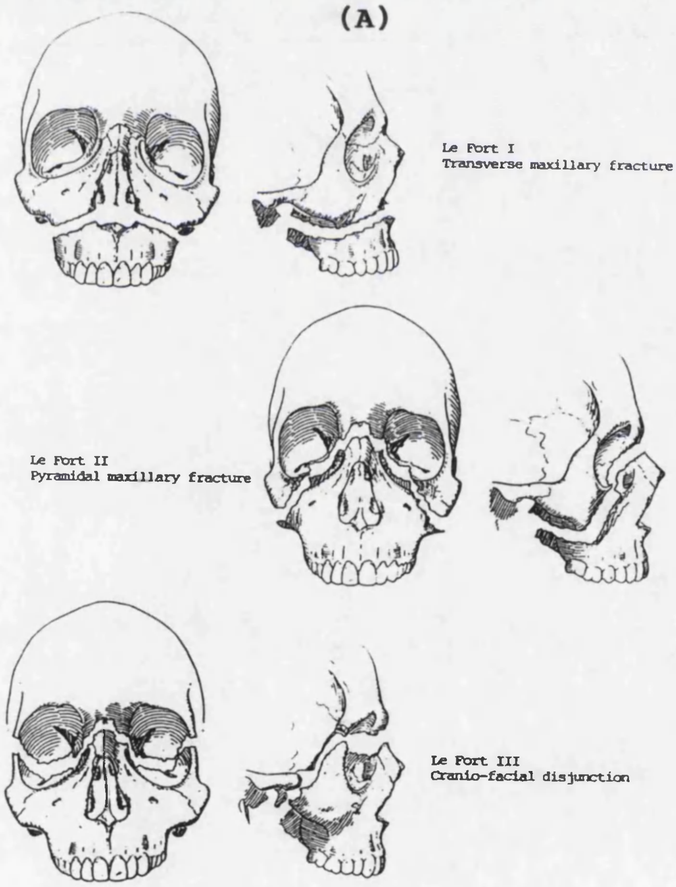
The bones most vulnerable to injury in the upper third of the face are those of the frontal sinuses which form part of the glabella. Fronto-temporal fractures commonly extend farther posteriorly. They may, however, extend into the superior or the lateral orbital walls. supra-orbital ridge fractures may extend into both the frontal sinuses and the roof of the orbit. Such injuries are often complicated by traumatic optic neuropathy (Katz et al, 1983; Anderson et al, 1982; Wolter, 1979; Ketchum, 1976).

Fractures of the maxilla and related bones are frequently referred to as fractures of the middle third of the face

(Haskell, 1985). In addition to the maxilla, the midface comprises the zygoma, lacrimal, nasal, palatine, inferior nasal concha and vomer bones (Schultz, 1988; Haskell, 1985; Hollinshead, 1968). Although the sphenoid, frontal and ethmoidal bones are not classically facial skeleton bones, they are frequently traumatised in midfacial fractures and thus should be considered as part of the midfacial skeleton (Frost & Kendell, 1991). The calvarium, which contains the brain, is not considered a part of the midface. This region may, therefore, be more accurately defined as that area bounded above by a transverse line connecting the two zygomatico-frontal sutures, passing through the frontonasal sutures, and limited below by the incisal and occlusal plane of the upper teeth. Posteriorly, the region is demarcated by the spheno-ethmoidal junction, but includes the free margin of the pterygoid laminae of the sphenoid bone inferiorly. For convenience, fractures of the middle third of the facial skeleton may be divided into those involving the central middle third and those of the lateral middle third (zygomatico-maxillary) (Fig 1.1) (Haskell, 1985). The central middle third fractures (Le Fort II and III types) may be further subdivided into alveolar fractures, fractures of the dento-alveolar complex (Le Fort I type), naso-maxillary fractures, and naso-ethmoidal fractures (Haskell, 1985). The majority of the skeleton of the central middle third is composed of wafer thin sheets of



(A)



(B)

Figure 1.1 A: Bones of the midface divided into thirds.
B: Le Fort fractures.
(Reproduced by kind permission of the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital, Glasgow)

cortical bone with stronger bony reinforcement (Fried, 1980).

Fractures of the middle third of the face are the most difficult to diagnose, demonstrate, and treat (Schultz, 1988). The midface contains the greatest concentration of functional bone structures: the orbits, the nose, the palate, and the maxillary sinuses. The most prominent facial bones would be expected to be the most commonly injured, and has been proved true statistically (Schultz, 1988). The zygoma (malar) fracture is next in frequency, with fractures of the orbital floor commonly occurring in association.

The lower third of the face comprises the mandible and teeth. The superior aspects of the mandible, the condyle and the coronoid process, are considered as part of the lower face (McVay, 1984; Hollinshead, 1968).

The facial bones form a compressible, energy absorbing shield for those vital organs which lie within and behind them. Such tissues - the eye, the lacrimal apparatus, the pharynx, the cervical spine and the brain are of prime concern to any surgeon responsible for treating facial injuries.

1.3.2 Soft Tissue of the Face

Muscles of Facial Expression

Although this group of muscles does not have a strong influence on the displacement of fractures, soft tissue damage found in major maxillofacial trauma as well as various surgical approaches necessary to repair deeper structures will invariably affect these muscles (Frost & Kendell, 1991).

The Facial Nerve

Damage to the facial nerve is possible in severe maxillo-facial injuries or penetrating wounds in the area of the course of the nerve and with fractures of the base of the skull and would result in ipsilateral paralysis of the muscles of facial expression (Frost & Kendell, 1991). In general, surgical repair of a divided facial nerve is only partially successful (Schultz, 1988).

Sensory Nerve Supply

The skin overlying the posterior portion of the masseter muscle receives its sensory supply from the posterior and anterior branches of the upper cervical nerves. Otherwise, facial sensation is provided entirely by branches of the trigeminal nerve (McVay, 1984; Hollinshead, 1968). Injury to the branches of this nerve rarely results in permanent anaesthesia of the face (Schultz, 1988). This is partially due to nerve regeneration and in part to the rich overlapping system of undamaged nerves. It is not necessary to repair these small sensory nerves (Piddle, 1968).

Vascular Supply

Arteries

The superficial arterial supply of the face is entirely from the external carotid artery while the upper face and the nasal cavity are supplied by branches of the internal carotid artery (McVay, 1984). Despite this fact, unilateral or even bilateral ligation of the external carotid artery usually is ineffective in permanently controlling facial wound haemorrhage. This is because of the rich anastomoses with branches of the internal carotid artery (Haskell, 1985).

Veins

Venous bleeding from facial wounds seldom causes serious problems (Schultz, 1988). However, venous haemostasis may be problematic following fractures or surgical procedures involving the ethmoidal sinuses due to the lack of access to bleeding points (Schultz, 1988). The potential retrograde spread of infection in the post operative phase in the trauma patient is an uncommon but grave complication of venous disruption (Frost & Kendell, 1991).

1.3.3 The Orbit and its Contents

Introduction

The orbit contains the eye and its accessory structures. The globe of the eye takes up one quarter of the volume of the orbit. The configuration of the bony structure of the orbit is that of a pyramid with its base facing anteriorly having a volume of approximately 35 cc (McVay, 1984).

The medial wall is the thinnest wall of the orbit, but it derives some increased strength by the trabeculization of the ethmoid air cells (Wolff, 1968). The orbital rim is the strongest portion and is derived from the frontal, maxillary, and zygomatic bones. It is thickest laterally and inferiorly where it comprises part of the zygomatic bone, becoming more prominent as it passes superiorly to form the external angular process. Both superiorly and inferiorly the orbital margin becomes progressively thinner and weaker as it becomes related to the frontal and maxillary sinuses respectively. Medially, the rim merges imperceptibly with the nasal and lacrimal bones where it is totally devoid of support because of the underlying nasal cavity (Wolff, 1968).

Orbital Connective Tissue

A definite structural organisation is demonstrable in human orbital connective tissue and the adipose tissue compartments lying in between (Koornneef, 1992). All the eye muscles have their own specific connective tissue systems the morphology of which has a complex three dimensional matrix (Koornneef, 1976).

The photographic reconstruction technique developed by Los (1970) and used by Koornneef (Koornneef & Los, 1975) to reproduce the spatial architecture of the human connective tissue has revealed that each ocular muscle has its own characteristic large attachment area to the relevant orbital wall in addition to the areas of insertion.

Orbital connective tissue can be considered as an important additional locomotor mechanism closely integrated with the ocular muscles, the optic nerve, the central nervous system, the eyeball, the eyelids, the lacrimal gland, and the intraorbital fat, all forming a functional anatomical entity (Koornneef, 1978). Hernia of the orbital contents into the maxillary antrum may contain muscles along with the entire motility apparatus near the orbital floor (i.e, the connective tissue septa of the muscles and the fat cushions in the fracture region) (Koornneef, 1992). This probably explains why, in cases of blow-out fractures of the orbital floor, the vertical motility disturbances nearly always have the characteristics of incarceration of the inferior rectus and inferior oblique muscles together (Koornneef, 1992). In some cases, however, the clinical picture of a blow-out fracture is seen when entrapment of fascial septa alone is demonstrable.

The Orbit in Maxillofacial Injuries

The orbit is invariably involved in depressed fractures of the zygomatic bone and the Le Fort II and III level fractures of the central middle third of the face (Converse et al, 1961). It may also be involved in fractures of the frontal bone and naso-ethmoidal complex. A fracture of the orbit without involvement of the rim is termed a 'blow-out fracture' (Converse & Smith, 1957) which is most common in the thin area of bone of the orbital floor medial to the inferior orbital fissure (Converse & Smith, 1960). The fracture is almost invariably caused by a blunt object of greater circumference than the orbital aperture, such as a fist or a cricket ball, striking the eye and creating a rapid increase of pressure within the orbit (DeHaven & Harle, 1966; Smith & Regan, 1957). However, a direct compression or buckling force on an orbital rim has also been suggested as a causative factor in the production of a blow-out fracture (Fujino & Makino, 1980; Fujino, T, 1974). Recently, Phalen and associates (1990) were able to simulate a focal injury to the face by striking the inferior orbital rim of dried skulls with a lead weight causing segmented fracture of the orbital floor without the pressure of the eyeball.

Blow-out fracture of the orbital floor may be accompanied by herniation of fat, or more rarely, the inferior rectus or the inferior oblique muscle into the maxillary sinus. The entrapment of the orbital tissue and fascial septa in the antrum causes imbalance of the extraocular muscles and diplopia (Heller, 1970). It may also cause enophthalmos (Berkowitz et al, 1981) and infra-orbital anaesthesia (Piddle, 1968).

Blow-out fracture of the medial wall is frequently seen in association with fracture of the floor of the orbit (de Visscher & van der Wal, 1988; Miller & Glaser, 1966) and has been attributed to an increase in the intra-orbital pressure (Linhart, 1943). The reported incidence of medial orbital wall fractures varies considerably in the literature. Cadaver studies into the mechanism of orbital floor blow-out fractures indicated an incidence of isolated medial wall fractures varying from 18% (Jones &

Evans, 1967) to 31% (Pearls & Vistnes, 1978) and of concomitant medial and inferior wall blow-out fractures between 12% (Jones & Evans, 1967) and 38% (Pearls & Vistnes, 1978). An isolated fracture of the medial orbital wall with entrapment of the medial rectus muscle is uncommon (Rumlet & Ernest, 1972). Blow-out fractures of the orbital roof are rare (Curtain et al, 1982).

Isolated mandibular and nasal fractures are rarely associated with ophthalmic complications (Holt & Holt, 1983). The optic foramen, situated within the lesser wing of the sphenoid bone and surrounded by relatively dense bone, is usually protected from involvement in fracture lines (Haskell, 1985). For this reason, direct injury to the optic nerve is unusual. The optic nerve and its surrounding membranes constitute a direct extension of the brain. Any injury to these structures is particularly serious since early blindness may occur (Wolin & Lavin, 1990).

The Extra-ocular Muscle Movements

Eye movements are frequently impaired following facial and head injury and muscle imbalance is a common complication in zygomatico-orbital fractures (Yee et al, 1975). Muscle involvement can be direct; it may be caused by entrapment in the fracture line of the muscle, the tendon, or more commonly the adjacent fascial septa. Disorders of ocular motility may also be due to central or peripheral damage to the cranial nerves supplying the muscles, or massive orbital oedema or haemorrhage (Smith et al, 1971; Cole & Smith, 1963).

1.4 FACIAL TRAUMA: CLASSIFICATION AND MANAGEMENT

1.4.1 Classification

The reconstruction of the midface following trauma demands uncompromising care. The face is intimately bound up with one's self image and is a region that is responsible for the senses of vision and smell and for providing the voice and its resonance through the presence of the air sinuses (Lew & Sinn, 1991).

Injuries to the face may be broadly divided into two groups: injuries to soft tissue and injuries to bone. The eye is a specialised organ, injury to which is considered as associated trauma (Schultz, 1988).

Soft Tissue Injuries

Soft-tissue injuries of the face may be clean lacerations, contused lacerations, abrasions, puncture wounds, shaved wounds or wounds associated with thermal or blast damage (Davis & Shaheen, 1985).

Windscreen injuries are likely to contain glass which may or may not be radio-opaque. Note should be made of the material causing a facial abrasion. If this is asphalt, coal, or other dusty material, particles of dirt may well be embedded in the wound but, because of the blood, are not always initially obvious (Davis & Shaheen, 1985). An underlying bony injury may or may not be present but should be suspected if any of the previously described signs and symptoms are observed.

Facial Bone Fractures

Various classifications are available for frontal and mid-facial fractures. For the purpose of this study, we have used the classification adopted by the Oral and Maxillo-facial Surgery Unit at Canniesburn Hospital in Glasgow, which is as follows:

Malar Fractures

The classification of malar (zygomatico-orbital) fractures was designed by Mr Henderson of Canniesburn Hospital, Glasgow in 1973 (unpublished) to simplify identification of these fractures and to enable treatment modalities for each type to be instituted, computerised, and later analysed (Ellis et al, 1985). Henderson divided malar fractures into seven anatomical types (Table 1.1).

Type	Description of Fracture
I	Undisplaced fracture (any site)
II	Zygomatic arch fracture only
III	Tripod fracture with undistracted F-Z suture
IV	Tripod fracture with distracted F-Z suture
V	Pure blow-out fracture of the orbit
VI	Fracture of the orbital rim only
VII	Comminuted fracture or other than the above

Table 1.1 Henderson's classification of malar fractures.

Maxillary Fractures

Identification of these fractures is according to the Le Fort classification (Le Fort, 1900), subsequently extended by the Oral and Maxillofacial Unit at Canniesburn Hospital (Table 1.2).

Other Fracture Sites

These comprise nasal, naso-orbito-ethmoidal (NOE) and frontal sinus fractures (Converse & Smith, 1963; Stranc, 1970 a & b).

1.4.2 Management

The management of maxillofacial injury has been extensively reviewed by Fonseca & Walker (1991), Schultz (1988) and Rowe & Williams (1985).

Diagnosis

After assessment and management of the patient's overall injuries, the facial injury is evaluated and treated. A comprehensive diagnosis and classification of facial injuries is made before starting treatment. The diagnosis

Type	Description of Fracture
Le Fort I	Transverse maxillary fracture
Le Fort II	Pyramidal maxillary fracture
Le Fort III	Cranio-facial disjunction
Additional classifications include:	
Any combination of Le Fort I, II or III	
Fracture of the tuberosity of maxilla	
Alveolar fracture excluding tuberosity fracture	

Table 1.2 Classification of maxillary fractures.

is established by observation, palpation and radiological examination (Schultz, 1988).

Observation

The face is observed for evidence of lacerations and the globe is examined for ocular injury. The symmetry of the face is observed, and any asymmetry which is greater than normally expected is recorded.

Palpation

Palpation of the bony prominences of the face is carried out and comparison of the relative heights of the malar eminences is performed. Tenderness can usually be elicited at the site of a facial bone fracture.

Radiological Examination

Gross facial bone fracture can be diagnosed clinically. Nevertheless, radiological studies play a definite part in the evaluation and documentation of facial injury. The most informative radiographic views and the structures they show most clearly are as in Table 1.3.

More precise or additional information can sometimes be obtained by use of linear tomography (laminography), which

Structures seen	**Radiological view**
Occipito-mental plane	Waters' view
Facial bones	(1) Posterior - anterior view of facial bones (2) Lateral view of facial bones
Nasal bones	(1) Lateral view of nasal bones (2) Occlusal view of nasal bones
Mandible	(1) Posterior - anterior view of mandible (2) Oblique view of mandible (3) Occlusal view of mandible (4) Towne view for ascending rami and condyles
Zygomatic arch	Tangential views

Table 1.3 Radiological views in facial fracture.

was the radiographic procedure of choice in the diagnosis of fractures of the middle third of the face until the introduction of computed tomography. It is a technique which brings into focus a given plane onto x-ray film whilst the other planes are deliberately blurred (Marsh et al, 1988). However, its definition of soft tissue is usually poor (Gould & Titus, 1966).

Hypocycloidal tomography (polytomography) differs from linear tomography in that the x-ray tube and film describe a hypocycloidal movement rather than a linear arc. It produces thinner slices giving more detail and less artefact than laminography. The efficiency and accuracy of this technique in evaluation of orbital floor fracture is said to be better than plain radiography or linear tomographic techniques (Dodick et al, 1969).

The technique of positive contrast orbitography which involves the injection of a solution of contrast medium along the orbital floor, external to the muscle cone has been deemed useful in the diagnosis of blow-out fractures of the orbital floor (Milauskas et al, 1966), though it is now rarely, if ever used.

Ultrasonography uses high frequency sound waves to produce echoes as they strike interfaces between acoustically different structures. The B-scan produces a two-dimensional picture of orbital structures and can be employed to accurately visualise breaks in the normal contour of the orbital wall and demonstrate soft tissue prolapse (Ord et al, 1981). There is no known radiation hazard.

Computed tomography (CT) has given an entirely new scope to the diagnostic aspect of head and maxillofacial injuries. CT scanning is a non-invasive technique which has the unique facility to give clear anatomical pictures of hard and soft tissues in axial, coronal and sagittal planes of section (Yamamoto et al, 1983). A three dimensional pictorial assessment of all the tissues of the head and face is now feasible. A definite place has been established for CT scanning in the diagnosis of major maxillofacial, orbital, and naso-ethmoidal injuries, especially when associated with head injury (Sinn & Karas, 1991).

CT examinations are most valuable in the diagnosis of midface fractures which extend from the supraorbital rim to the maxilla (Gerlock et al, 1981). Furthermore, CT can demonstrate air or haemorrhage in the cranium, orbits, and/or soft tissues; suspected cervical spine injuries may also be examined at the same time as cranial or facial views are taken (Sinn & Karas, 1991). Injuries associated with the orbits and paranasal sinuses may require coronal views, which show the horizontal displacement of the zygoma, to establish the full extent of the injury (Leib, 1986). Fractures of the roof of the orbit and the cribriform plate are also shown better on coronal views (Leib, 1986).

Magnetic resonance imaging (MRI) is a technique with great potential. Experiments describing the phenomenon of magnetic resonance were first reported in 1946 by Bloch and associates. MRI produces tomographic images of the body which have excellent spatial resolution. The technique involves placing a patient within a strong, uniform magnetic field, delivering a pulse of radio waves and recording the emitted signal (Underwood & Firmin, 1987).

Both CT and MRI scanning rely on reconstructive tomography and modern computing technology for the representation of anatomical cross-sections. However, CT has definite advantages over MRI in the diagnosis of facial fractures. The x-ray-generated CT allows better detailing of bony anatomy which is extremely helpful in the evaluation of fractures (Sinn & Karas, 1991).

If after exhausting all the available means of investigation, the surgeon remained uncertain as to the exact nature of fracture, surgical exploration provides the only definitive means of diagnosis.

Treatment

It is the rule at the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital in Glasgow to operate within two weeks of injury. The line of treatment of individual facial fractures and the techniques used and procedures followed are those generally accepted as standard by the profession. Details of treatment and surgical procedures employed are out of the scope of this study and have been reviewed by Fonseca & Walker (1991) and Rowe & Williams (1985). A short account is given here.

Unless associated with soft tissue injury or other complications, undisplaced fractures of the zygomatico-orbital complex are left without intervention. They usually heal well. On the other hand, displaced fractures are reduced and depressed fractures elevated. Fixation of these fractures may or may not then be required for stabilization. Various materials and methods are available for this purpose. Pins, wires, K wires and antral packs, have all been used. Sometimes a combination of two or more of these methods is needed.

Blow-out fractures do not constitute an emergency (Catone et al, 1988) and are sometimes left untreated unless complicated by orbital tissue entrapment and ocular or motility disorders. In such cases the orbital floor is exposed through the lower lid (transpalpebral), the conjunctiva (transconjunctival) or via the maxillary antrum (Caldwell-Luc procedure). Trapped soft tissue is then freed. The orbital floor is reconstructed by inserting a bone graft taken from the patient's iliac crest, or an alloplastic material such as a silastic sheet which can be cut to shape and size needed. Alternatively, it can be supported from below by an antral pack. The forced duction test is then performed to ensure free motility of the eye.

1.5 BLUNT TRAUMA TO THE EYE: PATHOGENESIS AND CONSEQUENCES

The association of ocular injuries with facial trauma, especially fractures of the orbital floor, is well recognised (Petro, 1979). A number of factors protect the globe from injury. These include the prominence of the bones of the orbit and the natural reflexes of self-protection, namely blinking, averting the head, and protecting the eye with hand or forearm. Despite these factors, the eye may sustain injury, but the resilient structure of the globe allows it to withstand blows of considerable force without rupture. The following description reviews the salient clinical features of such eye injuries and motility disorders.

1.5.1 Blunt Ocular Trauma

It is essential to perform a complete ophthalmic examination in every case of blunt trauma to the eye and orbit (Benson et al, 1991). All trauma to the face, particularly above the level of the mouth, requires a careful ocular examination, including an estimation of the visual acuity of each eye if consciousness permits (Duguid, 1985). This is important for future decision making and to elicit evidence of ocular damage for medico-legal purposes. If a Snellen chart is not available, the ability to read a newspaper with an appropriate spectacle correction gives some indication of the visual function. A careful assessment of pupillary reactions is necessary. The examiner can detect retinal or optic nerve damage by using the swinging flashlight test (Slamovits & Glaser, 1991) to demonstrate an afferent pupillary defect.

Mechanisms Ocular Damage

Blunt trauma causes ocular damage by the coup mechanism, by the contre-coup mechanism, or by ocular compression and distortion.

Coup and contre-coup

Courville introduced the concept of coup and contre-coup injury to explain brain damage caused by trauma to the head (Courville, 1962; 1942). Coup refers to local trauma at the site of impact. Contre-coup refers to distant injuries caused by shock waves that traverse the brain to the opposite side of the skull. Wolter later used these concepts to explain eye injuries (Wolter, 1963). Examples of coup injuries are corneal abrasions, subconjunctival haemorrhages, and retinal necrosis (Blight & Hart, 1977). The best example of contre-coup ocular injury is commotio retinae (Hart & Frank, 1975).

Anterior-posterior Compression and Horizontal Expansion

The volume of a fluid filled indistensible enclosed space cannot be changed. Therefore, when the eye (which has limited distensibility) is compressed along its antero-posterior axis, it must either expand in its horizontal plane or rupture. Using high speed photography, Delori and associates studied blunt trauma in enucleated pig eyes (Delori et al, 1969). The cornea was indented by up to 8.5 mm, reducing the original antero-posterior axis by 41% and bringing the posterior surface of the cornea into contact with the iris and the lens. The equatorial plane circumference was expanded to 128% of its original length. In a similar study, Weidenthal & Schepens (1966) subjected enucleated pig eyes, which were mounted in 10% gelatin, to contusion. The eyes exhibited intraocular changes similar to those in eyes traumatised in situ. Apart from damage to the lens, these changes included detachment of the nasal ora serrata, nasal retinal dialysis, festoon formation, peripheral pigmentary changes, and tears in the ciliary epithelium of the pars plana. Damage to the nasal ora serrata was most common when the site of impact was near the centre of the cornea. It was necessary to strike the globe near the temporal limbus to produce a temporal retinal dialysis. It was concluded that much of the damage which was produced experimentally in the fundus periphery was the result of rapid equatorial expansion and oscillation of the ocular coats following impact. Temporal retinal dialysis was probably caused by local deformation of the sclera near the temporal ora serrata.

Eyelids

Haemorrhage

Bruising of the eyelids may arise from local or remote extravasation of blood spreading into the palpebral region because of the tissue laxity. This gradually absorbs but rarely infection arises. Such bruising is usually benign, but it may be related to severe ophthalmic injury (Culbertson, 1983). Careful examination of the eye is thus required in each case.

Haemorrhage and oedema of the eyelid may cause ptosis; this is essentially a mechanical problem which, by virtue of the extra bulk or weight of the involved eyelid, causes it to droop. With spontaneous absorption of the oedema and haemorrhage, the eyelid should gradually rise to its normal level without treatment (Arden & Moore, 1989).

Ptosis due to traumatic third nerve paresis requires no immediate treatment but should be left for about six months, as spontaneous recovery ensues in many instances (Russell, 1960).

Laceration

Lacerations of the eyelids are divided into two groups: those that are parallel to the eyelid margin and hence do not gape, and those that involve the eyelid margin and are drawn apart by traction of the fibres of the orbicularis oculi muscle (Newell, 1986). Suturing is generally desirable with careful apposition of the wound edges using a fine suture material such as 5/0 collagen for deep tissue and 6/0 silk for the skin. Wounds of the lid margins merit special attention to avoid the sequel of a notch or defect (Culbertson, 1983).

Conjunctiva

Damage to the conjunctiva is rarely serious, but great care should be taken during examination because perforating or lacerating injuries of the bulbar conjunctiva may conceal a perforating or penetrating wound of the underlying sclera (Lichenstein, 1991; Martin, 1988).

Blunt injuries to the conjunctiva usually cause oedema (chemosis) but this resolves spontaneously. A breach of one or more conjunctival capillaries gives rise to subconjunctival haemorrhages. Although unsightly, these haemorrhages gradually absorb without treatment. Haemorrhage from osseous fracture cannot be visualised at its posterior border irrespective of the ocular position. There is usually a latent period of several hours following the fracture before such haemorrhage becomes subconjunctival.

Lacerations of the conjunctiva usually merit suturing unless they are small, using fine suture materials such as 6/0 collagen, 6/0 cat gut or 8/0 vicryl.

While oedema and haemorrhage into the soft tissues are prominent features of orbital fractures, the escape of air from the maxillary or ethmoid sinuses into the orbit (orbital emphysema) is not infrequent. This air may pass forward below the conjunctiva giving a bulbous appearance and crepitation on palpation (Jordan et al, 1988; Coppeto, 1987).

Cornea

Abrasions of the corneal epithelium (Fig 1.2) are common, but since they heal rapidly, they are of little long term consequence unless complicated by recurrent abrasion. Endothelial damage can be more serious (Cibis et al, 1978). A local concussion (coup effect) can rupture endothelial cells and the tight junctions between them. In addition, in severe injuries the endothelium can be further damaged by being pushed against the iris and the lens. The transient corneal oedema usually clears promptly, but endothelial damage can be permanent (Slingsby & Forstot, 1981). Hyphaema (Fig 1.3) probably results, in most cases, from tearing of blood vessels at the root of the iris (Tonjum, 1966). Follow up studies of patients with traumatic hyphaema show endothelial cell loss that correlates with the severity of the initial injury (Cibis et al, 1978; Bourne et al, 1976). When a total hyphaema and a high intraocular pressure are present, the damaged corneal endothelium permits erythrocyte fragments to enter the corneal stroma. The resultant corneal blood staining clears spontaneously, but this may require many months. The central cornea clears last (Slingsby & Forstot, 1981).

In injuries severe enough to rupture the corneal stroma, the wound usually crosses the limbus into the adjacent sclera (Cherry, 1978; Cherry, 1972).

Lacerations of the cornea vary greatly in their extent. A small superficial tear will often heal without intervention but rupture of Bowman's membrane leaves opacification. Deep corneal lacerations usually require suturing especially if associated with a flat anterior chamber or incarcerated tissue. All incarcerated tissue should be removed or freed from the wound, the corneal wound repaired meticulously, and the anterior chamber re-formed to prevent synechiae.

Figure 1.2 Corneal erosion and diffuse suconjunctival haemorrhage.

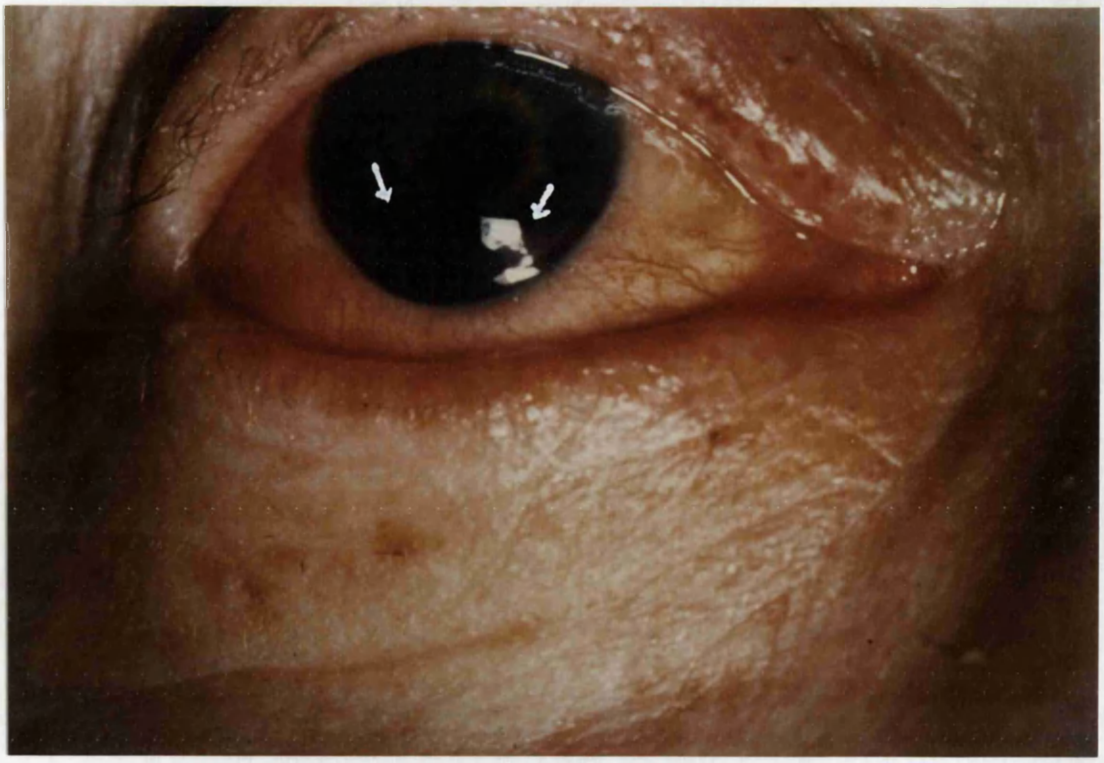


Figure 1.3 Hyphaema secondary to blunt eye injury.

Stellate lacerations with retraction of corneal stroma should be accurately sutured. If there is leakage of aqueous, surgical adhesive and a bandage lens may be applied. If such treatment is not sufficient, or if there is evidence of corneal tissue loss, a patch graft is indicated (Goldberg and Paton, 1976; Troutman, 1974).

Anterior Uvea

When the cornea is abruptly forced backward by severe blunt trauma, it presses the iris against the lens preventing the escape of aqueous into the posterior chamber. If enough force is applied, the entrapped aqueous is thought to dissect into the ciliary body, most commonly into its anterior face resulting in angle recession (Kaufman & Tolpin, 1974). Tearing of the ciliary body is responsible for about 90% of the hyphaemas seen after blunt trauma (Read and Goldberg, 1974; Tonjum, 1966). Haemorrhage may also occur if the iris is separated from the ciliary body (iridodialysis, fig 1.4) or if the sphincter muscle is ruptured.

After blunt trauma to the eye the intraocular pressure may be normal, increased, or decreased. Aqueous out-flow may be decreased because of angle recession, inflammatory trabeculitis, or hyphaema. Ciliary body injury tends to decrease aqueous inflow. The intraocular pressure depends on the balance of these factors (Benson et al, 1991).

It is thought that the pressure rise is caused by trabecular damage and not by the angle recession itself (Yanoff & Fine, 1975). However, the larger the area of recessed angle the higher the incidence of glaucoma. Nine percent of patients with angle recession develop glaucoma (Kaufman and Tolpin, 1974). It is important to be aware of the fact that the glaucoma may not develop until many years following the injury. For that reason, Kaufman and Tolpin advocated that all patients with extensively recessed angles (180° or more) should be examined periodically for late glaucoma. However, the number of patients in their study (n=33) is too small to allow such generalisations to be made.

Blunt injuries to the eye may give rise to traumatic miosis or mydriasis of the pupil. Such a defect is usually permanent (Duguid, 1985). A mydriasis, which is due to pupil sphincter rupture (Fig 1.5), is prominent after severe concussion of the eye and is usually accompanied by paresis of accommodation (Martin, 1988). A spastic miosis is common after less severe injury to the globe. In such cases, miosis may be accompanied by a transient spasm of accommodation brought about by axonal reflexes. This spasm

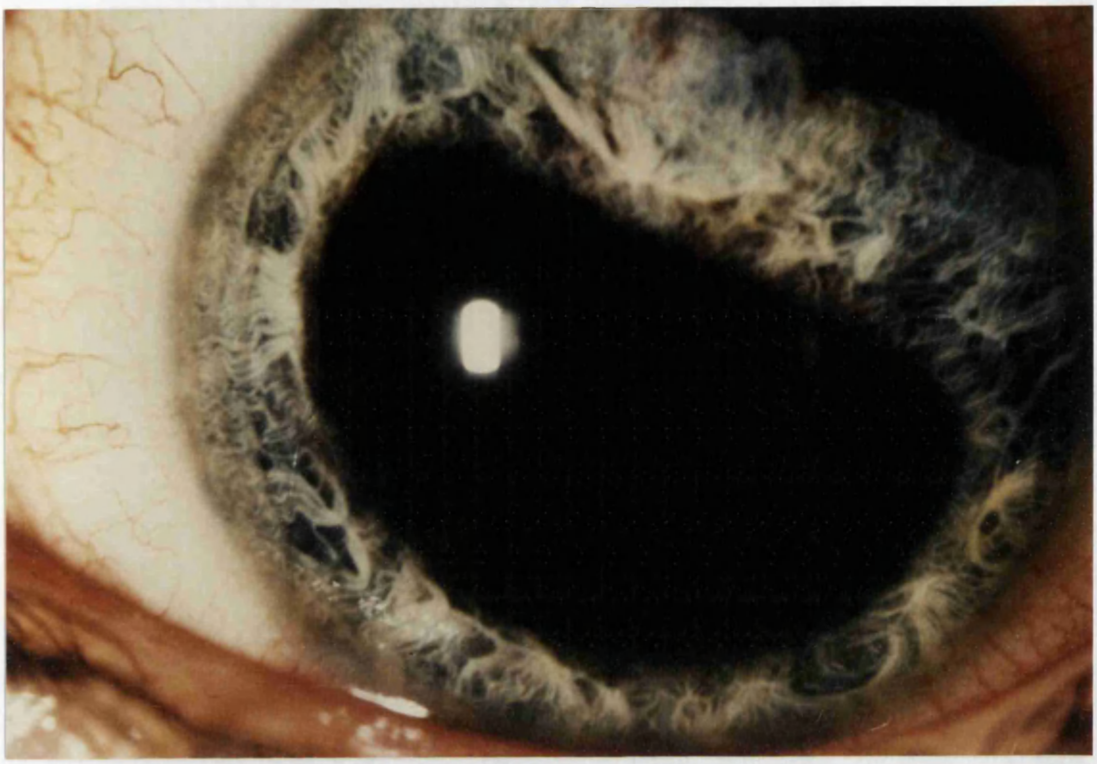


Figure 1.4 Iridodialysis.

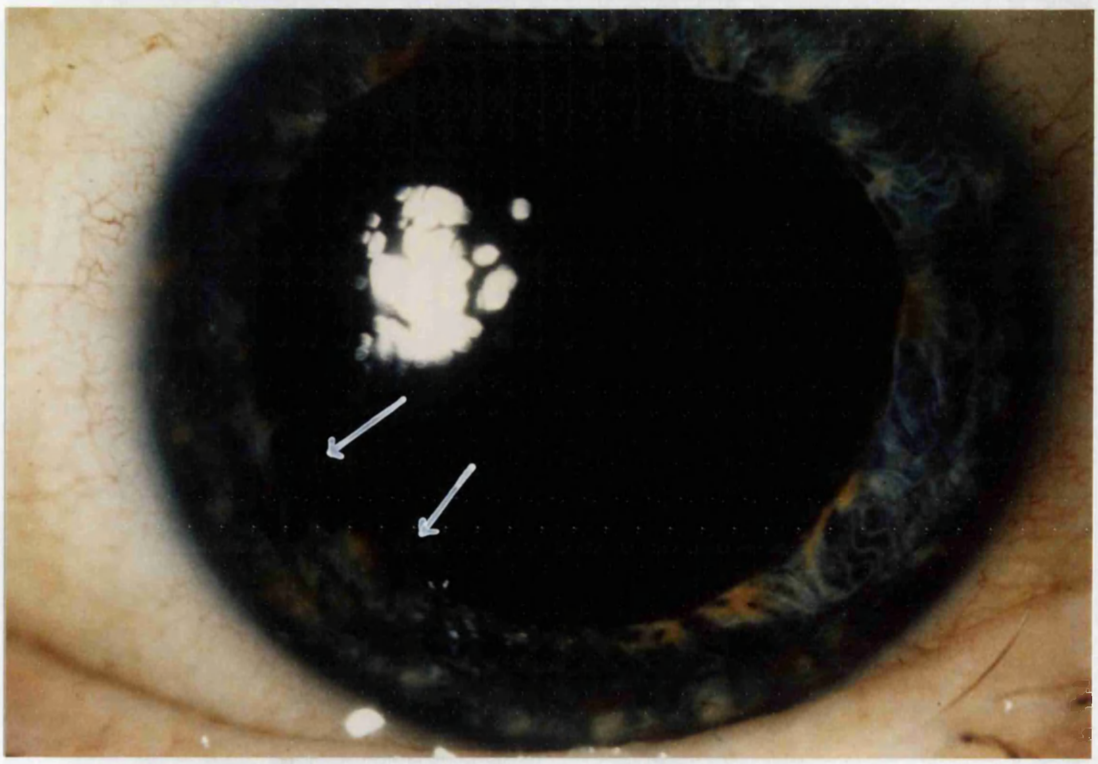


Figure 1.5 Traumatic mydriasis.

may be followed within minutes or hours by paralytic mydriasis (Benson et al, 1991).

Care should be taken to exclude other causes of pupillary dilatation, such as antecedent Adie's pupil and, in the unconscious patient, an oculomotor nerve palsy resulting from intracranial injury with tentorial herniation of the cerebrum.

Prolapse of the iris through a perforating wound leads to distortion and peaking of the pupil towards the perforation which requires immediate surgical intervention.

Lens

Trauma is the most common cause of unilateral cataract (Fig 1.6) in young individuals and concussion injuries may lead to the development of Vossius' ring due to an 'imprinting' of iris pigment onto the anterior lens capsule (Britten, 1965). A concussion injury may also cause a transient anterior subcapsular cataract, known as rosette cataract. This may progress to involve the whole lens (Ioannides et al, 1988).

Blunt trauma to the eye may cause subluxation (Fig 1.7) or total dislocation of the lens (Ioannides et al, 1988;

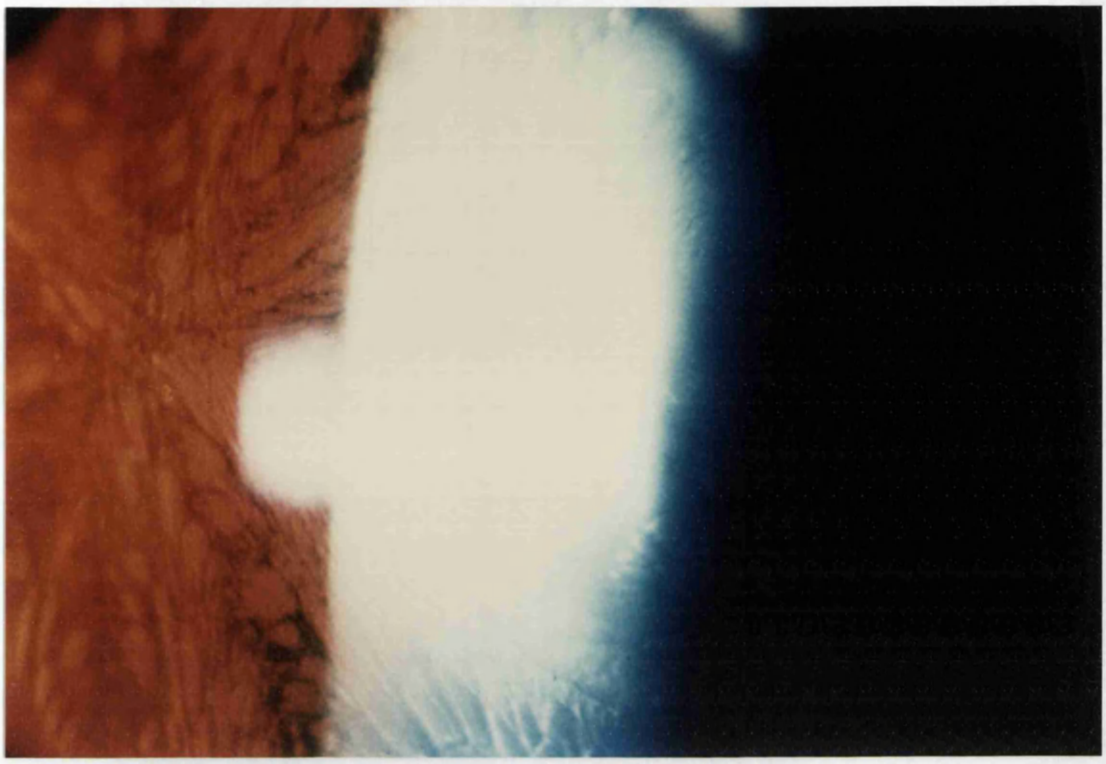


Figure 1.6 Traumatic posterior subcapsular cataract.

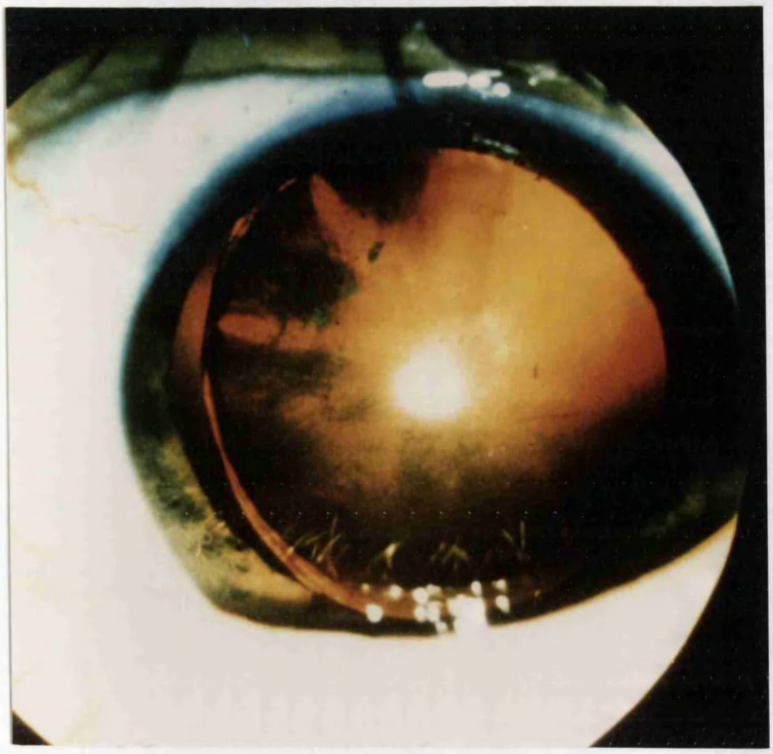


Figure 1.7 Traumatic subluxation of the lens.

Milauskas & Fueger, 1966). In some cases the lens itself can rupture and cause phacolytic glaucoma (Martin, 1988).

Ciliary Body

Damage to the ciliary body can result in impairment in the formation of aqueous and a consequent reduction in intraocular pressure. Moreover, injury to the ciliary muscle gives rise to impairment in accommodation, which is a fairly common short-term sequela of eye injury. There is no specific treatment for either of these conditions. The intraocular pressure is normally restored shortly after injury, and accommodation similarly recovers spontaneously in most cases.

Traumatic cyclodialysis is unusual and results from severe blunt ocular injury (Read & Crouch, 1991). A persistent very low intraocular pressure may indicate that cyclodialysis has taken place.

Retinal Injuries

Commotio Retinae (Fig 1.8)

This is a contre-coup injury. It is a retinal oedema which can occur peripherally or centrally, at the macula. When transient and reversible, it may be called Berlin's oedema

(Berlin, 1873). Immediately, and for several hours after trauma, the retina may appear normal, although the patient may complain of decreased vision. Thereafter the affected area becomes opalescent with an ill defined periphery.

The visual acuity in commotio retinae varies from 6/6 to 6/60 and does not always correlate with the degree of retinal oedema. The majority of patients report a rapid improvement in vision during the first 40 minutes or so after injury (Hart & Frank, 1975). Prognosis is usually excellent except in cases with associated subfoveolar choroidal rupture. Poor visual recovery can also be expected in cases with severe retinal pigment epithelial damage. This, usually is preceeded by serous retinal detachment, which can be confirmed by leakage of fluorescein into the subretinal space (Friberg, 1979; Hart & Frank, 1975). Experimental studies have shown that following severe injury, produced by firing a pellet from a modified airgun, fragmentation of the photoreceptor outer segments and damage to the retinal pigment epithelium occur the site of impact and gradual regeneration of the photoreceptor segments subsequently takes place (Blight & Hart, 1975).

The late manifestations of blunt injury to the retinal pigment epithelium (RPE) vary from minor atrophic changes to massive hyperplasia and migration of the RPE cells.

This latter condition results in bone corpuscular and granular pigmentation that resembles retinitis pigmentosa (Bastek, 1981; Crouch and Apple, 1974; Cogan, 1969). Total retinal destruction of all retinal layers can occur in some cases (Crouch and Apple, 1974).

In rare cases, retinal contusion causes cystoid macular oedema which may, in turn, progress to a macular cyst or hole (Friberg, 1979). A macular hole (Fig 1.9) can also be caused by acute posterior vitreous detachment with foveal traction (Friberg, 1979). In this case an overlying operculum is seen.

Retinal Tears

Necrotic Tears

If a blunt object strikes the eye posterior to the ora serrata, the direct concussive effects on the retina (coup effect) may cause full thickness retinal necrosis with subsequent retinal detachment (Cox & Freeman, 1979).

Vitreous Traction Tears

Weidenthal and Schepens (1966) concluded from their study of non-perforating trauma to enucleated pig eyes that rapid equatorial expansion is responsible for tears at the

Figure 1.8 Commotio retinae (Traumatic retinal oedema).

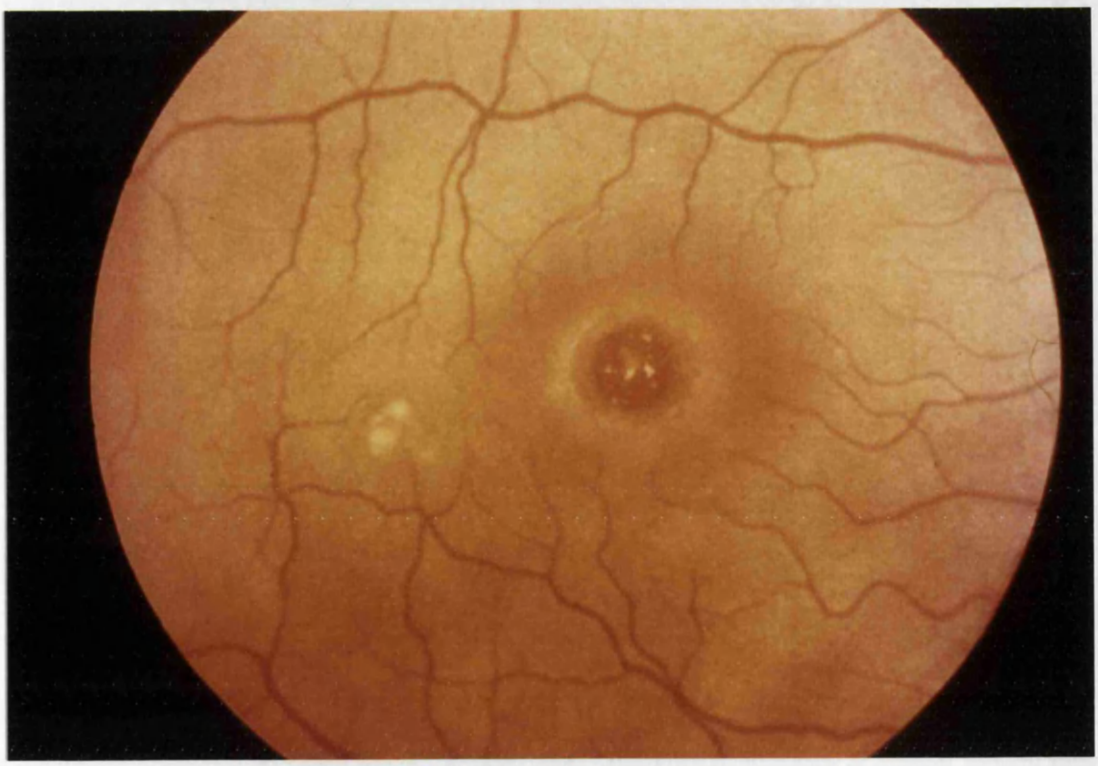


Figure 1.9 Macular hole.

anterior and posterior borders of the vitreous base. When the eye is rapidly compressed, the eye does not have time to stretch. As a consequence, there is strong traction at the vitreous base that may cause tears at its anterior and posterior borders. Nasal retinal tears were most common when the site of impact was near the centre of the cornea whilst temporal tears were probably caused by local deformation of the sclera near the temporal ora serrata.

Whatever the mechanism, a portion of the vitreous base can be avulsed from the retina and pars plana. The appearance of this avulsion is pathognomonic of blunt trauma and may have considerable medico-legal importance (Weidenthal & Schepens, 1966). In most cases of severe trauma, the vitreous base does not separate clearly from the retina and pars plana epithelium. It remains adherent, thus tearing these structures. The retina can be torn along the posterior margin of the vitreous base, or the non-pigmented pars plana epithelium can be torn along the anterior margin of the base, or both can be torn simultaneously. Tears along the anterior and posterior margins of the vitreous base are common inferotemporaly and superonasaly. When located superonasaly, they are usually caused by trauma (Hagler & North, 1968). There is some controversy as to whether inferotemporal tears along the vitreous base are all traumatic or whether they can be familial and developmental (Ross, 1981; Verdaguer et al, 1975). Tasman's clinical study (1972), in which he prospectively followed up, for two years, 52 patients who had sustained ocular contusion, confirmed Weidenthal's experimental evidence that tears caused by blunt trauma nearly always occur at the time of injury. Nine of 52 patients had dialysis diagnosed within three weeks of injury while a tenth patient was found to have a dialysis when his vitreous haemorrhage cleared four months after injury. Other investigators have confirmed this conclusion (Sellors & Mooney, 1973)

Stretch Tears

If the retina is too inelastic to stretch when the eye expands horizontally, retinal breaks concentric to the optic nerve are produced. These breaks have the unusual tendency to be partially self-sealing. In some cases the retina remains attached. In others it detaches (Benson et al, 1991).

Retinal and Vitreous Haemorrhage

If the retina is torn, blood vessels which bridge the tears may bleed into the vitreous (Cox et al, 1966). Vitreous haemorrhage can also result from acute posterior vitreous detachment (PVD) and avulsion of superficial retinal vessels (Cox & Freeman, 1979). A third possible mechanism is rupture of the ciliary body (Benson, et al, 1991). In many cases the source of bleeding is never found.

Retinal haemorrhages may be small or large, single or multiple and varied in position. They usually absorb slowly either spontaneously or with proliferative neovascularisation from the retinal vasculature. Occasionally, fibrosis in the vitreous can result in a tractional retinal detachment (Cox & Freeman, 1979).

Retinal Detachment

The distortional effects of concussive injury may cause retinal detachment (Fig 1.10). The vitreous gel is firmly adherent to the peripheral retina, and the acute coronal distension that accompanies anteroposterior blunt eye injury can result in a retinal tear (Delori et al, 1969). This condition causes a retinal dialysis (Fig 1.11) or the formation of a retinal hole because the distortional forces produce vitreous traction at the retinal periphery. Laboratory experiments indicate that the majority of retinal breaks caused by contusion are formed at the time of injury (Delori et al, 1969; Weidenthal & Schepens, 1966). It has been suggested that virtually all cases of retinal dialysis are secondary to trauma (Ross, 1981).

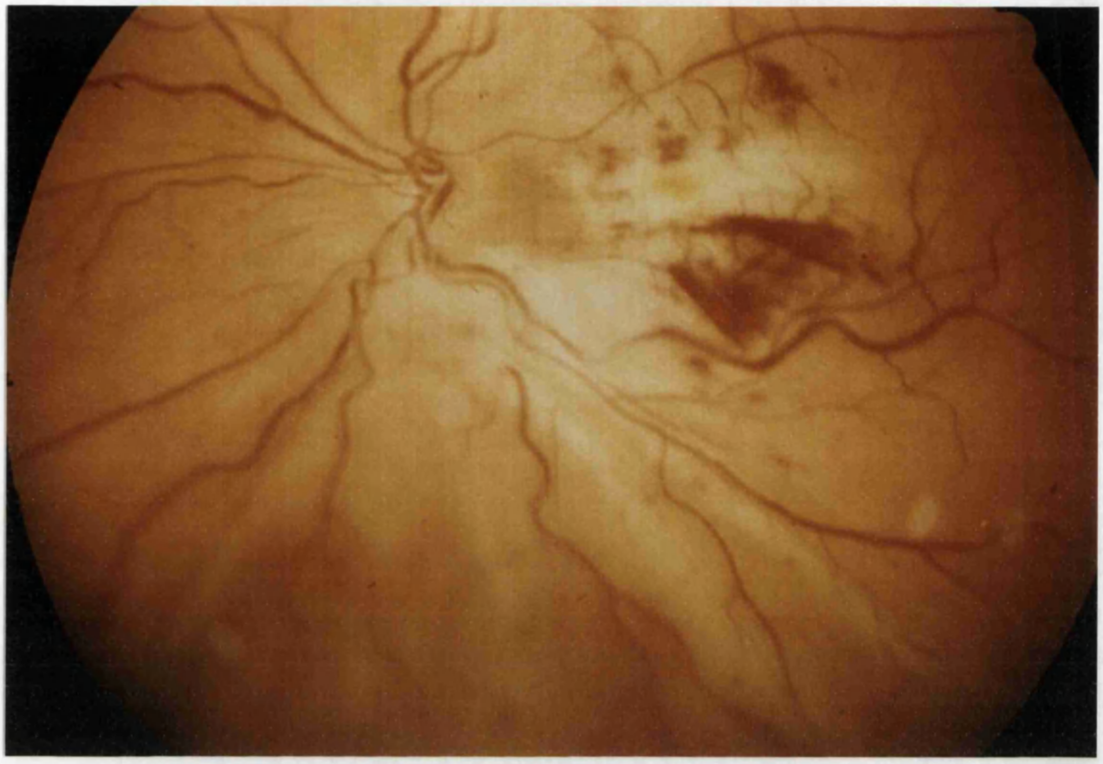


Figure 1.10 Retinal detachment. A severe blunt injury has resulted in intraretinal haematoma and retinal detachment.

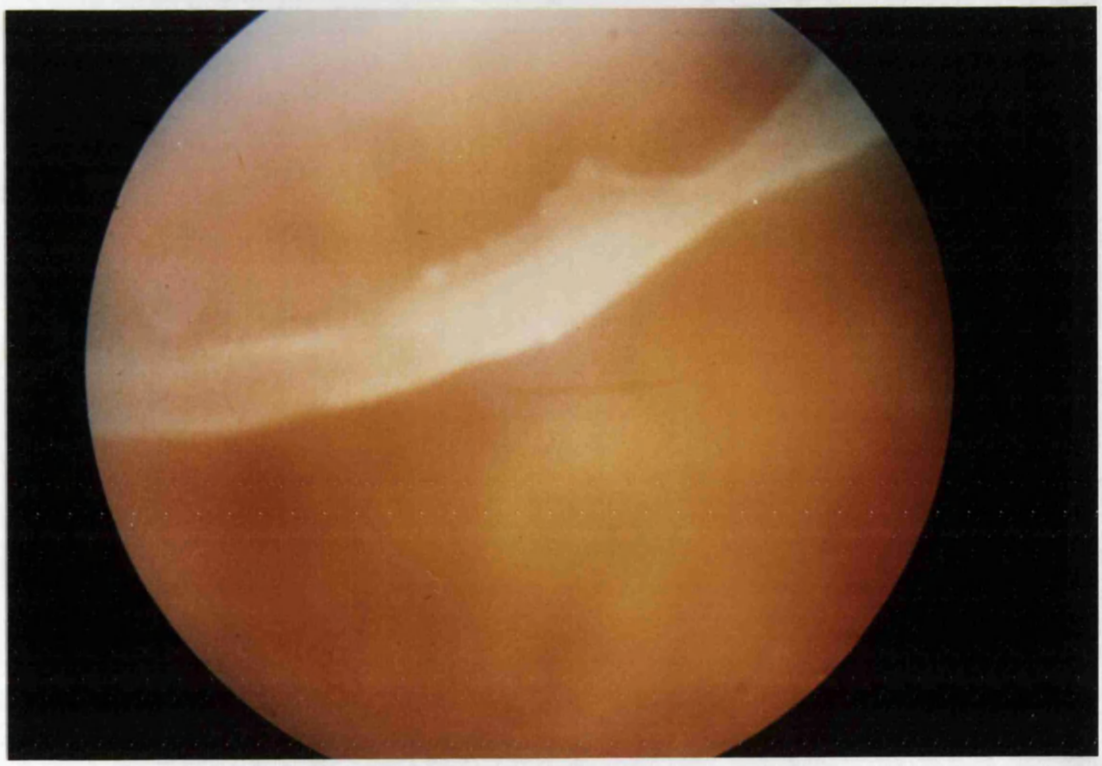


Figure 1.11 Retinal dialysis.

Traumatic retinal breaks are treated with cryotherapy or photocoagulation, otherwise detachment may later develop. Demarcation lines, atrophy of the underlying pigment epithelium, subretinal precipitates and extensive vitreous 'tobacco dust' are all commonly seen following traumatic retinal detachment (Cox & Freeman, 1979; Cox et al, 1966). Because these detachments rarely progress to proliferative vitero-retinopathy, the prognosis for re-attachment is excellent provided, of course, that all breaks are found. Blunt trauma is the leading cause of retinal detachment in children and adolescents (Winslow and Tasman, 1978).

Traumatic Retinal Angiopathy (Purtscher's Retinopathy)

This is an indirect ophthalmic consequence of injury. Multiple discrete, superficial infarcts of the retina (Fig 1.12) manifesting as loss of central vision, accompanied by the development of multiple cotton-wool spots adjacent to the optic disc, may appear as a result of severe skull fractures, chest compression, and long bone fractures (Purtscher, 1910). Although the pathogenesis is unknown, a sudden increase in pressure within the retinal venules has been incriminated (Archer, 1986), as have fat emboli. There is no specific treatment but the prognosis is good with spontaneous, gradual recovery of vision during the ensuing 2 to 3 months in most cases.

Figure 1.12 Traumatic retinal angiopathy (Purtscher's retinopathy). Multiple cotton-wool spots in the retina of this patient, who sustained a severe head injury.

Choroidal Injuries

Choroidal Tear (Fig 1.13, 1.14)

Oedema of the choroid does not usually threaten vision, nor does a rupture or tear of the choroid except when it involves the macular area when loss of central vision results (Duguid, 1985). Posterior choroidal ruptures are probably caused by antero-posterior compression and horizontal expansion. The retina is relatively elastic and the sclera is relatively tough. Both resist rupture. Bruch's membrane, on the other hand, is inelastic and more prone to rupture. Rupture of the adjacent choriocapillaris often results in subretinal haemorrhage. Patients with angioid streaks and other conditions known to be associated with a fragile Bruch's membrane are especially vulnerable to choroidal rupture (Wood & Richardson, 1990[a]). Fluorescein angiography can be useful in detecting small ruptures. Choroidal tears frequently result from severe contusion to the eye. They are commonly crescentic to the optic disc, vertical, and of variable length. They often lie between the disc and macula (Wood & Richardson, 1990[b]). Only rarely is a rupture radially oriented. Such ruptures are usually temporal to the disc and single, although nasal and multiple ruptures can also occur (Wood & Richardson, 1990[b]). No treatment is usually needed. However, a choroidal tear can be complicated by the development of

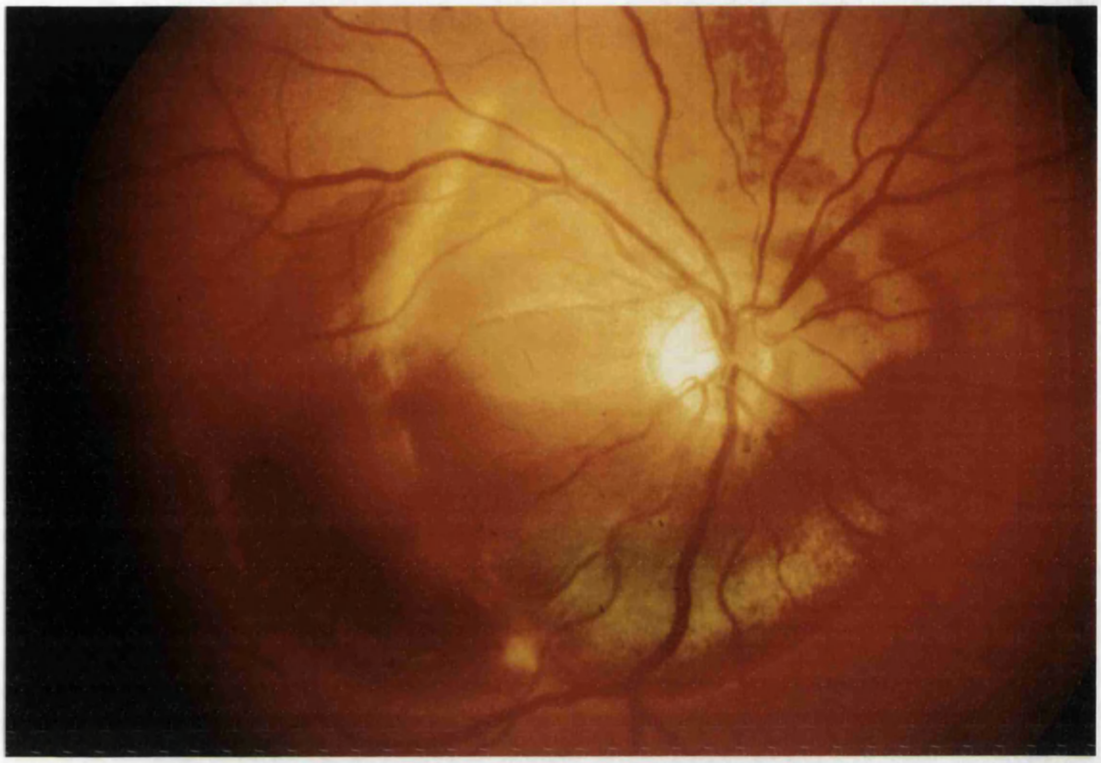


Figure 1.13 A traumatic choroidal tear has resulted in subretinal haematoma, which is clearing to reveal the tear passing through the macula.

Figure 1.14 Chorioretinal necrosis and haematoma due to an airgun pellet injury to the sclera.

subretinal new blood vessels, which may themselves bleed (Wood & Richardson, 1990[a]). In some cases, such a lesion may warrant laser photocoagulaion. All patients with a choroidal tear should, therefore, be evaluated and followed up by an ophthalmologist.

Choroidal Effusion

In cases of ocular hypotony, the choroid may detach from the underlying sclera owing to the accumulation of underlying plasma-like fluid. Treatment for the hypotony (eg. by repairing a scleral rupture) usually results in spontaneous reapposition of the choroid.

Scleral Injuries

Severe trauma can rupture the sclera (Cherry, 1972). It is important to recognise that this rupture may be "silent" and that sometimes the only clinical sign is ocular hypotony. The two most common sites for indirect rupture are the superonasal quadrant close to the limbus and parallel to the muscle insertions between them and the equator (Cherry, 1972). Radial and posterior ruptures are relatively uncommon (Slingsby & Forstot, 1981; Cherry, 1972). In one series of 34 cases of scleral rupture, 18% were associated with orbital fracture (Cherry, 1972). The hallmarks of scleral rupture are hypotony (although this is not always present), an abnormally deep anterior chamber, decreased ocular movements, and severe subconjunctival oedema (Cherry, 1972). If a scleral rupture is not repaired, persistent hypotony or the ingrowth of fibrous tissue into the eye may develop. In addition, sympathetic ophthalmia may rarely complicate scleral rupture (Rao et al, 1983; Rao & Wong, 1981; Lubin et al, 1980).

When the diagnosis is clinically difficult, ultrasonography and CT scanning can be helpful. The prognosis for useful vision in a ruptured globe is poor (Cherry, 1978; Milauskas & Fueger, 1966). Surgical exploration and repair is indicated in the majority of cases of persistent hypotony in which there is no alternative explanation, as repair of the scleral rupture is usually accompanied by restoration of intraocular pressure and prevents the complication of fibrous ingrowth (Cherry, 1978).

Optic Nerve Injuries

Rowbotham (1964) has classified traumatic optic neuropathy on the basis of a review of 3.6% (66 cases) of 1800 patients with facial injury.

Anterior Marginal Tear (12%)

This tear is diagnosed on the basis of the following signs. The development of a nerve fibre bundle visual field defect associated with early optic nerve pallor in one sector is suggestive of anterior optic nerve injury. This problem may be accompanied by spasm of the retinal vessels and, in some cases, haemorrhage at the optic disc margin that is associated with subsequent pigmentation. The disturbance of visual acuity and visual field depends upon the site and severity of the injury. Loss of vision is incomplete, but only little recovery takes place. The mechanism of damage is probably torsion of the globe with respect to the optic nerve.

Damage to the Anterior Optic Nerve (13%)

This condition follows frontal head injury and is usually accompanied by a local skull fracture. There is evidence of injury to the optic nerve behind the level of the optic disc but anterior to the entry of the central retinal artery.

Clinical examination of the posterior pole reveals the appearance of a central retinal artery occlusion with retinal oedema. Severe and complete loss of vision was seen in the majority of Rowbotham's cases.

Canalicular Optic Nerve Damage (65%)

In most cases (70%), this damage follows frontal injury. The patient is subject to immediate loss of vision. The prognosis for vision is poor, with fewer than 20% of patients developing useful recovery of vision. Pallor of the optic disc ensues with an average time of onset of 23 days. In Rowbotham's series, visual field loss occurred primarily in the lower visual field in 70% of cases, which lent support to a vascular aetiology for this injury, as it mirrored the clinical features of presumed hypertensive and arteritic posterior ischaemic optic neuropathy (Rowbotham, 1964). Such injury is probably due to stretching, thrombosis, or tearing of the small vessels supplying the optic nerve, and surgical decompression is probably not indicated. The indication for surgical exploration is that of initial recovery of vision with late deterioration, in which case the deterioration may occasionally be reversed by dividing the traumatic arachnoid adhesions that may develop.

Heinze (1969) carried out a study of the pathology of the optic nerve in patients who sustained traumatic optic neuropathy and subsequently died. In the majority of cases, haemorrhage into the optic nerve sheath was seen. In only 1 of 21 cases was laceration of the nerve observed, and partial division of the optic chiasm occurred in one other case. Perhaps intra-sheath haemorrhage compresses and occludes small blood vessels to the nerve fibres, or haemorrhage or thrombosis within the nerve causes an ischaemic atrophy (Anderson, 1982; Edmund and Godtfredsen, 1963; Gordon and Macrae, 1950). Contusion necrosis of the optic nerve has also been suggested (Shammas and Matta, 1974). Severe intra-orbital haemorrhage may result in a preventable post-traumatic atrophy of the optic nerve (Butt, 1979).

Comparison of groups of patients treated with or without operative decompression of the optic nerve reveals that the overall prognosis for visual recovery is the same in both groups, with improvement in vision in 25% to 40% and no recovery in the remainder (Walsh & Hoyt, 1969). However, when a course of systemic steroids was given as standard to all patients, the rate of visual improvement rose to 56% on steroids alone with additional improvement of 33% following optic nerve decompression in selected cases (Mauriello et al, 1992). Recovery of vision following removal of haematoma in the optic nerve sheath without pre-operative steroid treatment has also been reported (Gjerris, 1976). Spontaneous recovery from traumatic optic neuropathy following blunt head trauma has also been described (Wolin & Lavin, 1990).

The role of decompression procedures remain controversial, and the indications are not clear. It has been suggested that a narrowed or dislocated optic foramen, accompanied by slowly progressive loss of vision, provides a relative indication for surgical exploration (Walsh & Hoyt, 1969). A lucid interval of vision after injury and an enlarged optic nerve sheath have also been implied to be associated with improved prognosis following optic nerve sheath decompression (Mauriello et al, 1992).

Unlike with rapid loss of vision, for which there is probably no effective treatment, high-dose steroids appear to be of benefit if there is gradual, progressive loss of vision (Spoor et al, 1990; Lam & Weingeist, 1990; Anderson et al, 1982).

A more severe complication of blunt trauma is avulsion of the optic nerve from the eye (Fig 1.15). Although this usually is accompanied by severe damage to other ocular tissues, it can be the sole manifestation of an apparently minor ocular trauma following midfacial fracture (Ketchum et al, 1976).

Chiasmal Injury

Frontal injury close to the midline is most likely to give rise to chiasmal injury. Although occasional cases of

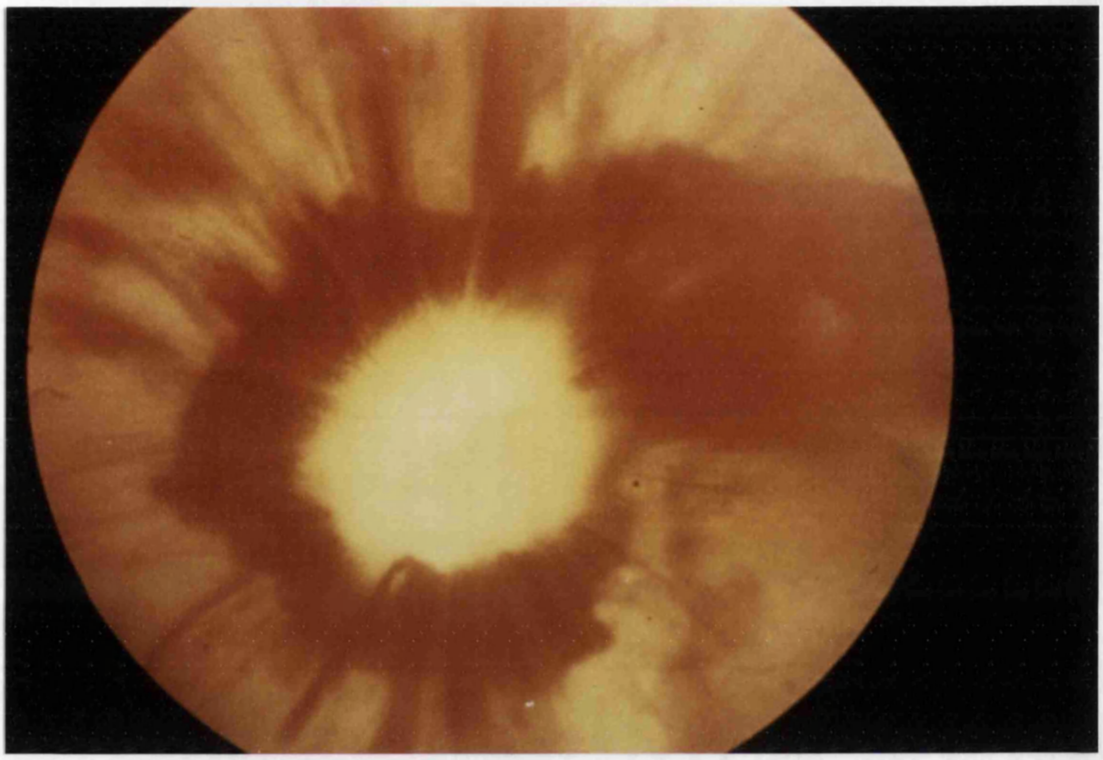


Figure 1.15 Optic nerve avulsion following severe facial trauma.

chiasmal tears have been reported (Heinze, 1969), in the majority of instances, no microscopic injuries are seen. The result is immediate bitemporal hemianopia, which may be explained by occlusion of the central chiasmal artery (Russell, 1960). In some cases, optochiasmal injuries may occur in which the ipsilateral eye is blind and the contralateral eye has temporal hemianopic field loss. Patients complaining of subtle impairment in visual function following head injury should undergo a detailed examination of the visual field, as subtle upper quadrantic bitemporal field defects may occur that would otherwise be missed.

Retrobulbar Haemorrhage

Retrobulbar haemorrhage is an uncommon sequel to facial fractures (Ord et al, 1986). In the majority of cases, this problem resolves spontaneously, with no adverse sequelae. However, blindness attributed to retrobulbar haemorrhage has been reported following ocular trauma (Babajews & Williams, 1986), malar fracture reduction (Ord, 1981; Gordon & Macrea, 1950), orbital floor repair (Nicholson & Guzak, 1971), orbital surgery (Ord, 1981) and blepharoplasty (Putterman, 1975). Venous haemorrhage probably causes no problem, but arterial haemorrhage can lead to compromise of optic nerve function and central retinal artery obstruction (Wood, 1986; Katz et al, 1983).

The patient usually complains of a severe aching pain accompanied by progressive loss of vision. Proptosis occurs and is accompanied by raised intraocular pressure, marked subconjunctival haemorrhage, and gross eyelid swelling (Wood, 1986). As vision is lost, the pupil becomes fixed and dilated. Immediate surgical decompression is required (Katz et al, 1983).

Lacrimal Passages

Disorders of the lacrimal passages are not uncommon in crushing injuries (Mewe, 1980). However, damage to the lacrimal drainage system has been reported in only 0.5% of 337 midface fractures (McCoy et al, 1962). The infrequent occurrence of epiphora following nasal fractures is probably due to the protective influence of the medial canthal ligament, which may prevent tearing of the upper lacrimal pathways (Stranc, 1970[b]). Avulsion of the medial canthal ligament following trauma can lead to displacement of the lower lacrimal punctum and the lacerated ligament should be sutured in the correct position as soon as possible.

Postoperative epiphora is frequently due to malposition of the lower eyelid and eversion of the punctum (Gruss et al, 1985). In severe nasal-orbital-ethmoid fracture, open reduction is usually preferable to avoid malpositioning of the bone fragments, which can lead to nasolacrimal apparatus obstruction (Gruss et al, 1985). In most cases of nasolacrimal injury, it is the bony canal that is obstructed.

Dacryocystorhinostomy is indicated no sooner than 3 months following injury, as spontaneous recovery of function may take place. In addition, some individuals do not develop epiphora in the long term and, therefore, do not warrant surgery.

1.5.2 Disorders of Ocular Motility

Disturbances in ocular motility may arise from:

1. Disorders of the central control of eye movement.
2. Injury to cranial nerves III, IV, or VI; or
3. Orbital injury with associated muscle injury or entrapment.

1. Disorders of Central Control of Eye Movement

Impairment of Accommodation and Convergence

Weakness of accommodation and convergence may follow head injury (Doden & Bunge, 1965). Most cases show parallel defects of the two functions as would be expected from their synergistic relationship. Occasionally, either may be deficient separately (Cross, 1945). Sometimes a brain stem or third nerve lesion may be responsible for causing either the combined defect or a weakness of either action alone. In many cases no organic cause can be detected (Duke-Elder, 1972).

Failure of convergence has also been reported following whiplash injuries to the cervical spine (Anderson, 1961). In some cases, it may be difficult to determine whether failure of convergence results from organic pathology or is functional in origin.

In the majority of cases, spontaneous resolution takes place. Occasionally, accommodation and convergence do not recover (Candler, 1945).

Loss of Fusion

Head injury may lead to loss of fusion of the images from both eyes resulting into diplopia (Stanworth, 1974; Hart, 1969). Trauma is also a recognised cause of the breakdown of a pre-existing heterophoria (Lee, 1983). Both cerebral contusion with or without skull fracture and whiplash injury may cause this problem. Spontaneous recovery may or may not occur.

Lateral Gaze Palsy

Lesions of either the frontal cortex or the pons may cause lateral gaze palsy, with or without persistent conjugate deviation of the eyes. In cerebral contusion, the eyes are deviated towards the damaged side, and spontaneous recovery of function usually occurs within 1 to 4 weeks (Lee, 1983). Pontine trauma, on the other hand, manifest a range of other clinical signs which tend to persist.

Skew Deviation

Skew deviation, where there is a vertical disparity in the position of the eyes on eccentric gaze, may result from a closed-head injury. However, the sign is not of localising value (Lee, 1983).

Parinaud's Syndrome

Parinaud's Syndrome results from midbrain trauma. The pupils are mid-dilated and fixed, and there is impairment in convergence and paresis of elevation (Lee, 1983).

Post-Traumatic Nystagmus

This may occur as a result of trauma to the petrous part of the temporal bone, with associated damage to the labyrinthine system, or may be due to brainstem trauma (Lee, 1983).

2. Cranial Nerve Injury

Up to 25% of all maxillofacial fractures are associated with head injuries (Walsh & Hoyt, 1969). Thus, in addition to direct trauma, various degrees of CNS damage with altered levels of consciousness as well as possible trauma to cranial nerves can present a difficult and complex diagnostic problem (Barr, 1970).

Typically, traumatic palsies of cranial nerves III, IV, and VI are caused by closed head injury, and a major cause is road traffic accident (Lee, 1983). The reported incidence of ocular palsies associated with head injuries ranges from 3% (Russell, 1960) to 7% (Hughes, 1964) although these figures must reflect referral criteria. Almost half of traumatic ocular palsies are due to road traffic accidents (Rucker, 1958). There is little relationship between the actual site of the trauma and the motility disturbances which ensues. Typically, the rootlets of origin of the III, IV, and VI nerves may be avulsed at their origin from the brain stem irrespective of the actual site of trauma (Heinze, 1969).

In a series of 1000 cases of ocular nerve palsies (Rucker, 1958), 16.8% were found to be traumatic in origin (30% 3rd cranial nerve, 14.5% 4th cranial nerve, 34% 6th cranial nerve, and 21.5% combinations). In a detailed neuropathic assessment of 21 patients in whom cranial nerve palsies developed following road traffic accidents but who failed to survive, cranial nerve III injuries comprised avulsion from the brainstem or contusion necrosis and intraneural haemorrhage in the region of the superior orbital fissure in equal proportion. There were six cases of abducens nerve injury. In five cases the nerve had been avulsed, and in only one was damage found at the point where the abducens nerve passes over the tip of the petrous temporal bone. Haemorrhage into the extraocular muscles close to the globe was found in 12 cases. This haemorrhage was commonly, but not invariably, associated with orbital fracture (Heinze, 1969).

Oculomotor Nerve Palsy

Oculomotor nerve palsy typically occurs following a frontal blow to the head, such as in a motorcycle accident. The condition is usually unilateral. There is ptosis, fixed dilation of the pupil, and abduction with slight depression of the globe. Three outcomes are possible: (1) no recovery, (2) recovery because of regeneration, and (3) aberrant regeneration. Recovery may take between 6 and 9 months to occur.

Trochlear Nerve Palsy

Trochlear nerve palsy may be unilateral or bilateral. Vertical diplopia, which may be accompanied by torsional diplopia, occurs. Torsion is especially problematic with bilateral cases. Frontal anteroposterior trauma is the most common cause (Burger et al, 1970). The presumed site of injury is the origin of the trochlear nerve from the posterior brainstem, perhaps following contre-coup injury upon the tentorium cerebelli. Spontaneous recovery may occur 3 to 6 months after injury. Surgery for squint is deferred to allow time for recovery to take place (Lee, 1983).

Abducens Nerve Palsy

The nerve may be damaged at its exit from the brainstem or within the cavernous sinus resulting in failure of abduction. Abducens palsy may also result from a traumatic caroticocavernous fistula. Surgery for squint is again deferred to allow spontaneous recovery to occur (Lee, 1983).

3. Mechanical Muscle Palsies

Diplopia is a common sequel to orbital injury. Causes of trauma-related motility disorders include entrapment of an extraocular muscle or its adjacent fascia in a fracture site, intramuscular haemorrhage and oedema, orbital haematoma, disinsertion of an extraocular muscle, and injury to the trochlea (von Noorden, 1979).

Blow-out Fracture

Anatomy of the orbit and pathogenesis of the orbital blow-out fracture as well as the appropriate radiological investigations have all been described (Section 1.3-1.5).

Clinical Signs

A blow-out fracture may be manifested by one or more of the following clinical signs:

1. Enophthalmos may initially be masked by tissue oedema. It may result from the combined effects of enlargement of the volume of the orbital cavity, prolapse of orbital fat, fat necrosis due to trauma or infection, and fibrotic shortening of extraocular muscles.

2. Impairment of ocular movement, which may cause diplopia (Crumley et al, 1977), may be due to muscle entrapment, fascial entrapment, injury to extraocular muscles, intraorbital or intramuscular haemorrhage, nerve damage, or the breakdown of a pre-existing heterophoria.

Typically, there is restriction of up and down gaze. Elevation of intraocular pressure on attempted elevation is suggestive of entrapment. Occasionally, retraction of the globe on upward gaze may be seen, owing to inferior rectus muscle entrapment. A positive forced duction test is helpful in confirming this diagnosis.

3. Pseudoptosis and deepening of the supratarsal fold accompany the enophthalmos. Attempted elevation of the affected lid leads to lid retraction on the opposite side.

4. Orbital emphysema may be seen shortly after injury but absorbs spontaneously.

5. Infraorbital nerve anaesthesia may occur. In 55% of cases, the symptoms resolve within one year of injury (Gillison & Oei, 1975), and many patients are symptom free in 2 years. However, persistent pain and dysaesthesia may be a sequel, especially to an orbital rim fracture.

Serious injury to the eye can also occur in up to 40% of cases (Leibsohn et al, 1976; Fradkin, 1971; Emery et al, 1971; Dodick et al, 1969; Milauskas & Fueger, 1966).

Fractures of the Orbital Roof

The incidence of orbital roof fractures in patients suffering from facial bone fractures is approximately 5% (McLachlan et al, 1982; Schultz, 1970). Undisplaced, blow-in, and blow-out fractures have all been described (Curtin et al, 1982; Sato et al, 1978). Bone fragments can interfere with superior rectus muscle function and cause impairment of elevation and ptosis of the upper lid. In addition, penetrating injuries may perforate the orbital roof and enter brain tissue (Flanagan et al, 1980).

The orbital roof is particularly weak at its posterior aspect and in the region of the superior orbital fissure and optic canal. Thus, the optic nerve and nerves passing through the superior orbital fissure may all be injured in various combinations. Epidural haematoma, depressed fracture of the superior orbital rim, persistent pneumocephalus with cerebrospinal fluid rhinorrhea, and tension pneumocephalus have all been reported to complicate such injuries (Wesley & McCord, 1982; Flanagan et al, 1980). Prolapse of orbital tissue into the frontal air sinus may also occur (Curtin et al, 1982).

A forced duction test may be positive, particularly in the upward direction, because the bones impinge on the muscle and mechanically restrict upward gaze. This finding can cause diagnostic confusion with an inferior blow-out fracture (McLurg & Swanson, 1976).

Trauma to the Trochlea

The superior oblique muscle passes through the trochlea close to the medial superior orbital margin. If the muscle is injured, subsequent fibrosis can result in tethering, which in turn leads to additional restriction of elevation of the eye. Entrapment of the superior oblique tendon in an orbital roof fracture has also been described (Al-Qurainy et al, 1988). Many patients recover spontaneously, but injection of steroids or surgical intervention may be required.

Displacement of the Globe

The eye may be displaced in any dimension following orbital injuries, namely, anteroposteriorly, horizontally, or vertically.

Proptosis

Proptosis is common initially, owing to haematoma and swelling of orbital tissues. In the majority of cases, this condition resolves spontaneously. Subperiosteal haematoma, notably of the orbital roof (Wolter, 1979), may give rise to persistent proptosis associated with downward displacement of the globe. Persistent proptosis may also result from inward displacement of orbital bone fragments.

Enophthalmos

Enophthalmos is a common late sequel that may initially be masked by intraorbital tissue swelling and haematoma. Expansion of the orbit, prolapse of soft tissue through a blow-out fracture, necrosis of soft tissue, and fibrosis may all contribute to the clinical picture (Converse et al, 1961).

Vertical Displacement

Vertical displacement commonly accompanies orbital fracture. In the acute phase, the eye may be displaced upward by haematoma. In the late phase, downward displacement of the globe is more common.

Horizontal Displacement

Horizontal displacement occurs laterally when the medial canthal ligament has been severed or the orbital bones have been laterally displaced (Mustardē & Rycroft, 1971).

1.6 TRAUMA TO THE FACE: REVIEW OF LITERATURE

1.6.1 Midfacial Trauma

Blow-out Fracture of the Orbital Floor

Lang (1889) was the first to suggest that a fracture of the orbit may be responsible for the condition he called 'Traumatic Enophthalmos'. This was emphasized and further described by Pfiffer in 1943 and Converse and Smith in 1956, who later added diplopia to its features and coined the term 'Blow-out Fracture'. King and Samuel (1944) reported a case of blow-out fracture of the orbital floor caused by a sudden increase in the intraorbital pressure which had resulted from a traumatic force to the soft tissue of the orbit. In 1957, Smith and Regan carried out experimental studies which suggested that blunt trauma to the orbit resulted in increased hydraulic pressure of the intraorbital contents thereby causing a downward fracture of the thin walled orbital floor. This theory was also supported by Converse et al (1961) who described muscle entrapment in an orbital floor as a cause of enophthalmos and diplopia.

In 1964, Dingman and Natvig, plastic surgeons, reported for the first time a case of blow-in fracture of the orbital floor due to the buckling effect of a severe blow to the orbital rim.

In 1966, Atiyeh and associates added paraesthesia of the infraorbital nerve distribution to the features of the true blow-out fracture of the orbital floor whilst DeHaven and Harle (1966) reported several cases of "black eyes" due to such fractures and called for careful examination of the orbit in such cases. In the same year, Milauskas, an ophthalmologist, and Fueger, a radiologist, reported a series of 84 consecutive patients with blow-out fractures, 14% of whom suffered severe ocular injuries. They concluded that complete ophthalmic examination is essential in all suspected cases of blow-out fractures.

Converse and colleagues (1967) were the first to describe the term "impure" in contrast to "pure" blow-out fracture, and reported the aetiological factors in 100 blow-out fractures. Around the same time, Jones and Evans, both maxillofacial surgeons, used fresh cadavers to produce experimental blow-out fractures in order to ascertain the common site of fracture. The relative thickness of different areas of the bony orbital floor and orbital plate of the ethmoid was measured, and the relation of the geometric orbital axis to the orbital walls investigated. They found that in 79% of cases, the main site of the fracture was situated in the thin posterior part of the orbital floor, medial to the inferior orbital fissure and canal.

In 1970, Dodick and his fellow ophthalmologists, reported two cases of concomitant blow-out fractures of the orbital floor and rupture of the globe, and questioned the hydraulic concept as a cause of such injuries. They suggested, therefore, a buckling mechanism as the major cause of blow-out fractures. This view was supported by Fujino (1974) whose experiment on a dried human skull without orbital contents produced a blow-out fracture of the orbital floor suggesting a direct compression force or buckling force as the causative factor. Fujino and Makino (1980) further emphasized this opinion in their report of the results of clinical analysis of 101 cases of pure blow-out fractures of the orbital floor in which they argued that the buckling force was the primary causative factor in the production of such fractures. However, this view was not shared by Raflo (1984) who suggested that both factors might be involved in the production of such orbital fractures, nor by Converse (1977) who believed that the buckling force alone cannot explain the entrapment mechanism of the orbital contents, unless the primary causative factor was increased hydraulic pressure.

Blow-out Fracture of the Medial Orbital Wall

These fractures are reported to accompany orbital floor blow-out fractures in between 21% (Dulley & Fells, 1974) and 70% (Jones & Evans, 1967; Pearl & Vistnes, 1978) of cases. It is important to recognise this condition because of the potential sequelae of enophthalmos or, more rarely, entrapment, resulting in restriction of lateral gaze, for which the results of late surgery are poor. However, entrapment of orbital tissue in a medial wall fracture is rare (Davidson et al, 1975). Medial orbital wall fracture with entrapment may occur as a single entity or, more commonly, is concomitant with an orbital floor fracture (Pearl and Vistnes, 1978).

Miller and Glaser (1966) were the first to present a case of traumatic medial wall fracture of the orbit with incarceration of orbital contents leading to retraction of the globe and narrowing of the palpebral fissure on attempted abduction. In 1968, Edwards and Ridley also described a case in which blunt trauma to the orbit was followed by entrapment of the medial rectus muscle with resulting diplopia and impairment of ocular motility. They highlighted the value of the forced duction test in the diagnosis of such disorders. This view was supported by other authors including Thering and Bogart (1979) who reported a case of fracture of the medial orbital wall in association with a large fracture of the orbital floor with entrapment of the medial rectus muscle and 6th nerve palsy for which the diagnosis was made primarily by the forced duction test. Rumlet and Ernest (1972) reported a similar case of muscle entrapment and so did Tokel and Potter (1969), Fischbein and Leski (1969), Magnus et al (1971) and Davidson et al (1975) who reported two additional cases and emphasized the role of hypocycloidal tomography in the diagnosis of medial rectus entrapment in a medial orbital wall.

In an experimental study on fresh cadavers, Pearl and Vistnes (1978) found isolated medial wall blow-out fractures in 4 cases out of 13 cases (31%) studied. The rest were associated with orbital floor fractures. All medial wall fractures were located in the ethmoid bone midway between the medial canthus and the lesser wing of the sphenoid bone. They concluded that the discrepancy between the high frequency of occurrence of medial wall fractures in their study and the rarity of clinical diagnosis derived from the limitations of standard radiography and advocated the use of computed tomography (CT scan) to delineate these fractures. This view was further emphasized and supported by Risco and colleagues who, in 1984, reported a case of prolapse of the globe into the ethmoid sinus which was diagnosed by CT scan, and by Rauch (1985) whose diagnosis of two cases of a blow-out fracture of the medial orbital wall with concomitant soft tissue entrapment was confirmed by forced duction and computed tomography.

Blow-out Fracture of the Orbital Roof

Blow-out fracture of the orbital roof is increasingly being recognised as a complication of orbital trauma (Bleeker et al, 1975). However, it may be missed on skull radiographs (Gould & Titus, 1966). Computerised tomography (CT) can be employed effectively to demonstrate such injuries and its increased usage in craniofacial trauma may be responsible for the increased reporting of such orbital fractures (Wesley & McCord, 1982). Blow-in fracture of the orbital roof is extremely rare. There is only one reported case of such a fracture of the orbital roof which was caused by a horizontal shear strain to the skull (Sato et al, 1978).

Smith and Blount (1971) were the first to report a case of a blow-out fracture of the orbital roof in a 3-year-old child who presented with pulsating exophthalmos due to the communication between the subarachnoid space and the posterior orbit.

Interdisciplinary collaboration may have led to the recognition of more cases of orbital roof fracture, by raising the index of suspicion, than would have been otherwise. Bleeker and co-workers (1975) working in collaboration with neurosurgeons reported a series of 8 cases presenting in one year and so did Flanagan et al (1980) and McLachlan et al (1982) who reported 4 cases and 9 cases respectively in a similar cooperative studies.

Soft tissue entrapment in an orbital roof fracture is very rare. McClurg and Swanson (1976) described a case in which a fracture of the roof of the orbit, involving the anterior cranial fossa, has led to entrapment of soft tissue with diplopia, whilst Curtin and associates (1982) reported two additional cases of isolated pure blow-out fracture of the orbital roof, both with intra-orbital emphysema and small herniation of soft tissue in the frontal sinus. Only one case of true muscle involvement has been reported in which the superior oblique muscle tendon was entrapped in an orbital roof fracture leading to an acquired traumatic Brown's syndrome which was clinically manifested by impairment of elevation in adduction of the eye and radiologically proven by CT scan (Al-Qurainy et al, 1988).

1.6.2 Ocular Trauma in Relation to Facial Trauma

By the early seventies, a growing interest started developing in ocular and other associated complications of facial trauma. Fradkin (1971) reported 53 patients with blow-out fractures of the orbital floor, 40% of whom suffered severe ocular injuries. In a similar population of study, Kreiger (1971) detected an equal rate of associated ocular injuries in 10 patients whilst Smith and associates (1971) observed that 25% of their maxillofacial fracture patients sustained various degrees of damage to the CNS and cranial nerves II, III, IV, VI and VII. A retrospective review by Crumley and Leibsohn (1976) of 324 patients with 363 orbital floor fractures in isolation (38) or with associated midfacial fractures, showed that, on initial examination, 30% of all patients were found to have diplopia and 4% enophthalmos rising to 11% after five months. Thirty-seven percent of patients had demonstrable ocular injury at the time of initial examination ranging from simple subconjunctival haemorrhage to retinal detachment and rupture of the globe. Other serious, though rare, ocular injuries, have also been reported. Mandelcorn and Hill (1973) reported a case in whom a dynamite blast produced blow-out fractures of both orbits leading to the herniatin of one eye into the maxillary antrum and loss of vision. Two other incidents of herniation of a globe have been followed by successful repositioning of the eye with recovery of vision (Berkowitz et al, 1981).

1.6.3 Motility Disorder in Relation to Facial Trauma

Restricted extraocular motility and diplopia after blow-out fractures have been attributed to a variety of causes. Helveston (1977) believed that they were due to soft tissue oedema or haemorrhage or from damage to the nerve supply to the muscles rather than muscle entrapment. This view, however, is not shared by the majority of authors (Waddell, 1982; Koutroupas and Meyerhoff, 1982; Friedburg, 1980; Silva, 1973; Piddle, 1968) who are of the opinion that muscle entrapment was a likely factor in displaced fractures and that orbital floor exploration is a safe and effective means of reducing the complications associated with such fractures.

CHAPTER 2

AIMS OF STUDY

2. AIMS OF STUDY

Ocular and motility disorders are common following blunt midfacial trauma. However, review of previous work in this area has highlighted the paucity of clinical studies which relate the mode and characteristics of facial trauma to the underlying ophthalmic pathology.

The aims of this prospective study are as follows:

1. To categorise the facial injuries sustained by a cohort of patients according to aetiology, fracture type and therapeutic strategy required.

2. To determine the incidence and type of ophthalmic injury which accompany midfacial fractures.

3. To relate the data obtained and thereby to determine the risk factors which predispose to sight threatening eye injuries.

4. To design a scoring system in which the data elicited by the maxillofacial surgeon can be employed to ascertain which patients are at risk of eye injury warranting ophthalmic referral.

5. To evaluate the scoring system by asking the maxillofacial surgeons to apply it to a new cohort of patients all of whom are examined by an ophthalmologist.

CHAPTER 3

PATIENTS AND METHODS

3. PATIENTS AND METHODS

3.1 PATIENTS

The Oral and Maxillofacial Surgery Unit at Canniesburn Hospital, Glasgow is a regional centre. It undertakes the care and treatment of maxillofacial patients for about one third of the west of Scotland which has a total population of three and a half million people. The unit is also involved in the treatment of craniofacial fractures in the Regional Neurosurgical Unit at Southern General Hospital, Glasgow, but these patients (42 per annum on average) were included in the study only if they have been transferred to or followed up in Canniesburn Hospital. (This may have biased the sample towards less severe injuries).

A total of 363 patients who had sustained midfacial and/or frontal trauma resulting in fracture between 1st April, 1985 and 31st March, 1987 were admitted to this study. All patients with persistent pathology were followed up for at least one year. Patients with simple nasal or pure mandibular fractures without involvement of the zygomatico-orbital region were not included in this study since they were unlikely to have been complicated by ophthalmic injury (Holt et al, 1983).

Patients who had sustained major trauma were evaluated within 48 hours following injury, and those in whom there was no manifest ocular disorder (as judged by the attending maxillofacial surgeon) were evaluated at the ensuing weekly ophthalmic clinic.

Every effort was made to ensure the referral to the eye clinic of all patients of the above description irrespective of whether or not they had presented with manifest eye injury. The project was discussed with the senior and junior members of the staff at the maxillofacial unit stressing the importance of referral of such patients. Further discussion took place each time the medical staff rota changed. We also ensured that we attended the morning round with the maxillofacial surgeons on the day of the weekly ophthalmic clinic in order to identify patients requiring referral.

3.2 METHODS

All data were collected according to a detailed protocol.

3.2.1 History

A full history of the incident, including time, cause, related factors (such as influence of alcohol and the use of seat belts) and whether there was any trauma to the eye, was taken. The influence of alcohol was assessed by virtue of the amount of alcohol consumed as indicated by the patient (more than 2 units during the hour preceding the incident) or the patient's clinical status on first examination. A detailed past ophthalmic history such as errors of refraction, squint, amblyopia, eye surgery, and previous eye injuries was obtained. The occurrence of amnesia, its type and duration was recorded (Jennet & Teasdale, 1981; Teasdale & Jennet, 1974). Subjective symptoms of diplopia and their duration were noted as well as pain on eye movement, its quality, severity, and relation to direction of gaze.

3.2.2 Classification of Facial Fracture

Facial fractures were classified by the attending maxillo-facial surgeon by clinical and radiological examination into three groups: malar fractures according to Henderson (1973, unpublished) (Table 1.1; Fig 3.1-3.7), maxillary fractures as described by Le Fort (1900) with further extension as adapted by the maxillofacial unit (Table 1.2), and naso-orbito-ethmoidal fracture (Stranc, 1970[a]; Converse & Smith, 1963). Due to strict regional policy limiting the number of referrals, it was not always possible to perform an ultrasound or CT scan examination. However, CT scanning was performed on all patients with clinical evidence of a blow-out fracture of the orbit but

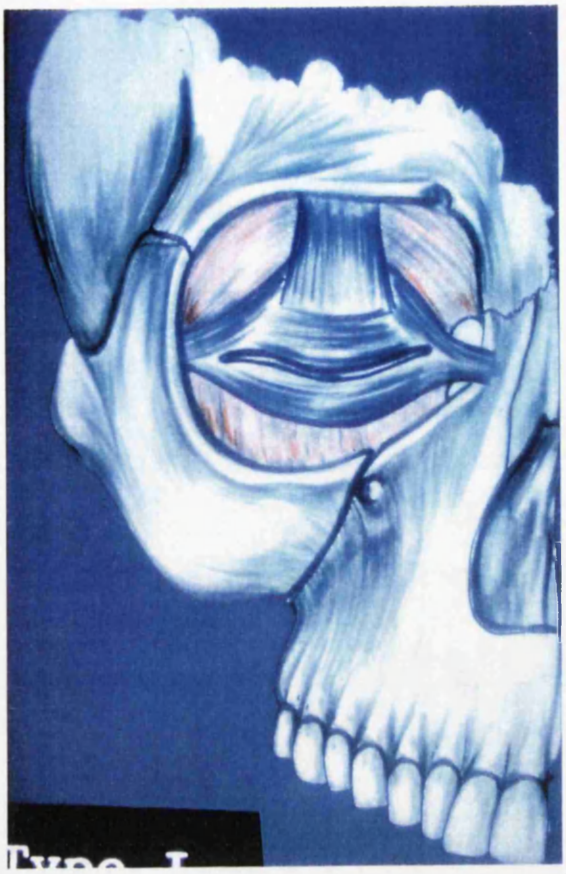


Figure 3.1 Type I #: undisplaced malar fracture.

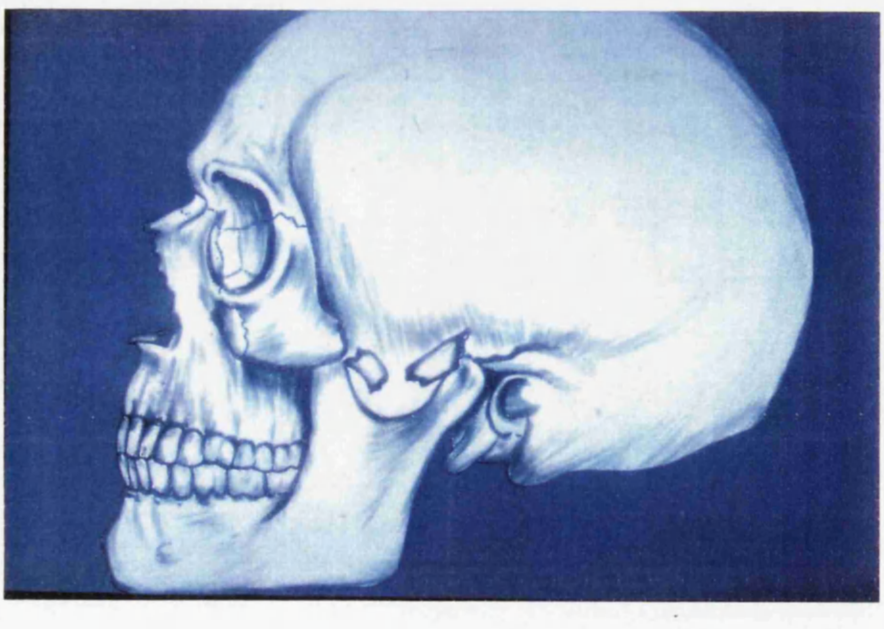


Figure 3.2 Type II #: zygomatic arch fracture only.

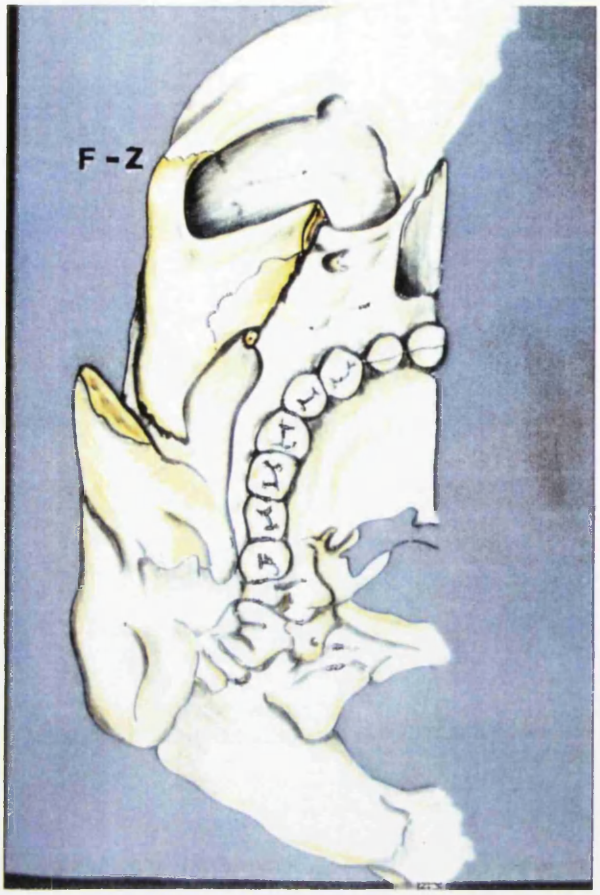


Figure 3.3 Type III #: Tripod fracture with undistracted fronto-zygomatic suture.

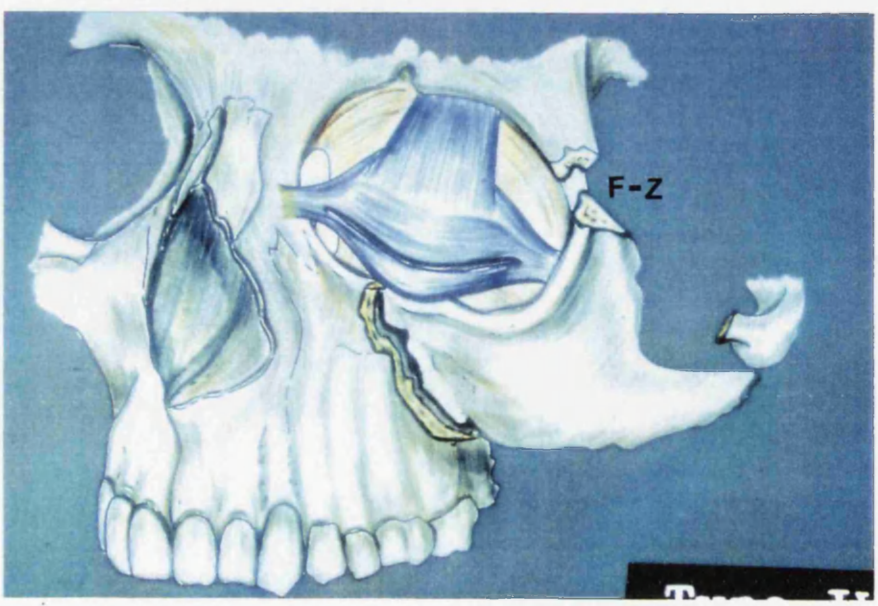


Figure 3.4 Type IV: Tripod fracture with a distracted fronto-zygomatic suture.

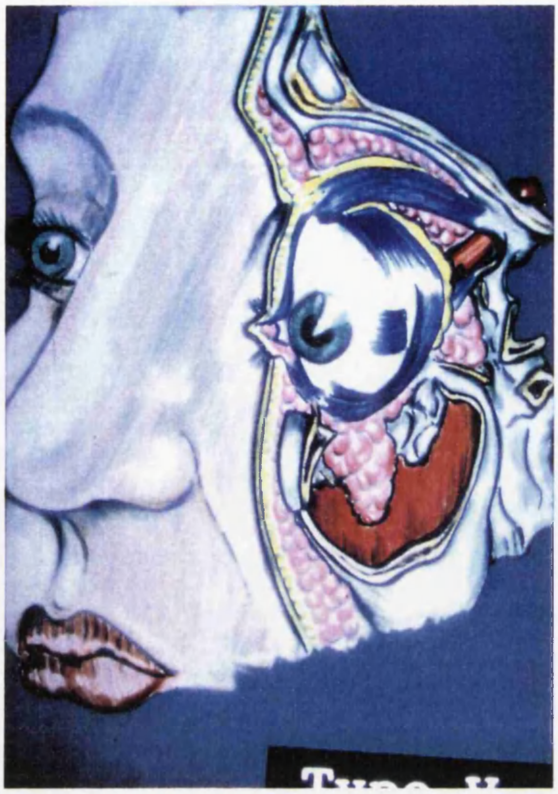


Figure 3.5 Type V #: Pure blow-out fracture of the orbit.

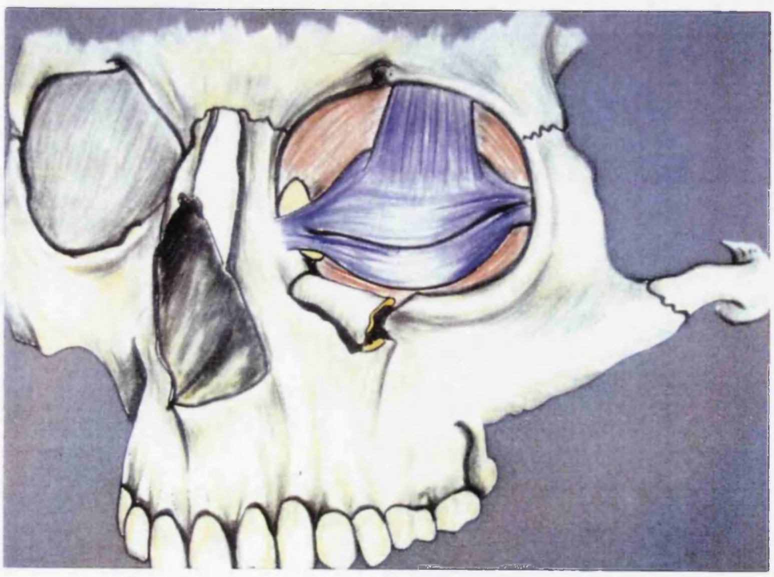


Figure 3.6 Type VI: Orbital rim fracture only.

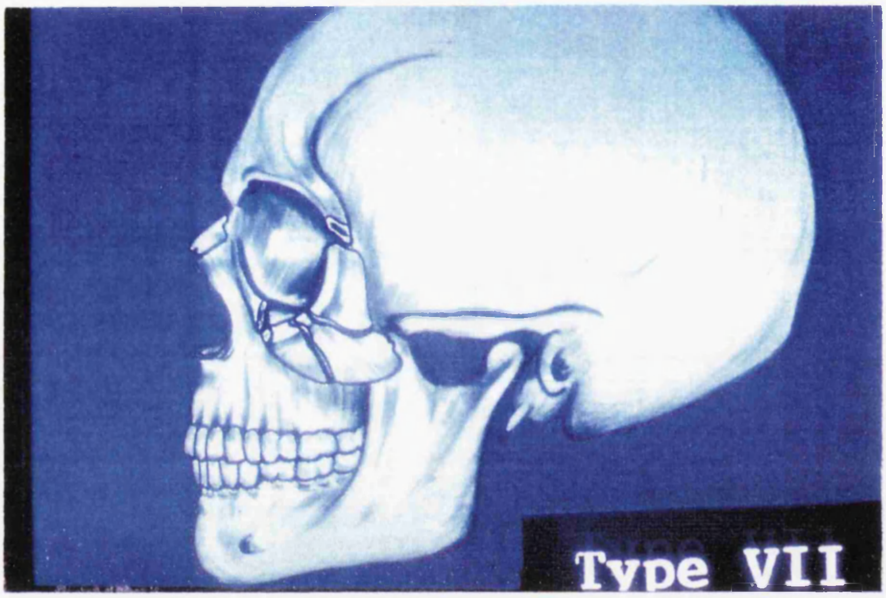


Figure 3.7 Type VII #: Comminuted fracture.

Figures 3.1-3.7 were reproduced by kind permission of the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital, Glasgow.

with negative plain radiographs except in those few in which exploration of the orbit was deemed necessary.

3.2.3 Treatment of Facial Fracture

Treatment of each facial fracture and the procedures employed were noted and recorded.

3.2.4 Ophthalmic Examination

All patients were subjected to full ophthalmic examination including detailed assessment of ocular motility.

Visual Acuity

All patients had their visual acuities tested, with appropriate spectacle correction when needed. A standard Snellen chart at 6-metres distance was used for this purpose. When the visual acuity was less than 6/6 in either or both eyes, assessment was carried out with the aid of a pinhole. Near vision was tested with a reduced Snellen chart. Failure of the pin hole to bring visual acuity up to 6/6 in an otherwise healthy looking eye indicated a possible macular dysfunction in which case the photo-stress test was performed (Glaser & Goodwin, 1991). Reduced corrected visual acuity was classified as folows:

Mild reduction: 6/9 - 6/12

Moderate reduction: 6/18 - 6/24

Severe reduction: 6/36 or less

Visual Fields

Assessment of red comparison in each quadrant was carried out and colour desaturation was also examined between the eyes. Visual fields were examined by confrontation for gross visual defects. The patient was seated to face the observer at arm's distance. He was then asked to fix the observer's eye while the fellow eye was covered. Using a small white pin, the field of vision to the moving target was compared with the observer's (normal) visual field. The test was then repeated for the other eye. Evidence of visual field loss in one or more areas called for more detailed assessment on a standard perimeter at the ophthalmic unit at the Western Infirmary.

Ocular Motility

All patients had their extra-ocular muscle function evaluated, and freedom or restriction of the individual movements noted and documented. The corneal light reflex test was performed and the pinpoint images of the reflected light by the two corneae were compared with respect to their position in relation to the pupil and to the cornea as a whole. The examination of ocular movements was carried out in all the principal positions of gaze and supplemented by the performance of the cover test in these positions.

The Cover Test

The cover uncover test was performed for near and for distance. When failed to elicit a manifest muscle imbalance, the alternate cover test was carried out. Each eye was covered in turn, the behaviour of each on uncovering was noted and finally both were uncovered. Attention was also paid to the speed of recovery of the binocular state.

The Hess Screen Test

All patients with suspected ocular muscle imbalance due to mechanical or neurological disorder with or without diplopia were tested on the Hess screen for assessment of the fields of binocular function. Lee's modification of the Hess screen was used and the patient was seated at a standard 0.5 metre distance from the screen. Each eye was tested in turn and the charts were plotted, interpreted and filed.

Assessment of Convergence and Accommodation

All patients were screened for evidence of convergence insufficiency and/or failure of accommodation. This was carried out by accurate measurement of the near points of convergence (NPC) and accommodation (NPA) on a standard R.A.F. near point rule and the results were recorded in centimetres. Normal figures for accommodation in relation to age were adopted from Duane (1922) and failure of accommodation was classified as follows:

Mild failure:	NPA equivalent to that of an older person by 1-5 years.
Moderate failure:	NPA equivalent to that of an older person by 6-10 years.
Severe failure:	NPA equivalent to that of an older person by more than 10 years.

Examination of the Globe

The external eye was thoroughly examined in anatomic sequence under diffuse light for evidence of gross ocular injury. The lids were examined for evidence of swelling, oedema, bruises, laceration, ptosis and abnormality of lid margin position (ectropion or entropion). The lacrimal drainage system was assessed for patency. External motility and sensory innervation about the eye was tested.

The pupils were examined for shape, size, equality and reactions to light and accommodation. The pupils of the two eyes are usually of about equal size and respond similarly to stimulation. Distortion and irregularity of the pupil may indicate injury to the sphincter (Newell, 1986). Manifest or suspected displacement of the globe following facial trauma was ascertained. Vertical and horizontal displacements were measured to the nearest millimetre. Antero-posterior displacement (exophthalmos or enophthalmos) was assessed using the Keeler frame in those patients in whom the lateral orbital margin was intact. Examination from above with careful delineation of the position of the globe with respect to the supra-orbital margin were carried out in all other patients.

Slit-lamp Examination

Detailed examination of the anterior segment of the eye was carried out using the slit-lamp microscope. A Zeiss microscope with facilities for low (10x) and high (25x) magnification was used for this purpose. The different parts of the anterior segment of the eye were examined in anatomic sequence for evidence of injury or disorder.

Gonioscopy was carried out with the Goldman three-mirror contact lens for examination of the angle of the anterior chamber when indicated, as in cases of suspected angle recession (hyphaema, distorted pupil, deep anterior chamber), iris dialysis and post-traumatic glaucoma.

Intraocular pressure was measured when clinically was suspected to be abnormal as in cases of corneal oedema or hyphaema, or likely to be abnormal in the future as in cases of angle recession. A hand held 'Perkins' tonometer was used after topical anaesthesia with amethocaine 1% drops.

Funduscopy

Funduscopy of both eyes following dilatation with tropicamide 1% drops was performed on all patients, first by direct and then by indirect ophthalmoscopy. Keeler's direct and indirect ophthalmoscopes were used and a 20-dioptre Nikon lens.

3.2.5 Data Coding

With the exception of the patient's name and reference number, all data whether general or clinical were given serial numbers for ease of recognition. The total number of parameters was 55. The code book employed comprises appendix 1.

Data regarding all patients were classified into three major categories:

1. **General data:** consisted of routine information readily available to the maxillofacial surgeon on initial examination and investigation. They included such information as sex and age of patient, and month of the incident. They also included details of the patient's present and past medical history, cause and type of facial and other fractures sustained and their definitive treatment in that sequence.

2. **Motility disorder:** dealt with symptoms, signs and investigations of motility disorder, as well as with treatment, recovery and complications of such ocular disorders.

3. **Eye abnormality:** included data on all ocular anatomical injuries sustained and their physiological sequelae, complications, treatment and recovery.

Details of each item of information were arranged in order of severity or frequency of occurrence in practice, or on an anatomical basis, and allotted numbers of rising value when a certain parameter did not need to be detailed further. Zero was given to non-occurrence (No) and (1) was given for occurrence (Yes). This served to identify at a glance those patients who suffered a particular abnormality from those who did not.

Data Sheets

Data sheets (Appendix 2) were designed to record coded data regarding patients who underwent ophthalmic consultation. This was done in a way as to permit coded information to be recorded in successive columns on a single raw. Recorded data appeared on the sheets in the same sequence of them in the code book (viz. Data Coding). Consequently, coded information regarding any parameter on all patients fell into the same column and hence be recognised immediately.

At the end of each working day, data sheets were completed with the coded information of the newly attending patients. Any additional information such as those concerning treatment, recovery, or complications would be added as they come. All coded data were stored in the mainframe university computer for future analysis (Appendix 3).

3.3 CLASSIFICATION OF OCULAR INJURIES ACCORDING TO THE NECESSITY FOR REFERRAL TO THE OPHTHALMOLOGIST

In their study, Holt et al (1983) classified eye injuries associated with facial fractures into three categories: minor or temporary, serious, and blinding. They defined minor eye injuries as those injuries without permanent visual or physiological sequelae; serious injuries were defined as those producing sustained visual loss or adnexal sequelae requiring subsequent reconstructive measures; and blinding injuries as those manifested by no light perception or surgical anophthalmos.

That classification served the purpose of Holt's study which aimed at highlighting the high risk of ocular involvement in cases of facial fractures. However, for a number of reasons, it is not entirely satisfactory for the purpose of determining guidelines for referral of such patients to the ophthalmologist by the attending maxillofacial surgeon. Blinding injuries represent extreme degrees of severity of ocular injury and therefore can fit into the category of serious eye injuries without being grouped separately. Moreover, the relation between serious eye injuries and blinding injuries is rather loose as the former can progress into the latter at a later stage. Furthermore, it is easy enough to categorise the minor and the serious eye injuries into well defined groups but in practice, the majority of eye injuries fall in between, being neither mild enough to be considered minor, nor severe enough at the time of initial examination to be considered serious eye injury.

For the purpose of this study (Chapters 6 and 7), ocular disorders were classified according to the necessity for referral to the ophthalmologist rather than the criteria of degree of severity of eye injury. In this context, patients have been classified into two major groups: those who need to be referred to the ophthalmologist on the basis of their ocular injuries, and those who need not. The referred cases are further subdivided into those who warrant referral soon on account of their need, and those who may be seen routinely. This system of classification was discussed with and evaluated by senior ophthalmic staff and a consensus view obtained.

DEFINITIONS

Non-Referral Cases (Table 3.1): Category of ocular injuries without permanent visual or physiological sequelae such as corneal abrasions and subconjunctival haemorrhages. These injuries heal spontaneously.

Routine-Referral Cases (Table 3.2): Category of ocular injuries which is unlikely to lead to permanent visual or physiological sequelae, but referral is indicated for the purpose of assessment or screening for pathology for which treatment may be indicated. Examples include minor eyelid and conjunctival lacerations and enophthalmos. Those patients may be referred to the ophthalmologist for a routine out-patient clinic assessment when convenient preferably within one month from the date of injury.

Early-Referral Cases (Table 3.3): Those ocular injuries, such as corneal lacerations and retinal damage, producing sustained visual loss or adnexal sequelae requiring advice at an early stage concerning management, therapeutic intervention or detailed appraisal for medico-legal reasons. Such patients require early referral as soon as their general condition permits to avoid the occurrence of potentially preventable damage or the risk of missing clinical signs of medico-legal significance.

Code	Eye Abnormality	Remarks
30	Post-traumatic neuralgia/ anaesthesia	Spontaneous recovery. No treatment needed.
31	Coronal eye displacement	Should respond to facial fracture treatment.
36	Orbital emphysema	Absorbs spontaneously.
37.1	Eyelid swelling/bruises	Clears spontaneously.
38.1	Conjunctival chemosis	Clears spontaneously.
38.2	Subconjunctival haemorrhage	Clears spontaneously.
39.1	Corneal abrasion	Heals spontaneously.
46.1	Mild failure of accommodation	Usually recovers spontaneously.
47.1	Mild reduction of visual acuity with recovery	May be due to corneal abrasion. No treatment is usually needed.
49.1	Mild commotio retinae	Spontaneous resolution.

Table 3.1 List of the 'non-referral' category of ophthalmic injuries. For each type of injury, the justification for the referral is given.

Code	Eye Abnormality	Remarks
34	Enophthalmos	Usually responds to orbital wall reconstruction.
37.2	Eyelid laceration	Can be treated by the maxillofacial surgeon.
38.3	Conjunctival lacerations	May or may not need suturing depending on extent and involvement of underlying tissue.
41	Traumatic pupillary changes	May indicate other pathology Observe and document.
42.1	Iridodialysis	May indicate other pathology Occasionally the iris may need to be repaired.
44.1	Lens damage: Vossius' ring	May indicate underlying pathology requiring treatment or follow-up, e.g. angle recession.
44.2	Lens dislocation/ subluxation	Treatment may be needed for post-traumatic complications e.g. glaucoma.
44.3	Traumatic cataract	Indicates need for future treatment
44.4	Ruptured lens capsule	As above
45.1	Positive photostress test	Macular oedema. Self limiting.
46.2	Moderate failure of accommodation.	No immediate treatment. May need refraction later.
46.3	Severe failure of accommodation.	As above.

Table 3.2 List of the 'routine-referral' category of ophthalmic injuries. For each type of injury, the justification for the referral is given.

Code	Eye Abnormality	Remarks
47.2	Moderate reduction of visual acuity	May be due to macular oedema corneal abrasion/oedema, traumatic mydriasis etc. Need to be investigated.
49.2	Moderate commotio retinae	To be documented and observed. If involving the macula, may progress into macular cyst/hole.
49.3	Severe commotio retinae	As above.
50.1	Choroidal tear: single	To be documented and observed.
50.2	Choroidal tear: multiple	As above.
51.1	Vitreous floater	Should be closely followed-up. May indicate underlying pathology, but treatment is not immediately indicated.
51.3	Traumatic pigmentary retinopathy	To be documented and observed.
53.	Naso-lacrimal damage	If not repaired by the maxillofacial surgeon.

Table 3.2 List of the 'routine-referral' category of ophthalmic injuries (Cont'd).

Code	Eye Abnormality	Remarks
33	Proptosis	May be due to retrobulbar haemorrhage leading to optic nerve compression.
35	Retrobulbar haemorrhage	As above.
39.2	Corneal damage: puncture wound	Needs urgent repair under microscope.
39.3	Corneal laceration	As above.
40	Scleral injury	Needs careful repair.
42.2	Hyphaema	May lead to rise of intraocular pressure.
43	Angle recession	To be followed-up closely for glaucoma. An acute pressure rise may occur.
47.3	Severe reduction in visual acuity	Optic nerve injury, retinal detachment, vitreous haem-orrhage etc. Aetiology must be established immediately.
47.4	*Loss of vision	As above.
48	*Visual field loss	To be investigated soon for retinal detachment and/or damage to the visual pathway
50.3	*Choroidal tear involving the macula	Document and observe prior to facial treatment for medico legal reasons.
51.2	*Vitreous haemorrhage	Needs urgent specialist examination.
51.4	*Flat retinal tear/hole	May need laser/cryotherapy.
51.5	*Retinal detachment with macula on	Needs urgent specialist treatment.

Table 3.3 List of the 'early-referral' category of ophthalmic injuries. For each type of injury, the justification for the referral is given.

Code	Eye Abnormality	Remarks
51.6	*Retinal detachment with macula off	As above.
51.7	*Total retinal detachment	As above.
52.1	*Optic nerve injury: contusion	Endangers sight. To be documented.
52.2	*Optic nerve: partial injury	As above.
52.3	*Optic nerve: total damage/avulsion	To be documented for medico-legal reasons.

Table 3.3 List of the 'early-referral' category of ophthalmic injuries (Cont'd).

* All of these signs indicate major injury. Accurate evaluation prior to surgical repair of the facial injuries is preferred so that loss of vision cannot be attributed to the surgical intervention and an appropriate sequence of management can be organised.

3.4 STATISTICAL METHODS

Various statistical tests were used to analyse data collected, the test employed in a particular case being noted as appropriate in the text.

The chi-squared (X^2) test was used with normally distributed data except when the low frequency of some occurrence rendered this test unreliable, Fisher's test was then employed.

For interval data, checks were made for normality of distribution. If the distributions were plausibly normal, the t-test was applied, while Welch's method was used if the sample variances seemed unequal. On the other hand, if data were non-normally distributed, the Mann-Whitney test or Wilcoxon rank sum test was chosen.

CHAPTER 4

THE CHARACTERISTICS OF MIDFACIAL FRACTURES AND THE ASSOCIATION WITH OCULAR INJURY

4. THE CHARACTERISTICS OF MIDFACIAL FRACTURES AND THE ASSOCIATION WITH OCULAR INJURY

4.1 INTRODUCTION

Facial fractures are frequently complicated by injury to the eye and its adnexae (Holt & Holt, 1983; Holt et al, 1983; Culbertson, 1983; Berine, 1981; Steidler et al, 1980; Petro et al, 1979). These injuries may result in loss of vision in one or both eyes (Babajews & Williams, 1986; Anderson et al, 1982; Steidler et al, 1980; Ketchum et al, 1976; Gwyn et al, 1971; Gordon & Macrae, 1950) or may compromise ocular function (Holt & Holt, 1983). When these injuries are severe, they may be detected with ease by any medical or maxillofacial practitioner but many injuries appear minimal and may be missed by the non-ophthalmologist (Holt et al, 1983; Records, 1979; Binder, 1978).

Ocular injury may result in preventable severe dysfunction of the visual apparatus, if not detected shortly after injury. In one published study by Jabaley et al (1975), the difference in the rate of detection of such ocular injury between a prospective evaluation, where an ophthalmologist was involved, and a retrospective evaluation, where only selective referral was made by the plastic surgeon, was 18% (29% and 11% respectively).

Ideally, all patients with a facial or craniofacial fracture should be reviewed by an ophthalmologist (Holt et al, 1983; Gwyn et al, 1979; Miller & Tenzel, 1967). Defects in the field of vision, ocular structure and ocular motility may be permanent (Stiedler et al, 1980; Turvey, 1977; Jabaley et al, 1975; Barclay, 1958). It is important to define such functional defects in order to treat any preventable ocular dysfunction such as retinal detachment, tears or holes, or angle recession glaucoma (Holt et al, 1983; Jabaley et al, 1975) and to advise the patient appropriately. The results of ophthalmic examination may lend weight to medico-legal claims.

Estimates (Table 4.1) of the incidence of ocular disorder following midfacial fracture vary considerably, being between 2.7% (Luce et al, 1979) and 67% (Holt et al, 1983). These estimates depend on referral practice, which specialty carried out the evaluation, and whether minor injuries were included in addition to major ones. The results also depend on whether the study was prospective (Jabaley et al, 1975; Miller & Tenzel, 1967) or retrospective (Holt & Holt, 1983; Steidler et al, 1980; Luce et al, 1979; Turvey, 1977; Jabaley et al, 1975; Morgan et al, 1972; Gwyn et al, 1971). Most studies which have been carried out have been retrospective and the details concerning the techniques of ophthalmic examination

Author(s)	Specialty	Eye Injury (No)	Eye Injury (%)	Total No. of Patients
McCoy et al (1962)	Plastic	8	1	855
Milauskas & Fueger (1966)	Ophthalmology	12	14	84
Converse et al (1967)	Plastic	5	10	50
Miller & Tenzel (1967)	Ophthalmology	5	17	30
Schultz (1967)	Plastic	36	9	400
Gwyn et al (1971)	Plastic	19	3.4	567
Morgan et al (1972)	Plastic	49	16.3	300
Turvey (1977)	Oral Surgery	24	4	593
Luce et al (1979)	Plastic	28	2.7	1020
Holt et al (1983)	Ophthalmology	487	67	727

Table 4.1 Estimates of the incidence of ocular disorder follow ing midfacial trauma in previous studies.

employed have not been described. Prospective studies have included only small numbers of patients (Miller & Tenzel, 1967).

This prospective study was set up to determine the incidence and types of ocular and motility disorders, as assessed by the ophthalmologist, in patients who had sustained midfacial fractures and who were under the care of a maxillofacial surgeon.

4.2 PATIENTS AND METHODS

Three hundred and sixty three patients with midfacial fractures due to blunt facial trauma who attended the Oral and Maxillofacial Unit at Canniesburn Hospital, Glasgow during the period 1 April 1985 to 31 March 1987 were admitted to this study. All patients were assessed pre-operatively and the minimum follow-up time was one year. The details of this group, the methods of assessment and Canniesburn's policy on timing of surgical intervention are described in chapter 3.

4.3 RESULTS

4.3.1 Type of Fracture

The study includes data concerning 363 patients with confirmed midface fractures who presented during a two year period. They had sustained a total of 438 midfacial fractures. The majority of fractures involved the malar bone either in isolation (280) or in combination with other midface fractures (61). Isolated fractures of the Le Fort type had been sustained by only 6 patients, while 29 others sustained Le Fort fractures in combination with various other midfacial fractures. Ten patients suffered an isolated naso-orbito-ethmoidal (N.O.E.) fracture while 3 others sustained a frontal bone fracture. Altogether, 64 of these 363 patients sustained fractures of two or more facial bones.

4.3.2 Age and Sex Distribution

Patients having midfacial fractures ranged in age from an 8-year old boy (who suffered a blow-out fracture when he hit a broken door handle with his closed left eye), to an 84-year old woman (who sustained a tripod malar fracture [Type III] in a fall). Males accounted for 81.3% (n=295) of all midfacial fracture patients, and females, 18.7% (n=68). The majority of patients were males between the

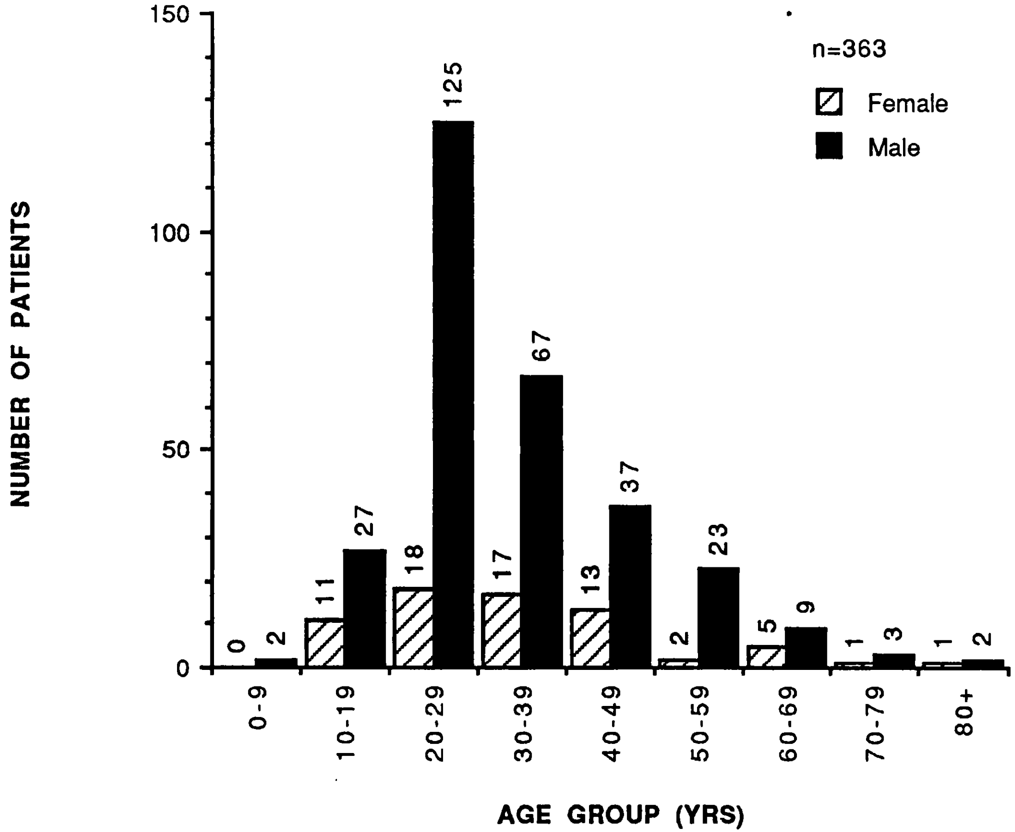


Figure 4.1 Age and sex distribution of the population of the study.

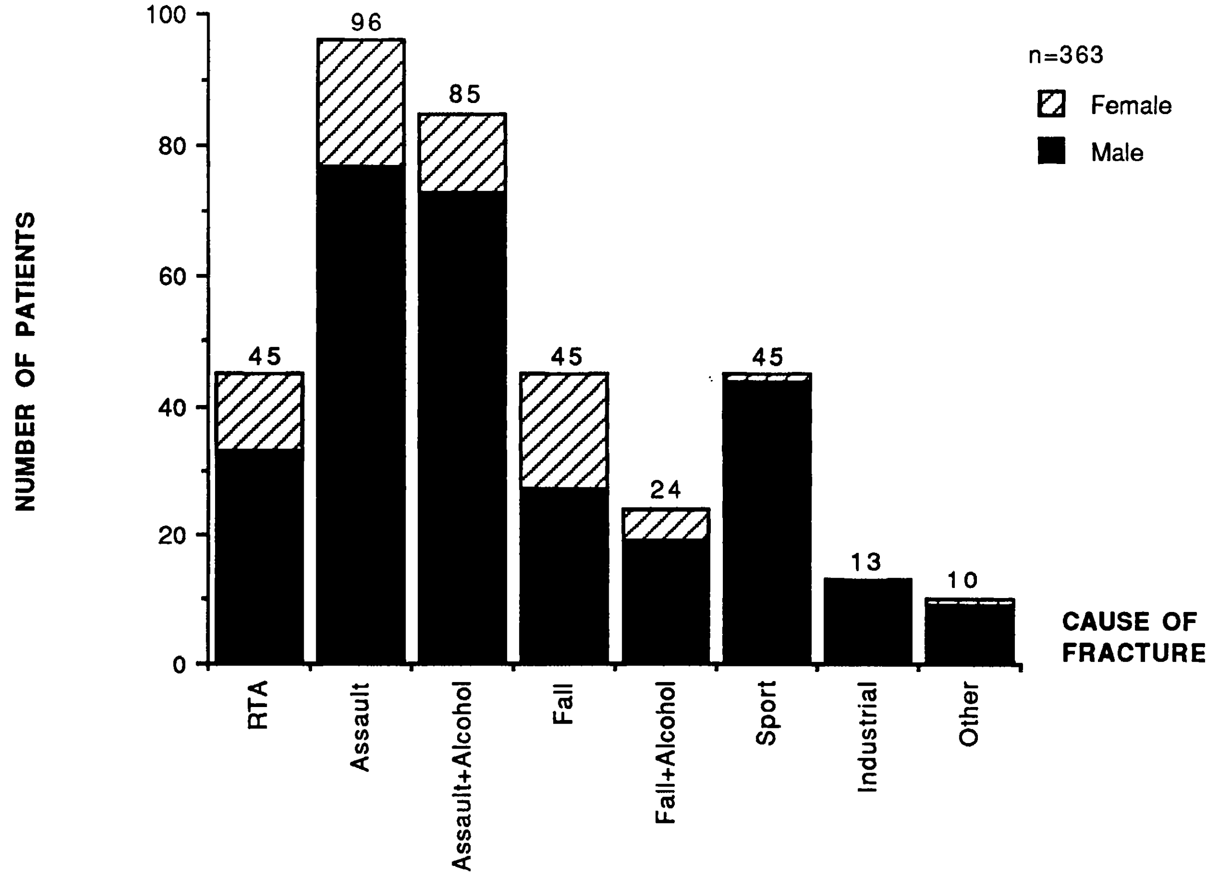


Figure 4.2 The aetiological distribution of midfacial fractures.

ages of 10 and 50 years with the peak incidence occurring in the 20 to 30-year age group for both sexes (Fig 4.1). In women, midfacial fractures were more prevalent in older age groups (60 to 80 years) than they were in men.

4.3.3 Monthly Distribution

The monthly distribution of incidence of injury showed June to be the month in which the greatest percentage of midfacial trauma occurred (14.3%), followed by March (12.4%), and May and September, (9.1%) each. June was the month in which most of the assaults, road traffic accidents and sporting accidents took place.

4.3.4 Cause of Injury

The major cause of midfacial trauma in this study was alleged assault, which accounted for 49.9% of all patients (181/363). Some of these allegedly assaulted individuals (n=85) were under the influence of alcohol when they sustained their trauma while others (n=96) were not. The second most common cause was falls (19% or 69 patients), of whom 24 (6.6%) had been under the influence of alcohol, followed by road traffic accidents and sporting accidents which affected 45 patients (12.4%) each. Figure 4.2 shows the frequency distribution of the causes of midfacial fractures in males and females.

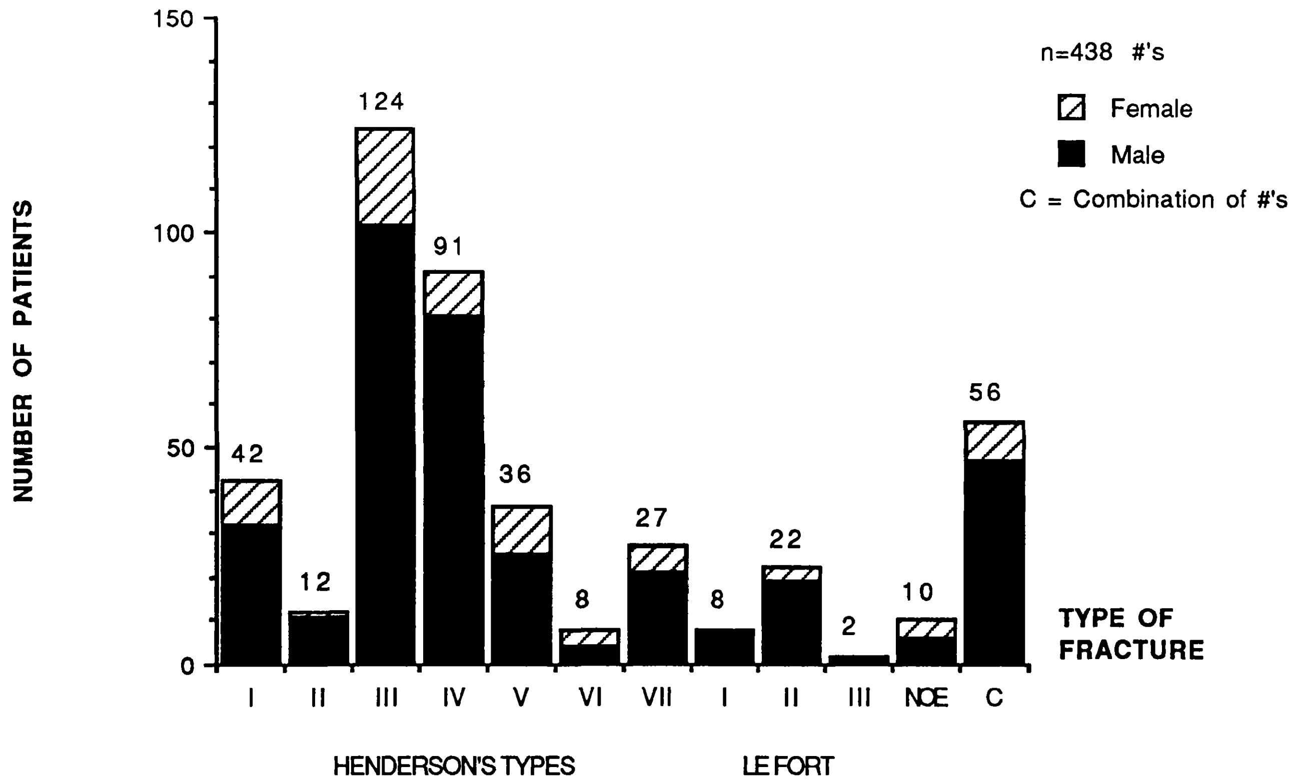


Figure 4.3 The site distribution of midfacial fractures.

Alleged assault was more common in males (50.9% of 295) than in females (45.6% of 68) while falls were more frequent in females (33.8%) than in males (15.6%). Road traffic accidents were also a more common cause of mid-facial trauma in females (17.6%) than in males (11.2%). Sporting accidents, on the other hand, were more than ten-fold more common in men (14.9% of 295) than in women (1.5% of 68).

Road traffic accidents were responsible for the injuries of 45 (12.4%) of the 363 patients. Within this group, 4 patients, all male, were involved in car accidents while not wearing seat belts, 16 in car accidents while wearing seat belts, 8 while sitting in a back seat of a car, 9 in motorcycle-related accidents and 8 in motor vehicle-pedestrian or other motor vehicle-related accidents.

Industrially-related accidents leading to midfacial fractures occurred only in males (n=13).

Figure 4.3 shows the distribution of midfacial fractures among the population studied. The most common anatomical type of malar fracture was the tripod zygomatico-orbital fractures with no distraction of the fronto-zygomatic suture (Type III) which affected 34.2% (n=124) of the patients, followed by tripod fractures with separation of the fronto-zygomatic suture (Type IV) in 25.1% (n=91) of the patients. Undisplaced zygomatic fractures (Type I) were third in prevalence with 11.6% (n=42). Less frequent fractures were pure blow-out fractures (Type V), (9.9%, n=36) and comminuted fractures (Type VII) which affected 27 patients (7.4%). Twelve patients (3.3%) sustained fractures of the zygomatic arch only (Type II), while another 8 patients (2.2%) suffered orbital rim fracture only (Type VI).

Table 4.2 demonstrates the different anatomical types of malar fractures in relation to the causal factors. In all causal groups except motorcycle injuries and motor vehicle-pedestrian related accidents, tripod fracture with no distraction to the fronto-zygomatic suture (Type III) was the most common type of zygomatico-orbital fracture. Motorcycle injuries and those related to pedestrians resulted in a larger percentage of tripod fractures in which the fronto-zygomatic sutures were distracted (Type IV).

4.3.5 Amnesia

Amnesia (fig 4.4) was common in association with midfacial trauma. One hundred and three patients (28.4% of 363) suffered from one type or another of amnesia, which was more common in males (89/295=30.2%) than in females (14/68 =20.6%). Twenty two males and 5 females suffered retro-

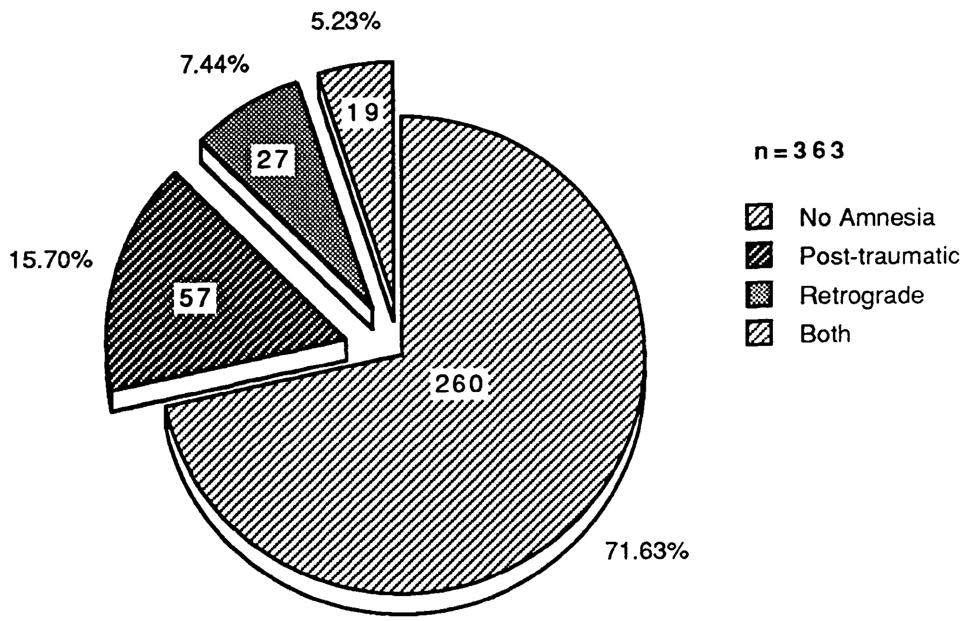


Figure 4.4 Types and frequency distribution of amnesia among the population of study.

	Number of Patients in Each Category of Malar Fracture									
Cause of Injury	I	II	III	IV	V	VI	VII	Other #'s	All	(%)
Car (no seat belt)	1	0	1	1	0	0	0	1	4	(1.1)
Car (seat belt on)	1	0	5	4	1	0	2	3	16	(4.4)
Car (back seat)	0	0	2	2	0	0	1	3	8	(2.2)
Motorcycle	0	0	1	3	1	0	1	3	9	(2.5)
Other RTA & Pedestrians	0	0	2	3	1	0	2	0	8	(2.2)
Alleged Assault	10	5	39	18	10	5	6	3	96	(26.5)
Assault under Alcohol	17	3	19	26	9	1	6	4	85	(23.4)
Fall	2	1	19	14	6	0	2	1	45	(12.4)
Fall under Alcohol	4	0	7	8	1	1	1	2	24	(6.6)
Sporting Accidents	5	3	21	8	3	1	4	0	45	(12.4)
Industrial Accidents	2	0	3	3	1	0	2	2	13	(3.6)
Black out	0	0	1	0	0	0	0	0	1	(0.3)
Epilepsy	0	0	1	0	0	0	0	1	2	(0.6)
Other Causes	0	0	3	1	3	0	0	0	7	(1.9)
All: (No)	42	12	124	91	36	8	27	23	363	
(%)	11.6%	3.3%	34.2%	25.1%	9.9%	2.2%	7.4%	6.3%	100%	

Other #'s = Fractures other than malar's.

Table 4.2 The different types of malar fracture in relation to the causal factors.

grade amnesia following their injuries, while 51 males and 6 females suffered the post-traumatic type of amnesia. Only 19 patients, 16 males and 3 females, suffered a combination of both types of amnesia. Amnesia was often associated with comminuted malar fractures (12/27=44.4%) and was most common with road traffic accidents (34/45=75.6%), (particularly when no seat belt was worn [4/4=100%]), and least common in association with sporting accidents where only one individual was affected (1/45=2.2%).

4.3.6 Ocular and Motility Disorders

Enophthalmos

Enophthalmos following midface fracture occurred in 8% of the patients (n=29) most of whom were male (n=23). The majority of patients were between the ages of 10 and 40 (n=24) with the peak incidence occurring in the 20 to 30-years age group (n=12). Nine of these 29 patients had also suffered an associated downward displacement of the globe which was not fully corrected by surgery in one case only.

All patients with enophthalmos had sustained either a pure (Type V; n=17) or an impure (Type IV or VII with floor defect; n=12) blow-out fracture of the orbit which was associated with soft tissue entrapment in 13 cases while the rest of patients (n=16) had significant displacement of orbital contents into the antrum but no functional evidence of entrapment. Eighteen of these 29 patients (62%) suffered an infra-orbital nerve anaes/paraesthesia.

Enophthalmos was most common with road traffic accidents (4/45=8.9%) and with assaults (16/181=8.8%) followed by simple falls (4/69=5.8%); and least common following sporting accidents (1/45=2.2%).

Motility Disorder

Motility disorder was common in association with enophthalmos. Twenty three of 29 patients (79.3%) exhibited symptoms and/or signs of ocular motility abnormality manifested by some limitation of ocular movement in one or more directions of gaze. Of these 23 patients, 6 complained of diplopia which was close to the midline, and 15 were symptomatic only in the extreme gaze. The other two patients had amblyopic eyes and, therefore, did not appreciate diplopia.

Ocular Injuries

Ocular injuries were common. Table 4.3 illustrates the incidence of these eye abnormalities, according to their severity, in the population of study. Tables 4.4 and 4.5 illustrate the same in relation to the type of midfacial

Eye Abnormality	Male (n=295) No. (%)	Female (n=68) No. (%)	All (n=363) No. (%)
Coronal eye displacement	24 (8.1)	5 (7.4)	29 (8.0)
Orbital emphysema	4 (1.4)	0 (--)	4 (1.1)
Eyelid swelling/bruising	198 (67.1)	54 (79.4)	252 (69.4)
Subconjunctival haemorrhage/chemosis	200 (67.8)	52 (76.5)	252 (69.4)
Corneal abrasion	0 (--)	0 (--)	0 (--)
Mild impairment of accommodation	5 (1.7)	1 (1.5)	6 (1.7)
Mild reduction of visual acuity (6/9 - 6/12)	20 (6.8)	9 (13.2)	29 (8.0)
Mild commotio retinae	16 (5.4)	8 (11.8)	24 (6.6)

Table 4.3a Incidence of mild ophthalmic disorders in the population of study.

Eye Abnormality	Male (n=295) No. (%)	Female (n=68) No. (%)	All (n=363) No. (%)
Enophthalmos	23 (7.8)	6 (8.8)	29 (8.0)
Eyelid laceration	10 (3.4)	0 (--)	10 (2.8)
Conjunctival laceration	0 (--)	0 (--)	0 (--)
Traumatic pupillary changes	6 (2.0)	3 (4.4)	9 (2.5)
Iridodialysis	0 (--)	0 (--)	0 (--)
Lens damage	1 (0.3)	0 (--)	1 (0.3)
Macular oedema	9 (3.1)	4 (5.9)	13 (3.6)
Moderate to severe impairment of accommodation	5 (1.7)	1 (1.5)	6 (1.7)
Moderate reduction of visual acuity (6/18 - 6/24)	10 (3.4)	5 (7.4)	15 (4.1)
Moderate to severe commotio retinae	2 (0.7)	0 (--)	2 (0.6)
Choroidal tear(s) not involving the macula	2 (0.7)	2 (2.9)	4 (1.1)
Vitreous floater/traumatic pigmentary retinopathy	7 (2.4)	1 (1.5)	8 (2.2)
Nasolacrimal damage	5 (1.7)	2 (2.9)	7 (1.9)

Table 4.3b Incidence of moderate ophthalmic disorders in the population of study.

Eye Abnormality	Male (n=295) No. (%)	Female (n=68) No. (%)	All (n=363) No. (%)
Gross proptosis	5 (1.7)	6 (8.8)	11 (3.6)
Retrobulbar haemorrhage	2 (0.7)	0 (--)	2 (1.5)
Corneal laceration	0 (--)	1 (1.5)	1 (0.3)
Hyphaema	4 (1.4)	1 (1.5)	5 (1.4)
Angle recession	7 (2.4)	1 (1.5)	8 (2.2)
Severe reduction/loss of vision (6/36 or less)	9 (3.1)	3 (4.4)	12 (3.3)
Visual field loss	7 (2.4)	1 (1.5)	8 (2.2)
Choroidal tear involving the macula	0 (--)	0 (--)	0 (--)
Vitreous haemorrhage or retinal tear(s)/detachment	8 (2.7)	1 (1.5)	9 (2.5)
Optic nerve Injury	7 (2.4)	2 (2.9)	9 (2.5)

Table 4.3c Incidence of severe ophthalmic disorders in the population of study.

Eye Abnormality	Type I (n=42) No. (%)	Type II (n=12) No. (%)	Type III (n=124) No. (%)	Type IV (n=91) No. (%)	Type V (n=36) No. (%)	Type VI (n=8) No. (%)	Type VII (n=27) No. (%)	All (n=341) No.
Reduced Visual Acuity	4 (9.5)	0 (--)	14 (11.3)	16 (17.6)	8 (22.2)	1 (12.5)	11 (40.7)	54
Visual Field Loss	0 (--)	0 (--)	3 (2.4)	3 (3.3)	0 (--)	0 (--)	1 (3.7)	7
Impaired Accommodation	4 (9.5)	0 (--)	4 (3.2)	3 (3.3)	1 (2.8)	0 (--)	0 (--)	12
Eyelid Injury	1 (2.4)	7 (58.3)	90 (72.6)	69 (75.8)	28 (77.8)	7 (87.5)	24 (88.9)	226
Nasolacrimal Damage	0 (--)	0 (--)	1 (0.8)	2 (2.2)	0 (--)	0 (--)	2 (7.4)	5
Eye Displacement	1 (2.4)	0 (--)	3 (2.4)	11 (12.1)	8 (22.2)	0 (--)	5 (18.5)	28
Proptosis	2 (4.8)	0 (--)	1 (0.8)	4 (4.4)	2 (5.6)	0 (--)	2 (7.4)	11
Enophthalmos	1 (2.4)	0 (--)	0 (--)	7 (7.7)	17 (47.2)	0 (--)	4 (14.8)	29
Orbital Emphysema	1 (2.4)	0 (--)	0 (--)	0 (--)	3 (8.3)	0 (--)	0 (--)	4
Conjunctival Injury	26 (61.9)	5 (41.7)	86 (69.4)	68 (74.7)	28 (77.8)	6 (75.0)	23 (85.2)	242
Corneal Damage	0 (--)	0 (--)	1 (0.8)	0 (--)	0 (--)	0 (--)	0 (--)	1
Pupillary Changes	0 (--)	0 (--)	3 (2.4)	2 (2.2)	2 (5.6)	0 (--)	1 (3.7)	8
Iris Damage	2 (4.8)	0 (--)	1 (0.8)	2 (2.2)	0 (--)	0 (--)	0 (--)	5
Angle Recession	1 (2.4)	0 (--)	1 (0.8)	4 (4.4)	1 (2.8)	0 (--)	1 (3.7)	8
Lens Damage	0 (--)	0 (--)	1 (0.8)	0 (--)	0 (--)	0 (--)	0 (--)	1
Macular Oedema	1 (2.4)	0 (--)	2 (1.6)	5 (5.5)	2 (5.6)	0 (--)	4 (14.8)	14
Commotio Retinae	3 (7.1)	0 (--)	5 (4.0)	8 (8.8)	5 (13.9)	1 (12.5)	3 (11.1)	25
Retinal/Vitreous Damage	0 (--)	0 (--)	2 (1.6)	6 (6.6)	0 (--)	1 (12.5)	3 (11.1)	12
Choroidal Injury	0 (--)	0 (--)	0 (--)	1 (1.1)	1 (2.8)	1 (12.5)	1 (3.7)	4
Optic Nerve Injury	0 (--)	0 (--)	3 (2.4)	2 (2.2)	1 (2.8)	0 (--)	2 (7.4)	8
Retrobulbar Haemorrhage	0 (--)	0 (--)	0 (--)	0 (--)	1 (2.8)	0 (--)	1 (3.7)	2
Total number of eye abnormalities:								706

Table 4.4 Incidence of ophthalmic disorder in relation to malar fractures.

Eye Abnormality	Le Fort I (n=8)	Le Fort II (n=22)	Le Fort III (n=2)	N.O.E (n=10)
Reduced Visual Acuity	1	7	0	1
Visual Field Loss	0	1	0	0
Impaired Accommodation	0	0	0	0
Eyelid Injury	6	17	1	7
Nasolacrimal Damage	0	2	0	2
Eye Displacement	1	6	0	3
Proptosis	0	1	0	1
Enophthalmos	1	3	0	1
Orbital Emphysema	0	0	0	0
Conjunctival Injury	5	16	1	7
Corneal Damage	0	0	0	0
Pupillary Changes	1	1	1	0
Iris Damage	0	0	0	0
Angle Recession	0	1	0	0
Lens Damage	0	0	0	0
Macular Oedema	0	2	0	1
Commotio Retinae	0	3	0	0
Retinal/Vitreous Damage	0	1	0	0
Choroidal Injury	0	0	0	0
Optic Nerve Injury	0	3	0	0
Retrobulbar Haemorrhage	0	0	0	0

NB Le Fort type fractures were isolated in only 6 cases and the rest were combined with other midfacial fractures.

Table 4.5 Incidence of ophthalmic disorders in relation to Le Fort and NOE fractures.

Eye Abnormality	RTA (n=45) No.(%)	Assault (n=181) No. (%)	Fall (n=69) No.(%)	Sport (n=45) No.(%)	Industrial (n=13) No.(%)	Other (n=10) No.(%)	All (n=363) No. (%)
Reduced Visual Acuity	13(28.9)	29(16.0)	4(5.8)	5(11.1)	3(23.1)	2(20.0)	56(15.4)
Visual Field Loss	4(8.9)	1(0.6)	1(1.4)	0(--)	1(7.7)	1(10.0)	8(2.2)
Impaired Accommodation	0(--)	10(5.5)	1(1.4)	0(--)	0(--)	1(10.0)	12(3.3)
Eyelid Injury	30(66.7)	138(76.2)	48(69.6)	29(64.4)	9(69.2)	8(80.0)	262(72.2)
Nasolacrimal Damage	4(8.9)	2(1.1)	1(1.4)	0(--)	0(--)	0(--)	7(1.9)
Eye Displacement	6(13.3)	13(7.2)	5(7.2)	1(2.2)	3(23.1)	1(10.0)	29(8.0)
Proptosis	1(2.2)	6(3.3)	3(4.3)	0(--)	0(--)	1(10.0)	11(3.0)
Enophthalmos	4(8.9)	16(8.8)	4(5.8)	1(2.2)	2(15.4)	2(20.0)	29(8.0)
Orbital Emphysema	0(--)	1(0.6)	0(--)	1(2.2)	0(--)	2(20.0)	4(1.1)
Conjunctival Injury	31(12.3)	138(76.2)	40(58.0)	26(57.8)	8(61.5)	9(90.0)	252(69.4)
Corneal Damage	0(--)	0(--)	0(--)	0(--)	0(--)	1(10.0)	1(0.3)
Pupillary Changes	4(8.9)	2(1.1)	1(1.4)	2(4.4)	0(--)	0(--)	9(2.5)
Iris Damage	1(2.2)	3(1.7)	0(--)	0(--)	0(--)	1(10.0)	5(1.4)
Angle Recession	0(--)	5(2.8)	0(--)	1(2.2)	2(15.4)	0(--)	8(2.2)
Lens Damage	0(--)	1(0.6)	0(--)	0(--)	0(--)	0(--)	1(0.3)
Macular Oedema	6(13.3)	5(2.8)	2(2.9)	1(2.2)	0(--)	0(--)	14(3.9)
Commotio Retinae	4(8.9)	16(8.8)	2(2.9)	3(6.7)	0(--)	1(10.0)	26(7.2)
Retinal/Vitreous Damage	4(8.9)	5(2.8)	0(--)	1(2.2)	1(7.7)	1(10.0)	12(3.3)
Choroidal Injury	2(4.4)	1(0.6)	0(--)	1(2.2)	0(--)	0(--)	4(1.1)
Optic Nerve Injury	3(6.7)	2(1.1)	2(2.9)	0(--)	1(7.7)	1(10.0)	9(2.5)
Retrobulbar Haemorrhage	0(--)	1(0.6)	0(--)	0(--)	0(7.7)	1(10.0)	2(0.6)
Total Number of Eye Abnormalities	117	395	114	72	30	33	761

Table 4.6 Incidence of ophthalmic disorder in relation to the aetiology of midfacial trauma.

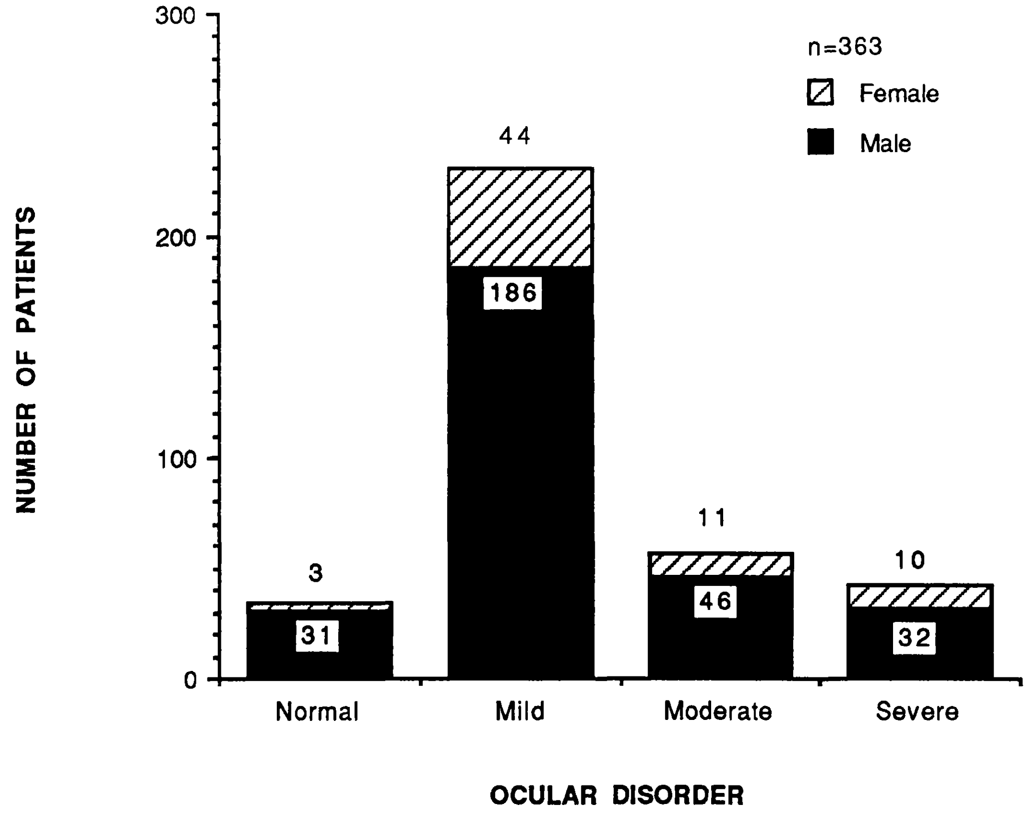


Figure 4.5 Grades of severity of ocular disorder in male and female.

fracture sustained, while table 4.6 relates these ocular abnormalities to the cause of injury.

The right eye was affected in 152 patients (152/363=41.9%), of whom 125 were male (125/295=42.4%) and 27 were female (27/68=39.7%). The left eye was involved in 153 cases (42.2%), 120 males (42.1%) and 33 females (48.5%). In 33 patients (9.1%), 29 males (9.8%) and 4 females (5.9%) both eyes were injured while the rest of the patients, 21 males and 4 females, sustained no direct ocular trauma whatsoever.

Figure 4.5 illustrates the frequency distribution of ocular injuries in relation to their severity for both males and females. Only thirty four patients (9.4%) did not exhibit any detectable eye injury at all. Most patients (63.4%, n=230) suffered only minor or transient ocular injuries such as lid bruises, subconjunctival haemorrhages and mild impairment of accommodation. Fifty seven patients (15.7%) suffered moderately severe ocular injuries which were unlikely to have led to permanent visual or physiological sequelae. Examples include minor eyelid and conjunctival lacerations and enophthalmos. Forty two patients (11.6%) sustained severe eye injuries such as angle recession, retinal or vitreous injury, or optic nerve damage. Of those 42 patients, 9 (2.5%) lost vision in the affected eye.

Severe ocular disorder was more common in females (14.7%) than in males (10.8%). However, the difference was not found to be statistically significant. The severity of ocular abnormality was not found to be significantly related to age or to season.

Severe ocular disorder was most common following road traffic accidents (Fig 4.6). Nine of 45 patients (20%) with confirmed midfacial fractures due to RTA suffered a severe ocular abnormality of one type or another. Assaults were associated with 11% of severe ocular abnormalities (20/181) and falls with 7.2% (5/69) whilst only 4.4% (2/45) of those individuals who were involved in sporting accidents sustained severe ocular injury. Industrial accidents were associated with a 23% rate of severe eye abnormality (3/13); however, the size of this group is too small to allow accurate interpretation.

Road traffic accidents caused the highest proportion of impaired vision (28.9%), (X^2: $0.001<p<0.01$), while simple falls caused the lowest proportion (5.8%), (X^2: $0.01<p<0.02$). Other causes of facial trauma such as assaults, sports and industrial accidents were associated with intermediate frequencies of reduced vision (15%).

Figure 4.7 demonstrates the relationship of ocular injury categories to the different types of malar fracture

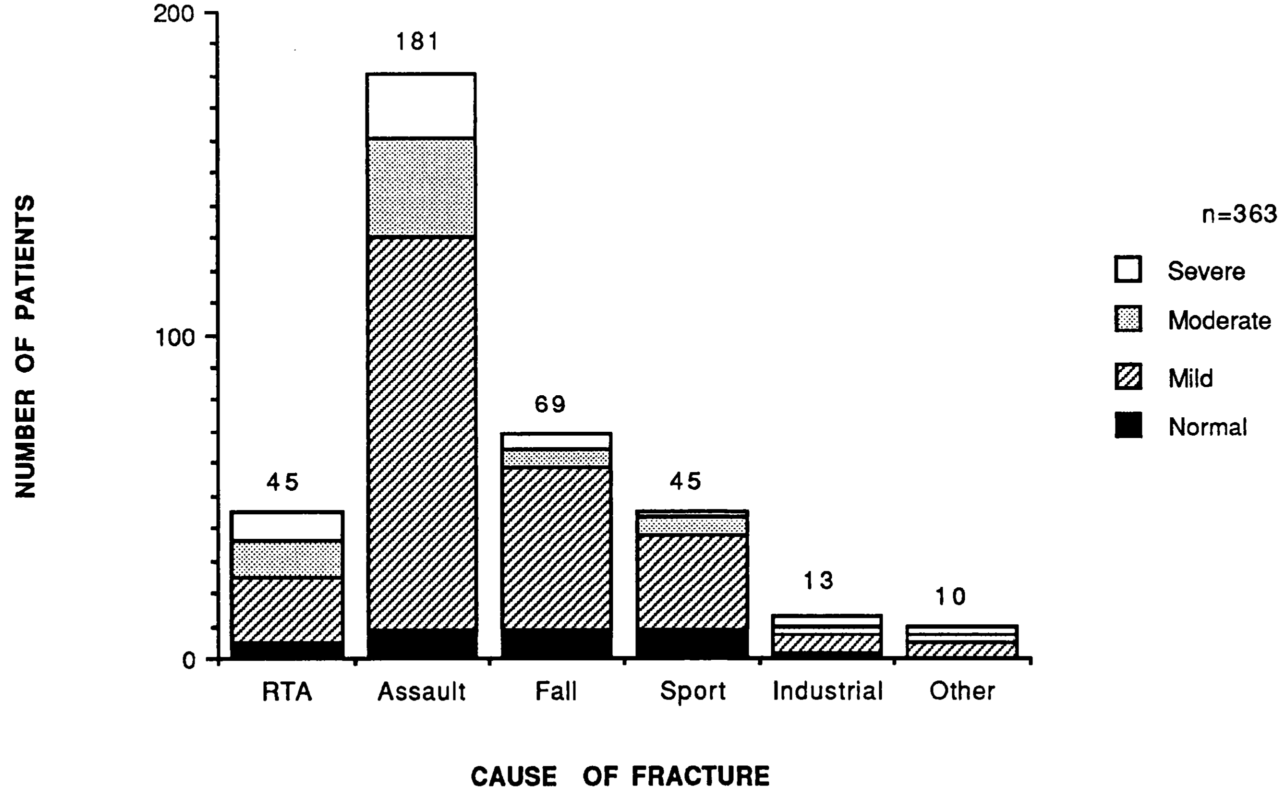


Figure 4.6 Ocular disorder, according to severity, in relation to aetiology of facial fracture.

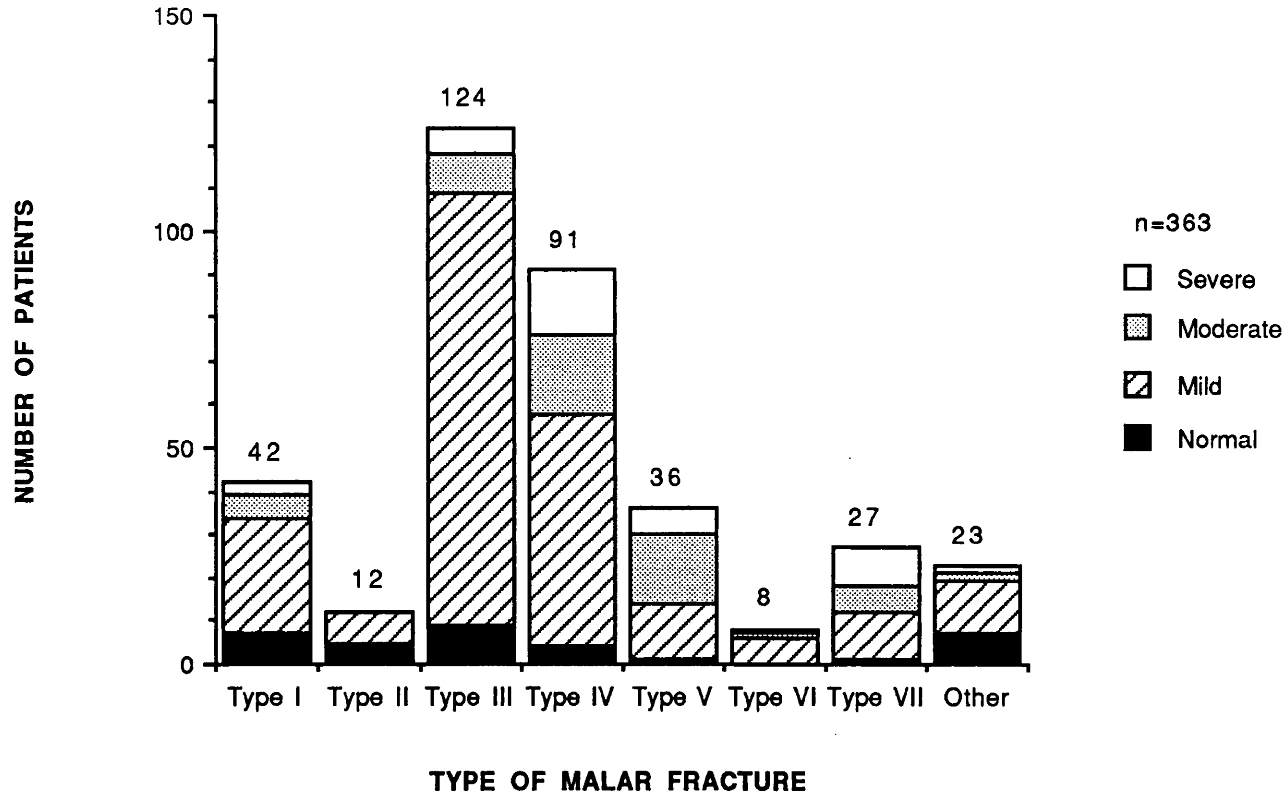


Figure 4.7 Ocular disorder, according to severity, in relation to type of malar fracture.

sustained. One third of all patients with comminuted malar fracture (Type VII) suffered a severe ocular disorder (9/27=33.3%) while blow-out fractures (Type V) came second (6/36=16.7%). No severe ocular disorders were observed with fractures of the zygomatic arch only (Type II).

Fifty six of 363 patients (15.4%) had suffered transient or permanent visual loss. Causes of impaired vision varied from minor, self healing corneal injuries to optic nerve avulsion. Permanent loss of vision, ensued in 9 cases and was due to traumatic optic neuropathy in each case.

The type of facial fracture was found to be an important factor in relation to visual deterioration. Comminuted malar fractures were associated with a 41% incidence of visual impairment in contrast to 15% for the whole series ($p<0.001$).

Visual loss occurred most frequently to the 30 to 40-year age group ($0.02<p<0.05$) and least frequently to the 20 to 30-year age group patients ($p<0.001$). Females were significantly more likely to suffer visual loss than males of the same age group ($0.01<p<0.02$).

4.4 DISCUSSION

To my knowledge, this report is the largest prospective survey of the ocular sequelae of midfacial fracture currently available. Ocular injury of varying severity is a common sequel to midfacial fracture (Paton & Emery, 1973). The severity of the facial fractures is dependent upon the nature of the injury and is directly related to the likelihood and severity of trauma to the eye. Disruption of the orbital walls deprives the eye and its adnexae from the protection they are normally afforded (Jabaley et al, 1975).

In the present study, patients with the most severe head or facial injuries warranting neurosurgical intervention are managed in a separate unit and have not been included in this report. It can be anticipated, however, that such individuals are highly susceptible to injury of the visual system.

Disturbance of ocular motility was also a common sequel. The details of the motility disorders seen in this series of patients are described separately (Chapter 5).

The major cause of maxillofacial trauma in this study was alleged assault. The patients assessed were from a large industrialised area with a high rate of unemployment

(Ellis et al, 1985). This may be related to the fact that the majority of injured patients were young adult males who sustained their injuries in alleged assaults. This cause of fracture and type of patient correspond to the results found in comparable studies (Altonen et al, 1976; Lundin, 1973).

Although alleged assaults were more common in males than in females, the difference was not found to be statistically significant. Falls, however, were far more common in females than in males ($0.001<p<0.01$).

In general, fractures were almost equally shared between both sides of the face. However, fractures sustained due to assaults were predominantly left-sided which relates with the fact that the majority of people are righthanded. This finding is in agreement with the investigations of Muller and Schoeman (1977), Altonen et al (1976) and Hitchin and Shuker (1973) who also found that assault victims displayed a predominance of left-sided fractures.

June was the month in which the greatest incidence of injury occurred. This, probably, is expected in a summer month where most of the assaults, RTA's and sporting events take place.

It was interesting to find that patients who sustained a head injury severe enough to cause amnesia were more likely to suffer a disturbance to their visual system. As expected, amnesia was most common following road traffic accidents. The protective effect of seat belts is evidenced by the fact that, in this series, all unrestrained patients developed concussion manifested by amnesia.

Traumatic enophthalmos is usually due to enlargement of the orbital cavity subsequent to a floor or, less commonly, medial wall fracture (Converse & Smith, 1956); although dislocation of the trochlea, atrophy of orbital fat, cicatricial contraction of retrobulbar tissue and rupture of orbital ligaments or fascial bands had all been previously incriminated (Benedict, 1936).

Pfeiffer (1943) reviewed 53 patients with traumatic enophthalmos following facial bone fractures and noted orbital bone involvement in each case. Of these, 24 patients sustained pure blow-out fractures which were due to assaults in 58% (n=14), and road traffic accidents in 17% (n=4) of cases. The present study's figures are closely in accordance with those of Pfeiffer. Enophthalmos following facial trauma should always raise suspicion of an orbital wall fracture and a CT scan is advisable when plain x-rays are negative.

There is a number of reports with large series of facial fractures in which associated injuries are reported (Steidler et al, 1980; Morgan et al, 1972; Gwyn et al, 1971) but most of these reports only mention briefly or fail to characterise specific ocular complications.

The very low incidence of ophthalmic injury reported by non-ophthalmic authors (Luce et al, 1979; Turvey, 1977; Morgan et al, 1972; Gwyn et al, 1971; Schultz, 1967; Converse et al, 1967; McCoy et al, 1962) probably indicates that significant ophthalmic pathology may have gone undetected. On the other hand, the low incidence of ophthalmic injury reported by some ophthalmic surgeons (Miller & Tenzel, 1967; Milauskas & Fueger, 1966) may indicate that only the more serious ocular injuries had been reported while mild and less serious injuries were not mentioned.

The overall incidence of ophthalmic injury of any severity (including sub-conjunctival haemorrhages) following maxillofacial trauma in this series is 90.6% (329/363). This figure is significantly higher than those previously published in the literature including the 67% rate reported by Holt et al (1983). However, Holt's series was retrospective, included only 51% of facial fracture patients potentially available for assessment and provided limited follow-up documentation. It was therefore selective on the part of the attending maxillofacial surgeon and allowed no definite conclusions to be drawn concerning the possibility of undetected eye injuries in the patients who did not have formal eye examination.

The results of this study reinforce the contention that road traffic accidents cause more severe ocular injuries than any other cause of facial trauma (Holt et al, 1983; Petro, et al, 1979). Similarly, they demonstrate that comminuted malar fractures are associated with the highest incidence of visual dysfunction.

Fortunately, many of the ocular injuries in this series were transient and of no permanent consequence. However, the incidence of 11.6% (42/363) of patients with serious or blinding injuries is significant. Of particular importance are those cases of optic nerve contusion or compression (2.5%). Total loss of vision in quite healthy looking eyes is not uncommon following facial trauma. Ketchum et al (1976) presented two cases of bilateral blindness without direct injury to the globe and postulated that a haematoma, ischaemia, or direct bone fragment penetration of the optic nerve may have been the cause. In the light of the recent study by Mauriello and associates (1992) illustrating the proper timing of surgical decompression of the optic nerve and response to 'mega-dose' corticosteroid therapy, such injuries may deserve immediate and aggressive management especially if the initial examination demonstrates some vision immediately following the injury.

4.5 CONCLUSIONS

The results of the eye examination, while they do not often alter the type of fracture repair, may influence the indications and timing of the repair as treatment of certain ocular injuries, such as optic nerve compression, must be instituted at once. The proper time for an eye examination, especially visual acuity testing, is at the time of initial assessment of the patient's trauma. All patients sustaining midfacial fracture associated with a significant decrease in visual acuity either pre- or post-operatively should, ideally, have an early ophthalmological review.

Many patients sustaining facial injury make recourse to legal or insurance claims. The results of detailed ophthalmic assessment often add weight to such cases and significantly affect levels of compensation paid.

The data accruing from this study have been subjected to discriminant function analysis and has resulted in the development of a scoring system which allows the maxillofacial surgeon to determine which patients warrant ophthalmic referral and with what urgency (Chapter 6).

CHAPTER 5

DIPLOPIA FOLLOWING MIDFACIAL FRACTURES

5. DIPLOPIA FOLLOWING MIDFACIAL FRACTURES

5.1 INTRODUCTION

Diplopia resulting from extra-ocular muscle imbalance is a common complication of fractures of the zygomatico-maxillary complex (Yee et al, 1975). The reported incidence of diplopia ranges between 15% of recent to 56% of late presentations of zygomatic fractures (Tajima et al, 1974). Blow-out fractures of the orbit comprise the most common cause of diplopia (Helveston, 1977) which occurs in between 36 and 86% of such cases (Leibsohn et al, 1976; Converse et al, 1967).

Vertical diplopia on up and down gaze commonly results from the soft tissue incarceration which follows an orbital floor blow-out fracture; whilst impairment of abduction may be a sequel to the less common medial wall blow-out fracture (Piddle, 1968) or a traumatic VI nerve palsy. Orbital rim fractures without blow-out fractures may also result in diplopia (Converse et al, 1961) which may be transitory. This diplopia has been attributed to impairment of movement due to oedema, haematoma or injury to the extra-ocular muscles or their nerve supply (Converse et al, 1961; Barclay, 1958).

Entrapment of orbital tissue, extra-ocular muscle, check ligament injury, intra-orbital haemorrhage or oedema, or paresis due to cranial nerve injury, must be differentiated from one another as they may give rise to similar clinical features but their management differs (Thaller-Antlanger, 1980). A detailed history, thorough clinical examination and appropriate radiological evaluation is therefore required in order to establish the cause of double vision and to plan appropriate treatment.

The present prospective study has been carried out in order to relate the type of injury sustained to the incidence and severity of subsequent eye movement disorder and thereby to identify the risk factors involved and to determine the prognosis for the restoration of binocular single vision.

5.2 PATIENTS AND METHODS

Three hundred and sixty three patients with midfacial fractures due to blunt facial trauma, the details of which have already been described (Chapter 3), were admitted to this study. All patients were assessed preoperatively and the follow-up time was up to 2 years for those patients with motility disturbances. At each consultation patients were asked whether they were troubled by double vision.

The duration of diplopia from the time of injury was classified into six categories:

1. Less than one day.
2. One day to one week.
3. One week to one month.
4. One month to six months.
5. Six months to one year, and
6. More than one year.

Diplopia was classified according to the following sub-categories:

1. Close to midline (within 15° of fixation).
2. In eccentric gaze (outside 15° of fixation), or
3. Diplopia, of either category, which developed following surgery.

5.3 RESULTS

Three hundred and sixty three patients (295 males and 68 females) sustained a total of 438 midfacial fractures. The frequency distributions of the types and causes of these fractures have already been illustrated in chapter 4.

Seventy two of 363 patients (19.8%) complained of diplopia at the time of injury (Fig 5.1). Fifty eight were male

(19.7% of the male population) and 14 were female (20.6% of the female population). They had sustained a total of 94 midfacial fractures. Eighteen out of these 72 patients (25%) had sustained multiple fractures at 2 or 3 facial sites. However, the difference between multiple injury and single injury as a risk factor in the causation of diplopia was not found to be statistically significant.

5.3.1 Cause of Trauma

Diplopia was most common in association with road traffic accidents (14/45=31.1%), and least common with simple falls (7/69=10.1%), while assaults and industrial accidents yielded 20.4% (37/181), and 23% (3/13) of diplopia patients respectively (fig 5.2).

5.3.2 Type of Fracture

The incidence of diplopia was related to the type of fracture sustained (fig 5.3). Diplopia was most common following blow-out fractures of the orbit (Type V), whilst it did not occur with isolated fractures of the orbital rim (Type VI). Twenty one (58.3%) of the patients with an orbital blow-out fracture had diplopia, all but one had their orbital floors explored. Eleven of these patients were found at surgery to have soft tissue entrapment in the orbital floor whilst one exhibited entrapment in the

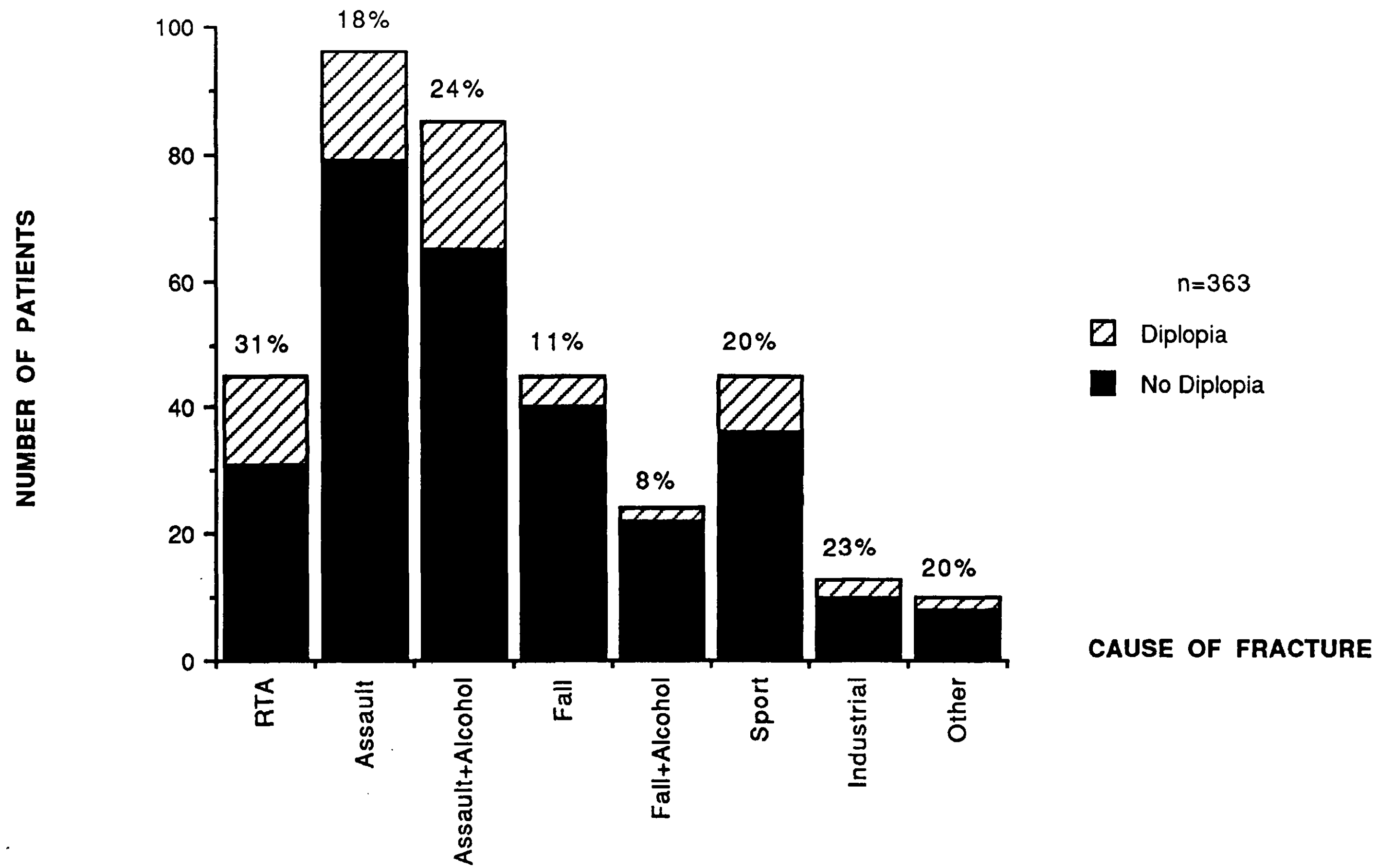


Figure 5.2 The proportion of patients who suffered with diplopia (percentage figures) compared with those who did not, in each aetiological category.

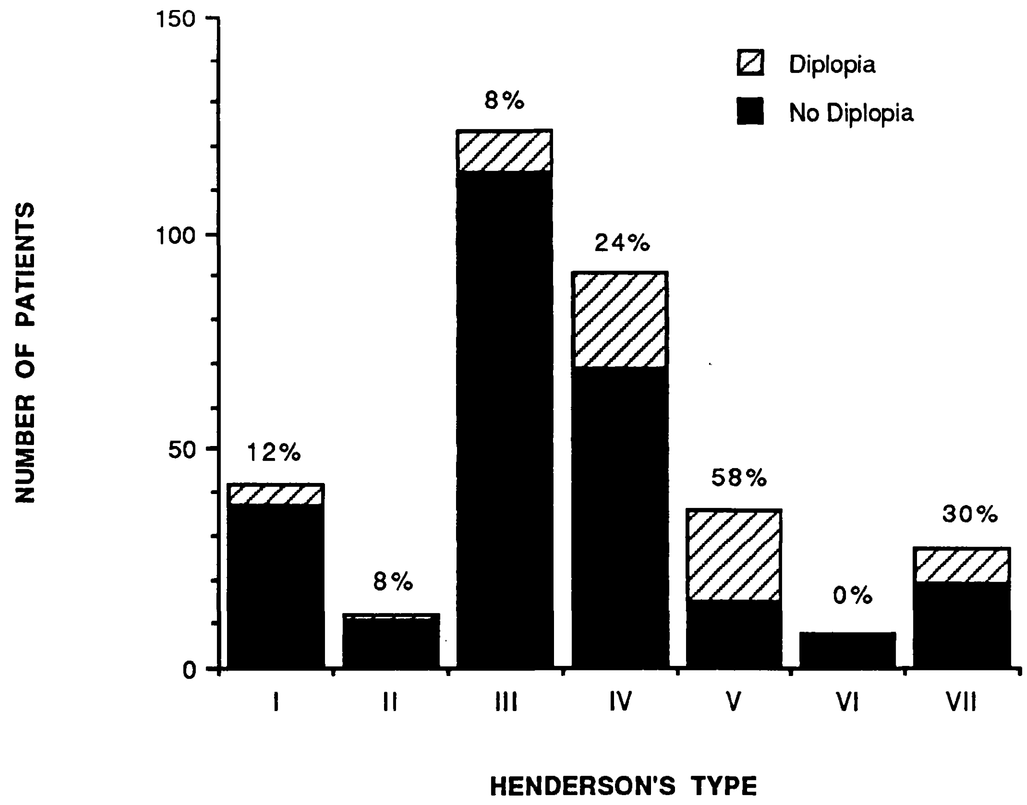


Figure 5.3 The distribution of patients with diplopia in each category of malar fracture.

floor and medial wall. Eight patients had displacement of orbital contents into the antrum but no entrapment was elicited at the time of surgery. The remaining patient demonstrated entrapment of the superior oblique tendon in an orbital roof fracture verified by CT scan (Al-Qurainy et al, 1988).

Almost 30% of patients (8 out of 27) with a comminuted malar fracture (Type VII) suffered of diplopia, while 22 of the 91 patients (24%) who had sustained a malar fracture with a distracted fronto-zygomatic (F-Z) suture (Type IV) were aware of double vision. A smaller proportion of patients (10 out of 124), (8.1%) complained of diplopia when the F-Z suture was undistracted (Type III) or when the fracture involved only the zygomatic arch (Type II) (1/13 = 8.3%). Malar fractures which were classified as undisplaced (Type I) led to diplopia in 11.9% of cases (5 out of 42). With regard to maxillary fractures, 1 of the 2 patients with a Le Fort III type fracture and 8 of the 22 patients with a Le Fort II type fracture (36.4%) reported diplopia. However, all these patients with Le Fort fractures had also sustained other facial fractures.

5.3.3 Types of Diplopia

On examining the 72 patients with diplopia (Fig 5.4), 19 (26.4%) of them had diplopia close to the midline whilst 46 (63.9%) only experienced diplopia in extreme (eccentric) gaze. Seven (9.7%) patients who initially reported symptoms of diplopia which had been confirmed on first examination were found to have recovered by the time they were seen in the eye clinic within a week of injury.

Fifteen of the 19 patients with diplopia close to the midline had sustained a pure (6) or an impure (9) blow-out fracture. Nine of these 15 patients had demonstrable soft tissue entrapment at surgery, the other 6 patients had significant orbital content displacement but no true entrapment. The remaining patients had sustained a range of facial fractures; N.O.E. (2), Le Fort II (1), type I malar fracture (1).

5.3.4 Recovery of Diplopia

Diplopia was transient, lasting only for a few seconds or minutes in 2 out of the 72 (2.8%) diplopia patients (Fig 5.5). A quarter of the patients (18) recovered in a matter of few hours to one week. Twenty one patients (29.2%), however, recovered within one to four weeks, and another 18 (25%) suffered from diplopia for between one to six

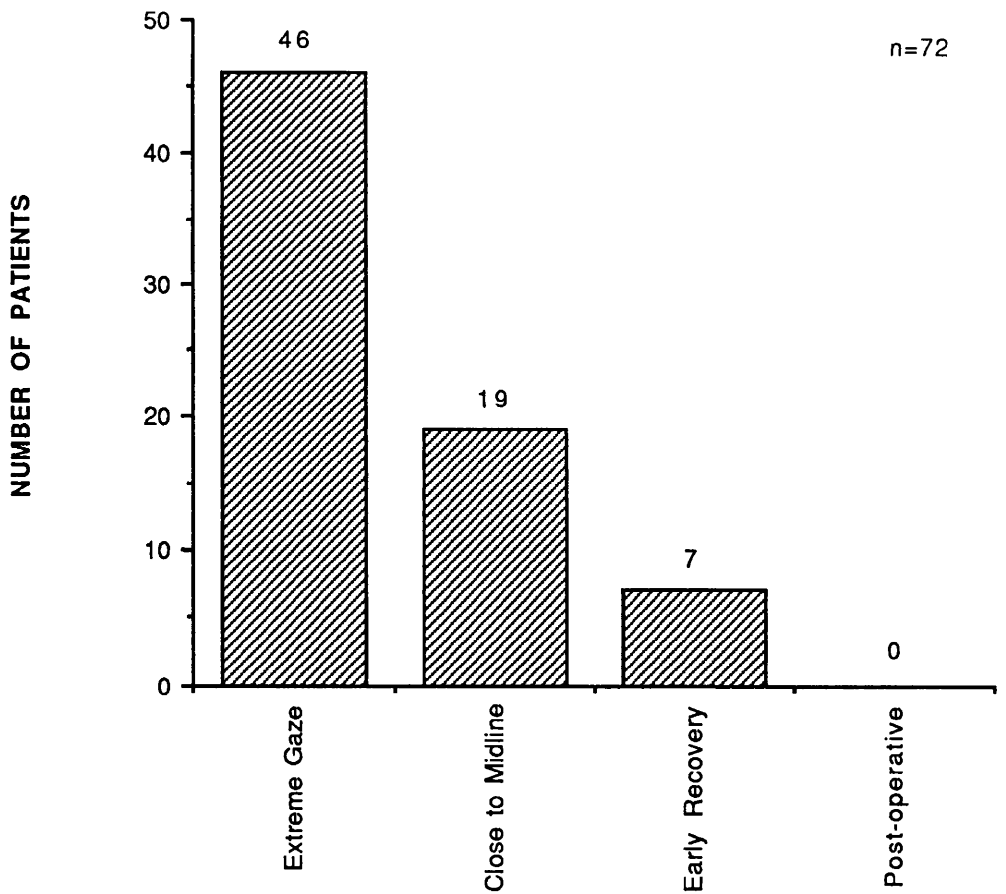


Figure 5.4 The proportion of patients with each category of diplopia.

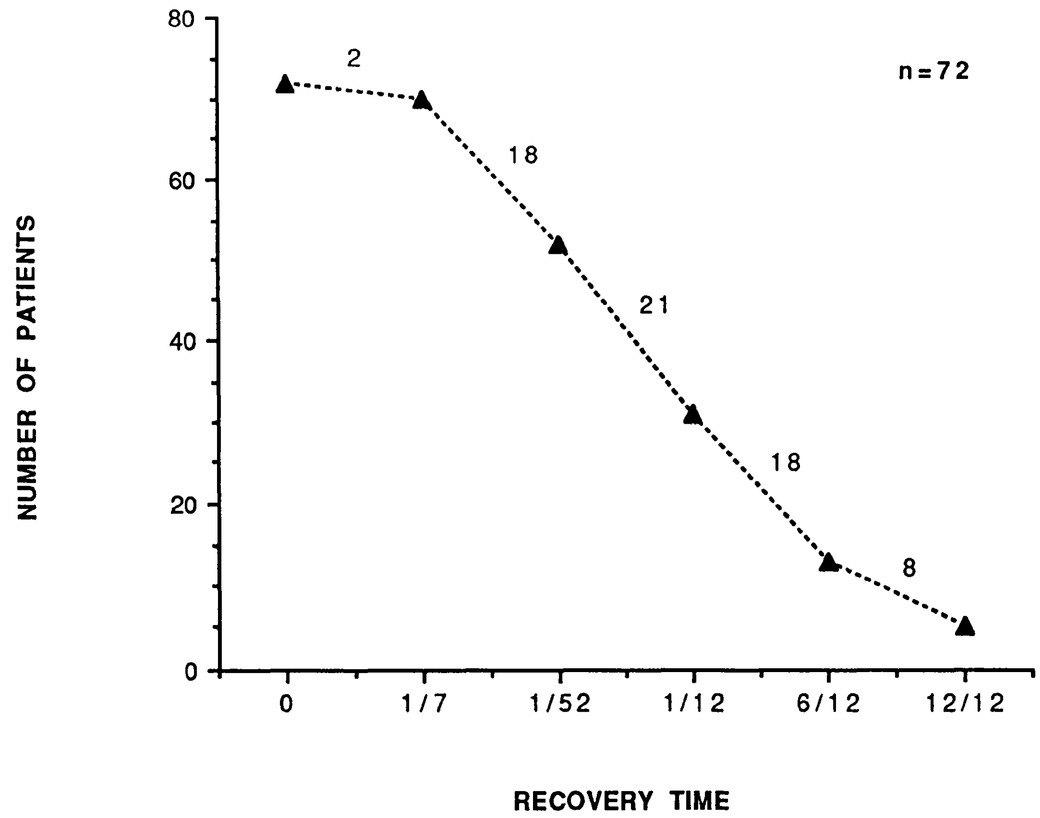


Figure 5.5 The rate of recovery of diplopia among the affected patients. Figures on the dotted line indicate the number of patients who recovered during that period.

months. Eight (11.1%) of the patients with diplopia required 7 to 12 months to recover, and 5 (6.9%) complained of diplopia for greater than 12 months from the date of injury.

Four of the 13 patients with diplopia for more than 6 months had sustained a tripod fracture with a distracted F-Z suture (Type IV), 6 patients had sustained a blow-out fracture (Type V) and 3 had suffered comminuted fractures of the zygomatico-orbital complex (Type VII) with associated skull fractures.

With regard to the 5 patients who suffered diplopia for more than a year, 2 had sustained pure blow-out fractures of the orbital floor and another 2 had received tripod fractures with distraction of the F-Z suture. One patient had sustained a combination of a comminuted zygomatico-orbital fracture, a maxillary fracture at Le Fort II level and an associated skull fracture.

The causes of injury in the 13 patients with diplopia for greater than 6 months were RTA (3 patients) and assault (7 patients), the rest of the patients suffered an industrial accident (1), a sporting injury (1) and a home accident in an 8-year old child (1). There does not appear to be a relationship between the aetiology of the injury and persistence of diplopia.

Diplopia was seen only in extreme (eccentric) gaze in 12 out of the 13 patients with persistent diplopia and it did not cause them concern. One patient had troublesome diplopia in the primary position of gaze for which he required squint surgery.

In this series of 363 patients, 10 patients (2.8%) developed a III, VI or VII nerve palsy. Five of them followed road traffic accidents, 4 were due to assault and one was due to an industrial accident. There were 2 cases of abducent nerve palsy and 1 case of an oculomotor nerve paralysis. In these cases spontaneous recovery took place within 7 months. Seven patients developed a facial nerve palsy. No case of trochlear nerve palsy was identified.

Six of the patients with cranial nerve palsy developed diplopia. Three of these were patients with abducent and oculomotor nerve palsies. The other three patients had facial nerve palsies, the cause of diplopia in these cases was a consequence of the orbital fracture. The type of fracture and cause of injury was different in each case.

5.4 DISCUSSION:

Diplopia resulting from facial fractures has frequently been reported. However, a review of the literature shows that little attention has been paid to the course of recovery and the prognosis. Moreover, no attempt has been made to link diplopia to the cause or type of injury contributing to it. The reported incidence of diplopia following facial trauma (Table 5.1) is variable. It ranges between 1% (McCoy et al, 1962) and 86% (Converse et al, 1967). In this study, 19.8% of 363 patients suffered diplopia. This is comparable to the work of Tajima (1974) and Hakelius and Ponten (1973) who reported figures of 21% and 15% respectively. However, figures of incidence of diplopia rise to high levels when the population of study is pre-selected as for example in the case of blow-out fractures (Greenwald et al, 1974; Converse et al, 1967).

Road traffic accidents were the most common cause of diplopia in this study (fig 5.2). Thirty one percent of all patients involved in RTA complained of diplopia. The second most common cause was assault associated with alcohol consumption (23.5%). However, I have seen more patients with diplopia overall due to assault than in any other category because of the high number of assaulted patients in this series. The incidence of diplopia was higher among patients who were assaulted whilst under the influence of alcohol (23.5%) than among those who were sober (17.7%). However, the difference between these two groups was not statistically significant. Industrial accidents led to diplopia in 23% of cases. Smaller percentages of diplopia were seen following sporting

Author(s)	Incidence	Population of Patients
McCoy et al (1962)	1.0%	Middle third fractures
Converse et al (1967)	86.0%	Blow-out fractures
Hakelius & Ponten (1973)	21.8%	Midface fractures
Tajima et al (1974)	15.0%	Recent zygomatic fractures
Tajima et al (1974)	56.0%	Late zygomatic fractures
Greenwald et al (1974)	70.0%	Blow-out fractures
Leibsohn et al (1976)	36.0%	Blow-out fractures
Steidler et al (1980)	32.1%	Middle third fractures

Table 5.1 Incidence of diplopia following facial fractures as quoted in other studies.

accidents (19.1%) or following a fall (8.3%). I find no such detailed classification of diplopia in relation to aetiology to compare these results with. Patients with Type IV, Type V and Type VII (pure or impure blow-out) fractures were found to be more likely to suffer from persistent diplopia (>6 months).

Diplopia is most frequently associated with blow-out fractures of the orbit. Converse et al (1967) reported the incidence to be 86% and Greenwald (1974) reported 70%. The more recent work by Leibsohn (1976) showed the incidence to be 36%. In this study, blow-out fractures contributed to diplopia in 58.3% of cases. The present study's figures probably represent a relatively unbiased spectrum of mid-facial fractures. By contrast, it is possible that an element of pre-selection contributed to the high proportions quoted by Greenwald (1974) and Converse et al (1967).

Maxillary fractures at the Le Fort III and Le Fort II levels associated with other facial fractures were common causes of diplopia, as were the naso-orbito-ethmoidal fractures. This probably represents the severity of the injury. This study does not, however, accurately represent the incidence of diplopia in this group of patients as many of the more serious Le Fort fractures are associated with head injuries and therefore treated by the

Canniesburn maxillofacial staff in the neurosurgical unit and have not been included in this study.

Maxillofacial fractures are associated with a significant head injury in 25% of cases (Converse & Smith, 1963). Therefore, in addition to direct orbital trauma affecting ocular motility, various degrees of damage to the central nervous system, as well as possible trauma to the cranial nerves can take place (Smith et al, 1971). Loss of the ability to fuse the images from both eyes after trauma has been described by Stanworth (1974) and Hart (1969), and trauma is a recognised cause for the breakdown of an existing heterophoria (Lee, 1983). A cranial nerve palsy may affect ocular motility. A review of literature (Table 5.2) shows that motor cranial nerve palsy following head and/or facial trauma ranges between 3% in closed head injuries (Russell, 1960) and 71% following fatal RTA (Heinze, 1969). No case of IVth nerve palsy was identified in this study. However, trochlear nerve injuries are usually less common than those affecting the oculomotor or abducens nerves (Burger et al, 1970).

Orbital fractures commonly give rise to double vision. Not surprisingly, severe injuries resulting in comminution or fronto-zygomatic distraction or blow-out fractures pose the greatest risk. Eight percent of patients with zygomatic arch fractures developed diplopia on eccentric gaze

Author(s)	Incidence	Basis for Selection
Rucker (1958)	16.2%	Blunt trauma to the head
Russell (1960)	3.0%	Closed head injuries
Scharder & Schletzinger (1960)	2.9%	VIth nerve palsy only
Hughes (1964)	7.0%	Head injuries
Rucker (1966)	14.3%	Blunt trauma to the head
Heinze (1969)	71.0%	Fatal RTA only
Burger et al (1970)	39.0%	IVth nerve palsy only
Robertson et al (1970)	20.0%	IVth nerve in children

Table 5.2 Incidence of oculomotor palsy following head and/or facial trauma as cited by other authors. Note that Scharder & Schletzinger's study was exclusively on patients with isolated abducens nerve paralysis to show the relative frequency of the various aetiologies of which head and/or face trauma was responsible for 2.9% of cases. Similarly, Burger's study was carried out exclusively on patients with acquired isolated trochlear nerve palsy of which 39% of cases was due to trauma.

but this settled rapidly. Diplopia in these cases may have been caused simply by oedema.

The present policy in the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital is to operate early (within 2 weeks) on patients who have sustained facial trauma. Only 8 out of the 72 patients were treated conservatively, all of whom had sustained undisplaced fractures, and whose diplopia resolved spontaneously.

Resolution of diplopia is not well documented in the literature. Often, diplopia has been given as a reason to advocate early (Harley, 1975) or late (Langnickel & Munich, 1978) surgical treatment of maxillofacial trauma. Retrospective studies are biased in their patient selection since those patients who were not treated surgically were either considered not to warrant treatment or they had declined surgery. Putterman (1974) retrospectively reviewed the clinical course of 28 patients and prospectively assessed 29 patients following blow-out fractures of the orbital floor which had not been treated surgically. The relatively benign outcome in these patients demonstrated, in that author's opinion, the feasibility of managing blow-out fractures without surgery. This opinion, however, has not been shared by many other authors (Reeh & Tsujimura, 1966; Patterson et al, 1962; Smith & Regan, 1957; Smith & Converse, 1957) who have recommended an early exploratory and active surgical approach for the fear of persistent diplopia and disfiguring enophthalmos.

The risk factors for the development of diplopia are: road traffic accidents, blow-out fracture and comminuted malar fracture. For persistent diplopia however, the only risk factor which is statistically significant is the pure or impure blow-out fracture.

Only one out of 363 patients in the present series required squint surgery for diplopia and he had suffered a medial wall blow-out fracture that had initially been overlooked. Early surgical reconstruction, especially of the orbital floor, and conservative management of motility disorders may, in this series, have contributed to a satisfactory outcome for the patients.

5.5 CONCLUSIONS

It is my belief that all patients with diplopia in the primary position of gaze should be evaluated by an ophthalmologist in order to establish the cause and to determine the optimum plan of management. Mild diplopia may be caused by oedema, haematoma, nerve palsies or entrapment. These causes can be differentiated with appropriate investigations such as the forced duction and force generation tests.

Early surgery for blow-out fractures with manifest clinical and radiological features has, in this study, not been associated with long standing motility disorder, and the belief that diplopia may be aggravated permanently by surgery is disproved. In this study no patient developed persistent diplopia as a direct result of surgery.

CHAPTER 6

MIDFACIAL FRACTURES AND THE EYE: THE DEVELOPMENT OF A SYSTEM FOR DETECTING PATIENTS AT RISK OF EYE INJURY

6. MIDFACIAL FRACTURES AND THE EYE: THE DEVELOPMENT OF A SYSTEM FOR DETECTING PATIENTS AT RISK OF EYE INJURY

6.1 INTRODUCTION

Maxillofacial fractures are commonly complicated by injuries to the visual system (Holt & Holt, 1983; Culbertson, 1983; Petro et al, 1979). Such injuries may be difficult to detect without the requisite expertise and equipment. However, it is impractical to refer all such patients for ophthalmic assessment. In order to determine the optimum criteria for referral of appropriate patients to the ophthalmologist, a scoring system, based upon a prospectively assembled data base of patients with maxillofacial fractures, has been devised.

6.2 PATIENTS AND METHODS

The data-base comprises a cohort of 363 patients with a variety of midfacial fractures who had been sent as secondary referrals to the Oral and Maxillofacial Unit at Canniesburn Hospital during a two year period. The details of this group and the methods of assessment have already been described in chapter 3.

Table 6.1 lists the criteria chosen as 'predictors' for potential ophthalmic injury. The predictors comprise the

1. Basic patient data

Sex
Age
Month of injury

2. Cause of injury

3. Site of injury - determined clinically and radiologically

Malar fracture site
Maxillary fracture site
Other fracture site
Entrapment of soft tissue (floor or medial wall blow-out)
Injury in the region of the trochlea

4. Method(s) of surgical treatment

5. Ophthalmic data

Affected eye/side
Visual acuity

6. Ocular motility data

Symptoms: Double vision

History of transient double vision with recovery

Pain on eye movement

Signs: Abnormal head posture to compensate for double vision

Manifest deviation of the eye in the primary position of gaze

Restriction of extra-ocular movements found on clinical examination

Convergence insufficiency

7. Evidence of associated injuries

Amnesia
Cranial nerve palsies

Table 6.1 List of information available to the maxillofacial surgeon which was tested as comprising predictors for the outcome of ophthalmic injury.

data which are immediately available to the maxillofacial surgeon. Tables 3.3, 3.4, and 3.5 (Chapter 3) list the eye injuries as 'outcomes' divided into the categories of non-referral, routine referral and early referral. A brief justification for selecting each parameter as an outcome is also given.

The methods of statistical analysis were as follows:

(a) Selection of Risk Factors

Each predictor was initially considered separately. The data were cross-classified according to the type of referral required and the value of each predictor. From each two-way contingency table which resulted the Chi-squared statistic was computed and predictors were noted for which the statistic was significant at or near the 5% level of confidence (Armitage, 1971).

(b) Construction of a Scoring System

The data concerning referral type (non-referral = 0; routine-referral = 1; early-referral = 2) were combined with the data obtained for the predictors which had been selected as risk factor in the manner outlined above. These were analysed using a stepwise linear regression analysis. This was carried out using the 'STEPWISE' command in Minitab with forward selection of variables (Minitab Reference Manual, Release 7, pp. 7-15 to 7-19). Incomplete cases are omitted at this stage, although assessment of the final method used all cases for which all necessary variables were available. The precise values of the regression coefficients have had a nominal p-value of less than 0.05. This procedure comprises a means of selecting a subset of the predictors which extracts the meaningful predictive information contained by all the predictors. The resulting subset therefore provides a parsimonious but efficient means of prediction. The coefficients in the corresponding regression equation were then rounded off to more convenient numbers to provide the formula for the raw score shown in the 'Results' section. This raw score was, in turn, converted to a refined score suitable for use in every day practice. Most statistical analyses were carried out by Professor Titterington (Department of Statistics, University of Glasgow) using the statistical package MINITAB (Ryan et al, 1985).

6.3 RESULTS

The principal risk factors which, in combination, successfully detected the largest numbers of patients deemed to warrant referral to the ophthalmologist were as follows: (a) Impairment of visual acuity; this emerged as the most sensitive single predictor for ophthalmic injury (see Table 6.2 for the associated contingency table). (b) Comminuted malar fractures were associated with the most severe ocular and motility defects, no doubt because the protection afforded by an intact lateral orbital wall was lost and the eye and orbital tissues were subjected to compressive injury. (c) Pure blow-out fractures commonly produced motility defects which were also deemed to warrant ophthalmic referral. (d) Abnormalities of eye movement can, in most cases, be easily detected by the maxillofacial surgeon primarily on the basis of double vision. Although this predictor 'overlaps' with comminuted and blow-out fractures it emerged as a predictor which identified additional cases deemed to warrant referral. (e) Finally, those patients who were injured severely enough to cause amnesia had sustained severe injuries which commonly affected the visual system.

The scoring system developed from the risk factors was tested by determining the sensitivity and specificity for the detection of the ophthalmic conditions listed in Tables 3.3, 3.4 and 3.5 (Chapter 3), and by computing the value of the Chi-squared statistic. The basic scoring system produced by the regression analysis involved the creation, and testing, of many similar but distinct

V.A. CODE	0	1	2	3	4
REFERRAL CATEGORY	NUMBER OF PATIENTS ENTERING EACH CATEGORY				
Non-referral	250	13	(4)	(0)	(0)
Routine referral	39	9	6	2	0
Early referral	18	7	5	2	8

Scores: 0-4 refer to the values given to visual acuity.

Table 6.2 The categorisation of 363 patients according to their referral criteria based on changes in visual acuity only. The numbers underlined identify those patients who should have been referred on clinical ground but were not (false negatives) because the visual acuity was normal or only mildly reduced. The numbers in parentheses denote false positives. (The visual acuity code comprises 0=6/6 or better; 1=6/9-6/12; 2=6/18-6/24; 3=less than 6/24; 4=no perception of light).

scoring rules. This led to the following system which provided the highest value of Chi-squared.

(a) Visual acuity variable (X_1)
where 0 = normal visual acuity (6/4-6/6)
1 = visual acuity mildly reduced (6/9-6/12)
2 = visual acuity moderately reduced (6/18-6/24)
3 = visual acuity severely reduced (<6/24)
4 = loss of vision

(b) Malar fracture type (X_2)
1 = if fracture is comminuted or blow-out
0 = if otherwise

(c) Motility disorder (X_3)
1 = if motility disorder present
0 = if motility disorder absent

(d) Amnesia (X_4)
1 = if amnesia (retrograde and post-traumatic amnesia) present
0 = if otherwise

From these parameters, the following scoring system was computed:

$$\text{Score} = (4 \times X_1) + (3 \times X_2) + (3 \times X_3) + (5 \times X_4)$$

The "round number" weights in the scoring formula were created as approximations to the actual values that came out of the stepwise regression analysis. In relative terms, the rounded numbers were in proportion to the actual numbers, they therefore served effectively in terms of sensitivity and specificity and they are clearly convenient in their simplicity.

The score values were then interpreted by the decision rule as follows:

If score = 0	(non-referral)
If score = 1-4	(non-referral)
If score = 5-8	(routine-referral)
If score = 9-11	(routine-referral)
If score = 12 or more	(early-referral)

When this system, based on visual acuity changes only, is applied to the population of 363 patients it gives a sensitivity value of 80% and the categorisation of results shown in Table 6.2 is obtained.

Table 6.3 shows that the scoring system described above results in appropriate referral in the majority of cases when the rest of the variables is taken into account, although a total of 37 patients (Table 6.4) received scores of 0 or 1 but should have been referred and a total of 26 patients received scores of 5 or more but would not have warranted referral. The tests of validity for these scores are as follows:

Sensitivity value = 89%

Specificity value = 90%

Predictive value of a positive test = 92%

Predictive value of a negative test = 87%

REFINED SCORE	0	1-4	5-8	9-11	12+
REFERRAL CATEGORY	NUMBER OF PATIENTS ENTERING EACH CATEGORY				
Non-referral	173	69	(22)	(3)	(1)
Routine referral	10	12	24	5	4
Early referral	5	10	5	7	13

Table 6.3 The referral categorisation of the same patients as in Table 6.2, taking into account the factors shown in Table 6.5. The numbers underlined comprise the false negative population where as the numbers in parentheses denote the false positives.

No. of Cases	Eye Abnormality	Desired Outcome
2	Flat retinal hole	Early Referral
1	Vitreous Haemorrhage	Early Referral
6	Angle recession	Early Referral
5	Proptosis	Early Referral
3	Uncomplicated vitreous floater	Routine Referral
1	Lens damage (traumatic cataract)	Routine Referral
4	Enophthalmos	Routine Referral
6	Eyelid laceration	Routine Referral
2	Nasolacrimal damage	Routine Referral
4	Moderate/severe failure of accommodation	Routine Referral
3	Traumatic pupillary changes	Routine Referral
37	Patients	

Table 6.4 The characteristics of patients whose eye abnormalities were falsely predicted not to warrant referral to the ophthalmologist (false negatives).

As shown in Table 6.4, the patients which this system failed to identify as referrals included five patients with proptosis. However, as in each of these cases the visual acuities were not impaired, sight-threatening retro-bulbar haemorrhage was not implicated and referral may not have resulted in any tangible benefits for the patients. Four patients with impaired accommodation were also missed by this system. However, the visual acuities in these patients were measured for distance and were normal. The majority of bedside examinations by maxillofacial surgeons would utilise near visual acuities and would therefore identify patients with impaired accommodation as having impaired vision and warranting referral. The major cause for concern in this series, however, centred on six patients with peripheral retinal/vitreous damage and six patients who were found to have angle recession. It would appear that, short of carrying out prospective examination of such patients, they are liable to 'slip through the net' of the scoring system described because they could only be identified by specialised slit-lamp and ophthalmoscopic examination.

6.4 DISCUSSION

The scoring system described above is designed to be applied at the time the patient first presents and has a high predictive accuracy for the patient cohort from which it was derived. It requires to be tested upon a new group of patients in order to determine whether or not the values reported still hold for such a group (Chapter 7). The clinical data sheet being employed for this purpose is illustrated in Table 6.5.

The method for constructing the scoring system from the selected class of informative predictors is undoubtedly informal from a statistical point of view. In particular, the application of linear regression analysis to data of this type, using the arbitrary scores of 0, 1 or 2 assigned to the three types of referral is, in itself, not above criticism. It should be emphasised, however, that this technique was used here as a 'stepping stone' to the scoring system which forms the major product of this chapter. The scoring system itself has been validated legitimately and has been identified as being empirically as good as any similar system so far evaluated. On the other hand, the system is not sacrosanct, in that any of a wide class of alternative minor variants perform almost as well. A comparatively informal approach for the construction of the scoring system has been used because of the unwieldily nature of the data and partly because regression analysis with an ordered categorical response variable (such as the scores assigned to the referral type) combined with the incorporation of stepwise selection of predictors remains a topic of basic statistical research.

Clinical Feature	Initial Score	Final Score
Visual Acuity		
6/6 or better	0	
6/9 - 6/12	4	
6/18 - 6/24	8	
6/36 or less	12	
No light perception	16	( )
Malar Fracture Type		
Comminuted (VII)	3	
Blow-out (V)	3	
Otherwise	0	( )
Motility Abnormality		
Present (eg diplopia or squint)	3	
Absent	0	( )
Amnesia		
Retrograde + PTA	5	
Otherwise	0	( )
Total Score		( )

Consider adding (1) to the final score if one or more of the following factors exist and total score is (11):-

If sex is female
If Age is 30-39 years
If cause of injury is RTA

Referral to the Ophthalmologist

If total score = 0 - 4 : Do not refer
= 5 - 11 : Consider routine referral
= 12 + : Consider urgent referral

Table 6.5 Scoring sheet used by the maxillofacial surgeon to determine which patients with midface injuries warrant referral to an ophthalmologist.

The patient cohort upon which this study was based does not include individuals with additional head injuries warranting neurosurgical admission. However, the database upon which the scoring system is based is sufficiently broad to ensure its reliability. Moreover, it was the ophthalmic **OUTCOME** which determined the general nature of the **PREDICTORS** which thereby ensures reliability of the system when it is applied to different patient groups.

The selection of 'predictors' and 'outcomes' was inevitably empirical as was the selection of referral criteria. However, these were discussed with a number of maxillofacial surgeons and ophthalmologists before being chosen and hopefully provide a consensus from both specialties. It may be considered that other predictors can be identified which will effectively select additional patients for referral. Other predictors which were considered, and which in isolation were found to have a fair predictive value, comprised sex (females were more likely to have sustained significant ocular injury than males, perhaps because they did not defend themselves so well), age (individuals between the ages of 30-39 were more likely to have sustained ophthalmic injury than in any other age group) and road traffic accidents (those patients involved in road traffic accidents were more likely to have sustained ophthalmic injuries than from any other cause). However, when these predictors were considered within the above system they failed to identify any additional individuals warranting referral.

Holt et al (1983) classified eye injuries associated with facial fractures into three categories, minor or temporary, serious and blinding. They defined minor injuries as those injuries without permanent, visual or physiological sequelae; serious injuries as those producing sustained visual loss or adnexal sequelae requiring subsequent reconstructive measures; and blinding injuries as those resulting in either no light perception or loss of the eye. Although this classification effectively serves to highlight the fact that a large proportion of patients with maxillofacial injuries are subject to significant ocular sequelae, it does not identify those individuals for whom an ophthalmic referral may be indicated.

6.5 CONCLUSIONS

In conclusion, the principal predictor for the detection of ophthalmic injury is visual acuity. It is therefore of the utmost importance that this parameter be assessed in all patients sustaining orbital injuries sufficient to cause facial fracture. The application of the scoring system described, when taken in conjunction with a detailed clinical appraisal of the patient, is likely to result in a large proportion of 'appropriate' referrals to the ophthalmologist and will lessen the likelihood of significant ophthalmic pathology being missed.

CHAPTER 7

THE SCORING SYSTEM: A PROSPECTIVE EVALUATION

7. THE SCORING SYSTEM: A PROSPECTIVE EVALUATION

7.1 INTRODUCTION

A simple scoring system and chart has been designed for the maxillofacial surgeon to determine which patients with midfacial injuries warrant referral to an ophthalmologist (Chapter 6). The sensitivity and specificity of this evaluation system approximated to 90% when it was applied to the population of 363 patients upon which it was based. Clearly, however, this did not provide a reliable reflection of the sensitivity and specificity for detection in further practice, since the system will inevitably be "biased" in favour of the original patients upon whom it was based.

In order to determine whether this system is applicable and effective in the management of patients who sustain midfacial fracture, it was essential to carry out a further prospective evaluation of the scoring system upon a new cohort of patients.

7.2 PATIENTS AND METHODS

7.2.1 Patients

One hundred patients who sustained midfacial trauma leading to fracture were assessed. Patients with isolated nasal, mandibular or skull fractures were not included.

7.2.2 Methods

Patients who attended the maxillofacial unit at Canniesburn Hospital for the first time between December 1989 and August 1990, with radiologically proven midfacial fractures, were assessed at the time of initial presentation according to a written protocol. Each patient was examined by the attendant maxillofacial surgeon and was accorded a score from which the patients were categorised as requiring non-referral, routine referral or early referral. In addition, the reason for referral, as elicited by the maxillofacial surgeon, was appended to the score sheet (Chapter 6).

All patients were then evaluated by the ophthalmologist according to the same protocol used for the original study (Chapter 3). In brief, this comprised an assessment of visual function, including the visual acuity as tested on the standard or the reduced Snellen's chart, a detailed evaluation of ocular motility including the use of Hess charts, pupillary dilatation and examination of the anterior and posterior segments of both eyes. Data accruing from that examination were then coded according to the score sheet appended.

The criteria as to what ophthalmic conditions warrant referral to an ophthalmologist and for what reasons have already been described (Chapter 3).

It was essential to ensure that reduced visual acuity, when it occurred, was a sequel to the midfacial trauma sustained and not to a previous ophthalmic disorder such as amblyopia, uncorrected errors of refraction, non-traumatic cataract or previous trauma affecting the eye or the visual pathway. Similarly, amnesia scored positively only when it included both retrograde and post-traumatic loss of memory.

7.3 RESULTS

A total of 100 patients with radiological evidence of midfacial fracture was assessed for the purpose of evaluating the validity of the scoring system. Of these, 17 patients were correctly identified by the system as warranting referral to the ophthalmologist (true positive) and another 73 as unlikely to warrant such referral (true negative). Nine patients were incorrectly identified as requiring referral (false positive) whilst only one patient was incorrectly classified as not justifying referral (false negative). The tests for validity for these scores are as follows:

Sensitivity value = 94.4%

Specificity value = 89.0%

Predictive value of a positive test = 65.4%

Predictive value of a negative test = 98.6%

7.4 DISCUSSION

A screening test should provide a good preliminary indication of which individuals actually have the disease and which do not (Mausner & Bahn, 1974). This is referred to as the validity of the test. Validity has two components: sensitivity and specificity. Sensitivity is the ability to identify correctly those who have the disease. Specificity is the ability to identify correctly those who do not have the disease.

An ideal screening test would be 100 percent sensitive and 100 percent specific. In practice this does not occur; sensitivity and specificity are usually inversely related. Naturally, we want a sensitive test, one which will identify a high proportion of those individuals who actually have the disease in question. The results of this pilot study clearly demonstrate the high sensitivity of the scoring system. This confirms the prediction that the application of the scoring system is likely to result in a large proportion of "appropriate" referrals to the ophthalmologist, thereby lessening the likelihood of significant ophthalmic pathology being missed.

Only one patient in the whole series was incorrectly identified as not warranting referral (false negative). However, his only ocular problem was a 3 mm enophthalmos which was unlikely to have led to visual complications and would have been dealt with by the maxillofacial surgeon in any case.

With a specificity value of less than 100%, inevitably, some "inappropriate" referrals are bound to occur. In this study, 9 patients with healthy eyes scored 5-10 on the scoring system and, therefore, were incorrectly categorised as warranting routine referral to the ophthalmologist. Of those, 6 patients scored 6 because they had sustained either a blow-out or a comminuted malar fracture (score=3) leading to diplopia or other motility disorder (score=3) whilst two others scored 10 since they had suffered a temporary mild reduction of visual acuity (score=4). The ninth patient scored 5 because of the combined type of amnesia he had sustained. However, because of the small size of this category of patients (1 patient per month in this series) and the fact that their scores on the system placed them on the routine rather than the early list of referrals, this improper referral was unlikely to have disturbed or overloaded the routine work at the local eye clinic at all. Moreover, it is preferable to make a few unnecessary referrals than to miss potentially sight threatening pathology.

7.5 CONCLUSIONS

In conclusion, the scoring system which has been devised and tested appears to be efficient enough for routine use. It is hoped that maxillofacial surgeons will find it helpful in the assessment of their patients' needs, especially when an ophthalmologist is not immediately available.

The principal predictor of an adverse ophthalmic prognosis is impaired visual acuity. This study re-affirms the tenet that the assessment of acuity, or alternative tests of central visual function should be performed in all patients sustaining midfacial fracture. Failure to do so could be deemed negligent.

CHAPTER 8

CONVERGENCE INSUFFICIENCY AND FAILURE OF ACCOMMODATION FOLLOWING MIDFACIAL TRAUMA

8. CONVERGENCE INSUFFICIENCY AND FAILURE OF ACCOMMODATION FOLLOWING MIDFACIAL TRAUMA

8.1 INTRODUCTION

Defects of convergence and accommodation are common sequels of head injuries, the two functions failing usually to a commensurate degree although either may occasionally be separately deficient (Duke-Elder, 1972). Sometimes a lesion in the brain stem or 3rd nerve is causal, very occasionally in the supranuclear mechanism, while in other cases the disability is functional (Duke-Elder, 1972).

Jampolsky (1971) reported that convergence insufficiency was often the result of poor accommodation whilst Raskind (1976) noted that it might have been secondary to head trauma. Head trauma has also been found to result in weakness of accommodation or accommodative insufficiency in the post-traumatic period (Matsuzaki, 1964). Cooper and Duckman (1978) considered trauma a rare cause of convergence insufficiency while Davis (1956) and Krohel et al (1986) thought it was more frequent. Paralysis of accommodation and convergence have also been described following cerebral concussion (Doden & Bunge, 1965; Franceschetti & Klinger, 1943).

The anatomical localisation of convergence insufficiency secondary to head trauma remains uncertain, although lesions in the occipital lobe and upper midbrain seem capable of producing this syndrome (Krohel et al, 1986). No matter what the cause of the defect is, the result causes considerable discomfort, and is therefore of clinical significance (von Noorden, 1985).

Sometimes spontaneous resolution occurs (Cross, 1945). Occasionally, however, patients are encountered whose fusional amplitude and near point of convergence cannot be improved in spite of prolonged and intensive therapy (von Noorden et al, 1973). In uncomplicated cases the prognosis is good, but unfortunately in many cases the changes in psychology and personality following head injuries of any severity may be profound and lasting (Paterson, 1944 & 1942).

8.2 AIMS OF STUDY

Impaired accommodation and convergence is a recognised complication of head injury and was seen in a number of patients in Canniesburn hospital prior to the commencement of this trial. As the main study proceeded, it became clear that the incidence of these defects was higher than previously expected. It was, therefore, deemed appropriate to perform a separate prospective study aimed at investigating these problems.

The aims of the present study are as follows:

1. To prospectively examine convergence and accommodation in a group of patients sustaining midfacial fracture in order to determine the incidence of such disorders.

2. To determine the aetiology and type of injury which predispose to impairment in these functions.

8.3 PATIENTS AND METHODS

Fifty-two patients who had sustained frontal and/or midfacial fracture and were treated at the Oral and Maxillofacial Unit at Canniesburn Hospital over a period of six months were admitted to this study. All patients were subjected to full ophthalmic examination. The criteria for timing of this ophthalmological assessment were along the same lines adopted in the main study.

In addition to full ophthalmic examination, evaluation included routine work-up for muscle imbalance, including visual acuity determination, cover and prism cover tests, and measurement of the near points of convergence and accommodation on a standard R A F near point rule. Accommodation was checked binocularly and then monocularly with the patient wearing his correction for distance when applicable. Prism fusion range was also measured. Factors which could have affected accommodation and/or convergence other than those related to facial injury such as refractive errors, poor visual acuity, drugs, previous squint or other ocular operations, ill health and debility, were eliminated by careful history taking and examination.

The data accruing from each consultation was documented on a separate "data collection sheet" and stored in alphabetical order of surname for future analysis. Patients who had been found to have normal near points of accommodation and convergence on their first visit were not reassessed in that particular area, and only those individuals with significant reduction were reviewed regularly on each subsequent visit at appropriate periods and had their responses assessed.

Published normal values relate to very large cohorts and are, therefore, better than what can be assembled from a smaller group of control subjects. Hence, normal values for accommodation appropriate to age were adopted from Duane's study (1922) in which he examined 4200 normal volunteers. Table 8.1 describes accommodation in terms of distance and dioptric power. To my knowledge, there is no recognised classification for the severity of convergence insufficiency. Following consultation with fellow ophthalmologists and orthoptists, the severity of convergence insufficiency was classified into mild, moderate, severe, and absolute failure as defined in Table 8.2.

Age (Years)	ACCOMMODATION Minimum D (cm)	Mean D (cm)	Maximum D (cm)
10	11.1 (09.0)	13.4 (07.5)	15.7 (06.4)
15	10.1 (09.9)	12.3 (08.1)	14.5 (06.9)
20	08.9 (11.2)	11.1 (09.0)	13.4 (07.5)
25	07.8 (12.8)	09.9 (10.1)	12.2 (08.2)
30	06.5 (15.4)	08.7 (11.5)	10.8 (09.3)
35	05.2 (19.2)	07.3 (13.7)	09.3 (10.8)
40	03.4 (29.4)	05.8 (17.2)	07.9 (12.7)
45	01.9 (52.6)	03.6 (27.3)	05.9 (17.0)
50	01.0 (100)	01.9 (52.6)	03.2 (31.3)
55	0.8 (125)	01.3 (76.9)	01.9 (52.6)
60	0.7 (143)	01.2 (83.3)	01.7 (58.8)
65	0.6 (167)	01.1 (90.9)	01.6 (62.5)
72		01.0 (100)	

D: Dioptre, cm: centimetre

Table 8.1 Normal accommodation values (Reproduced from Duane, 1922).

Near Point of Convergence	Severity of Insufficiency
6- 7 cm	Normal
8-12 cm	Mild failure
13-24 cm	Moderate failure
25-30 cm	Severe failure
More than 30 cm	Absolute failure

Table 8.2 Grades of convergence insufficiency (produced following consultations with fellow ophthalmologists and orthoptists).

8.4 RESULTS

8.4.1 General Results

Fifty two patients with midfacial fracture and associated head injury were included in this study irrespective of whether or not they had complained of any untoward visual symptoms. Of those, 11 patients were found to have convergence and/or accommodative disturbances. The findings in this group of patients are summarised in Table 8.3.

The population of study included 35 males (67.3%) and 17 females (32.7%). Their ages ranged from 5 to 72 years with a mean age value of 31.4 years. The right side of the face was injured in 28 patients (53.8%) while the left was affected in another 21 patients (40.4%). Only one patient had sustained a bilateral fracture while another two had suffered of a nasal fracture.

The most common cause of facial trauma was alleged assault which affected 37 patients (71%) followed by simple falls which involved another 8 patients (15.4%). Five patients sustained their injuries following road traffic accidents (9.6%) while one patient suffered of a sporting accident and another of an industrial accident(1.9% each).

Case No	Age/ Sex	Cause of Injury	Type & Side of Fracture	Visual Acuity Right Eye Distance	Visual Acuity Right Eye Near	Visual Acuity Left Eye Distance	Visual Acuity Left Eye Near
01	32 Male	Assault	R+L III	6/6	6/6	6/6	6/6
02	13 Male	Assault	R V	6/6	6/6	6/6	6/6
03	72 Female	Fall	Nasal	6/9	6/12	6/12	6/12
04	27 Female	Assault	R IV	6/6	6/9	6/6	6/6
05	48 Female	Assault	L III	6/6	6/12	6/6	6/9
06	17 Female	Fall	R V	6/6	6/6	6/6	6/6
07	47 Male	Assault	Nasal	6/6	6/6	6/6	6/12
08	30 Female	Assault	L V	6/9	6/12	6/9	6/18
09	30 Male	Assault	L III	6/6	6/6	6/6	6/6
10	33 Male	Assault	L IV	6/6	6/6	6/6	6/6
11	32 Male	Assault	L VII	6/6	6/6	6/6	6/6

Table 8.3 The clinical findings in the population of study.

Case No	Cover Test Near	Cover Test Distance	NPC	Accommodation RE	Accommodation LE	Accommodation BE	Prism Fusional Range CN	Prism Fusional Range DN	Prism Fusional Range CD	Prism Fusional Range DD	Management
01	exo	NAD	7	11	11	10	14	10	10	4	Exercises
02	hyper	eso	6	7	7	7	30	6			Exercises
03	exo	NAD	10	*	*	*					No exercises
04	exo+hypo	hypo	6	10	9	8	20	10	10	2	Exercises
05	exo	NAD	6	25	26	24	40	10	8	2	No exercises
06	NAD	NAD	8	13	10	10	18	2	14	2	No exercises
07	eso	NAD	10	31	32	30	40	10	20	1	Exercises
08	hyper	hyper	28	29	30	29	14	6	10	4	Exercises
09	NAD	NAD	26	20	14	20	25	10	16	2	No exercises
10	NAD	NAD	8	9	8	8	20	12	8	4	No exercises
11	exo	exo	11	17	16	16	20	8	10	2	Exercises

NPC = Near Point of Convergence
RE = Right Eye LE = Left Eye BE = Both Eyes
CN = Convergence at Near (1/3m) DN = Divergence at Near (1/3m)
CD = Convergence at Distance (6m) DD = Divergence at Distance (6m)
NAD = No Abnormality Detected
exo = Exophoria eso = Esophoria
hyper = hyperphoria hypo = hypophoria
* = Minimal accommodation

Table 8.3 The clinical findings in the population of study (Cont'd).

Types of fracture were variable. Pure blow-out fractures (Type V) and tripod fractures with an intact F-Z suture (Type III) were the most common fractures encountered and had affected 12 patients each. The second most common fractures were comminuted malar fractures (Type VII) and tripod fractures with distracted F-Z sutures (Type IV) which affected 8 patients each. Less common fractures were type I (n=7), type VI (n=2), and nasal fractures (n=2).

Twenty patients complained of diplopia at near, seven complained of blurred vision and reading difficulties, one had headaches while 24 patients were symptomless.

8.4.2 Patients with Accommodation/Convergence Disturbances

Of 52 patients in this study, 11 suffered of accommodation and/or convergence disturbances. They included 6 males (17% of the male population) and 5 females (29% of the female population). Their ages ranged from 13 to 72 years with a mean age value of 30.2 years. The right eye/side of face was involved in 3 patients and the left in 5 patients. One patient had a bilateral fracture and another two sustained nasal fractures.

Nine of these 11 patients sustained their injuries due to alleged assaults (24.3% of all assaulted patients) while another two had their injuries following simple falls (25%

of all fall victims). Road traffic accidents, industrial, and sporting accidents did not yield any accommodative or convergence disturbances in this group of patients.

The fractures sustained included a tripod fracture with an intact F-Z suture in 3 patients (25% of all fractures of this type), a tripod fracture with a distracted F-Z suture in 2 patients (again 25% of all similar fractures), and a pure blow-out fractures in another 3 patients (25% of all type V fractures). Only one patient had sustained a comminuted fracture (12.5% of all type VII fractures), and another 2, nasal fractures.

Five patients complained of diplopia at near (5/11=45.5%) and another 4 of blurred vision and/or difficulty with reading (4/11=36.4%). The remaining 2 patients did not complain of any symptoms. All patients who complained of diplopia had sustained either a pure (Type V; n=4) or impure (Type VII; n=1) blow-out fracture of the orbit. Diplopia was more common among assault victims (n=4) than among simple fall victims (n=1). Similarly, the incidence of difficulty with reading and/or blurred vision was higher among the assaulted group (n=3) than among those who sustained a simple fall (n=1).

Four patients suffered of a reduced accommodation, reduced near point of convergence (NPC) and reduced prism fusion range. Four patients had normal accommodation and NPC but reduced prism fusion range. Three patients had normal accommodation but reduced NPC and prism fusion range. All patients but one, had reduced prism fusion range. All patients with normal NPC (n=4) had normal accommodation as well. Table 8.4 lists the convergence and accommodation values for each patient while Table 8.5 describes the interpretation of these measurements.

All patients, except those with isolated nasal fractures, were operated on for correction of their midfacial fractures within 2 weeks of injury. They all had pre- and postoperative ophthalmological assessments and were found to have maintained their accommodative and/or convergence disturbances in the immediate postoperative period.

The follow-up period was up to 6 months for each patient either from the date of operation for those who underwent surgery or the date of first ophthalmological examination for those who did not undergo surgery. Six patients (No 1, 2, 6, 7, 8 & 11) were randomly selected to receive orthoptic exercises/treatment while the other five were only monitored for signs of spontaneous recovery.

Patient (Age & Sex)	Accommodation RE	LE	BE (Centimetre)	NPC cm	Fusion Range 1/3m	6m (Prism Dioptre)
No 1 (32, M)	11	11	10	7	14	10
No 2 (13, M)	7	7	7	6	30	15
No 3 (72, F)	*	*	*	10	20	10
No 4 (27, F)	10	9	8	6	20	10
No 5 (48, F)	25	26	24	6	40	8
No 6 (17, F)	13	10	10	8	18	14
No 7 (47, M)	31	32	30	10	40	20
No 8 (30, F)	29	30	29	28	14	10
No 9 (20, M)	20	14	20	26	25	16
No 10 (33, M)	9	8	8	8	20	8
No 11 (32, M)	17	16	16	11	20	10

* Minimal accommodation.

Table 8.4 Accommodation and convergence values for each patient.

Patient	Accommodation	NPC	Fusion Range
No 1	Normal	Normal	Reduced for 1/3 & 6m
No 2	Normal	Normal	Reduced for 1/3 & 6m
No 3	Normal	Reduced (M)	Reduced for 1/3 & 6m
No 4	Normal	Normal	Reduced for 1/3 & 6m
No 5	Normal	Normal	Reduced for 6m only
No 6	Reduced	Reduced (M)	Reduced for 1/3 & 6m
No 7	Normal	Reduced	Normal
No 8	Reduced	Reduced (S)	Reduced for 1/3 & 6m
No 9	Reduced	Reduced (S)	Reduced for 1/3 & 6m
No 10	Normal	Reduced (M)	Reduced for 1/3 & 6m
No 11	Reduced	Reduced	Reduced for 1/3 & 6m

(M) Mild
(S) Severe

Table 8.5 Clinical interpretation of the valued results in in Table 8.4.

Of those who received orthoptic treatment, 5 patients recovered to within the normal range of convergence/accommodation values (5/6=83%) while the sixth (No 11) did not, although he showed some improvement. Among the 5 patients who did not receive orthoptic treatment (No 3, 4, 5, 9 & 10), four recovered from their disturbances (4/5=80%) while the fifth patient (No 9) did not though, again, he did show signs of a limited recovery. The difference in the incidence of recovery between orthoptically treated and untreated patients is not statistically significant.

The duration of recovery for those patients who received orthoptic exercises/treatment ranged from 1 to 6 months with a mean value of 3.6 months. In contrast, the mean duration of recovery for those individuals who did not receive orthoptic treatment was 2.2 months. The difference was not statistically significant.

8.5 DISCUSSION

As expected, the composition of the population of this study (n=52) is comparable to that of the main study (n=363) with regard to age and sex distribution, and in respect to the cause of injury and type of facial fracture sustained.

It appears that convergence and accommodative disturbances are commoner among the young-age group especially females. This, however, may only be a reflection of the composition of the population of midfacial trauma patients which tends to be young with the females sustaining a higher incidence of visual complications than males and as such, is in line with the findings in the main study.

Weakness of convergence and accommodation is very common following head injury. Most cases show commensurate defect of the two functions, as would be expected from their synergic relationship. Occasionally, either may be deficient separately (Cross, 1945). In this study, 21% of patients (11/52) developed convergence and/or accommodative defects which included 4 patients with full set of combination of defects while the rest of patients (n=7) exhibited either another combination (n=1) or simply an individual defect.

Twenty-four percent of all allegedly assaulted patients, and 25% of fall victims suffered with convergence and/or accommodative failure. No other cause of injury contributed to such defects. The difference in the incidence of convergence/accommodation disturbances between the two causes is statistically insignificant. It does not appear that there is any significant correlation between the cause of injury sustained and the occurrence of these visual defects.

Similarly, it does not appear that the type of midfacial fracture sustained had any bearing on the incidence of failure of accommodation or convergence. Twenty-five percent of patients with each of type III, IV, and V have developed such defects. It is important to note, however, that these fractures are among the most serious zygomatico-orbital fractures and require a great force of impact to take place. The occurrence of convergence and accommodation defects may, therefore, be related to the severity and force of impact and the associated closed head trauma rather than to the actual fracture itself. However, the severity of the visual defect itself does not necessarily correlate positively with the severity of the head trauma (Krohel et al, 1986). It is also interesting to note that both patients with nasal fractures suffered from convergence and accommodation impairment. Bearing in mind that these fractures were simple enough as not to require surgical treatment, one can only assume that the visual defects may have either been related to the associated head trauma and its presumed antero-posterior vector or, less likely, to local bruises/haematoma of the soft tissue affecting the functions of the medial recti. Extraocular muscle imbalance is a common sequel to head injury (Cross, 1945) which is also a recognised cause of convergence and/or accommodative failure (Krohel et al, 1986; Andree & Klein, 1983; Neely, 1945).

Simultaneous failure of convergence and accommodation in a patient is common (Cross, 1945) and has taken place in 4 patients while the rest of patients had either function impaired separately. Some patients complained of diplopia for near and/or difficulty with reading or blurred vision in spite of their normal NPC and accommodation values (n=4). The common feature in most of them, however, was a reduced fusion range which affected 10 of our 11 patients. It appears that the prism fusion range is a more reliable index than the NPC in the diagnosis of convergence insufficiency. This opinion had also been expressed by Krohel and associates (1986) and Brinkley and Walonker (1983) who noted that some of their patients had symptoms of convergence insufficiency with a normal NPC but reduced fusional reserves.

Spontaneous recovery of accommodation and convergence following head trauma is well recognised in the literature although, some authors believed that the process can be hastened by orthoptic exercises (Duke-Elder, 1972; Candler, 1945). In this study, 9 of 11 patients (82%) recovered from their accommodation/convergence failure within 6 months of injury or facial bone surgery. Of 6 patients who received orthoptic exercises, 5 recovered

(83%) while 4 of 5 other patients (80%) achieved spontaneous recovery. The difference in the recovery rate between the two groups is not statistically significant.

The mean time for recovery was 3.6 months for the treated group and 2.2 months for the spontaneous recovery group. In spite of the apparent difference in the recovery time between the two groups, this difference is not statistically significant. This, however, may be due to the small number of patients in these groups.

8.6 CONCLUSIONS

In conclusion, it appears that the incidence of accommodation and/or convergence failure following midfacial trauma is not significantly related to the cause nor to the type of fracture sustained. It may, however, be related to the severity of impact and the associated closed head trauma.

Recovery of function does not appear to be more favourable following a course of orthoptic exercises than without. Consequently, recovery of convergence and/or accommodative functions seems to be spontaneous and is not hastened with exercises.

Prism fusional range emerged as a more reliable tool for diagnosis of convergence insufficiency than measurement of the near point of convergence.

CHAPTER 9

MIDFACIAL TRAUMA AND THE EYE:
PROSPECTIVE VERSUS RETROSPECTIVE ANALYSIS

9. MIDFACIAL TRAUMA AND THE EYE: PROSPECTIVE VERSUS RETROSPECTIVE ANALYSIS

9.1 INTRODUCTION

Direct injury to the eyeball is relatively rare compared to the frequency of orbital trauma, but the sensitivity of the eye as a visual receptor may make such injuries especially disabling to the patient. When an injury to the eye occurs in association with an orbital fracture, its detection becomes doubly important as treatment may, sometimes, have to be instituted at once, and the presence of ocular injury documented prior to treatment of the fracture.

Ocular trauma itself has been abundantly described in the literature (Paton & Emery, 1973). However, there have been only sporadic reports of individual cases of eye injury in patients with facial fractures (Edmund & Godtfredsen, 1963; Gordon & Macrea, 1950).

The present study was designed to allow both ophthalmologists and maxillofacial surgeons to examine all patients with midfacial fractures prospectively during a 2-year period. For comparison, a retrospective study of all patients treated for zygomatico-maxillary fractures in the preceding 2 years was performed. The purposes of the study were to determine the nature and incidence of ocular injury in patients with midfacial fracture and to compare the frequency of detection of ocular and motility disorders in both series.

9.2 PATIENTS AND METHODS

A list of 555 patients with midfacial fractures due to blunt trauma, who had attended the Oral and Maxillofacial Unit during the two years immediately preceding the beginning of this study, has been drawn from the computer database (Appendix 4) at Canniesburn Hospital. The majority of patients in this group had been managed by the maxillofacial surgeon only. Ophthalmic consultation and formal pre-operative ophthalmic assessment was carried out only in 34 cases of midfacial fracture (6%).

9.3 RESULTS

The study included 555 patients who had sustained a total of 530 malar or orbital fractures and 60 maxillary fractures. Of these maxillary fractures, 25 were in isolation and 35 in association with malar fractures. In the majority of cases malar or orbital fractures were unilateral. Only 14 patients suffered from bilateral fracture.

9.3.1 Age and Sex Distribution

The population of study included 453 males (81.6%) and 102 females (18.4%). The age and sex distribution (Fig 9.1) shows the highest incidents (n=201 or 36%) to be among the 20 to 29 year age group of patients tapering steadily on both sides of the curve.

9.3.2 Cause of Midfacial Trauma

The major cause of midfacial trauma was alleged assault, which accounted for 62.3% of all patient (346/555). The second most common cause was falls (15.5% or 86 patients) followed by road traffic accidents and sporting accidents (Fig 9.2). No distinction was made between individuals who sustained their trauma while under the influence of alcohol and those who were not.

9.3.3 Types of Midfacial Fracture

The most common malar fracture sustained was a tripod fracture with an intact F-Z suture (Type III) which accounted for 37% of patients (192) followed by un-displaced fractures (Type I) and tripod fractures with a distracted F-Z suture (Type IV) which accounted for 24% and 18% of patients respectively (Fig 9.3).

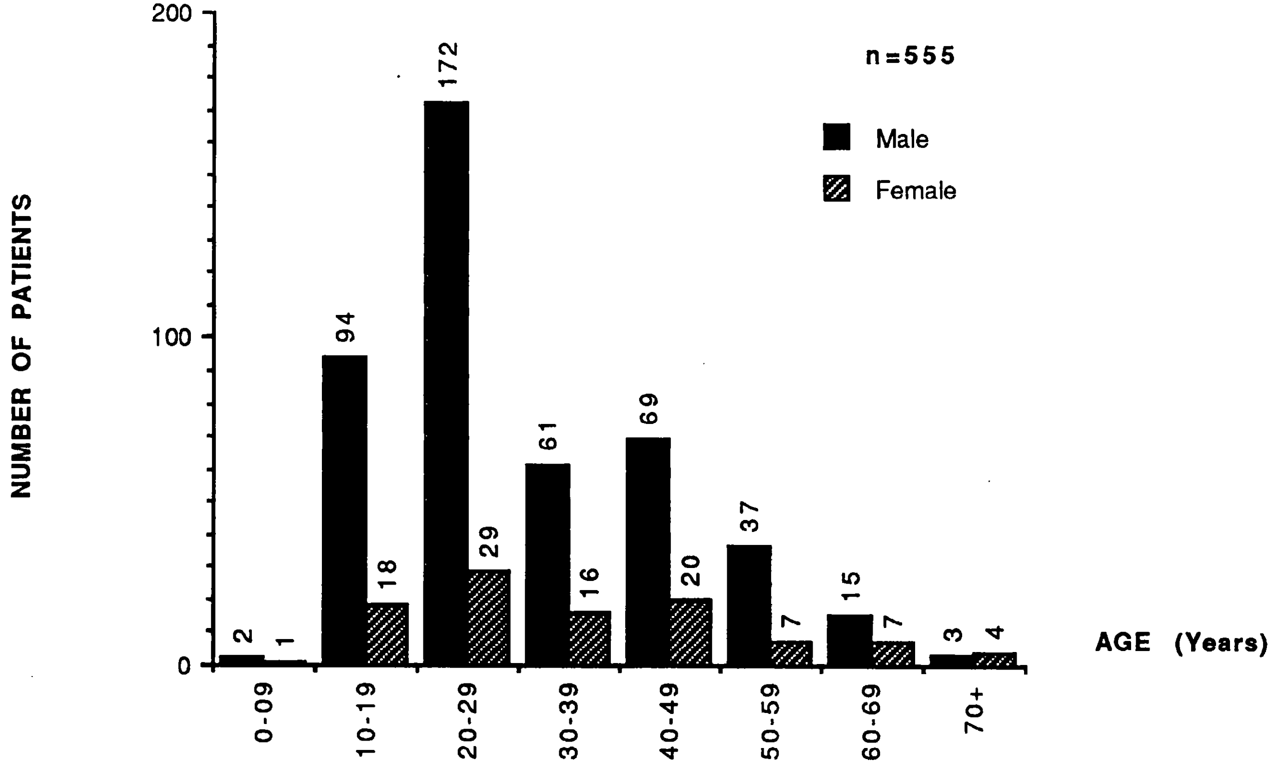


Figure 9.1 Age and sex distribution of the population of the study.

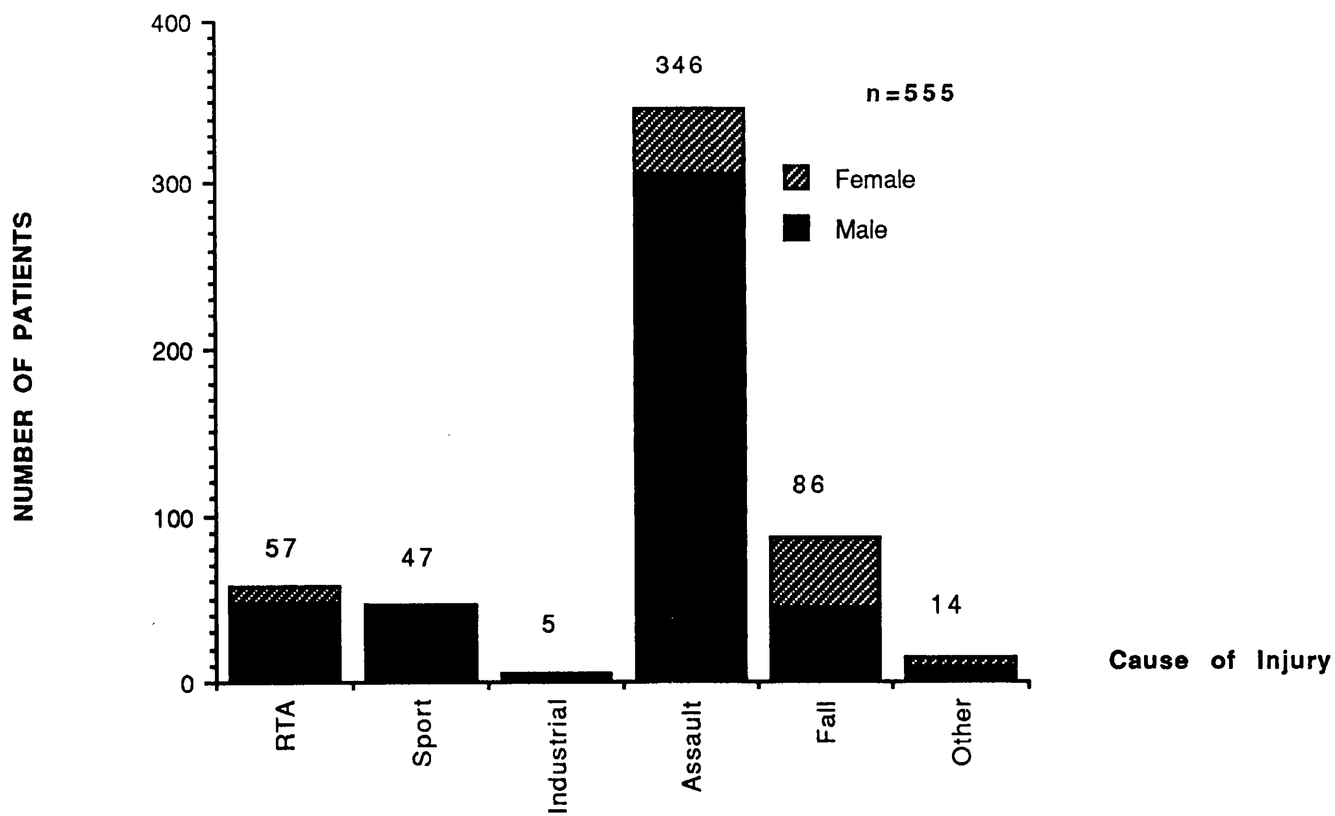


Figure 9.2 The aetiological distribution of midfacial fractures.

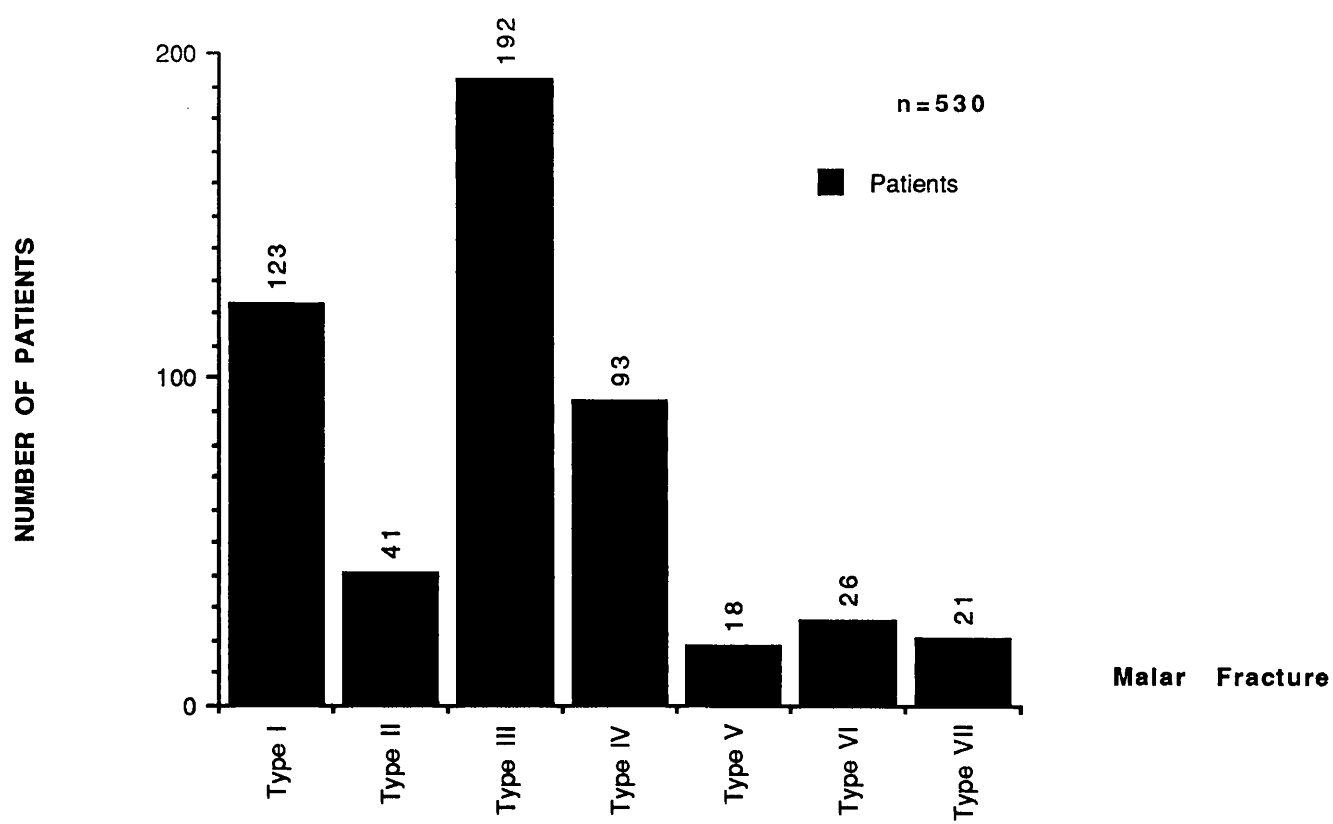


Figure 9.3 The site distribution of malar fractures.

The orbit was fractured in 116 cases (21%), 54 were of the right and 62 the left orbit. No patient presented with a bilateral orbital fracture. Fifty four of these orbits, 30 right and 24 left, required surgical exploration for diagnosis and/or management.

9.3.4 Amnesia

Sixty patients (10.8%) suffered from post-traumatic, retrograde, or both types of amnesia. No details of which type of amnesia sustained are available from this data base.

9.3.5 Enophthalmos

Enophthalmos was a feature in 20 patients following midfacial trauma (3.6%). The number was equally shared between the right and left sides.

9.3.6 Diplopia

Seventy patients (12.6%) complained of diplopia following their facial trauma. However, clinical examination revealed abnormal eye movements in only 35 of them (6.3%). No details are available on the nature of this abnormality of eye movement nor of the direction and extent of double vision. Only 3 of these 70 patients (4.3%) continued to suffer with diplopia by the time they were discharged from the follow-up clinic. However, it is not clear how long it normally took before a patient was deemed to have had a persistent diplopia.

9.3.7 Ocular Injuries

Injury to the Conjunctiva

Subconjunctival ecchymosis, swelling or haemorrhage was the most frequently detected eye injury which affected 275 patients (49.5%). It involved the right eye in 101 patients and the left eye in another 159 patients. Fifteen patients sustained a bilateral conjunctival injury.

Pupil Reactions

Pupils were tested for reaction to light and found to be either slow or non reactive in 2% of all patients (n=12). However, no comment was made on the shape or reaction to accommodation of these pupils nor about any future signs of recovery if any.

Other Ocular Injuries

Unspecified injuries to the globe were reported in 3 cases only accounting to 0.5% of all patients.

No other ocular or motility disorders were described.

Table 9.1 illustrates the prospective/retrospective relationship with regard to the ophthalmic complications identified in the retrospective series.

9.4 DISCUSSION

9.4.1 Previous Studies

The treatment of facial fractures has been well described in the surgical literature (Paton & Emery, 1973), as has the management of ocular injuries (Paton & Emery, 1973; Milauskas & Fueger, 1966). The simultaneous occurrence of these two entities has rarely been approached in a systematic fashion. There are reports of isolated cases of blindness (McCoy, 1958), optic nerve atrophy (Edmund & Godtfredsen, 1963), and other ocular injuries (Holt & Holt, 1983) resulting in reduced vision following orbital fractures. McCoy and associates (1962), plastic surgeons, retrospectively reviewed 855 patients with facial fractures, including 337 fractures involving the middle third of the face. They noted only 4 patients (0.5%) with ocular injuries leading to blindness in 3 cases and traumatic mydriasis in the fourth case. Similarly, Luce et al (1979), Turvey (1977), Morgan et al (1972), Gwyn et al (1971), Converse et al (1967), Schultz (1967), all plastic

Post-traumatic Abnormality	Retrospective Series (n=555)		Prospective Series (n=363)	
	No.	%	No.	%
Amnesia	60	10.8%	103	28.4%
Enophthalmos	20	3.6%	29	8.0%
Diplopia	70	12.6%	72	19.8%
Conjunctival injuries	275	49.5%	252	69.4%
Pupil reactions	12	2.0%	9	2.5%
Unspecified ocular injuries	3	0.5%	-	-

Table 9.1 Illustrates the prospective/retrospective relationship with regard to the ophthalmic complications identified in the retrospective series.

surgeons, reported incidences of ocular injuries following orbital or midfacial trauma which ranged between 2.7% (Luce et al, 1979) and 16% (Morgan et al, 1972). On the other hand, Miller and Tenzel (1967), ophthalmologists, described 5 major ocular injuries in a series of 30 consecutive facial fractures giving a detection rate of 17%. Milauskas and Fueger (1966) reported a 14% incidence of ocular complications in 84 blow-out fractures, again reflecting the experience of ophthalmologists. The largest series of patients with facial fracture resulting in ocular trauma, where an ophthalmologist and a maxillofacial surgeon are involved, is that of Holt and Holt (1983). They analysed retrospectively the clinical data of 727 patients who had sustained a blunt maxillofacial trauma and found that 67% of them (487 patients) had suffered of ocular injuries ranging from temporary (79%) to serious (18%) through to blinding injuries (3%).

9.4.2 The Present Study

Cause of Injury

Retrospective examination of data from 555 patients with midfacial trauma shows that the majority of patients were male (81.6%) between the ages of 20 and 29 years (36%) whose main cause of injury was alleged assaults. The second most common cause of trauma was simple falls which was also the most common cause of injury for females (n=42). Almost an equal number of females (n=41) was allegedly assaulted. These findings are in agreement with those obtained from the prospective study and several other comparable studies (Ellis et al, 1985; Altonen et al, 1976; Lundin et al, 1973). Of interest is the obvious fall in the number of injuries due to car accidents while the patient is not restraint with a seat belt from 2.7% (15/555) in the retrospective series to only 1.1% (4/363) in the prospective series, a fall of nearly 60%. Though statistically insignificant, the difference probably demonstrates the beneficial effect of the compulsory wear of seat belts.

Type of Fracture

The most common malar fractures were Type III (192/555= 37.4%) and Type IV (93/555=18%) respectively. This is in accordance with the prospective series and a previous study on a similar population of patients (Ellis et al, 1985).

Enophthalmos

Enophthalmos following midfacial fracture occurred in 3.6% of patients (n=20) and showed no predominance towards either side. This figure is significantly lower than the

8% from the prospective study (p=0.006). Enophthalmos can easily be missed if it is of a minor degree or if the lateral orbital wall is fractured with loss of a useful reference point or, simply during the early stages of trauma where tissue bruises and haematoma may lead to a transient exophthalmos rather than enophthalmos.

Amnesia

Amnesia was common following midfacial fracture and had affected 10.8% of patients (60/555). However, this figure is significantly lower (p<001) than that obtained from the prospective series (103/363=28.4%). Since amnesia is a transient functional disorder with no obvious physical features, it can easily be missed if not positively look for as part of a routine examination.

Conjunctival Injury

Conjunctival and subconjunctival injury is easy to detect by the ophthalmologist and the non-ophthalmologist alike, and had been reported in 275 patients (49.5%) including 15 where injury was bilateral. While subconjunctival haemorrhage is not a serious injury in itself, it may, however, conceal a more serious ocular injury such as scleral rupture. In fact, the only presenting feature of some blow-out fractures of the orbit may be a black eye or a subconjunctival haemorrhage (BMJ Editorial, 1975; DeHaven & Harle, 1966). It is, therefore, surprising that only 3 patients with some unspecified "Injury to the Globe" were detected, a mere 0.5% of the population of study! The incidence of the reported subconjunctival haemorrhage is also significantly lower than that in the prospective study (69.4%) and other studies on similar populations (Ellis, 1985; Holt & Holt, 1983). One can reasonably assume that the less severe conjunctival injuries and some possible underlying pathology may have been missed or ignored.

Pupillary Changes

Two percent of patients presented with abnormal pupillary shape or reaction. This figure is in agreement with the prospective figure of 2.5%. However, no further description of these pupillary changes has been reported which greatly limits the significance of this clinical finding.

Visual Acuity

Of particular importance is the absence of information on visual acuity in the database even though visual acuity testing is a recognised part of the routine clinical examination and appears as one of the variables on the computer data collection card. In the prospective study, visual acuity emerged as the most important single factor in the detection of eye injury. No examination, therefore, is complete without testing and recording visual acuity.

Diplopia

Diplopia was common following midfacial fracture in the retrospective series and amounted to 12.6% of patients. However, the absence of details on direction of gaze and the field and extent of diplopia limit the usefulness of this piece of information. The incidence of double vision as reported is again significantly lower (p=0043) than that in the prospective series. It may indicate that some patients have recovered from diplopia by the time they have been examined in hospital or simply ignored by the patient when it was away from central gaze. Examination of ocular movement and active seeking for signs of double vision by the examiner are, therefore, important parts of any accurate assessment of the midface fracture patient.

9.5 CONCLUSIONS

The retrospective portion of this study is probably an accurate reflection of the type of fracture and the cause of injury as it exists in this major referral centre and is in accordance with other major studies on similar populations. Patients with facial fractures frequently arrive intoxicated with alcohol, confused, or both. Many are unable to give an accurate history, and the facial examination is performed while other major injuries are being examined and treated simultaneously.

In the prospective portion of the study, a careful ophthalmological examination was a routine portion of the preoperative evaluation of every patient. As a result, more subtle diagnoses were made.

Examination of the data from both series confirms that the index of detection of ophthalmological disorder is higher, and the examination is more useful, when an ophthalmologist is involved in the clinical assessment of patients. This, inevitably, would be reflected on the quality of management and, hopefully, prevent a potentially sight threatening ocular injury being missed.

GLASGOW

British Journal of Oral and Maxillofacial Surgery (1991) 29, 291-301
© 1991 The British Association of Oral and Maxillofacial Surgeons

BRITISH JOURNAL OF ORAL & MAXILLOFACIAL SURGERY

The characteristics of midfacial fractures and the association with ocular injury: a prospective study

I. A. Al-Qurainy, L. F. A. Stassen, G. N. Dutton, K. F. Moos, A. El-Attar

Tennent Institute of Ophthalmology, Western Infirmary, and Department of Oral and Maxillofacial Surgery, Canniesburn Hospital, Glasgow.

SUMMARY. **Ocular injuries commonly occur in patients with facial fractures. This prospective study was set up to determine the incidence of ocular injuries, as assessed by an ophthalmologist, in patients who had sustained midfacial fractures. Over a 2-year period, a study of 363 patients who had sustained midfacial trauma sufficient to lead to a facial bone fracture (438 fractures) was undertaken and patients received a comprehensive examination by an ophthalmologist and an orthoptist within 1 week of injury. The characteristics of the eye injuries sustained were related to the aetiology of the fracture, the type of fracture, and the sex and age of each patient. Ninety percent of patients sustained ocular injuries of various severities. Sixty three percent of patients sustained only minor or transient ocular injuries, 16% suffered moderately severe ocular injury and 12% experienced severe eye injuries. Road traffic accident was associated with the highest incidence of severe ocular disorder (9/45=20%) whilst assaults had the second highest incidence at 11% (20/181). One third of all patients with comminuted malar fracture suffered a severe ocular disorder (9/27) whilst blow-out fracture came second at 16·7% (6/36). Fifty six patients (15·4%) had a decrease in their visual acuity and 9 patients (2·5%) had significant traumatic optic neuropathy.**

Decrease in visual acuity was the main clinical finding accompanying the majority of significant eye injuries. When ocular injuries were related to aetiology, it was apparent that road traffic accidents and assaults associated with alcohol abuse showed the highest incidence of major ocular dysfunction. It is suggested that all patients sustaining midfacial fracture associated with a significant decrease in visual acuity either pre- or postoperatively should have an early ophthalmological review.

GLASGOW UNIVERSITY LIBRARY

INTRODUCTION

Facial fractures are frequently complicated by injury to the eye and its adnexae (Petro *et al.*, 1979; Steidler *et al.*, 1980; Beirne *et al.*, 1981; Culbertson, 1983; Holt *et al.*, 1983; Holt & Holt, 1983). These injuries may result in loss of vision in one or both eyes (Gordon & Macrae, 1950; Gwyn *et al.*, 1971; Ketchum *et al.*, 1976; Steidler *et al.*, 1980; Anderson *et al.*, 1982; Babajews & Williams, 1986) or may compromise ocular function (Holt & Holt, 1983). When these injuries are severe, they may be detected with ease by any medical or maxillofacial practitioner but many injuries appear minimal and may be missed by the non-ophthalmologist (Binder, 1978; Records, 1979; Holt *et al.*, 1983). Ocular injury may result in preventable severe dysfunction of the visual apparatus, if not detected shortly after injury. In one published study by Jabaley *et al.* (1975), the difference in the rate of detection of such ocular injury between a prospective evaluation, where an opthalmologist was involved, and a retrospective evaluation, where only selective referral was made by the plastic surgeon, was 18% (29% and 11% respectively).

Ideally, all patients with a facial or craniofacial fracture should be reviewed by an ophthalmologist (Miller & Tenzel, 1967; Gwyn *et al.*, 1971; Holt *et al.*, 1983). Defects in the field of vision, ocular structure and ocular motility may be permanent (Barclay, 1958; Jabaley *et al.*, 1975; Turvey, 1977; Steidler *et al.*, 1980). It is important to define such functional defects in order to treat any preventable ocular dysfunction such as retinal detachment, tears or holes, or angle recession glaucoma (Jabeley *et al.*, 1975; Holt *et al.*, 1983) and to advise the patient appropriately. The results of ophthalmic examination may lend weight to medico-legal claims. Estimates of the incidence of ocular disorder following midfacial fracture vary considerably, being between 2·7% (Luce *et al.*, 1979) and 67% (Holt *et al.*, 1983). These estimates depend on referral practice, which specialty carried out the evaluation, and whether minor injuries were included in addition to major ones. The results also depend on whether the study was prospective (Miller & Tenzel, 1967; Jabaley *et al.*, 1975) or retrospective (Gwyn *et al.*, 1971; Morgan *et al.*, 1972; Jabaley *et al.*, 1975; Turvey, 1977; Luce *et al.*, 1979; Steidler *et al.*, 1980; Holt & Holt, 1983). Most studies which have been carried out have been retrospective and the details concerning the techniques of ophthalmic examination employed have not been described. Prospective studies have included only small numbers of patients (Miller & Tenzel, 1967).

This prospective study was set up to determine the incidence and types of ocular and motility disorders, as assessed by the opthalmologist, in patients who had sustained midfacial fractures and who were under the care of a maxillofacial surgeon.

PATIENTS AND METHODS

The Oral and Maxillofacial Unit at Canniesburn Hospital, Glasgow, Scotland is a regional centre. It undertakes the care and treatment of maxillofacial patients for about one third of the West of Scotland, which has a total population of three and a half million people. Patients who had sustained midfacial or frontal bone trauma and who were treated at Canniesburn Hospital between 1 April 1985 and 31 March 1987, were admitted to this study. All patients with persistent pathology were followed up for at least 1 year. Canniesburn surgeons are also involved in the treatment of craniofacial fractures in the regional neurosurgical unit at the Southern General Hospital, but these patients (42 per annum on average) were included in the study only if they had been transferred to or followed up in Canniesburn Hospital. Patients with simple nasal or mandibular fractures were not included in the study.

Every effort was made to ensure the referral to the eye clinic of all patients of the above description irrespective of whether or not they had presented with manifest eye injuries. Patients who had sustained major trauma were evaluated ophthalmologically within 48 h following injury, and those in whom there was no manifest ocular disorder (as judged by the attending maxillofacial surgeon) were evaluated at the ensuing weekly opthalmic clinic. A full history of the incident including time, cause, related factors (such as influence of alcohol and the use of seat belts) and whether there was any trauma, was taken. The influence of alcohol was assessed by virtue of the amount of alcohol consumed as indicated by the patient (more than 2 units during the hour preceding the incident) or the patient's clinical status on first examination. A detailed past ophthalmic history such as errors of refraction, squint, amblyopia, eye surgery and previous eye injuries was obtained. The occurrence of amnesia, its type and duration was recorded (Teasdale & Jennett, 1974; Jennett & Teasdale, 1981). Subjective symptoms of diplopia and their duration, as well as pain on eye movements, were noted.

Facial fractures were classified by clinical and radiological examination into three groups: malar fractures according to Henderson (1973, unpublished) (Table 1); maxillary fractures as described by Le Fort (1900); and nasal, naso-orbito-ethmoidal (NOE) and frontal sinus fractures (Converse & Smith, 1963; Stranc, 1970). Computerised tomographic (CT) scanning was performed on all patients with clinical evidence of a blow-out fracture of the orbit but with negative plain radiographs, except in those few in which exploration of the orbit was deemed necessary.

A detailed assessment of eye structure and function was carried out by an ophthalmologist and an orthoptist in all cases at each visit. All data accruing from each opthalmic consultation was divided into three categories:

Table 1 – Henderson's classification of malar fractures

Type	Description of fracture
I	Undisplaced fracture, any site
II	Zygomatic arch fracture only
III	Tripod fracture with undistracted fronto-zygomatic suture
IV	Tripod fracture with distracted fronto-zygomatic suture
V	Pure blow-out fracture of the orbit
VI	Fracture of the orbital rim only
VII	Comminuted fracture or other than the above

1. General: All routine information.
2. Eye abnormality: All ocular injuries sustained and their functional consequences, complications, treatment and recovery.
3. Motility disorder: Examination of ocular motility comprised assessment of movement in all directions of gaze and the elicitation of diplopia. All motility abnormalities were quantified by means of the prism cover test, the synoptophore, the field of binocular single vision and/or the Hess chart as appropriate for the type of motility disorder detected.

Enophthalmos was assessed using the Keeler frame in those patients in whom the orbital margin was intact. Examination from above with careful delineation of the positions of the globe with respect to the supra-orbital margin were carried out in all other patients.

Table 3 provides a detailed classification of ophthalmic disorders according to their severity. All data were coded and stored in the mainframe university computer for future analysis. Each patient was discharged when ocular pathology became quiescent and of no further risk to the patient.

RESULTS

Types of injury

The study included 363 patients with confirmed midface fractures who presented during a 2-year period. They had sustained a total of 438 midfacial fractures. The majority of fractures involved the malar bone either in isolation (280) or in combination with other midface fractures (61). Isolated fractures of the Le Fort type had been sustained by only 6 patients, while 29 others sustained Le Fort fractures in combination with various other midfacial fractures. Ten patients suffered an isolated NOE fracture while 3 others sustained a frontal bone fracture. Altogether, 64 of the 363 patients sustained fractures of two or more facial bones.

Patients having midfacial fractures ranged in age from an 8-year-old boy who suffered a blow-out fracture when he hit a broken door handle with his

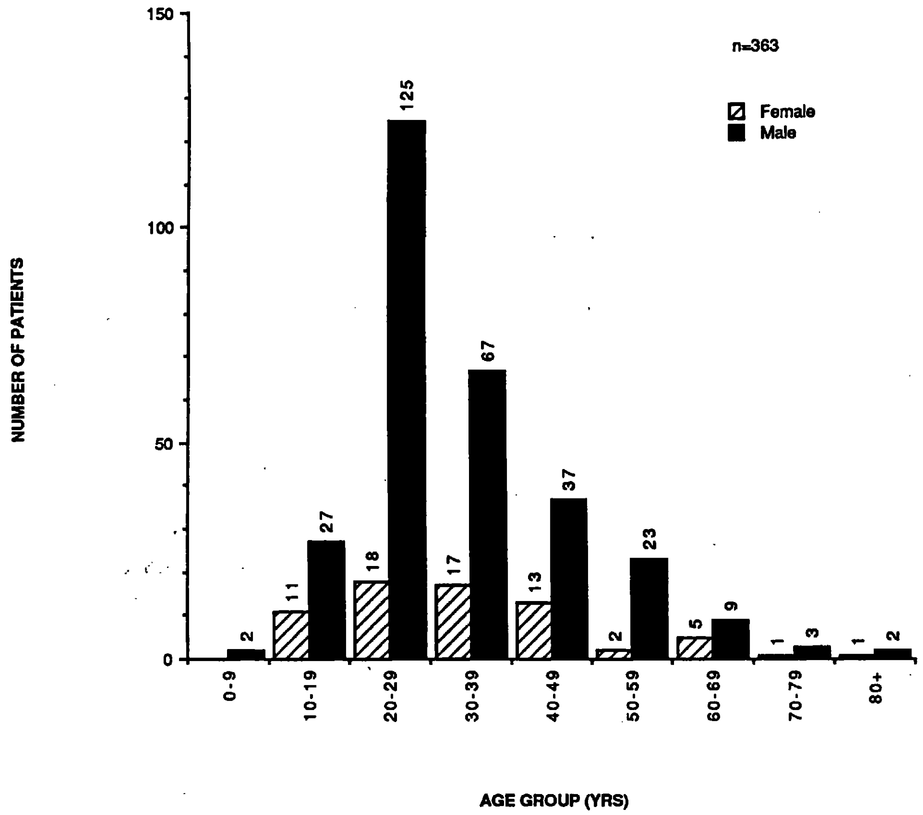


Fig. 1–Age and sex distribution of the population of the study.

closed left eye, to an 84-year-old woman who sustained a tripod malar fracture (Type III), in a fall. Males accounted for 81·3% (n=295) of all midfacial fracture patients, and females, 18·7% (n=68). The majority of patients were males between the ages of 10 and 50 years with the peak incidence occurring in the 20 to 30 year age group for both sexes (Fig. 1). In women, midfacial fractures were more prevalent in older age groups (60–80 years) than they were in men. The monthly distribution of incidence of injury showed June to be the month in which the greatest percentage of midfacial trauma occurred (14·3%), followed by March (12·4%), and May and September (9·1% each). June was the month in which most of the assaults, road traffic accidents (RTA) and sporting accidents took place.

The major cause of midfacial trauma in this study was alleged assault, which accounted for 49·9% of all patients (181/363). Some of these allegedly assaulted individuals (n=85) were under the influence of alcohol when they sustained their trauma while others (n=96) were not. The second most common cause was falls (19% or 69 patients), of whom 24 (6·6%) had been under the influence of alcohol, followed by RTA and sporting accidents which affected 45 patients (12·4%) each. Figure 2 shows the frequency distribution of the causes of midfacial fractures in males and females. Alleged assault was more common in males (50·9% of 295) than in females (45·6% of 68) while falls were more frequent in females (33·8%) than in males (15·6%). RTA were also a more common cause of midfacial trauma in females (17·6%) than in males (11·2%). Sporting accidents, on the other hand, were more than 10-fold more common in men (14·9% of 295) than in women (1·5% of 68). RTA were responsible for the injuries of 45 (12·4%) of our 363 patients. Within this group, 4 patients, all male, were involved in car accidents while not wearing seat belts, 16 in car accidents while wearing seat belts, 8 while sitting in a back seat of a car, 9 in motorcycle-related accidents and 8 in motor vehicle–pedestrian or other motor vehicle–related accidents. Industrially-related accidents leading to midfacial fractures occurred only in males (n=13).

Figure 3 shows the distribution of midfacial fractures among the population studied. The most common anatomical type of malar fracture was the tripod zygomatico-orbital fracture with no distraction of the fronto-zygomatic (F–Z) suture (Type III) which affected 34·2% (n=124) of the patients, followd by tripod fractures with separation of the F–Z suture (Type IV) in 25·1% (n=91) of the patients. Undisplaced zygomatic fractures (Type I) were third in prevalence with 11·6% (n=42). Less frequent fractures were isolated blow-out fractures (Type V), (9·9%, n=36) and comminuted fractures (Type VII) which affected 27 patients (7·4%). Twelve patients (3·3%) sustained fractures of the zygomatic arch only (Type II), while another 8 patients (2·2%) suffered orbital rim fracture only (Type VI). Table 2 demonstrates the different anatomical types of malar fractures in relation to the causal factors. In all causal groups except motorcycle

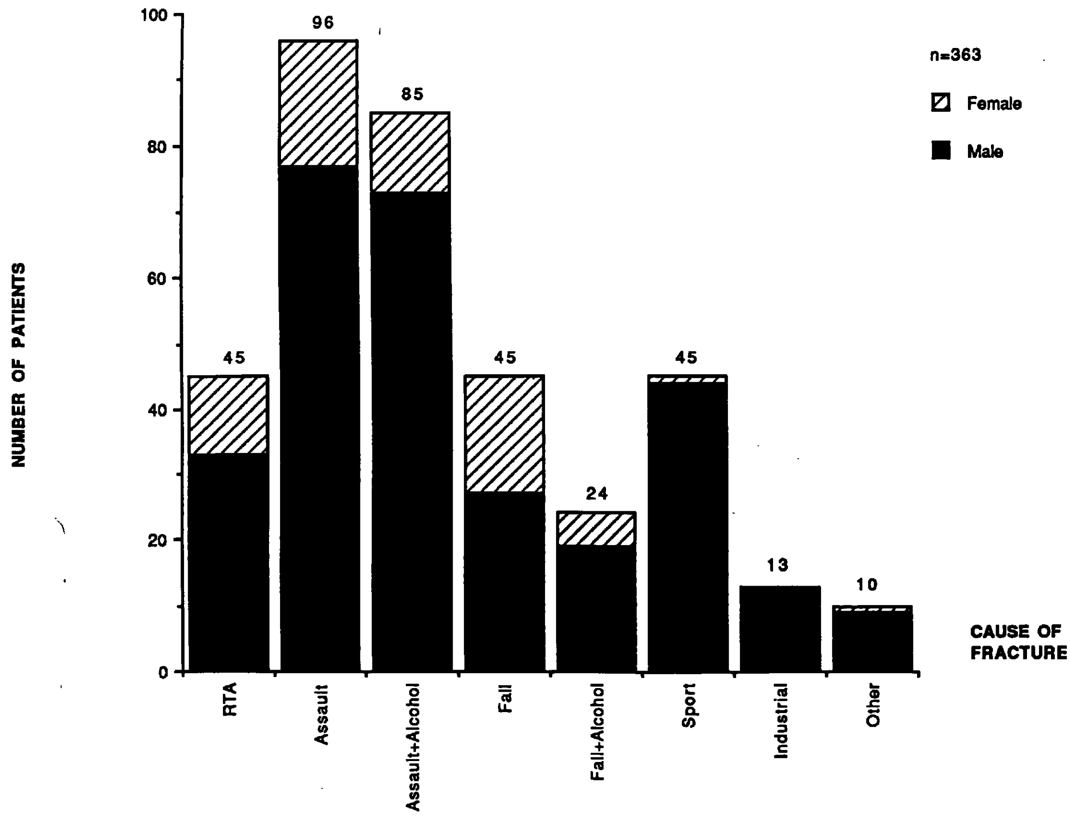


Fig. 2–The aetiological distribution of midfacial fractures.

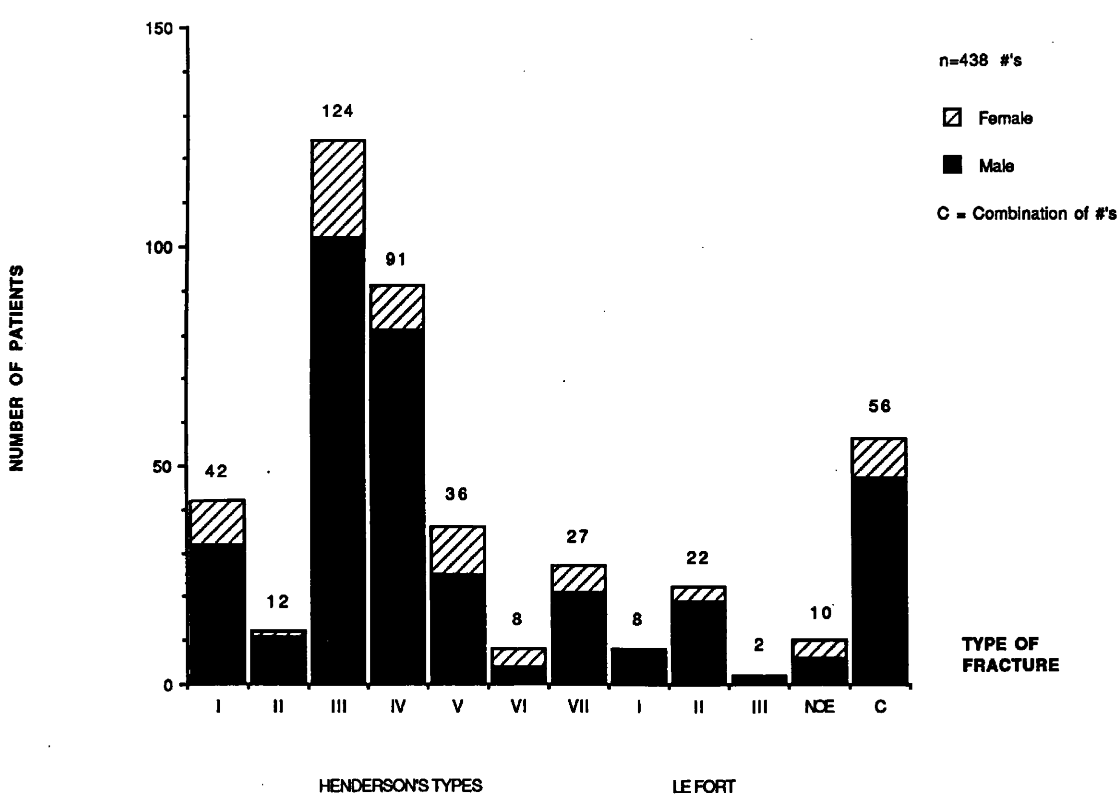


Fig. 3–The site distribution of midfacial fractures. C=combination of fractures.

Table 2 – The different types of malar fracture in relation to the causal factors

	Number of patients in each category of malar fracture								
Cause of injury	I	II	III	IV	V	VI	VII	Other #'s	All (%)
Car (no seat belt)	1	0	1	1	0	0	0	1	4 (1.1)
Car (seat belt on)	1	0	5	4	1	0	2	3	16 (4.4)
Car (back seat)	0	0	2	2	0	0	1	3	8 (2.2)
Motorcycle	0	0	1	3	1	0	1	3	9 (2.5)
Other RTA and pedestrians	0	0	2	3	1	0	2	0	8 (2.2)
Alleged assault	10	5	39	18	10	5	6	3	96 (26.5)
Assault under alcohol	17	3	19	26	9	1	6	4	85 (23.4)
Fall	2	1	19	14	6	0	2	1	45 (12.4)
Fall under alcohol	4	0	7	8	1	1	1	2	24 (6.6)
Sporting accidents	5	3	21	8	3	1	4	0	45 (12.4)
Industrial accidents	2	0	3	3	1	0	2	2	13 (3.6)
Blackout	0	0	1	0	0	0	0	0	1 (0.3)
Epilepsy	0	0	1	0	0	0	0	1	2 (0.6)
Other causes	0	0	3	1	3	0	0	0	7 (1.9)
All: (no)	42	12	124	91	36	8	27	23	363
(%)	11.6%	3.3%	34.2%	25.1%	9.9%	2.2%	7.4%	6.3%	100%

Other #'s=fractures other than malar's.

injuries and motor vehicle–pedestrian related accidents, tripod fracture with no distraction of the F–Z suture (Type III) was the most common type of zygomatico–orbital fracture. Motorcycle injuries and those related to pedestrians resulted in a larger percentage of tripod fractures in which the F–Z sutures were distracted (Type IV).

Amnesia

Amnesia was common in association with midfacial trauma. One hundred and three patients (28·4% of 363) suffered from amnesia, which was more common in males (89/295=30·2%) than in females (14/68=20·6%). Twenty two males and 5 females suffered retrograde amnesia following their injuries, while 51 males and 6 females suffered the post-traumatic type of amnesia. Only 19 patients, 16 males and 3 females, suffered a combination of both types of amnesia. Amnesia was often associated with comminuted malar fractures (12/27=44·4%) and was most common with RTA (34/45=75·6%), particularly when no seat belt was worn (4/4=100%), and least common in association with sporting accidents where only one individual was affected.

Ocular injuries

Enophthalmos following midface fracture occurred in 8% of the patients (n=29) most of whom were male (n=23). The majority of patients were between the ages of 10 and 40 (n=24) with the peak incidence occurring in the 20- to 30-year age group (n=12). Nine of these 29 patients had also suffered an associated downward displacement of the globe which was not fully corrected by surgery in 1 case only. All patients with enophthalmos had sustained either a pure (Type V; n=17) or an impure (Type IV or VII with floor defect; n=12) blow-out fracture of the orbit which was associated with soft tissue entrapment in 13 cases while the rest of patients (n=16) had significant displacement of the orbital contents into the antrum but no functional evidence of entrapment. Eighteen of these 29 patients (62%) suffered infra-orbital nerve anaesthesia or paraesthesia. Enophthalmos was most common with RTA (4/45=8·9%) and with assaults (16/181=8·8%) followed by simple falls (4/69=5·8%); and least common following sporting accidents (1/45=2·2%).

Motility disorder was common in association with enophthalmos. Twenty three of 29 patients (79·3%) exhibited symptoms and/or signs of ocular motility abnormality manifested by some limitation of ocular movement in one or more directions of gaze. Of these 23 patients, 6 complained of diplopia which was close to the midline, and 15 were symptomatic only in the extreme gaze. The other 2 patients had amblyopic (lazy) eyes and therefore did not appreciate diplopia.

Ocular injuries were common. Table 3 illustrates the incidence of these eye abnormalities, according to their severity, in the population of the study. Tables 4 and 5 illustrate the same in relation to the type of midfacial fracture sustained, while Table 6 relates these ocular abnormalities to the cause of injury. The right eye was affected in 152 patients (152/363=41·9%), of whom 125 were male (125/295=42·4%) and 27 were female (27/68=39·7%). The left eye was involved in 153 cases (42·2%), 120 males (42·1%) and 33 females (48·5%). In 33 patients (9·1%), 29 males (9·8%) and 4 females (5·9%) both eyes were injured while the rest of the patients, 21 males and 4 females, sustained no direct ocular trauma whatsoever. Figure 4 illustrates the frequency distribution of ocular injuries in relation to their severity for both males and females. Only 34 patients (9·4%) did not exhibit any detectable eye injury at all. Most patients (63·4%, n=230) suffered only minor or transient ocular injuries such as lid bruise, sub-conjunctival haemorrhage and mild impairment of accommodation. Fifty seven patients (15·7%) suffered moderately severe ocular injuries which were unlikely to have led to permanent visual or physiological sequelae. Examples include minor eyelid and conjunctival lacerations and enoph-

Table 3 – Incidence of ophthalmic disorder in the population of study

Eye abnormality	Male (n=295) no. (%)	Female (n=68) no. (%)	All (n=363) no. (%)
Mild ophthalmic disorders			
Coronal eye displacement	24 (8.1)	5 (7.4)	29 (8.0)
Orbital emphysema	4 (1.4)	0 (—)	4 (1.1)
Eyelid swelling/bruising	198 (67.1)	54 (79.4)	252 (69.4)
Subconjunctival haemorrhage/chemosis	200 (67.8)	52 (76.5)	252 (69.4)
Corneal abrasion	0 (—)	0 (—)	0 (—)
Mild impairment of accommodation	5 (1.7)	1 (1.5)	6 (1.7)
Mild reduction of visual acuity (6/9–6/12)	20 (6.8)	9 (13.2)	29 (8.0)
Mild commotio retinae	16 (5.4)	8 (11.8)	24 (6.6)
Moderate ophthalmic disorders			
Enophthalmos	23 (7.8)	6 (8.8)	29 (8.0)
Eyelid laceration	10 (3.4)	0 (—)	10 (2.8)
Conjunctival laceration	0 (—)	0 (—)	0 (—)
Traumatic pupillary changes	6 (2.0)	3 (4.4)	9 (2.5)
Iridodialysis	0 (—)	0 (—)	0 (—)
Lens damage	1 (0.3)	0 (—)	1 (0.3)
Macular oedema	9 (3.1)	4 (5.9)	13 (3.6)
Moderate to severe impairment of accommodation	5 (1.7)	1 (1.5)	6 (1.7)
Moderate reduction of visual acuity (6/18–6/24)	10 (3.4)	5 (7.4)	15 (4.1)
Moderate to severe commotio retinae	2 (0.7)	0 (—)	2 (0.6)
Choroidal tear(s) not involving the macula	2 (0.7)	2 (2.9)	4 (1.1)
Vitreous floater/traumatic pigmentary retinopathy	7 (2.4)	1 (1.5)	8 (2.2)
Nasolacrimal damage	5 (1.7)	2 (2.9)	7 (1.9)
Severe ophthalmic disorders			
Gross proptosis	5 (1.7)	6 (8.8)	11 (3.6)
Retrobulbar haemorrhage	2 (0.7)	0 (—)	2 (1.5)
Corneal laceration	0 (—)	1 (1.5)	1 (0.3)
Hyphaema	4 (1.4)	1 (1.5)	5 (1.4)
Angle recession	7 (2.4)	1 (1.5)	8 (2.2)
Severe reduction/loss of vision (6/36 or less)	9 (3.1)	3 (4.4)	12 (3.3)
Visual field loss	7 (2.4)	1 (1.5)	8 (2.2)
Choroidal tear involving the macula	0 (—)	0 (—)	0 (—)
Vitreous haemorrhage or retinal tear(s)/detachment	8 (2.7)	1 (1.5)	9 (2.5)
Optic nerve injury	7 (2.4)	2 (2.9)	9 (2.5)

Table 4 – Incidence of ophthalmic disorder in relation to malar fractures

Eye abnormality	Type I (n=42) no. (%)	Type II (n=12) no. (%)	Type III (n=124) no. (%)	Type IV (n=91) no. (%)	Type V (n=36) no. (%)	Type VI (n=8) no. (%)	Type VII (n=27) no. (%)	All (n=341) no.
Reduced visual acuity	4 (9.5)	0 (—)	14 (11.3)	16 (17.6)	8 (22.2)	1 (12.5)	11 (40.7)	54
Visual field loss	0 (—)	0 (—)	3 (2.4)	3 (3.3)	0 (—)	0 (—)	1 (3.7)	7
Impaired accommodation	4 (9.5)	0 (—)	4 (3.2)	3 (3.3)	1 (2.8)	0 (—)	0 (—)	12
Eyelid injury	1 (2.4)	7 (58.3)	90 (72.6)	69 (75.8)	28 (77.8)	7 (87.5)	24 (88.9)	226
Nasolacrimal damage	0 (—)	0 (—)	1 (0.8)	2 (2.2)	0 (—)	0 (—)	2 (7.4)	5
Eye displacement	1 (2.4)	0 (—)	3 (2.4)	11 (12.1)	8 (22.2)	0 (—)	5 (18.5)	28
Proptosis	2 (4.8)	0 (—)	1 (0.8)	4 (4.4)	2 (5.6)	0 (—)	2 (7.4)	11
Enophthalmos	1 (2.4)	0 (—)	0 (—)	7 (7.7)	17 (47.2)	0 (—)	4 (14.8)	29
Orbital emphysema	1 (2.4)	0 (—)	0 (—)	0 (—)	3 (8.3)	0 (—)	0 (—)	4
Conjunctival injury	26 (61.9)	5 (41.7)	86 (69.4)	68 (74.7)	28 (77.8)	6 (75.0)	23 (85.2)	242
Corneal damage	0 (—)	0 (—)	1 (0.8)	0 (—)	0 (—)	0 (—)	0 (—)	1
Pupillary changes	0 (—)	0 (—)	3 (2.4)	2 (2.2)	2 (5.6)	0 (—)	1 (3.7)	8
Iris damage	2 (4.8)	0 (—)	1 (0.8)	2 (2.2)	0 (—)	0 (—)	0 (—)	5
Angle recession	1 (2.4)	0 (—)	1 (0.8)	4 (4.4)	1 (2.8)	0 (—)	1 (3.7)	8
Lens damage	0 (—)	0 (—)	1 (0.8)	0 (—)	0 (—)	0 (—)	0 (—)	1
Macular oedema	1 (2.4)	0 (—)	2 (1.6)	5 (5.5)	2 (5.6)	0 (—)	4 (14.8)	14
Commotio retinae	3 (7.1)	0 (—)	5 (4.0)	8 (8.8)	5 (13.9)	1 (12.5)	3 (11.1)	25
Retinal/vitreous damage	0 (—)	0 (—)	2 (1.6)	6 (6.6)	0 (—)	1 (12.5)	3 (11.1)	12
Choroidal injury	0 (—)	0 (—)	0 (—)	1 (1.1)	1 (2.8)	1 (12.5)	1 (3.7)	4
Optic nerve injury	0 (—)	0 (—)	3 (2.4)	2 (2.2)	1 (2.8)	0 (—)	2 (7.4)	8
Retrobulbar haemorrhage	0 (—)	0 (—)	0 (—)	0 (—)	1 (2.8)	0 (—)	1 (3.7)	2
Total number of eye abnormalities								706

Table 5 – Incidence of ophthalmic disorder in relation to Le Fort and NOE fractures

Eye abnormality	Le Fort I (n=8)	Le Fort II (n=22)	Le Fort III (n=2)	NOE (n=10)
Reduced visual acuity	1	7	0	1
Visual field loss	0	1	0	0
Impaired accommodation	0	0	0	0
Eyelid injury	6	17	1	7
Nasolacrimal damage	0	2	0	2
Eye displacement	1	6	0	3
Proptosis	0	1	0	1
Enophthalmos	1	3	0	1
Orbital emphysema	0	0	0	0
Conjunctival injury	5	16	1	7
Corneal damage	0	0	0	0
Pupillary changes	1	1	1	0
Iris damage	0	0	0	0
Angle recession	0	1	0	0
Lens damage	0	0	0	0
Macular oedema	0	2	0	1
Commotio retinae	0	3	0	0
Retinal/vitreous damage	0	1	0	0
Choroidal injury	0	0	0	0
Optic nerve injury	0	3	0	0
Retrobulbar haemorrhage	0	0	0	0

Le Fort type fractures were isolated in only 6 cases and the rest were combined with other midfacial fractures.

thalmos. Forty two patients (11·6%) sustained severe eye injuries such as angle recession, retinal or vitreous injury, or optic nerve damage. Of those 42 patients, 9 (2·5%) lost vision in the affected eye.

Severe ocular disorder was more common in females (14·7%) than in males (10·8%). However, the difference was not found to be statistically significant. The severity of ocular abnormality was not found to be significantly related to age or to season. Severe ocular disorder was most common following RTA (Fig. 5). Nine of 45 patients (20%) with confirmed midfacial fractures due to RTA suffered a severe ocular abnormality. Assaults were associated with 11% of severe ocular abnormalities (20/181) and falls with 7·2% (5/69) whilst only 4·4% (2/45) of those individuals who were involved in sporting accidents sustained severe ocular injury. Industrial accidents were associated with a 23% rate of severe eye abnormality (3/13), however, the size of this group is too small to allow accurate interpretation. RTA caused the highest proportion of impaired vision (28·9%), ($0{\cdot}001<p<0{\cdot}01$), while simple falls caused the lowest proportion (5·8%), ($0{\cdot}01<p<0{\cdot}02$). Other causes of facial trauma such as assaults, sports and industrial accidents were associated with intermediate frequencies of reduced vision (15%).

Figure 6 demonstrates the relationship of ocular injury categories to the different types of malar fracture sustained. One third of all patients with comminuted malar fracture (Type VII) suffered a severe ocular disorder (9/27=33·3%) while blow-out fractures (Type V) came second (6/36=16·7%). No severe ocular disorders were observed with fractures of the zygomatic arch only (Type II).

Fifty six of 363 patients (15·4%) had suffered transient or permanent visual loss. Causes of impaired vision varied from minor, self-healing corneal injuries to optic nerve avulsion. Permanent loss of vision ensued in 8 cases and was due to traumatic optic neuropathy in each case. The type of facial fracture was found to be an important factor in relation to visual deterioration. Comminuted malar fractures were associated with a 41% incidence of visual impairment in contrast to 15% for the whole series ($p<0{\cdot}001$). Visual loss occurred most frequently in the 30- to 40-year age group ($0{\cdot}02<p<0{\cdot}05$) and least frequently in the 20- to 30-year age group patients ($p<0{\cdot}001$). Females were significantly more likely to suffer visual loss than males of the same age group ($0{\cdot}01<p<0{\cdot}02$).

Table 6 – Incidence of ophthalmic disorder in relation to the aetiology of midfacial trauma

Eye abnormality	RTA (n=45) no. (%)	Assault (n=181) no. (%)	Fall (n=69) no. (%)	Sport (n=45) no. (%)	Industrial (n=13) no. (%)	Other (n=10) no. (%)	All (n=363) no. (%)
Reduced visual acuity	13 (28.9)	29 (16.0)	4 (5.8)	5 (11.1)	3 (23.1)	2 (20.0)	56 (15.4)
Visual field loss	4 (8.9)	1 (0.6)	1 (1.4)	0 (—)	1 (7.7)	1 (10.0)	8 (2.2)
Impaired accommodation	0 (—)	10 (5.5)	1 (1.4)	0 (—)	0 (—)	1 (10.0)	12 (3.3)
Eyelid injury	30 (66.7)	138 (76.2)	48 (69.6)	29 (64.4)	9 (69.2)	8 (80.0)	262 (72.2)
Nasolacrimal damage	4 (8.9)	2 (1.1)	1 (1.4)	0 (—)	0 (—)	0 (—)	7 (1.9)
Eye displacement	6 (13.3)	13 (7.2)	5 (7.2)	1 (2.2)	3 (23.1)	1 (10.0)	29 (8.0)
Proptosis	1 (2.2)	6 (3.3)	3 (4.3)	0 (—)	0 (—)	1 (10.0)	11 (3.0)
Enophthalmos	4 (8.9)	16 (8.8)	4 (5.8)	1 (2.2)	2 (15.4)	2 (20.0)	29 (8.0)
Orbital emphysema	0 (—)	1 (0.6)	0 (—)	1 (2.2)	0 (—)	2 (20.0)	4 (1.1)
Conjunctival injury	31 (12.3)	138 (76.2)	40 (58.0)	26 (57.8)	8 (61.5)	9 (90.0)	252 (69.4)
Corneal damage	0 (—)	0 (—)	0 (—)	0 (—)	0 (—)	1 (10.0)	1 (0.3)
Pupillary changes	4 (8.9)	2 (1.1)	1 (1.4)	2 (4.4)	0 (—)	0 (—)	9 (2.5)
Iris damage	1 (2.2)	3 (1.7)	0 (—)	0 (—)	0 (—)	1 (10.0)	5 (1.4)
Angle recession	0 (—)	5 (2.8)	0 (—)	1 (2.2)	2 (15.4)	0 (—)	8 (2.2)
Lens damage	0 (—)	1 (0.6)	0 (—)	0 (—)	0 (—)	0 (—)	1 (0.3)
Macular oedema	6 (13.3)	5 (2.8)	2 (2.9)	1 (2.2)	0 (—)	0 (—)	14 (3.9)
Commotio retinae	4 (8.9)	16 (8.8)	2 (2.9)	3 (6.7)	0 (—)	1 (10.0)	26 (7.2)
Retinal/vitreous damage	4 (8.9)	5 (2.8)	0 (—)	1 (2.2)	1 (7.7)	1 (10.0)	12 (3.3)
Choroidal injury	2 (4.4)	1 (0.6)	0 (—)	1 (2.2)	0 (—)	0 (—)	4 (1.1)
Optic nerve injury	3 (6.7)	2 (1.1)	2 (2.9)	0 (—)	1 (7.7)	1 (10.0)	9 (2.5)
Retrobulbar haemorrhage	0 (—)	1 (0.6)	0 (—)	0 (—)	0 (7.7)	1 (10.0)	2 (0.6)
Total number of eye abnormalities	117	395	114	72	30	33	761

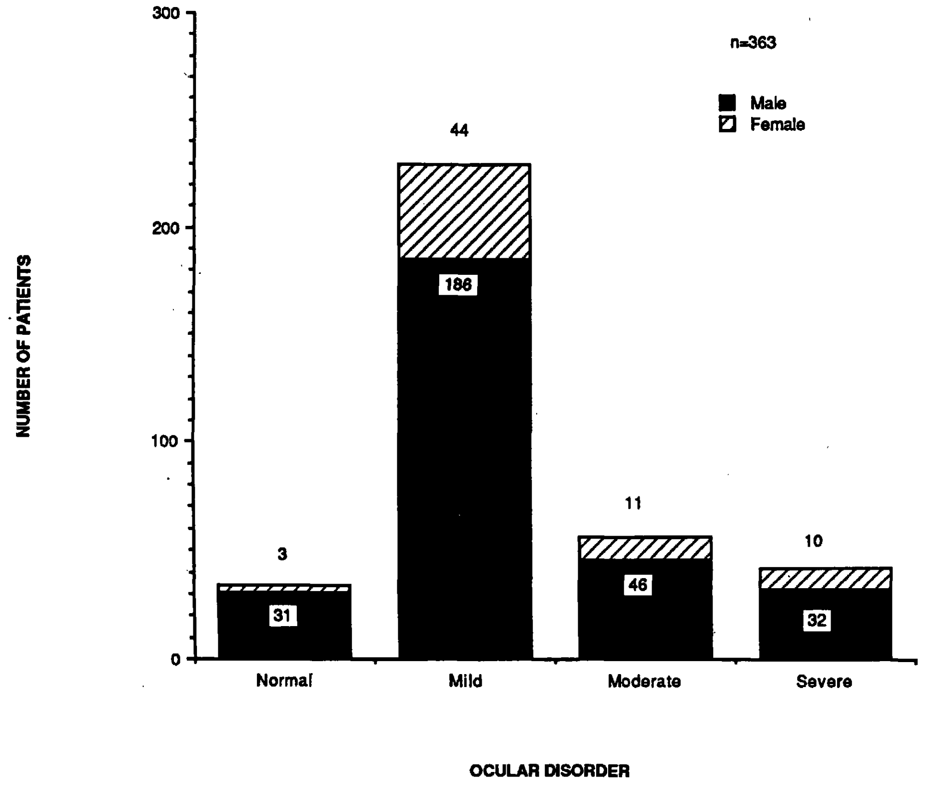


Fig. 4–Grades of severity of ocular disorder in male and female.

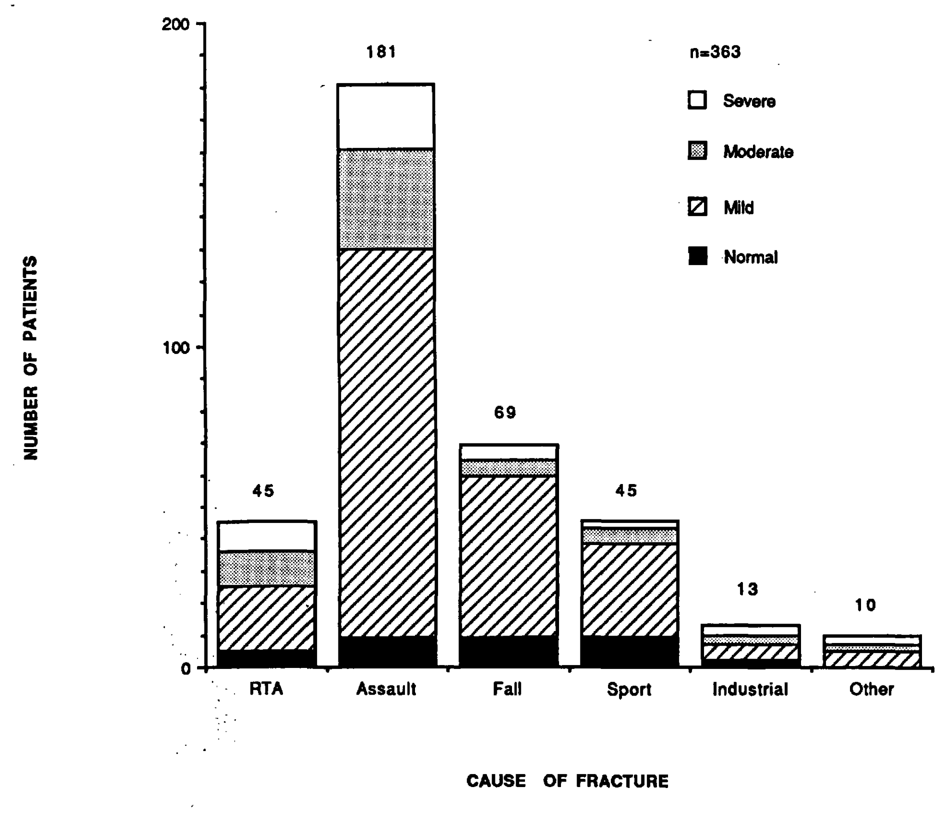


Fig. 5–Ocular disorder, according to severity, in relation to aetiology of facial fracture.

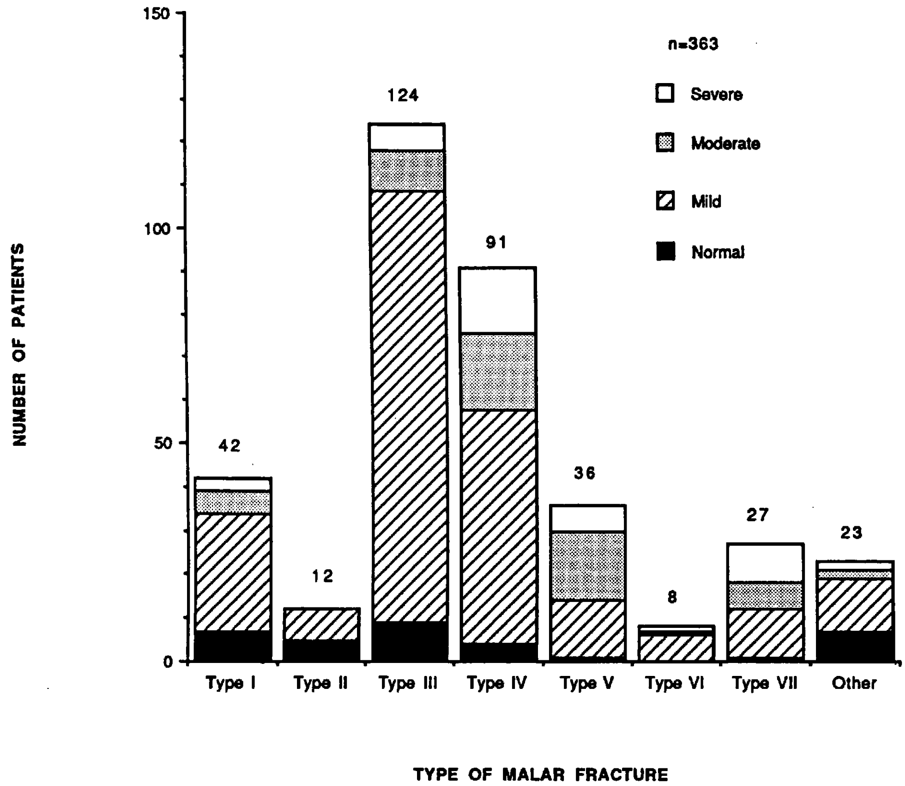


Fig. 6–Ocular disorder, according to severity, in relation to type of malar fracture.

DISCUSSION

This report is the largest prospective survey of the ocular sequelae of midfacial fracture currently available. Ocular injury of varying severity is a common sequel to midfacial fracture (Paton & Emery, 1973). The severity of the facial fractures is dependent upon the nature of the injury and is directly related to the likelihood and severity of trauma to the eye. Disruption of the orbital walls deprives the eye and its adnexae from the protection they are normally afforded (Jabaley *et al.*, 1975). In the present study, patients with the most severe head or facial injuries warranting neurosurgical intervention are managed in a separate unit and have not been included in this report. It can be anticipated, however, that such individuals are highly susceptible to injury of the visual system. Disturbance of ocular motility was also a common sequel. The details of the motility disorders seen in this series of patients are described separately (Al-Qurainy *et al.*, 1991a).

The major cause of maxillofacial trauma in this study was alleged assault. The patients assessed were from a large industrialised area with a high rate of unemployment (Ellis *et al.*, 1985). This may be related to the fact that the majority of injured patients were young adult males who sustained their injuries in alleged assaults. This cause of fracture and type of patient correspond to the results found in comparable studies (Lundin *et al.*, 1973; Altonen *et al.*, 1976). Although alleged assaults were more common in males than in females, the difference was not found to be statistically significant. Falls, however, were more common in females than in males ($0{\cdot}01<p<0{\cdot}001$). In general, fractures were almost equally shared between both sides of the face. However, fractures sustained due to assaults were predominantly left-sided which relates with the fact that the majority of people are right-handed. This finding is in agreement with the investigations of Hitchin and Shuker (1973), Altonen *et al.* (1976) and Muller and Schoeman (1977), who also found that assault victims displayed a predominance of left-sided fractures. June was the month in which the greatest incidence of injury occurred. This, probably, is expected in a summer month where most of the assaults, RTA and sporting events take place.

It was interesting to find that patients who sustained a head injury severe enough to cause amnesia were more likely to suffer a disturbance to their visual system. As expected, amnesia was most common following RTA. The protective effect of seat belts is evidenced by the fact that all unrestrained patients developed concussion manifested by amnesia.

Traumatic enophthalmos is usually due to enlargement of the orbital cavity subsequent to a floor or, less commonly, medial wall fracture (Converse & Smith, 1956); although dislocation of the trochlea, atrophy of orbital fat, cicatricial contraction of retrobulbar tissue and rupture of orbital ligaments or fascial bands have been previously incriminated (Benedict, 1936). Pfeiffer (1943) reviewed 53 patients with traumatic enophthalmos following facial bone fractures and noted orbital bone involvement in each case. Of these, 24 patients sustained pure blow-out fractures which were due to assaults in 58% (n=14), and RTA in 17% (n=4) of cases. Our figures are closely in accordance with those of Pfeiffer. Enophthalmos following facial trauma should always raise suspicion of an orbital wall fracture and a CT scan is advisable when plain X-rays are negative.

There is a number of reports of large series of facial fractures in which associated injuries were reported (Gwyn *et al.*, 1971; Morgan *et al.*, 1972; Steidler *et al.*, 1980) but most of these reports only mentioned briefly or fail to characterise specific ocular complications. The very low incidence of ophthalmic injury reported by non-ophthalmic authors (McCoy *et al.*, 1962; Converse *et al.*, 1967; Schultz, 1967; Gwyn *et al.*, 1971; Morgan *et al.*, 1972; Turvey, 1977; Luce *et al.*, 1979) probably indicates that significant ophthalmic pathology may have gone undetected. On the other hand, the low incidence of ophthalmic injury reported by some ophthalmic surgeons (Milauskas & Fueger, 1966; Miller & Tenzel, 1967) may indicate that only the more serious ocular injuries had been reported while mild and less serious injuries were not mentioned.

The overall incidence of ophthalmic injury of any severity (including sub-conjunctival haemorrhage) following maxillofacial trauma in the present series is 90·6% (329/365). This figure is much higher than those previously published in the literature, including the 67% rate reported by Holt *et al.* (1983). However, Holt's series was retrospective, included only 51% of facial fracture patients potentially available for assessment and provided limited follow-up documentation. It was therefore selective on the part of the attending maxillofacial surgeon and allowed no definite conclusions to be drawn concerning the possibility of undetected eye injuries in the patients who did not have formal eye examination.

The results of this study reinforce the contention that RTA cause more severe ocular injuries than any other cause of facial trauma (Petro *et al.*, 1979; Holt *et al.*, 1983). Similarly, comminuted malar fractures were associated with the highest incidence of visual dysfunction.

Fortunately, many of the ocular injuries were transient and of no permanent consequence. However, the incidence of 11·6% (42/363) of patients with serious or blinding injuries is significant. Of particular importance are those cases of optic nerve contusion or compression (2·5%). Total loss of vision in quite healthy looking eyes is not uncommon following facial trauma. Ketchum *et al.* (1976) presented 2 cases of bilateral blindness without direct injury to the globe and postulated that a haematoma, ischaemia, or direct bone fragment penetration of the optic nerve may have been the cause. In the light of the more recent study by Anderson *et al.* (1982) illustrating the proper timing of surgical decompression of the optic nerve and response to high dose corticosteroid therapy, such injuries may deserve immediate and aggressive management especially if the initial examination demonstrates some vision immediately following the injury.

The results of the eye examination, while they do not often alter the type of fracture repair, may influence the indications and timing of the repair because treatment of certain ocular injuries, such as optic nerve compression, must be instituted at once. The proper time for an eye examination, especially visual acuity testing, is at the time of initial assessment of the patient's trauma. We suggest that all patients sustaining midfacial fracture associated with a significant decrease in visual acuity either pre- or postoperatively should have an early ophthalmological review. Many patients sustaining facial injury make recourse to legal or insurance claims. The results of detailed ophthalmic assessment often add weight to such cases and affect levels of compensation paid.

The data accruing from this study have been subjected to discriminant function analysis and has resulted in the development of a scoring system which allows the maxillofacial surgeon to determine which patients warrant ophthalmic referral and with what urgency (Al-Qurainy *et al.*, 1991b.)

References

Al-Qurainy, I. A., Stassen, L. F. A., Dutton, G. N., Moos, K. F. & El-Attar, A. (1991a). Diplopia following midfacial fractures. *British Journal of Oral and Maxillofacial Surgery*, **29**, 302.

Al-Qurainy, I. A., Titterington, D. M., Dutton, G. N., Stassen, L. F. A., Moos, K. F. & El-Attar, A. (1991b). Midfacial fractures and the eye: the development of a system for detecting patients at risk of eye injury. *British Journal of Oral and Maxillofacial Surgery*, **29**, In press.

Altonen, M., Kohonen, A. & Dickhoff, K. (1976). Treatment of zygomatic fractures: internal wiring-antral packing-reposition without fixation. A comparative study. *Journal of Maxillo-Facial Surgery*, **4**, 107.

Anderson, R. L., Panie W. R. & Gross, C. E. (1982). Optic nerve blindness following blunt forehead trauma. *Ophthalmology*, **89**, 445.

Babajews, A. & Williams, J. Ll. (1986). Blindness after trauma insufficient to cause bony injury: Case report and review. *British Journal of Oral and Maxillofacial Surgery*, **24**, 7.

Barclay, T. L. (1958). Diplopia in association with fractures involving the zygomatic bone. British Journal of Plastic Surgery, **11**, 147.

Beirne, O. R., Schwartz, H. C. & Leake, D. L. (1981). Unusual ocular complications in fractures involving the orbit. *International Journal of Oral Surgery*, **10**, 12.

Benedict, W. L. (1936). Diseases of the orbit. In: *The Eye and Its Diseases*, C. Berens (ed.), p. 330. Philadelphia: W. B. Saunders.

Binder, P. S. (1978). Evaluation of the eye following periorbital trauma. *Head and Neck Surgery*, **1**, 139.

Converse, J. M. & Smith, B. (1956). Enophthalmos and diplopia in fractures of the orbital floor. *British Journal of Plastic Surgery*, **9**, 264.

Converse, J. M. & Smith, B. (1963). Naso-orbital fractures. *Transactions of the American Academy of Ophthalmology and Otolaryngology*, **67**, 622.

Converse, J. M., Smith, B., Obear, M. F. & Wood-Smith, D. (1967). Orbital blow-out fractures: A ten-year survey. *Plastic and Reconstructive Surgery*, **39**, 20.

Culbertson, W. W. (1983). Diagnosis and management of ocular injuries. *Otolaryngologic Clinics of North America*, **16**, 563.

Ellis, E. III, El-Attar, A. & Moos, K. F. (1985). An analysis of 2,067 cases of zygomatico-orbital fracture. *Journal of Oral and Maxillofacial Surgery*, **43**, 417.
Gordon, S. & Macrae, H. (1950). Monocular blindness as a complication of the treatment of a malar fracture. *Plastic and Reconstructive Surgery*, **6**, 228.
Gwyn, P. P., Carraway, J. H., Horton, C. E., Adamson, J. E. & Miladick, R. A. (1971). Facial fractures: associated injuries and complications. *Plastic and Reconstructive Surgery*, **47**, 225.
Henderson, D. (1973). Classification of malar fractures. Unpublished.
Hitchin, A. D. & Shuker, S. T. (1973). Some observations on zygomatic fractures in the eastern region of Scotland. *British Journal of Oral Surgery*, **2**, 114.
Holt, G. R. & Holt, J. E. (1983). Incidence of eye injuries in facial fractures: an analysis of 727 cases. *Otolaryngology–Head and Neck Surgery*, **91**, 276.
Holt, J. E., Holt, G. R. & Blodgett, J. M. (1983). Ocular injuries sustained during blunt facial trauma. *Ophthalmology*, **90**, 14.
Jabaley, M. E., Lerman, M. & Sanders, H. J. (1975). Ocular injuries in orbital fractures. A review of 119 cases. *Plastic and reconstructive Surgery*, **56**, 410.
Jennett, B. & Teasdale, G. (1981). *Management of Head Injuries*, p 89. Philadelphia: F. A. Davis.
Ketchum, L. D., Ferris, B. & Masters, F. W. (1976). Blindness without direct injury to the globe: a complication of facial fractures. *Plastic and Reconstructive Surgery*, **58**, 187.
Le Fort, R. (1900). Fractures de la machoire superieure. *Congres International de Medecine*, Paris, 275.
Luce, E. A., Tubb, T. D. & Moore, A. M. (1979). Review of 1000 major facial fractures and associated injuries. *Plastic and Reconstructive Surgery*, **63**, 26.
Lundin, K., Ridell, A., Sandberg, N. & Ohman, A. (1973). One thousand maxillofacial and related fractures at the ENT-Clinic in Gothenburg. A two-year prospective study. *Acta Otolaryngologica*, **75**, 359.
McCoy, F. J., Chandler, R. A., Magnan, C. G. Jr., Moore, J. R. & Siemsen, G. (1962). An analysis of facial fractures and their complications. *Plastic and Reconstructive Surgery*, **29**, 381.
Milauskas, A. T. & Fueger, G. F. (1966). Serious ocular complications associated with blow-out fractures of the orbit. *American Journal of Ophthalmology*, **62**, 670.
Miller, G. R. & Tenzel, R. R. (1967). Ocular complications of midfacial fractures. *Plastic and Reconstructive Surgery*, **39**, 37.
Morgan, B. G. D., Madan, D. K. & Bergerot, J. P. C. (1972). Fractures of the middle third of the face: a review of 300 cases. *British Journal of Plastic Surgery*, **25**, 147.
Muller, E. J. & Schoeman, H. S. (1977). Zygomatico-maxillary fractures: a statistical analysis of 1,233 cases. *Journal of the Dental Association of South Africa*, **32**, 585.
Paton, D. & Emery, J. M. (1973). Injuries of the eye, the lids, and the orbit. In: *Management of Trauma*, W. F. Ballinger (ed.), 2nd Ed., pp 219–254. Philadelphia: W. B. Saunders.
Petro, J., Tooze, F. M., Bales, C. R. & Baker, G. (1979). Ocular injuries associated with periorbital fractures. *Journal of Trauma*, **19**, 730.
Pfeiffer, R. L. (1943). Traumatic enophthalmos. *Archives of Ophthalmology*, **30**, 718.
Records, R. E. (1979). Primary care of ocular emergencies. I. Traumatic injuries. *Postgraduate Medicine*, **65**, 143.
Schultz, R. C. (1967). Facial injuries from automobile accidents: A study of 400 consecutive cases. *Plastic and Reconstructive Surgery*, **40**, 415.
Steidler, N. E., Cook, R. M. & Reade, P. C. (1980). Residual complications in patients with major middle third facial fractures. *International Journal of Oral Surgery*, **9**, 259.
Stranc, M. F. (1970). Primary treatment of naso-ethmoid injuries with increased intracanthal distance. *British Journal of Plastic Surgery*, **23**, 8.
Teasdale, G. & Jennett, B. (1974). Assessment of coma and impaired consciousness. *Lancet*, **2**, 81.
Turvey, T. A. (1977). Midfacial fractures: a retrospective analysis of 593 cases. *Journal of Oral Surgery*, **35**, 887.

The Authors

I. A. Al-Qurainy MBChB, MCOphth, DO
Senior Registrar
G. N. Dutton MD, FRCS, FCOphth
Consultant
Tennent Institute of Ophthalmology
Western Infirmary
38 Church Street
Glasgow G11 6NT
L. F. A. Stassen MA, FRCS, FDSRCS
Senior Registrar
K. F. Moos FRCS, FDSRCS, FDSRCPS
Consultant
A. El-Attar MBBS, FDSRCS, FDSRCPS
Consultant
Department of Oral and Maxillofacial Surgery
Canniesburn Hospital
Bearsden
Glasgow G61 1QL

Correspondence and requests for offprints to Dr I. A. Qurainy, Royal Eye Infirmary, Apsley Road, Plymouth PL4 6PL.

Paper received 25 July 1990
Accepted 7 May 1991

British Journal of Oral and Maxillofacial Surgery (1991) 29, 302-307
© 1991 The British Association of Oral and Maxillofacial Surgeons

BRITISH JOURNAL OF ORAL & MAXILLOFACIAL SURGERY

Diplopia following midfacial fractures

I. A. Al-Qurainy, L. F. A. Stassen, G. N. Dutton, K. F. Moos, A. El-Attar

Tennent Institute of Ophthalmology, Western Infirmary, and Department of Oral and Maxillofacial Surgery, Canniesburn Hospital, Glasgow.

SUMMARY. **Over a period of 2 years, 363 patients who had sustained a total of 438 midfacial fractures due to blunt trauma received a full ophthalmological examination within 1 week of injury. Of these, 72 patients (19·8%) developed diplopia. Diplopia was most common following road traffic accidents (31%) and least common with simple falls (10%). Blow-out fractures of the orbit led to double vision in 58% of cases. Eighty two percent of patients recovered from diplopia within 6 months of injury; only 1 patient required squint surgery for double vision. The principal risk factors for diplopia comprise road traffic accidents, blow-out fractures and comminuted malar fractures. Early surgical reconstruction of midfacial fractures with conservative management of concomitant motility disorders has, in our series, resulted in very few patients having diplopia in the long term.**

INTRODUCTION

Diplopia resulting from extra-ocular muscle imbalance is a common complication of fractures of the zygomatico-maxillary complex (Yee *et al.*, 1975). The reported incidence of diplopia ranges between 15% of recent to 56% of late presentations of zygomatic fractures (Tajima *et al.*, 1974). Blow-out fractures of the orbit comprise the most common cause of diplopia (Helveston, 1977) which occurs in 36 to 86% of such cases (Converse *et al.*, 1967; Leibsohn *et al.*, 1976).

Vertical diplopia on up and down gaze commonly results from the soft tissue incarceration which follows an orbital floor blow-out fracture; whilst impairment of abduction may be a sequel to the less common medial wall blow-out fracture (Piddle, 1968) or a traumatic VI cranial nerve palsy. Orbital rim fractures without blow-out fractures may also result in diplopia (Converse *et al.*, 1961) which may be transitory. This diplopia has been attributed to impairment of movement due to oedema, haematoma or injury to the extra-ocular muscles or their nerve supply (Barclay, 1958; Converse *et al.*, 1961).

Entrapment of orbital tissue, extra-ocular muscle, check ligament injury, intra-orbital haemorrhage or oedema, or paresis due to cranial nerve injury, must be differentiated from one another as they may give rise to similar clinical features but their management differs (Thaller-Antlanger, 1980). A detailed history, thorough clinical examination and appropriate radiological evaluation is therefore required in order to establish the cause of double vision and to plan appropriate treatment.

The present prospective study was carried out to relate the type of injury sustained to the incidence and severity of subsequent eye movement disorder and thereby to identify the risk factors involved and to determine the prognosis for the restoration of binocular single vision.

PATIENTS AND METHODS

Three hundred and sixty three patients with midfacial fractures due to blunt facial trauma who attended the Oral and Maxillofacial Unit at Canniesburn Hospital, Glasgow during the period 1 April 1985 to 31 March 1987 were admitted to this study. All patients were assessed preoperatively and the follow-up time was up to 2 years for those patients with motility disturbances. The details of this group, the methods of assessment and Canniesburn's policy on timing of surgical intervention have already been described (Al-Qurainy *et al.*, 1991). At each consultation patients were asked whether they were troubled by double vision.

The duration of diplopia from the time of injury was classified into six categories: less than 1 day; 1 day to 1 week; 1 week to 1 month; 1 month to 6 months; 6 months to 1 year and more than 1 year. Diplopia was classified according to the following sub-categories (1) close to midline (within 15° of fixation); (2) in eccentric gaze (outside 15° of fixation); or (3) diplopia, of either category, which developed following surgery.

RESULTS

Three hundred and sixty three patients (295 males and 68 females) sustained a total of 438 midfacial fractures. The frequency distributions of the types and causes of these fractures have already been

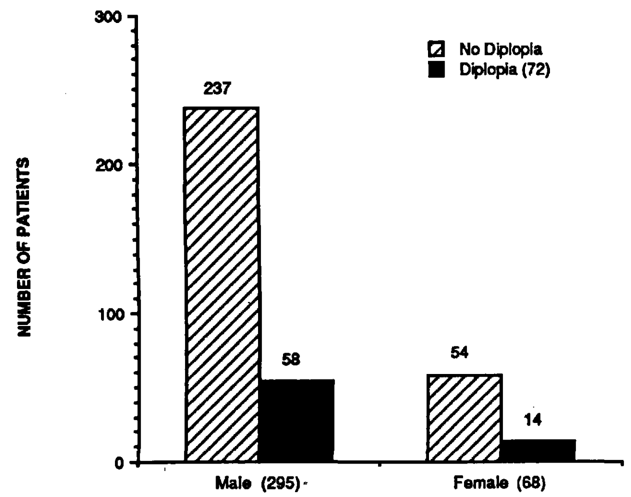


Fig. 1–The frequency distribution of diplopia in males and females.

illustrated (Al-Qurainy *et al.*, 1991). Seventy two of 363 patients (19.8%) complained of diplopia at the time of injury (Fig. 1). Fifty eight were male (19.7% of the male population) and 14 were female (20.6% of the female population). They had sustained a total of 94 midfacial fractures. Eighteen out of these 72 patients (25%) had sustained multiple fractures at two or three facial sites. However, the difference between multiple injury and single injury as a risk factor in the causation of diplopia was not found to be statistically significant.

Diplopia was most common in association with road traffic accidents (RTA) (14/45=31.1%), and least common with simple falls (7/69=10.1%), while assaults and industrial accidents yielded 20.4% (37/181), and 23% (3/13) of diplopia patients respectively (Fig. 2). The incidence of diplopia was related to the type of fracture sustained (Fig. 3). Diplopia was most common following blow-out fractures of the orbit (Type V), whilst it did not occur with isolated fractures of the orbital rim (Type VI). Twenty one (58.3%) of the patients with an orbital blow-out fracture had diplopia, all but one had their orbital floors explored. Eleven of these patients were found at surgery to have soft tissue entrapment in the orbital floor whilst one exhibited entrapment in the floor and medial wall. Eight patients had displacement of orbital contents into the antrum but no entrapment was elicited at the time of surgery. The remaining patient demonstrated entrapment of the superior oblique tendon in an orbital roof fracture verified by computerised tomogram (CT) scans (Al-Qurainy *et al.*, 1988).

Almost 30% of patients (8 out of 27) with a comminuted malar fracture (Type VII) suffered diplopia, while 22 of the 91 patients (24%) who had sustained a malar fracture with a distracted fronto-zygomatic (F–Z) suture (Type IV) were aware of double vision. A smaller proportion of patients (10 out of 124) (8.1%) complained of diplopia when the F–Z suture was undistracted (Type III) or when the fracture involved only the zygomatic arch (Type II)

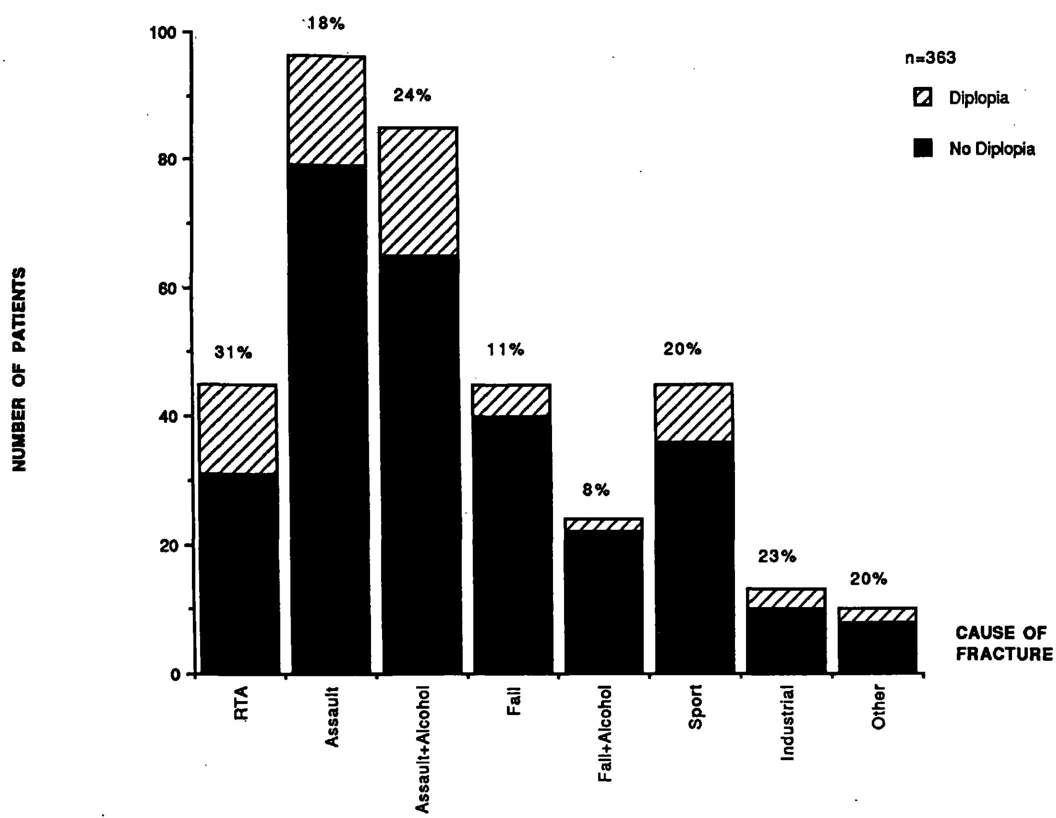


Fig. 2–The proportion of patients who suffered with diplopia (percentage figures) compared with those who did not, in each aetiological category.

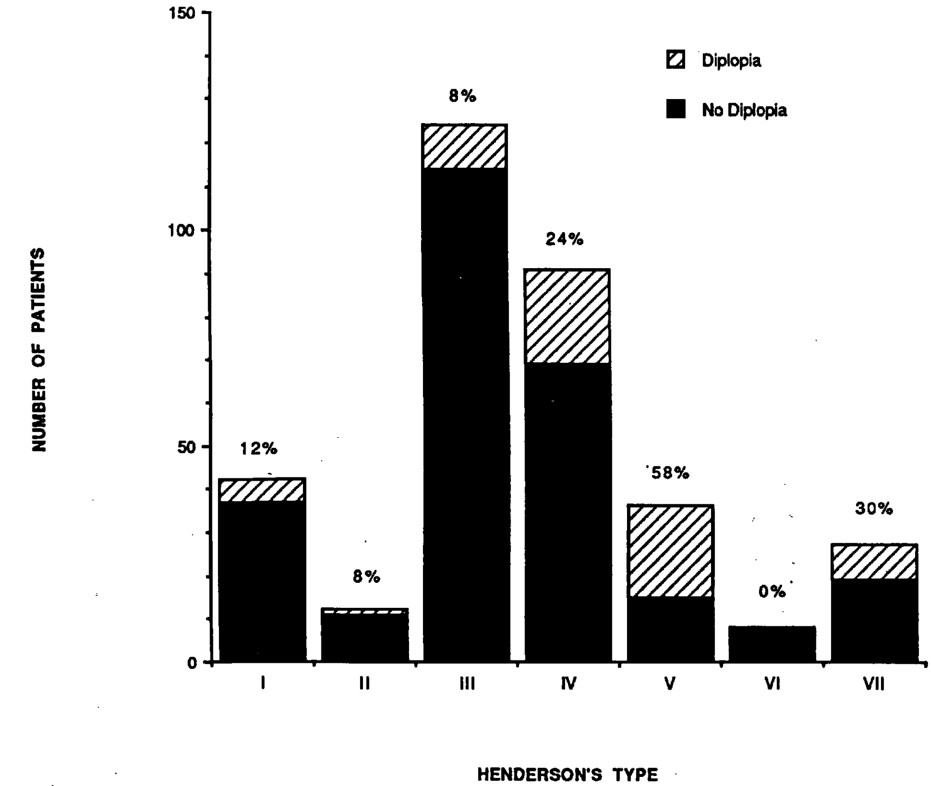


Fig. 3–The distribution of patients with diplopia in each category of malar fracture.

(1/13=8.3%). Malar fractures which were classified as undisplaced (Type I) led to diplopia in 11.9% of cases (5 out of 42). With regard to maxillary fractures, 1 of the 2 patients with a Le Fort III type fracture and 8 of the 22 patients with a Le Fort II type fracture (36.4%) reported diplopia. However, all these patients with Le Fort fractures had also sustained other facial fractures.

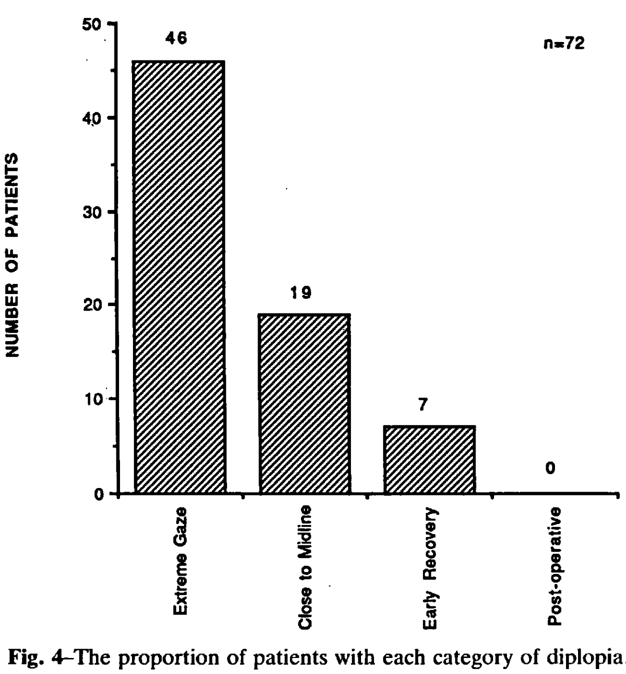


Fig. 4–The proportion of patients with each category of diplopia.

On examining the 72 patients with diplopia (Fig. 4), 19 (26.4%) of them had diplopia close to the midline whilst 46 (63.9%) experienced diplopia only in extreme (eccentric) gaze. Seven (9.7%) patients who initially reported symptoms of diplopia which had been confirmed on first examination were found to have recovered by the time they were seen in the eye clinic within 1 week of injury.

Fifteen of the 19 patients with diplopia close to the midline had sustained a pure (6) or an impure (9) blow-out fracture. Nine of these 15 patients had demonstrable soft tissue entrapment at surgery, the other 6 patients had significant orbital content displacement but no true entrapment. The remaining patients had sustained a range of facial fractures: naso-orbito-ethmoidal (NOE) (2), Le Fort II (1), type I malar fracture (1).

Diplopia was transient, lasting only for a few seconds or minutes in 2 out of the 72 (2.8%) diplopia patients (Fig. 5). A quarter of the patients (18) recovered in a matter of a few hours to 1 week. Twenty one patients (29.2%), however, recovered within 1 to 4 weeks, and another 18 (25%) suffered from diplopia for between 1 to 6 months. Eight (11.1%) of the patients with diplopia required 7 to 12 months to recover, and 5 (6.9%) complained of diplopia for greater than 12 months from the date of injury. Four of the 13 patients with diplopia for more than 6 months had sustained a tripod fracture with a distracted F–Z suture (Type IV), 6 patients had sustained a blow-out fracture (Type V) and 3 had suffered comminuted fractures of the zygomatico-orbital complex (Type VII) with associated skull

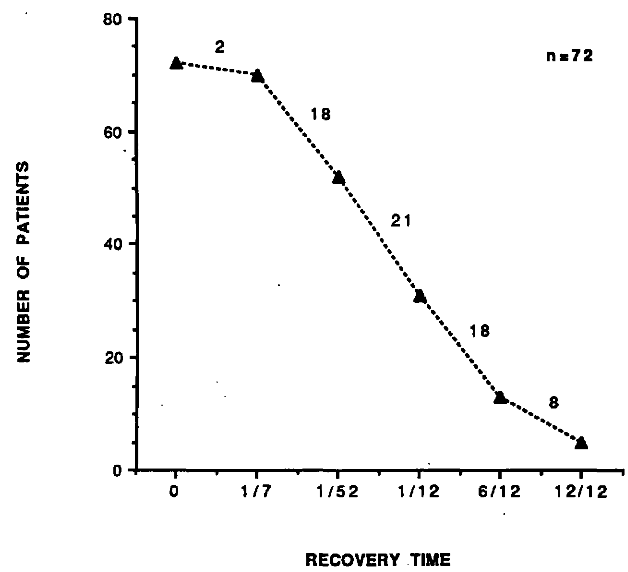


Fig. 5–The rate of recovery of diplopia among affected patients. Figures on the dotted line indicate the number of patients who recovered during that period.

fractures. With regard to the 5 patients who suffered diplopia for more than 1 year, two had sustained pure blow-out fractures of the orbital floor and another two had received tripod fractures with distraction of the F–Z suture. One patient had sustained a combination of a comminuted zygomatico-orbital fracture, a maxillary fracture at the Le Fort II level and an associated skull fracture.

The causes of injury in the 13 patients with diplopia for greater than 6 months were RTA (3 patients) and assault (7 patients), the rest of the patients suffered an industrial accident (1), a sporting injury (1) and a home accident in an 8-year-old child (1). There did not appear to be a relationship between the aetiology of the injury and persistence of diplopia.

Diplopia was seen only in extreme (eccentric) gaze in 12 out of the 13 patients with persistent diplopia and it did not cause them concern. One patient had troublesome diplopia in the primary position of gaze for which he required squint surgery.

In this series of 363 patients, 10 patients (2.8%) developed a III, VI or VII cranial nerve palsy. Five of them followed RTA, four were due to assault and one was due to an industrial accident. There were 2 cases of abducent nerve palsy and 1 case of an oculomotor nerve paralysis. In these cases, spontaneous recovery took place within 7 months. Seven patients developed a facial nerve palsy. No case of trochlear nerve palsy was identified.

Six of the patients with a cranial nerve palsy developed diplopia. Three of these were patients with abducent and oculomotor nerve palsies. The other three patients had facial nerve palsies, the cause of diplopia in these cases was a consequence of the orbital fracture. The type of fracture and cause of injury were different in each case.

DISCUSSION

Diplopia resulting from facial fractures has frequently been reported. However, a review of the literature shows that little attention has been paid to the course of recovery and the prognosis. Moreover, no attempt has been made to link diplopia to the cause or type of injury contributing to it. The reported incidence of diplopia following facial trauma (Table 1) is variable. It ranges from 1% (McCoy *et al.*, 1962) to 86% (Converse *et al.*, 1967). In this present study, 19·8% of 363 patients suffered diplopia. This is comparable to the work of Hakelius and Ponten (1973) and Tajima *et al.*, (1974) who reported figures of 21% and 15% respectively. However, the incidence of diplopia rises to high levels when the population of study is pre-selected, as for example in the case of blow-out fractures (Converse *et al.*, 1967; Greenwald *et al.*, 1974).

RTA were the most common cause of diplopia in this study (Fig. 2). Thirty one percent of all patients involved in RTA complained of diplopia. The second most common cause was assault associated with alcohol consumption (23.5%). However, we have seen more patients with diplopia overall due to assault than in any other category because of the high number of assaulted patients in our series. The incidence of diplopia was higher among patients who were assaulted whilst under the influence of alcohol (23.5%) than among those who were sober (17.7%). However, the difference between these two groups was not statistically significant. Industrial accidents led to diplopia in 23% of cases. Smaller percentages of diplopia were seen following sporting accidents (19.1%) or a fall (8.3%). No such detailed classification of diplopia in relation to aetiology is available for comparison. Patients with Type IV, Type V, and Type VII (pure or impure blow-out) fractures were more likely to suffer from persistent diplopia (for longer than 6 months).

Diplopia is most frequently associated with blow-out fractures of the orbit. Converse *et al.* (1967) reported the incidence to be 86% and Greenwald (1974) reported 70%. The more recent work by Leibsohn (1976) showed the incidence to be 36%. In our study, blow-out fractures contributed to diplopia in 58.3% of cases. Our figures probably represent a relatively unbiased spectrum of midfacial fractures. By contrast, it is possible that an element of pre-

Table 1 – Incidence of diplopia following facial fractures as quoted in other studies

Author(s)	Incidence	Population of patients
McCoy *et al.* (1962)	1.0%	Middle third fractures
Converse *et al.* (1967)	86.0%	Blow-out fractures
Hakelius & Ponten (1973)	21.8%	Midface fractures
Tajima *et al.* (1974)	15.0%	Recent zygomatic fractures
Tajima *et al.* (1974)	56.0%	Late zygomatic fractures
Leibsohn *et al.* (1976)	36.0%	Blow-out fractures
Greenwald *et al.* (1974)	70.0%	Blow-out fractures
Steidler *et al.* (1980)	32.1%	Middle third fractures

selection contributed to the high proportions quoted by Converse *et al.* (1967) and Greenwald (1974).

Maxillary fractures at the Le Fort III and Le Fort II levels associated with other facial fractures were common causes of diplopia, as were the NOE fractures. This probably represents the severity of the injury. Our study does not, however, accurately represent the incidence of diplopia in this group of patients because many of the more serious Le Fort fractures were associated with head injuries and therefore treated by the Canniesburn maxillofacial staff in the neurosurgical unit and have not been included in this study.

Maxillofacial fractures are associated with serious head injury in 25% of cases (Converse & Smith, 1963). Therefore, in addition to direct orbital trauma affecting ocular motility, various degrees of damage to the central nervous system, as well as possible trauma to the cranial nerves, can take place (Smith *et al.,* 1971). Loss of the ability to fuse the images from both eyes after trauma has been described by Hart (1969) and Stanworth (1974) and trauma is a recognised cause of the breakdown of an existing heterophoria (latent squint) (Lee, 1983). A cranial nerve palsy may affect ocular motility. A review of the literature (Table 2) shows that motor cranial nerve palsy following head and/or facial trauma ranges from 3% in closed head injuries (Russell, 1960) to 71% following fatal RTA (Heinze, 1969).

Orbital fractures commonly give rise to double vision. Not surprisingly, severe injuries resulting in comminution, F–Z distraction or blow-out fractures pose the greatest risk. Eight percent of patients with zygomatic arch fractures developed diplopia on eccentric gaze but this settled rapidly. Diplopia in these cases may have been caused simply by oedema.

The present policy in the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital is to operate early (within 2 weeks) on patients who have sustained facial trauma. Only 8 of the 72 patients were treated conservatively, all of whom had sustained undisplaced fractures, and whose diplopia resolved spontaneously. Resolution of diplopia is not well documented in the literature. Often, diplopia has been given as a reason to advocate early (Harley, 1975) or late (Langnickel & Munich, 1978) surgical treatment of maxillofacial trauma. Retrospective studies are biased in their patient selection because those patients who were not treated surgically were either considered not to warrant treatment or they had declined surgery. Putterman *et al.,* (1974) retrospectively reviewed the clinical course of 28 patients and prospectively assessed 29 patients following blow-out fractures of the orbital floor which had not been treated surgically. The relatively benign outcome in these patients demonstrated, in that author's opinion, the feasibility of managing blow-out fractures without surgery. This opinion, however, has not been shared by many other authors (Smith & Converse, 1957; Smith & Regan, 1957; Patterson & Depue, 1962; Reeh & Tsujimura, 1966) who have recommended an early exploratory and active surgical approach to avoid persistent diplopia and disfiguring enophthalmos.

Table 2 – Incidence of oculomotor palsy following head and/or facial trauma as cited by other authors

Author(s)	Incidence	Basis of selection
Rucker (1958)	16·2%	Blunt trauma to the head
Russell (1960)	3·0%	Closed head injuries
Schrader & Schlezinger (1960)	39·0%	VIth cranial nerve palsy only
Hughes (1964)	7.0%	Head injuries
Rucker (1966)	14.3%	Blunt trauma to the head
Heinze (1969)	71.0%	Fatal RTA only
Burger *et al.* (1970)	39.0%	VIth cranial nerve palsy only
Robertson *et al.* (1970)	20.0%	IVth cranial nerve in children

The risk factors for the development of diplopia are: RTA, blow-out fracture and comminuted malar fracture. For persistent diplopia, however, the only risk factor which is statistically significant is the pure or impure blow-out fracture. Only 1 of 363 patients in the series required squint surgery for diplopia and he had suffered a medial blow-out fracture which had initially been overlooked. Early surgical reconstruction, especially of the orbital floor, and conservative management of motility disorders may have contributed to a satisfactory outcome for our patients.

It is our belief that all patients with diplopia in the primary position of gaze should be evaluated by an ophthalmologist in order to establish the cause and to determine the optimum plan of management. Mild diplopia may be caused by oedema, haematoma, nerve palsies or entrapment. These causes can be differentiated with appropriate investigations. Early surgery for blow-out fractures with manifest clinical and radiological features has, in this study, not been associated with long-standing motility disorder, and the belief that diplopia may be aggravated permanently by surgery is disproved. In this study no patient developed diplopia as a direct result of surgery.

References

Al-Qurainy, I. A., Dutton, G. N., Moos, K. F., Reynolds, S. T. & McMillan, N. (1988). Orbital injury complicated by entrapment of the superior oblique tendon: A case report. *British Journal of Oral and Maxillofacial Surgery,* **26,** 336.

Al-Qurainy, I. A., Stassen, L. F. A., Dutton, G. N., Moos, K. F. & El-Attar, A. (1991). The characteristics of midfacial fractures and the association with ocular injury: A prospective study. *British Journal of Oral and Maxillofacial Surgery,* **29,** 291.

Barclay, T. L. (1958). Diplopia in association with fractures involving the zygomatic bone. British Journal of Plastic Surgery, **11,** 147.

Burger, L. J., Kalvin, N. H. & Smith, J. L. (1970). Acquired lesions of the fourth cranial nerve. *Brain,* **93,** 567.

Converse, J. M., Cole, G. & Smith, B. (1961). Late treatment of blow-out fracture of the floor of the orbit. *Plastic and Reconstructive Surgery,* **28,** 183.

Converse, J. M. & Smith, B. (1963). Naso-orbital fractures. In: Symposium: Midfacial Fractures. *Transactions of the American Academy of Ophthalmology and Otolaryngology,* Sept–Oct, 622.

Converse, J. M., Smith, B., Obear, M. F. & Wood-Smith, D. (1967). Orbital blow-out fractures: A ten-year survey. *Plastic and Reconstructive Surgery*, **39**, 20.
Greenwald, H. S., Keeney, A. H. & Shannon, G. M. (1974). A review of 128 patients with orbital fractures. *American Journal of Ophthalmology*, **78**, 655.
Hakelius, L. & Ponten, B. (1973). Results of immediate and delayed surgical treatment of facial fractures with diplopia. *Journal of Maxillo-Facial Surgery*, **1**, 150.
Harley, R. D. (1975). Surgical management of persistent diplopia in blow-out fractures of the orbit. *Annals of Ophthalmology*, **7**, 1621.
Hart, C. T. (1969). Disturbances of fusion following head injury. *Proceedings of the Royal Society of Medicine*, **62**, 704.
Heinze, J. (1969). Cranial nerve avulsion and other neural injuries in road accidents. *Medical Journal of Australia*, **2**, 1246.
Helveston, E. M. (1977). The relationship of extraocular muscle problems to orbital floor fractures: early and late management. *Transactions of the American Academy of Ophthalmology and Otolaryngology*, **83**, 660.
Hughes, B. (1964). The results of injury to special parts of the brain and skull. In: *Acute Injuries of the Head: Their Diagnosis, Treatment, Complications and Sequels*. G. F. Robotham (ed.), 4th Ed. Edinburgh & London: Churchill Livingstone.
Langnickel, R. & Munich, H. (1978). A contribution to the operative treatment of diplopia in patients with long-standing orbital floor fractures. A combined operative technique by an ophthalmologist is described. *HNO*, **26**, 301.
Lee, J. (1983). Ocular motility consequences of trauma and their management. *British Orthoptic Journal*, **40**, 26.
Leibsohn, J., Burton, T. C. & Scott, W. E. (1976). Orbital floor fractures: A retrospective study. *Annals of Ophthalmology*, **8**, 1057.
McCoy, F. J., Chandler, R. A., Magnan, C. G. Jr., Moore, J. R. & Siemsen, G. (1962). An analysis of facial fractures and their complications. *Plastic and Reconstructive Surgery*, **29**, 381.
Patterson, Jr., R. W. & Depue, Jr., R. V. (1962). Blow-out fracture of the orbit. *American Journal of Ophthalmology*, **53**, 841.
Piddle, W. J. (1968). Blow-out fractures of the orbit. *Manitoba Medical Review*, **48**, 322.
Putterman, A. M., Stevens, T. & Urist, M. J. (1974). Neurosurgical management of blow-out fractures of the orbital floor. *American Journal of Ophthalmology*, **77**, 232.
Reeh, M. J. & Tsujimura, J. K. (1966). Early detection and treatment of blow-out fracture of the orbit. *American Journal of Ophthalmology*, **62**, 79.
Robertson, D. M., Douglas Hines, J. & Rucker, C. W. (1970). Acquired sixth-nerve paresis in children. *Archives of Ophthalmology*, **83**, 574.
Rucker, C. W. (1966). The causes of paralysis of the third, fourth and sixth cranial nerves. *American Journal of Ophthalmology*, **61**, 1293.
Rucker, C. W. (1958). Paralysis of the third, fourth and sixth cranial nerves. *American Journal of Ophthalmology*, **46**, 787.
Russell, W. R. (1960). Injuries to cranial nerves and optic chiasm. In: *Injuries of the Brain and the Spinal Cord and their Coverings*, S. Brock (ed.), p. 121. London: Cassell.
Schrader, K. E. & Schlezinger, N. S. (1960). Neuro-ophthalmic evaluation of abducent nerve paralysis. *Archives of Ophthalmology*, **63**, 84.
Smith, B. & Converse, J. M. (1957). Early treatment of orbital floor fractures. *Transactions of the American Academy of Ophthalmology and Otolaryngology*, **61**, 602.
Smith, B. & Regan, W. F., Jr. (1957). Blow-out fracture of the orbit: Mechanism and correction of internal orbital fracture. *American Journal of Ophthalmology*, **44**, 733.
Smith, B. C., Barr, D. R. & Langham, E. J. (1971). Complications of orbital fractures. *New York State Journal of Medicine*, Oct., 2407.
Stanworth, A. (1974). Defects of ocular movement and fusion after head injury. *British Journal of Ophthalmology*, **58**, 266.
Steidler, N. E., Cook, R. M. & Reade, P. C. (1980). Residual complications in patients with major middle third facial fractures. *International Journal of Oral Surgery*, **9**, 259.
Tajima, S., Sugimoto, C., Tanino, R., Oshiro, T. & Harashina, T. (1974). Surgical treatment of malunited fractures of zygoma with diplopia and with comments on blow-out fracture. *Journal of Maxillo-Facial Surgery*, **2**, 201.
Thaller-Antlanger, H. (1980). Is there a typical motility disturbance in orbital wall fractures? *Klinische Monatsblatter fur Augenheilkunde*, **177**, 845.
Yee, K. F., Cravens, J. & Kotler, R. (1975). Traumatic ophthalmoplegia: a complication of fractures of the zygomatico-maxillary complex. Review of aetiology with report of an unusual case. *Laryngoscope*, **85**, 570.

The Authors

I. A. Al-Qurainy MBChB, MCOphth, DO
Senior Registrar
G. N. Dutton MD, FRCS, FCOphth
Consultant
Tennent Institute of Ophthalmology
Western Infirmary
38 Church Street
Glasgow G11 6NT
L. F. A. Stassen MA, FRCS, FDSRCS
Senior Registrar
K. F. Moos FRCS, FDSRCS, FDSRCPS
Consultant
A. El-Attar MBBS, FDSRCS, FDSRCPS
Consultant
Department of Oral and Maxillofacial Surgery
Canniesburn Hospital
Bearsden
Glasgow G61 1QL
Correspondence and requests for offprints to Dr I. A. Qurainy, Royal Eye Infirmary, Apsley Road, Plymouth PL4 6PL.

Paper received 25 July 1990
Accepted 7 May 1991

British Journal of Oral and Maxillofacial Surgery (1991) **29**, 363–367
© 1991 The British Association of Oral and Maxillofacial Surgeons

BRITISH JOURNAL OF ORAL & MAXILLOFACIAL SURGERY

Midfacial fractures and the eye: the development of a system for detecting patients at risk of eye injury

I. A. Al-Qurainy, D. M. Titterington, G. N. Dutton, L. F. A. Stassen, K. F. Moos, A. El-Attar

Tennent Institute of Ophthalmology, Western Infirmary, Department of Statistics, University of Glasgow, and Department of Oral and Maxillofacial Surgery, Canniesburn Hospital, Glasgow.

SUMMARY. **Maxillofacial trauma is often complicated by injury to the eye. Such injuries may be difficult to detect and may therefore be missed. Detailed ophthalmic examinations were carried out prospectively on 363 patients who had sustained midfacial fractures. Fifty four parameters comprising maxillofacial, radiological and ophthalmic data were recorded and coded for each patient. All encoded data were divided into predictors (the data potentially available to the maxillofacial surgeon) and outcome (the data potentially available to the ophthalmologist). Statistical methods of regression, and the analysis of contingency tables, led to the identification of the principal predictors indicative of underlying ophthalmic injury and thence to a scoring system which predicts the severity of such injuries. Impaired visual acuity is the principal predictor and when employed alone gives a sensitivity value of 80%. Pure blow-out fracture or comminuted facial fracture, double vision and amnesia emerged as additional factors which yielded an efficient scoring system with a sensitivity of 89% and specificity of 90% for the population upon which it was based. A score sheet is provided in the paper. These predictors can be remembered from the acronym *B*low-out fracture, *A*cuity, *D*iplopia, *A*mnesia, *C*omminuted *T*rauma. As many such injuries result from a BAD ACT, it is easily remembered. This scoring system requires to be tested upon a new population of individuals in order to determine its efficacy.**

INTRODUCTION

Maxillofacial fractures are commonly complicated by injuries to the visual system (Petro *et al.*, 1979; Culbertson, 1983; Hold & Holt, 1983). Such injuries may be difficult to detect without the requisite expertise and equipment. However, it is impractical to refer all such patients for ophthalmic assessment. In order to determine the optimum criteria for referral of appropriate patients to the ophthalmologist, we have devised a scoring system based on a prospectively assembled data base of patients with maxillofacial fractures (Al-Qurainy *et al.*, 1991a).

PATIENTS AND METHODS

The database comprises a cohort of 363 patients with a variety of midfacial fractures who had been sent as secondary referrals to the Oral and Maxillofacial Unit at Canniesburn Hospital during a 2-year period. The details of this group and the methods of assessment have already been described (Al-Qurainy *et al.*, 1991a). Table 1 lists the criteria as 'predictors' for potential ophthalmic injury. The predictors comprise the data which are immediately available to the maxillofacial surgeon. Tables 2, 3 and 4 list the eye injuries as 'outcomes' divided into the categories of non-referral, routine referral and early referral. A brief justification for selecting each parameter as an outcome is also given:

Table 1 – List of information available to the maxillofacial surgeon which was tested as comprising predictors for the outcome of ophthalmic injury

Basic patient data
Sex
Age
Month of injury

Cause of injury

Site of injury: determined clinically and radiologically
Malar fracture site
Maxillary fracture site
Other fracture site
Entrapment of soft tissue (floor or medial wall blow-out)
Injury in the region of the trochlea

Method(s) of surgical treatment

Ophthalmic data
Affected eye/side
Visual acuity

Ocular motility data
Symptoms: Double vision
History of transient double vision with recovery
Pain on eye movement

Signs: Abnormal head posture to compensate for double vision
Manifest deviation of the eye in the primary position of gaze
Restriction of extra-ocular movements found on clinical examination
Convergence insufficiency

Evidence of associated injuries
Amnesia
Cranial nerve palsies

Table 2 – List of the 'non-referral' category of ophthalmic injuries. For each type of injury the justification for the referral category is given

Eye abnormality	Remarks
Post-traumatic neuralgia/ anaesthesia	Spontaneous recovery. No treatment needed
Coronal eye displacement	Should respond to facial fracture treatment
Orbital emphysema	Absorbs spontaneously
Eyelid swelling/bruises	Clears spontaneously
Conjunctival chemosis	Clears spontaneously
Subconjunctival haemorrhage	Clears spontaneously
Corneal abrasion	Heals spontaneously
Mild failure of accommodation	Usually recovers spontaneously
Mild reduction of visual acuity with recovery	May be due to corneal abrasion. No treatment is usually needed
Mild commotio retinae	Spontaneous resolution

Table 3 – List of the 'routine-referral' category of ophthalmic injuries. For each type of injury the justification for the referral category is given

Eye abnormality	Remarks
Enophthalmos	Usually responds to orbital wall reconstruction
Eyelid laceration	Can be treated by the maxillofacial surgeon
Conjunctival lacerations	May or may not need suturing depending on extent and involvement of underlying tissue
Traumatic pupillary changes	May indicate other pathology. Observe and document
Iridodialysis	May indicate other pathology. Occasionally the iris may need to be repaired
Lens damage: Vossius' ring	May indicate underlying pathology requiring treatment or follow-up, for example, angle recession
Traumatic cataract	Indicates need for future treatment
Positive photostress test	Macular oedema. Self-limiting
Moderate failure of accommodation	No immediate treatment. May need refraction later.
Severe failure of accommodation	As above
Moderate reduction of visual acuity	May be due to macular oedema, corneal abrasion/oedema, traumatic mydriasis. Needs to be investigated
Moderate commotio retinae	To be documented and observed. If involving the macula, may progress into macular cyst/hole
Severe commotio retinae	As above
Choroidal tear: single	To be documented and observed
Choroidal tear: multiple	As above
Vitreous floater	Should be closely folowed up. May indicate underlying pathology, but treatment is not immediately indicated
Traumatic pigmentary retinopathy	To be documented and observed
Naso-lacrimal damage	If not repaired by the maxillofacial surgeon

Table 4 – List of the 'early referral' category of ophthalmic injuries. For each type of injury the justification for the referral category is given

Eye abnormality	Remarks
Proptosis	May be due to retrobulbar haemorrhage leading to optic nerve compression
Retrobulbar haemorrhage	As above
Corneal damage: puncture wound	Needs urgent repair under microscope
Corneal laceration	As above
Scleral injury	Needs careful repair
Hyphaema	May lead to rise of intraocular pressure
Angle recession	To be followed up closely for glaucoma. An acute pressure rise may occur
Lens dislocation/subluxation	Treatment may be needed for post-traumatic complications for example, glaucoma
Ruptured lens capsule	Indicates need for future treatment
Severe reduction in visual acuity	Optic nerve injury, retinal detachment, vitreous haemorrhage. Aetiology must be established immediately
*Loss of vision	As above
*Visual field loss	To be investigated soon for retinal detachment and/or damage to the visual pathway
*Choroidal tear involving the macula	Document and observe prior to facial treatment for medico-legal reasons
*Vitreous haemorrhage	Needs urgent specialist examination
*Flat retinal tear/hole	May need laser/cryotherapy
*Retinal detachment with macula on	Needs urgent specialist treatment
*Retinal detachment with macula off	As above
*Total retinal detachment	As above
*Optic nerve injury: contusion	Endangers sight. To be documented
*Optic nerve: partial injury	As above
*Optic nerve: total damage/avulsion	To be documented for medico-legal reasons

*All of these signs indicate major injury. Accurate evaluation prior to surgical repair of the facial injuries is preferred so that loss of vision cannot be attributed to the surgical intervention and an appropriate sequence of management can be organised.

Non-referral cases were defined as those patients with ocular injuries without permanent visual or physiological sequelae such as corneal abrasion or subconjunctival haemorrhage. These injuries heal spontaneously.

Routine referral cases were selected as having ocular injuries which are unlikely to lead to permanent visual or physiological sequelae but for whom referral may be indicated for the purpose of assessment or screening for pathology necessitating treatment. Such patients may be referred for a routine outpatient examination within, say, 1 month of injury.

Early referral cases comprised those individuals with ocular injuries such as corneal laceration or retinal damage likely to produce sustained visual loss and those patients with adnexal injuries requiring advice at an early stage concerning management, therapeutic intervention or detailed appraisal for medico-legal reasons.

The methods of statistical analysis were as follows:

Selection of risk factors

Each predictor was initially considered separately. The data were cross-classified according to the type of referral required and the value of each predictor. From each two-way contingency table which resulted, the Chi-squared statistic was computed and predictors were noted for which the statistic was significant at or near the 5% level of confidence (Armitage, 1971).

Construction of a scoring system

The data concerning referral type (non-referral=0; routine referral=1; early referral=2) were combined with the data obtained for the predictors which had been selected as risk factor in the manner outlined above. These were analysed using a stepwise linear regression analysis. This procedure comprises a means of selecting a subset of the predictors which extracts the meaningful predictive information contained by all the predictors. The resulting subset therefore provides a parsimonious but efficient means of prediction. The coefficients in the corresponding regression equation were then rounded off to more convenient numbers to provide the formula for the raw score shown in the 'Results' section. This raw score was, in turn, converted to a refined score suitable for use in every day practice. Most statistical analysis were carried out using the statistical package Minitab (Ryan *et al.*, 1985).

RESULTS

The principal risk factors which, in combination, successfully detected the largest numbers of patients deemed to warrant referral to the ophthalmologist were as follows: (a) Impairment of visual acuity; this emerged as the most sensitive single predictor for ophthalmic injury (see Table 5 for the associated contingency table). (b) Comminuted malar fractures were associated with the most severe ocular and motility defects, no doubt because the protection afforded by an intact lateral orbital wall was lost and the eye and orbital tissues were subjected to compressive injury. (c) Pure blow-out fractures commonly produced motility defects which were also deemed to warrant ophthalmic referral. (d) Abnormalities of eye movement can, in most cases, be easily detected by the maxillofacial surgeon primarily on the basis of double vision. Although this predictor 'overlaps' with comminuted and blow-out fractures it emerged as a predictor which identified additional cases deemed to warrant referral. (e) Finally, those patients who were injured severely enough to cause amnesia had sustained severe injuries which commonly affected the visual system.

Table 5 – The categorisation of 363 patients according to their referral criteria based on changes in visual acuity only. The numbers underlined identify those patients who should have been referred on clinical grounds but were not (false negatives) because the visual acuity was normal or only mildly reduced. The numbers in parentheses denote false positives. (The visual acuity code comprises 0=6/6 or better; 1=6/9–6/12; 2=6/18–6/24; 3=<6/24; 4=no perception of light)

Visual acuity code	0	1	2	3	4
Referral category	Number of patients entering each category				
Non-referral	250	13	(4)	(0)	(0)
Routine referral	39	9	6	2	0
Early referral	18	7	5	2	8

Scores: 0–4 refer to the values given to visual acuity.

The scoring system developed from the risk factors was tested by determining the sensitivity and specificity for the detection of the ophthalmic conditions listed in Tables 2, 3 and 4 and by computing the value of the Chi-squared statistic. The basic scoring system produced by the regression analysis involved the creation and testing of many similar but distinct scoring rules. This led to the following system which provided the highest value of Chi-squared.

(a) Visual acuity variable (χ_1)
where 0=normal visual acuity (6/4–6/6)
1=visual acuity mildly reduced (6/9–6/12)
2=visual acuity moderately reduced (6/18–6/24)
3=visual acuity severely reduced (worse than 6/24)
4=loss of vision

(b) Malar fracture type (χ_2)
1=if fracture is comminuted or blow-out
0=if otherwise

(c) Motility disorder (χ_3)
1=if motility disorder present
0=if motility disorder absent

(d) Amnesia (χ_4)
1=if amnesia (retrograde and post-traumatic amnesia) present
0=if otherwise

From these parameters, the following scoring system was computed:

$$\text{Score}=(4\times X_1)+(3\times X_2)+(3\times X_3)+(5\times X_4)$$

The score values were then interpreted by the decision rule as follows:

If score=0	(non-referral)
If score=1–4	(non-referral)
If score=5–8	(routine referral)
If score=9–11	(routine referral)
If score=12 or more	(early referral)

Table 6 – The referral categorisation of the same patients in Table 5, taking into account the factors shown in Table 8. The numbers underlined comprise the false negative population whereas the numbers in parentheses denote the false positives.

Refined score	0	1–4	5–8	9–11	12+
Referral category	Number of patients entering each category				
Non-referral	173	69	(22)	(3)	(1)
Routine referral	10	12	24	5	4
Early referral	5	10	5	7	13

The numbers underlined comprise the false negative population whereas the numbers in parentheses denote the false positives.

When this system, based on visual acuity changes only, is applied to our population of 363 patients it gives a sensitivity value of 80% and the categorisation of results shown in Table 5 is obtained.

Table 6 shows that the scoring system described above results in appropriate referral in the majority of cases when the rest of the variables is taken into account, although a total of 37 patients (Table 7) received scores of 0 or 1 but should have been referred and a total of 26 patients received scores of 5 or more but would not have warranted referral. The tests of validity for these scores are as follows:

Sensitivity value=89%
Specificity value=90%
Predictive value of a positive test=92%
Predictive value of a negative test=87%

As shown in Table 7, the patients which this system failed to identify as referrals included 5 patients with proptosis. However, as in each of these cases the visual acuities were not impaired, sight-threatening retrobulbar haemorrhage was not implicated and referral may not have resulted in any tangible benefits for the patients. Four patients with impaired accommodation were also missed by this system. However, the visual acuities in these patients were measured for distance and were normal. The majority of bedside examinations by maxillofacial surgeons would utilise near visual acuities and would therefore identify patients with impaired accommodation as having impaired vision and warranting referral. The major cause for concern in this series, however, centred on 6 patients with peripheral retinal/vitreous damage and 6 patients who were found to have angle recession. It would appear that, short of carrying out prospective examination of such patients, they are liable to 'slip through the net' of the scoring system described because they could only be identified by specialised slit-lamp and ophthalmoscopic examination.

Table 7 – Characteristics of patients whose eye abnormalities were falsely predicted not to warrant referral to the ophthalmologist (false negatives)

No. of cases	Eye abnormality	Desired outcome
2	Flat retinal hole	Early referral
1	Vitreous haemorrhage	Early referral
6	Angle recession	Early referral
5	Proptosis	Early referral
3	Uncomplicated vitreous floater	Routine referral
1	Lens damage (traumatic cataract)	Routine referral
4	Enophthalmos	Routine referral
6	Eyelid laceration	Routine referral
2	Nasolacrimal damage	Routine referral
4	Moderate/severe failure of accommodation	Routine referral
3	Traumatic pupillary changes	Routine referral
37	Patients	

DISCUSSION

The scoring system described above is designed to be applied at the time the patient first presents and has a high predictive accuracy for the patient cohort from which it was derived. It requires to be tested upon a new group of patients in order to determine whether or not the values reported still hold for such a group (Al-Qurainy *et al.*, 1991b). The clinical data sheet being employed for this purpose is illustratedin Table 8.

The method for constructing the scoring system from the selected class of informative predictors is undoubtedly informal from a statistical point of view. In particular, the application of linear regression

Table 8 – Scoring sheet used by the maxillofacial surgeon to determine which patients with midface injuries warrant referral to an ophthalmologist

Clinical feature	Initial score	Final score
Visual acuity		
6/6 or better	0	
6/9–6/12	4	
6/18–6/24	8	
6/36 or less	12	
No light perception	16	( )
Malar fracture type		
Comminuted (VII)	3	
Blow-out (V)	3	
Otherwise	0	( )
Motility abnormality		
Present (diplopia or squint)	3	
Absent	0	( )
Amnesia		
Retrograde + PTA	5	
Otherwise	0	( )
Total score		( )

Consider adding (1) to the final score if one or more of the following factors exist and total score is (11):

If sex is female
If age is 30–39 years
If cause of injury is RTA

Referral to the ophthalmologist
If total score= 0–4: Do not refer
= 5–11: Consider routine referral
=12+: Consider urgent referral

analysis to data of this type, using the arbitrary scores of 0, 1 or 2 assigned to the three types of referral is, in itself, not above criticism. It should be emphasised, however, that this technique was used here as a 'stepping stone' to the scoring system which forms the major product of this paper. The scoring system itself has been validated legitimately and has been identified as being empirically as good as any similar system so far evaluated. On the other hand, the system is not sacrosanct, in that any of a wide class of alternative minor variants perform almost as well. We have used a comparatively informal approach for the construction of the scoring system because of the unwieldy nature of the data and partly because regression analysis with an ordered categorical response variable (such as the scores assigned to the referral type) combined with the incorporation of stepwise selection of predictors remains a topic of basic statistical research.

The patient cohort upon which this study was based does not include individuals with additional head injuries warranting neurosurgical admission. However, the database upon which the scoring system is based is sufficiently broad to ensure its reliability. Moreover, it was the ophthalmic *outcome* which determined the general nature of the *predictors* which thereby ensures reliability of the system when it is applied to different patient groups.

The selection of 'predictors' and 'outcomes' was inevitably empirical as was the selection of referral criteria. However, these were discussed with a number of maxillofacial surgeons and ophthalmologists before being chosen and hopefully provide a consensus from both specialties. It may be considered that other predictors can be identified which will effectively select additional patients for referral. Other predictors which were considered, and which in isolation were found to have a fair predictive value, comprised sex (females were more likely to have sustained significant ocular injury than males, perhaps because they did not defend themselves so well), age (individuals between the ages of 30–39 years were more likely to have sustained ophthalmic injury than in any other age group) and road traffic accidents (RTA) (those patients involved in RTA were more likely to have sustained ophthalmic injuries than from any other cause). However, when these predictors were considered within the above system they failed to identify any additional individuals warranting referral.

Holt *et al.* (1983) classified eye injuries associated with facial fractures into three categories, minor or temporary, serious and blinding. They defined minor injuries as those injuries without permanent, visual or physiological sequelae; serious injuries as those producing sustained visual loss or adnexal sequelae requiring subsequent reconstructive measures; and blinding injuries as those resulting in either no light perception or loss of the eye. Although this classification effectively serves to highlight the fact that a large proportion of patients with maxillofacial injuries are subject to significant ocular sequelae, it does not identify those individuals for whom an ophthalmic referral may be indicated.

In conclusion, the principal predictor for the detection of ophthalmic injury is visual acuity. It is therefore of the utmost importance that this parameter be assessed in all patients sustaining orbital injuries sufficient to cause facial fracture. The application of the scoring system described, when taken in conjunction with a detailed clinical appraisal of the patient, is likely to result in a large proportion of 'appropriate' referrals to the ophthalmologist and will lessen the likelihood of significant ophthalmic pathology being missed.

References

Al-Qurainy, I. A., Stassen, L. F. A., Dutton, G. N., Moos, K. F. & El-Attar, A. (1991a). The characteristics of midface fractures and the association with ocular injury: a prospective study. *British Journal of Oral and Maxillofacial Surgery,* **29,** 291.

Al-Qurainy, I. A., Dutton, G. N., Ilankovan, V, Titterington, D. M., Moos, K. F. & El-Attar, A. (1991b). Midfacial fractures and the eye: the development of a system for detecting patients at risk of eye injury—a prospective evaluation. *British Journal of Oral and Maxillofacial Surgery,* **29,** 368.

Armitage, P. (1971). *Statistical Methods in Medical Research,* 4.8, 7.5. Oxford: Blackwell.

Culbertson, W. W. (1983). Diagnosis and management of ocular injuries. *Otolaryngologic Clinics of North America,* **16,** 563.

Holt, G. R. & Holt, J. E. (1983). Incidence of eye injuries in facial fractures: an analysis of 727 cases. *Otolaryngology—Head and Neck Surgery,* **91,** 276.

Holt, J. E., Holt, G. R. & Blodgett, J. M. (1983). Ocular injuries during blunt facial trauma. *Ophthalmology,* **90,** 14.

Petro, J., Tooze, F. M., Bales, C. R. & Baker, G. (1979). Ocular injuries associated with periorbital fractures. *Journal of Trauma,* **19,** 730.

Ryan, B. F., Joiner, B. L. & Ryan, T. A. (1985). *Minitab Handbook,* 2nd Ed. Boston: Duxbury Press.

The Authors

I. A. Al-Qurainy MBChB, MCOphth, DO
Senior Registrar
G. N. Dutton MD, FRCS, FCOphth
Consultant
Tennent Institute of Ophthalmology
Western Infirmary
38 Church Street
Glasgow G11 6NT
D. M. Titterington PhD
Professor
Department of Statistics
University of Glasgow
Glasgow G12 8QQ
L. F. A. Stassen MA, FRCS, FDSRCS
Senior Registrar
K. F. Moos FRCS, FDSRCS, FDSRCPS
Consultant
A. El-Attar MBBS, FDSRCS, FDSRCPS
Consultant
Department of Oral and Maxillofacial Surgery
Canniesburn Hospital
Bearsden
Glasgow G61 1QL
Correspondence and requests for offprints to Dr I. A. Al-Qurainy, Royal Eye Infirmary, Apsley Road, Plymouth PL4 6PL.

Paper received 25 July 1990
Accepted 7 May 1991

British Journal of Oral and Maxillofacial Surgery (1991) 29, 368–369
© 1991 The British Association of Oral and Maxillofacial Surgeons

BRITISH JOURNAL OF ORAL & MAXILLOFACIAL SURGERY

Midfacial fractures and the eye: the development of a system for detecting patients at risk of eye injury—a prospective evaluation

I. A. Al-Qurainy, G. N. Dutton, V. Ilankovan, D. M. Titterington, K. F. Moos, A. El-Attar

Tennent Insitute of Ophthalmology, Western Infirmary, Department of Oral and Maxillofacial Surgery, Canniesburn Hospital, and Department of Statistics, University of Glasgow, Glasgow

SUMMARY. **Midfacial trauma is often complicated by ocular disorder. A scoring system has been devised to help the maxillofacial surgeon identify patients who warrant referral to an ophthalmologist. A prospective pilot study was carried out on 100 patients with midfacial fractures to evaluate the effectiveness of this system in clinical practice. The sensitivity value was 94.4% and the specificity value was 89%. Only 1 patient, who clinically warranted referral to an ophthalmologist, was missed by the system whilst 9 others were incorrectly classified as warranting referral. The results of this evaluation demonstrate the competence of the system.**

INTRODUCTION

A simple scoring system and chart has been designed for the maxillofacial surgeon to determine which patients with midfacial injuries warrant referral to an ophthalmologist (Al-Qurainy *et al.*, 1991a). The sensitivity and specificity of this evaluation system approximated to 90% when it was applied to the population of 363 patients upon which it was based. Clearly, however, this did not provide a reliable reflection of the sensitivity and specificity for detection in further practice, since the system is inevitably 'biased' in favour of the original patients upon whom it was based.

In order to determine whether this system is applicable and effective in the management of patients who sustain midfacial fracture, it was essential to carry out a further prospective evaluation of the scoring system upon a new cohort of patients.

PATIENTS AND METHODS

Patients

One hundred patients who sustained midfacial trauma leading to fracture were assessed. Patients with isolated nasal, mandibular or skull fractures were not included.

Methods

Patients who attended the maxillofacial unit at Canniesburn Hospital for the first time between December 1989 and August 1990, with radiologically proven midfacial fractures, were assessed at the time of initial presentation according to a written protocol. Each patient was examined by the attendant maxillofacial surgeon and was accorded a score from which the patients were categorised as requiring non-referral, routine referral or early referral. In addition, the reason for referral, as elicited by the maxillofacial surgeon, was appended to the score sheet (Al-Qurainy *et al.*, 1991a).

All patients were then evaluated by an ophthalmologist according to the same protocol used for the original study (Al-Qurainy *et al.*, 1991b). In brief, this comprised an assessment of visual function, including the visual acuity as tested on the standard or the reduced Snellen's chart, a detailed evaluation of ocular motility including the use of Hess charts, pupillary dilatation and examination of the anterior and posterior segments of both eyes. Data accruing from that examination were then coded according to the score sheet appended. The criteria as to what ophthalmic conditions warrant referral to an ophthalmologist and for what reasons have already been described (Al-Qurainy *et al.*, 1991a).

It was essential to ensure that reduced visual acuity, when it occurred, was a sequel to the midfacial trauma sustained and not to a previous ophthalmic disorder such as amblyopia (lazy eye), uncorrected errors of refraction (short or long sightedness), non-traumatic cataract or previous trauma affecting the eye or the visual pathway. Similarly, amnesia scored positively only when it included both retrograde and post-traumatic loss of memory.

RESULTS

A total of 100 patients with radiological evidence of midfacial fracture was assessed for the purpose of evaluating the validity of the scoring system. Of these, 17 patients were correctly identified by the system as warranting referral to the ophthalmologist (true positive) and another 73 as unlikely to warrant such referral (true negative). Nine patients were incorrectly identified as requiring referral (false positive) whilst only 1 patient was incorrectly classified as not justifying referral (false negative). The tests for validity for these scores are as follows:

Sensitivity value=94·4%
Specificity value=89·0%
Predictive value of a positive test=65·4%
Predictive value of a negative test=98·6%

DISCUSSION

A screening test should provide a good preliminary indication of which individuals have the disease and which do not (Mausner & Bahn, 1974). This is referred to as the validity of the test. Validity has two components: sensitivity and specificity. Sensitivity is the ability to identify correctly those who have the disease. Specificity is the ability to identify correctly those who do not have the disease.

An ideal screening test would be 100% sensitive and 100% specific. In practice this does not occur; sensitivity and specificity are usually inversely related. Naturally, a sensitive test is desired to identify a high proportion of those individuals who have the disease in question. The results of this pilot study clearly demonstrate the high sensitivity of our scoring system. This confirms the prediction that the application of the scoring system is likely to result in a large proportion of 'appropriate' referrals to the ophthalmologist, thereby lessening the likelihood of significant ophthalmic pathology being missed.

Only 1 patient in the series was incorrectly identified as not warranting referral (false negative). However, his only ocular problem was a 3 mm enophthalmos which was unlikely to have led to visual complications and would have been dealt with by the maxillofacial surgeon in any case.

With a specificity value of less than 100%, inevitably, some 'inappropriate' referrals are bound to occur. In this study, 9 patients with healthy eyes scored 5–10 on the scoring system and, therefore, were incorrectly categorised as warranting routine referral to the ophthalmologist. Of those, 6 patients scored 6 because they had sustained either a blowout or a comminuted malar fracture (score=3) leading to diplopia or other motility disorder (score=3), while 2 others scored 10 because they had suffered a temporary mild reduction of visual acuity (score=4). The ninth patient scored 5 because of the combined type of amnesia he had sustained. However, because of the small size of this category of patients (1 patient per month in the series) and the fact that their scores on the system placed them on the routine rather than the early list of referrals, this improper referral was unlikely to have disturbed or overloaded the routine work at the local eye clinic. Moreover, it is far better to make a few unnecessary referrals than to miss potentially sight threatening pathology.

In conclusion, the new scoring system which has been devised and tested appears to be efficient enough for routine use. It is hoped that maxillofacial surgeons will find it helpful in the assessment of their patients' needs, especially when an ophthalmologist is not immediately available. The principal predictor of an adverse ophthalmic prognosis is impaired visual acuity. This study reaffirms the tenet that the assessment of acuity, or alternative tests of central visual function, should be performed in all patients sustaining midfacial fracture.

References

Al-Qurainy, I. A., Titterington, D. M., Dutton, G. N., Stassen, L. F. A., Moos, K. F. & El-Attar, A. (1991a). Midfacial fractures and the eye: the development of a system for detecting patients at risk of eye injury. *British Journal of Oral and Maxillofacial Surgery*, **29**, 363–367.

Al-Qurainy, I. A., Stassen, L. F. A., Dutton, G. N., Moos, K. F. & El-Attar, A. (1991b). The characteristics of midfacial fractures and the association with ocular injury: a prospective study. *British Journal of Oral and Maxillofacial Surgery*, **29**, 291–301.

Mausner, J. S. & Bahn, A. K. (1974). *Epidemiology: An Introductory Text*, p. 242. Philadelphia: Saunders.

The Authors

I. A. Al-Qurainy MBChB, MCOphth, DO
Senior Registrar
G. N. Dutton MD, FRCS, FCOphth
Consultant
Tennent Institute of Ophthalmology
Western Infirmary
38 Church Street
Glasgow G11 6NT
V. Ilankovan FRCS, FDSRCS
Senior Registrar
K. F. Moos FRCS, FDSRCS, FDSRCPS
Consultant
A. El-Attar MBBS, FDSRCS, FDSRCPS
Department of Oral and Maxillofacial Surgery
Canniesburn Hospital
Bearsden
Glasgow G61 1QL
D. M. Titterington PhD
Professor
Department of Statistics
University of Glasgow
Glasgow G12 8QQ

Correspondence and requests for offprints to
Dr I. A. Al-Qurainy, Royal Eye Infirmary, Apsley Road, Plymouth PL4 6PL

Paper received 24 August 1990
Accepted 7 May 1991

THE OPHTHALMOLOGICAL CONSEQUENCES

OF MIDFACIAL TRAUMA

VOLUME TWO

BY
Isam A Al-Qurainy
MB ChB MCOphth DO

A Thesis Submitted in Two Volumes for the
Degree of PhD in Ophthalmology

To

The University of Glasgow

On the Basis of Research Conducted at the Tennent
Institute of Ophthalmology and the Oral and Maxillofacial
Surgery Unit at Canniesburn Hospital, Glasgow.

Submitted October, 1992

Copyright (c) 1992, By I A Al-Qurainy

Thesis
9738
Copy 1
Vol 2

GLASGOW
UNIVERSITY
LIBRARY

PART TWO

THE ROLE OF QUANTITATIVE TESTS IN THE DIAGNOSIS OF OCULAR MOTILITY DISORDERS

C O N T E N T S

PART TWO

THE ROLE OF QUANTITATIVE TESTS IN THE DIAGNOSIS OF MOTILITY DISORDERS

SUMMARY

The active and passive behaviour of the extraocular muscles can be investigated by the forced duction and force generation tests. Review of previous work has highlighted the limitations of designs and methods used. A quantitative apparatus for carrying out both tests has been developed. Stages of development and test procedures are described. The test system described provides a reliable and acceptable means for carrying out both tests.

Test results from normal eyes fell within a relatively narrow range while patients' tests showed that specific ocular muscle deficits produced different patterns of results falling outside the normal range, and these were specific to the underlying pathology.

PREFACE

Part II of this thesis deals with the single subject of mechanical assessment of the extraocular muscles. Due to the length of the section, and for reasons of clarity and convenience, it is presented as five chapters:

Chapter 10: Introduction

Chapter 11: Aims of Study

Chapter 12: The Development of an Instrument for Quantitative Forced Duction and Force Generation Tests

Chapter 13: Results

Chapter 14: Discussion and Conclusions

CHAPTER 10

MECHANICAL ASSESSMENT OF THE EXTRAOCULAR MUSCLES: THE FORCED DUCTION AND FORCE GENERATION TESTS

10. MECHANICAL ASSESSMENT OF THE EXTRAOCULAR MUSCLES: THE FORCED DUCTION AND FORCE GENERATION TESTS

10.1 INTRODUCTION

Facial trauma is often associated with ocular disorder (Petro et al, 1979). Frequently, this presents as diplopia which is most common with blow-out fractures of the orbit (Miller and Tenzel, 1967). It may be due to mechanical restriction of an extra-ocular muscle; neurological, due to partial or complete damage to the nerve supply; or simply due to orbital haemorrhage or oedema (Helveston, 1977).

Orbital tissue incarceration often causes restriction of ocular movements, significant discomfort on attempted rotation of the eye, and deviation from the primary position. Paresis of an extra-ocular muscle and orbital haemorrhage and oedema may produce similar findings (Metz et al, 1974). It may therefore be difficult to assess the functional status of an extra-ocular muscle solely on the clinical findings. The differentiation of the contribution of the different causative factors of the ophthalmoplegia, would be valuable in planning surgical procedures (Yee, 1983).

Quantitative studies, such as the forced duction and the force generation tests, are helpful in detecting mild, subclinical impairment of eye movements and in following the clinical course of motility disorders, such as cranial nerve palsies (Yee, 1983). As mechanical tethering and muscle weakness can coexist, investigation of both components is needed.

Over the past 20 years, a group of tests has been developed which have added to our understanding of strabismus and have assisted in formulating a rational treatment plan. New instrumentation has helped to quantify these procedures for more meaningful analysis. The tests include forced duction, active force generation, differential intraocular pressure, and saccadic velocity studies.

10.2 LITERATURE REVIEW OF THE FORCED DUCTION AND FORCE GENERATION TESTS

10.2.1 The Forced Duction Test

Qualitative Evaluation

The forced duction or traction test, first described by Wolff (1900) in a patient with Duane's syndrome and then by Jaensch (1929), is the primary diagnostic determinant of mechanical restriction. The test is based on the qualitative feeling of resistance to passive ocular rotation when the examiner grasps the eye at the limbus and rotates the globe in the direction of rotational limitation.

Dunnington and Berke (1943) described the forced duction test in patients with exophthalmos caused by chronic orbital myositis. They indicated that when the test was positive, it ruled out paralysis and disclosed the restrictive nature of the limitation of ocular motility.

O'Connor (1943) reported that a positive forced duction test indicated the contracture of an extraocular muscle following paralysis of its antagonist muscle.

Scobee (1948) also recognised the importance of mechanical factors in strabismus in young children. His technique for rotating the globe was to place a muscle hook in the conjunctival fornix and depress it.

More recently, Goldstein (1964) concluded that the forced duction test would indicate the presence of mechanical factors in any case of limited ocular rotation. He emphasized the need to perform preoperative forced ductions where limitation is noted and to perform an intraoperative forced duction after the suspected muscle and surrounding tissue have been detached from the globe.

Quantitative Evaluation

Schillinger (1966) was the first to describe an instrument to quantitate forced duction measurements. The instrument consisted of a spring attached to a hook. The hook was connected to sutures sewn through the limbal sclera and was used to rotate the globe. Schillinger believed that over- and under-corrections in horizontal strabismus surgery could be minimized by balancing the mechanical forces necessary to rotate the globe laterally and medially. Although this contention has never been proved, this work presented an interesting method of quantitating forced duction measurements.

Shortly thereafter, Stephens and Reinecke (1967) described a more accurate method of quantitating forced ductions. They used a Storz perilimbal suction cup on the globe attached by means of a silk suture to a small hook on the diaphragm of a force-displacement transducer which had been calibrated in grams. Following the instillation of a local anaesthetic drop, the suction cup with the surgical silk is placed on the eye and is connected to 70-80 mmHg of negative pressure by means of an aneroid device. A Sanborn 150 amplifier and writer was used to obtain permanent records. The transducer was held in such a way that a direct pull could be exerted on the cup and eye. This was then pulled via the hand held force-transducer until the point under observation moved the desired distance of 0.5 cm.

The force required to rotate the normal eye 0.5 cm medially or laterally from the primary position ranged from 24-98g, while a force of over 100g was required in the individuals with restricted eye movements. The working angle of the instrument was small and the standard deviation of the recordings in normal subjects was large. The eye was rotated by a linear rather than a rotational movement and the procedure required a significant amount of cooperation on the part of the subject which limited its use in children.

Robinson et al (1969) and Collins et al (1969) reported on quantitative forced duction measurements in strabismus patients undergoing surgery under topical anaesthesia. They used a strain gauge connected to the lateral rectus and then to the orbital tissues through a surgical silk thread after both the medial and lateral rectus muscles had been detached from the globe. They carried out measurements on five patients prior to strabismus corrective surgery. Stiffness of the orbital tissues to rotation was found to be nearly linear and measured approximately 0.5g/deg of rotation.

As this technique was used during surgical management, the forces measured were for the detached muscle and under anaesthesia.

In Japan, Kaneko and associates (1971) have also devised an instrument for quantitative forced ductions. They analysed the length-tension relationships in normal individuals and compared this curve with measurements taken in patients with mechanical restrictions. A very steep curve was found as a result of these mechanical restrictions.

Strachan and associates (1975) described the development of an apparatus for measuring muscle forces in strabismus. A suction cup without a flange, to reduce slippage, was used to fix the globe by applying negative pressure using a syringe and a fine polyethylene tube. The forces were measured with two strain-gauges which were wired into a balanced bridge circuit. The reading from this instrument was displayed on a storage oscilloscope. The angular displacement of the eye was measured by using a small pocket flashlight attached to the handle of the apparatus with a condensing lens in front of it so that the filament of the bulb is focussed on a tangent screen to act as a light pointer during passive duction testing.

The suction cup is applied to the eye after surface anaesthesia is induced. A force generation test is performed by asking the patient to make a saccade with the fellow eye to a particular point on the tangent screen while holding the eye under test in a fixed position. The force duction test is performed either with the free eye in the primary position or with the muscle under test in a fully relaxed state. The rotation of the globe is measured with the light pointer on the tangent screen and a reading of the force required to displace the globe is taken for each point investigated. A graph may be constructed of force against displacement. Values of 1-1.5g/deg were found for horizontal excursions in normal subjects.

The suction device for creation of the negative pressure was inadequate and did not have the facility for measurement. The number of normal subjects used to obtain the data was not specified, and no patients were studied to show the usefulness of the device.

Knox (1974) also described a suction cup device to move the eye passively in a desired direction. Topical anaesthetic drops were used and the suction cup was placed against the eye and held in place by -70 mmHg suction. Occasionally, suction was increased to -90 or -100 mmHg in an attempt to increase adherence and to move the eye in cases of great limitation of movement. Usually this did not produce any better movement. At that time, no method for quantitating this test had been developed.

Rosenbaum and Myer (1980) developed a spring gauge device which could be utilised for quantitative forced duction measurements. The instrument attaches to the limbal conjunctiva with the end of a self-retaining Pierce forceps. This is connected through a lever arm to a spring gauge which displays directly the force produced in grams. The instrument was easy to use and claimed to be accurate. However, the self-retaining forceps will not maintain its hold beyond 50g of force, a disadvantage in certain restrictive disorders where the tension required to produce rotation may be above this level.

Collins et al (1981) utilised a specially instrumented forceps to obtain a measurement of force up to 110g and forceps-positioned eye displacement up to 20 mm. Semiconductor strain gauges were mounted on the narrow beams of a large Pierce straight fixation forceps (Storz Model E 1942-13). Measurements of the applied forces at right angles to the long axis of the forceps were obtained. A small microphone with an opening near the tip of the forceps detected inaudible sound pulses from an ultrasonic source attached to the head of the subject. This ultrasonic device served as a sonar system and provided a constant indication of the distance between the ultrasonic source, which was mounted on the frame of a pair of glasses, and the forceps tip. Position accuracy was within +/-3% over the range of +/-50 degrees of eye position.

An X-Y oscilloscope was used to display force as a function of eye position. These data were simultaneously recorded on a multitrack FM tape recorder for later play-back on an X-Y chart recorder to provide a permanent record. The force and displacement channels of the length-tension forceps were zeroed following the instillation of topical anaesthetic drops. For a measurement of the orbital load due to a temporal rotation, the subject was directed to fix the gaze of his right eye on a 30° target to the right. The nasal limbus of the left eye was grasped with the forceps. The left eye was then slowly rotated from its 30° right position temporally to 30° or more left and returned to its starting position. This slow (10 deg/sec) duction movement resulted in a graphic plot of the load (stiffness) characteristics of the left ocular motility.

To measure active medial rectus forces, the left eye was held steadily in the extreme 50° abducted position while the subject tracked a smoothly moving target with his free right eye. The target was a small bright red spot projected from a 0.3 mm helium-neon laser source and was moved electronically, at a constant rate of 10 deg/sec, from the 50° left gaze position to the extreme 50° right gaze position and was then returned to the primary position. This procedure was performed two or three times. The left eye was then released. The forces required for a nasal rotation were obtained in a similar fashion.

Forces varied from 0.8 to 1.7g/deg for nasal rotation, and 0.77 to 1.2g/deg for temporal rotations. The active force for the medial rectus at maximum excursion (100 deg) ranged between 48 and 103g, while for the lateral rectus it was between 45 and 92g. The study measured the stiffness associated with innervated muscles, not passive ones. Again, the use of forceps creates trauma. Only linear rather than rotational displacement was measured, and it is not clear how the authors managed to maintain a steady displacement at 10 deg/sec.

More recently, Metz and Cohen (1984) have developed a strain gauge force transducer for quantitative forced duction measurements during surgery under general anaesthesia. A forceps is mounted in a thick plastic holder with a stainless steel hook clamped at the flexible tipped end. The hook engages a small loop of silk suture sewn through partial thickness sclera at the limbus (at the 9 and 3 o'clock meridians for horizontal movements and at the 12 and 6 o'clock meridians for vertical movements). The horizontal or vertical projection of the angular rotation is measured by a millimetre scale with the limbus as the index of movement. An ink mark on the lid and brow is used as a point of reference. The force in grams necessary to achieve the movement is read directly from a digital readout. The strain gauge force transducer is gas sterilised for use in the operating theatre. Tension was measured as the globe was rotated horizontally (or vertically) 3 mm, 5 mm, 8 mm and 10 mm from the resting position. Distance was measured visually by aligning the limbus with a ruler and three separate trials were made. Measurements were made in children and adults who had eye surgery other than strabismus repair (cataract, glaucoma, nystagmus surgery) to obtain normal values. In strabismus patients, measurements were made prior to operation, after muscle disinsertion and following a recession or resection procedure. The length-tension curve in various restrictive problems such as thyroid ophthalmopathy and orbital floor fracture was steep when compared with that of patients having non-restrictive types of deviation.

Modification of this equipment using a frame and track has led to more accurate measurements of the distance rotation. Computer interface hardware and associated software was introduced later. However, this modified instrument was bulky and proved more difficult to use.

In the same year, 1984, Miller and Kalina designed a simple suction cup which allowed convenient and comfortable forced duction testing. The device used a small rubber suction bulb connected directly to a specially designed transparent scleral contact lens which vaulted the cornea but was applied firmly at the corneoscleral limbus. The suction bulb shaft was angled 20° to avoid the orbital rim or the nose.

After instillation of a topical anaesthetic, the eyelids are separated and the suction cup is centred over the eye with the shaft angled away from the direction of impaired duction. While holding the scleral surface of the device against the globe with gentle pressure, the examiner slowly squeezes and then releases the suction bulb. Squeezing the suction bulb releases the vacuum and allows removal of the instrument at the end of the test. However, there is no control over the amount of negative pressure created between the suction cup and the eye. Again, there was no quantification in the measurement procedure.

10.2.2 Active Force Generation Test

Review of Past Experience

The forced duction test provides information about mechanical limitations to ocular rotation but not about active voluntary forces. Extraocular muscle weakness may be inferred when the globe can be rotated significantly further by forced duction than by voluntary gaze. However, when significant restrictions are present, the forced duction test does not assess the innervational status of a muscle.

In 1970, Madroszkiewics described the technique of oculodynamometry, which he introduced in 1952, to measure the strength of the extraocular muscles (force generation test) under local anaesthesia. Four-muscle-tendon sutures are performed and 4 loops, about 2 cm in length each, are made. The oculodynamometer is hooked to one of the suture loops and held immobile and slightly stretched while the eye is in the primary position of gaze. The patient is then instructed to try to move his eye to the extreme gaze in the direction of action of the muscle in question while the dynamometre measures the active forces generated by the muscle. The author investigated the contraction of the normal active extraocular muscle and recorded the initial strength at about 54-55g and the maximum strength at 60-61g. In convergent squint, he found a great difference in the strength between the medial rectus and its antagonist which may reach 20 or more grams. He concluded that it was possible to calculate the work performed by normal and abnormal muscles. He felt that this was an important aid in determining the proper operative therapy of convergent strabismus.

Although the test system was for force generation measurement, it was not comfortable for patients, and did not provide a facility to perform the forced duction test.

Scott and associates (1972) described the development of load-sensing forceps, which were modified in a manner akin to a torque-wrench, for quantitative assessment of the active muscle forces which could also be used to quantify the forced duction test. The forced duction test was performed when the patient's eye was in the primary position. The eye was grasped by forceps, held perpendicular to the conjunctiva at the limbus, and moved in the direction of choice. A ruler, taped near the eye, was used to estimate the distance of eye movement in the frontal plane. The forceps scale reading was recorded in a cassette recorder at each measurement of 1 mm of eye movement, and the length and tension subsequently plotted.

For the force generation test, the eye was again grasped at the limbus with the forceps held perpendicular to the globe. The contralateral eye followed a target presented by means of a perimeter to provide an accurate measurement of the intended angular rotation. The eye under examination was restrained from moving and the change in scale reading was recorded as a function of position of the contralateral eye. The test was performed under topical anaesthesia.

The average resistance to passive rotation was 1g/deg (range=0.5-1.75g/deg) in normal subjects and in strabismus patients without gross restriction. When restriction was present, stiffness was measured at between 1.5g/deg and 5g/deg of passive rotation. Active force generated by normal muscles for extreme lateral or vertical gaze ranged from 60 to 80g. Supranormal active forces were measured in muscles working against restrictions of the antagonist muscle as in cases of endocrine exophthalmos and Duane's syndrome. Scott suggested that that information was a helpful guide in planning strabismus surgery. The problems, however, were that the forceps used could cause trauma and measuring the displacement with a ruler was not an accurate method.

10.2.3 Alternative Methods

Differential Intraocular Pressure

A drawback of the forced duction test in the awake subject is that patient cooperation is required as there may be minor discomfort with the technique. In addition, without quantitative instrumentation, the method is dependant on the examiner's experience, interpretation and bias.

Helveston (1980) and Zappia et al (1971) reported on a differential intraocular pressure test to overcome these difficulties. It was thought that in the presence of restriction, the healthy agonist muscle contracted and the tight antagonist muscle or the surrounding tissue failed to slacken, thus exerting increased pressure on the globe. A paralysed muscle fails to move the eye in the intended direction when it acts as an agonist, but the intraocular pressure remains the same. This pressure was measured with the pneumotonometer, comparing measurements made in the primary position with those made with the eye attempting rotation in the vertical gaze at 15° up and 15° down.

The average change in pressure in 54 normal eyes from the primary to 15° upward gaze was 0.38 mmHg (SD=+/-0.8 mmHg), while from the primary position to downward gaze the corresponding values were -0.34 mmHg (SD=+/-0.9 mmHg).

An increase of 3 to 11 mmHg was found in patients with orbital myositis, orbital floor fractures with entrapment, and thyroid ophthalmoplegia.

Six patients with orbital blow-out fractures, two of them with entrapment, had increases of intraocular pressure between 7 and 10 mmHg, while four others exhibited elevations of up to 3 mmHg. In one third of the eyes of patients with thyroid ophthalmoplegia in one eye, which clinically had no limitation of motion, the differential intraocular pressure test showed a rise of 3 mmHg or more, indicating mild forms of muscle involvement.

The method described was limited to vertical eye movement only and was only effective in delineating gross restriction. Furthermore, it was difficult to carry out the test in infants and young children.

Saccadic Velocity Test

Saccades are rapid eye movements from one fixation point to another. This can be horizontal, vertical, or oblique. The rectus muscles appear primarily responsible for saccadic eye movements (Scott, 1975).

Electromyographic and mechanical recordings during a saccade show a large motor unit input (Robinson, 1969). Rectus muscle weakness diminishes this large input resulting in a proportionally slower saccade (Metz, 1983). When restrictions limit ocular rotation, saccades remain rapid (Metz, 1979). This has been shown in thyroid ophthalmopathy (Metz, 1977), orbital floor fracture (Metz et al, 1974), Brown's syndrome (Metz, 1979) and rectus muscle contracture following paresis of the antagonist muscle (Metz, 1983).

Saccadic velocity has been measured as a means of assessing paralysed musculature by Metz (1985) and by Francois and Derouk (1955) who were the first to suggest the clinical usefulness of the saccadic velocity with regard to the severity of muscle paralysis and the recognition of the return of function during recovery. Mackensen (1966) pointed out similar findings.

Saccadic velocity was measured with electrooculography (Metz et al, 1985). Voluntary saccades were made by asking the patient to look back and forth between two targets. The targets were separated vertically or horizontally at a known distance apart, allowing calibration of the eye movement recording.

Normal saccadic eye movements at least 20° in amplitude having velocities between 200° and 350° per second were recorded while reduced amplitude of rotation and a corresponding slowness of saccades were noted in patients with sixth-nerve paresis and others with third-nerve palsy. Patients with inferior rectus muscle palsy were 46% to 275% faster for upward saccades than downward saccades (Metz, 1980).

Although saccadic velocity measurement was easy to use, it did not give an indication about the passive behaviour of the globe and the surrounding tissue.

10.2.4 Summary of Previous Experience

Table 10.1 summarizes the previous investigative attempts to quantify the forced duction and force generation tests.

Source	Coupling	Device	Measured Forces
Wolff (1900)	-	-	-
Jaensch (1929)	Forceps	-	-
Dunnington & Berke (1943)	-	-	-
Scobee (1948)	Muscle Hook	-	-
Goldstein (1964)	-	-	-
Schillinger (1966)	Muscle Hook	Calibrated spring	-
Stephens & Reineck (1967)	Contact lens	Force displacement transducer on a linear track.	N: 10-42 mN/deg P:Over 42 mN/deg
Robinson et al (1967)	Contact lens	Force transducer	N:12 mN/deg
Robinson et al (1969)	Suture	Force transducer	Strabismus 4.9 mN/deg while both recti detached.
Madroszkiewicz (1970)	Suture	Spring + Hook	N:589-598 mN FGT Strabismus 785 mN
Scott et al (1972)	Forceps	Load sensing forceps + Millimetre ruler.	N:5-17mN/deg FGT 13.7mN/deg, P:15-49 mN/deg
Knox (1974)	Contact lens	-	-
Strachan et al (1975)	Contact lens	Strain gauge mounted on a syringe + flashlight on the apparatus handle.	N: 9.8 mN/deg.
Rosenbaum (1980)	Forceps	Spring dynamometer	N:3-4 mN/deg, P:7-20 mN/deg.

Table 10.1 Continue

Collins et al (1981)	Forceps	Force transducer on a forceps + ultrasound transducer.	N:8-17 mN/deg med, 7.6-12 mN/deg lat. FGT:0.942 mN/deg med, 1.049 mN/deg lat.
Metz & Cohen (1984)	Suture	Calibrated forceps + millimetre scale.	N:9.81 mN/deg and P: 14.7 mN/deg. while FGT
Miller & Kalina (1984)	Contact lens	-	-
Zappia et al (1971) & Helveston (1980)	Contact lens	Pneumotonometer.	
Metz (1985)	Electrodes	Electro-Oculography.	IR palsy 46-275% up faster than downward.

N: Normal control subjects P: Patients

Table 10.1 Previous attempts to develop FDT and FGT equipments.

CHAPTER 11

AIMS OF STUDY

11. AIMS OF STUDY

11.1 LIMITATIONS OF PREVIOUS QUANTITATIVE TESTS

The aim of the forced duction test is to determine the force required to rotate the eye passively in different directions of gaze. This requires a comfortable firm attachment to interface the eye with the force sensing device.

Review of work in this field has highlighted severe limitations with many of the methods previously used. Classically, the test is carried out using a pair of forceps to grasp the limbal conjunctiva in order to rotate the eye away from the direction of function of the muscle in question. This procedure is uncomfortable and frequently leads to subconjunctival haemorrhage or laceration (Metz and Cohen, 1984; Collins et al, 1981; Scott et al, 1972). The use of a self-closing clip (Rosenbaum & Myer, 1980) is equally harmful. Using surgical sutures instead (Madroszkiewics, 1970; Schillinger, 1966) can only be performed under general anaesthesia and therefore has its limitations.

To overcome this problem, a variety of suction cups were devised which can be applied to the anterior surface of the globe and maintained in position by creating vacuum in between (Robinson, 1975; Strachan et al, 1975; Knox, 1974; Stephen & Reinecke, 1967). This method has the advantage of firm coupling without the risk of eye trauma. However, none of the previous suction cups used was entirely satisfactory. Miller & Kalina (1984), for example, developed a suction cup which was huge and therefore allowed only a limited amount of displacement of the eye. Moreover, the system for creating, maintaining and monitoring negative pressure was not entirely adequate.

Accurate recording of tension and distance of rotation is another important point in the development of a reliable system. Pulling the eye through a suction cup using a surgical suture (Metz, 1985) does not allow accurate measurement of the direction or distance of rotation. Furthermore, the procedure may inadvertently lead to the eye being pushed backwards simulating full rotation by retraction. This fact has been noted by other authors who emphasised the point that the design of the forced duction apparatus should take into consideration the natural arc of rotation of the eye (Metz, 1976).

The force generation test poses lesser problems than the forced duction test (Scott et al, 1972). Still, accuracy cannot be guaranteed with a purely mechanical instrument which requires regular calibration against a more reliable equipment.

As the review in the previous chapter shows, most of the described techniques and designs were not entirely successful in producing painless, accurate and reproducible measurements. This was due to the lack of appropriate engineering concepts to overcome the obstacles of coupling between the eye and the force-sensing device, the rotational eye movement, the anatomical barriers of the brow and bridge of the nose, and the achievement of uniformity of test speed.

11.2 OBJECTIVES OF THE PRESENT STUDY

On account of the above shortfalls of previous work, it was decided to attempt to develop a painless, accurate and calibrated equipment which allows these tests to be performed as an outpatient procedure both by the ophthalmologist and, hopefully, the non-ophthalmologist.

The main objectives of the work described in this chapter are:

1. To design an integrated clinical system for quantification of the forced duction and force generation tests , which would:

   a. Provide a method of non-traumatic coupling between the eye and the force-sensing device which does not influence the intraocular pressure to an extent which places the eye at risk of acute ocular hypertension.

b. Follow the natural arc of eye movement.

c. Incorporate the facility for measurement in a full range of all meridians.

d. Ensure that only the intended (and measured) forces are applied to the eye, and with minimum artefact.

e. Allow recordings to be made quickly, both from the standpoint of clinical utility and, (with regard to the use of the suction cup), to prevent a prolonged intraocular pressure rise. A 10-minute limit has been recommended (Knox, 1974; Stephen & Reinecke, 1967).

f. Provide real-time force-rotation measurements to give the examiner an immediate indication of the success of individual tests as they are performed.

g. Have an on-line recording facility for the collected data which can be coded with the subject's details and used for comparison/examination on future occasions.

2. To use the equipment to carry out:

a. Pilot studies on normal control subjects to establish a database of normal ranges of force-displacement response.

b. Investigations which allow comparisons to be made of the similarities and differences between normal subjects and patients with a range of specific abnormalities.

CHAPTER 12

THE DEVELOPMENT OF AN INSTRUMENT FOR QUANTITATIVE FORCED DUCTION AND FORCE GENERATION TESTS

12. THE DEVELOPMENT OF AN INSTRUMENT FOR QUANTITATIVE FORCED DUCTION AND FORCE GENERATION TESTS

The system which has been developed was designed and produced in collaboration with Mr M F Al-Hinnawi from the department of Bioengineering at Strathclyde University, Glasgow. Each component of the prototype design was developed as a sequel to observations made at previous design stages.

12.1 CONTACT LENS AND VACUUM SYSTEM

The system consisted of a transparent 15 mm suction cup contact lens (Storz) with a 5 mm peripheral flange which was later reduced to 3 mm to prevent slippage (Fig 12.1). To ensure good contact between the lens and the globe and to minimize relative slippage, negative pressure between the cup and the eye is created via a hollow plastic evacuation tube inserted at a 25° angle through the lens. Later versions incorporated a hollow central plastic tube (Fig 12.2). The tube is connected via a soft Y-shaped silicone rubber tube to a 40 ml syringe through a reservoir on one end (Fig 12.3) while the other end is connected to port B of an electronic strain-gauged pressure transducer (SCX DN - Rugby, UK). The pressure between the contact lens and the eye can be reduced by depressing the plunger of the syringe, and maintained by

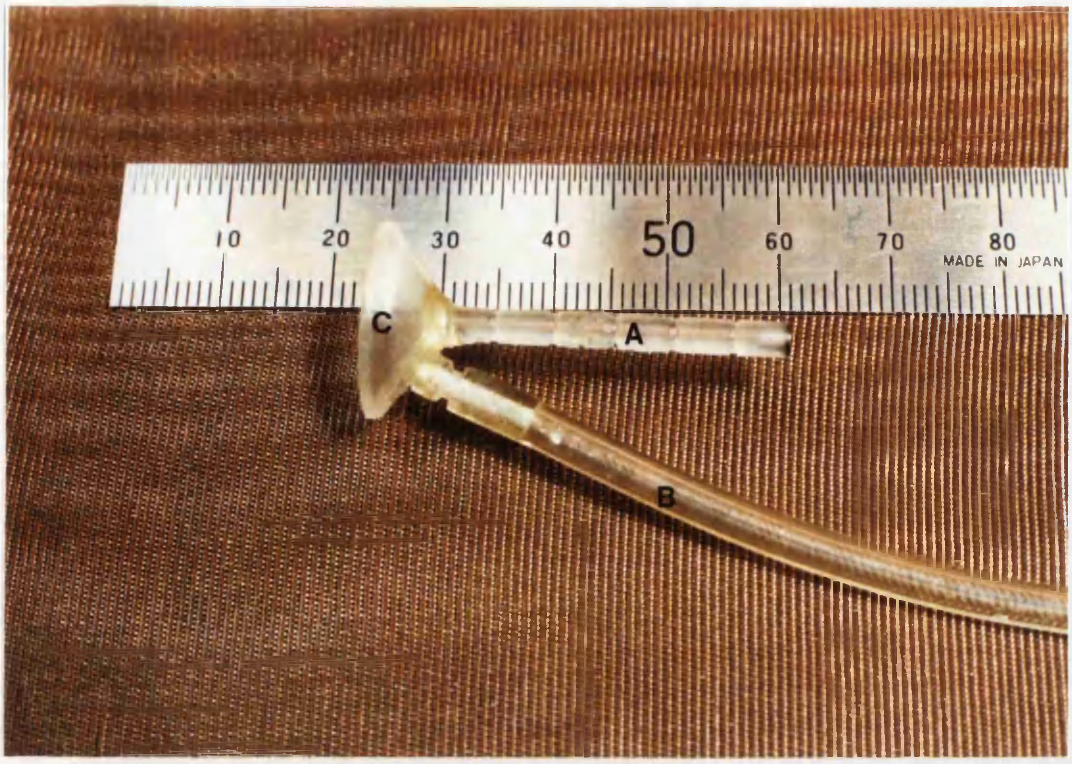


Figure 12.1 The suction cup (contact lens): A-stem, B-suction tube, C-lens.

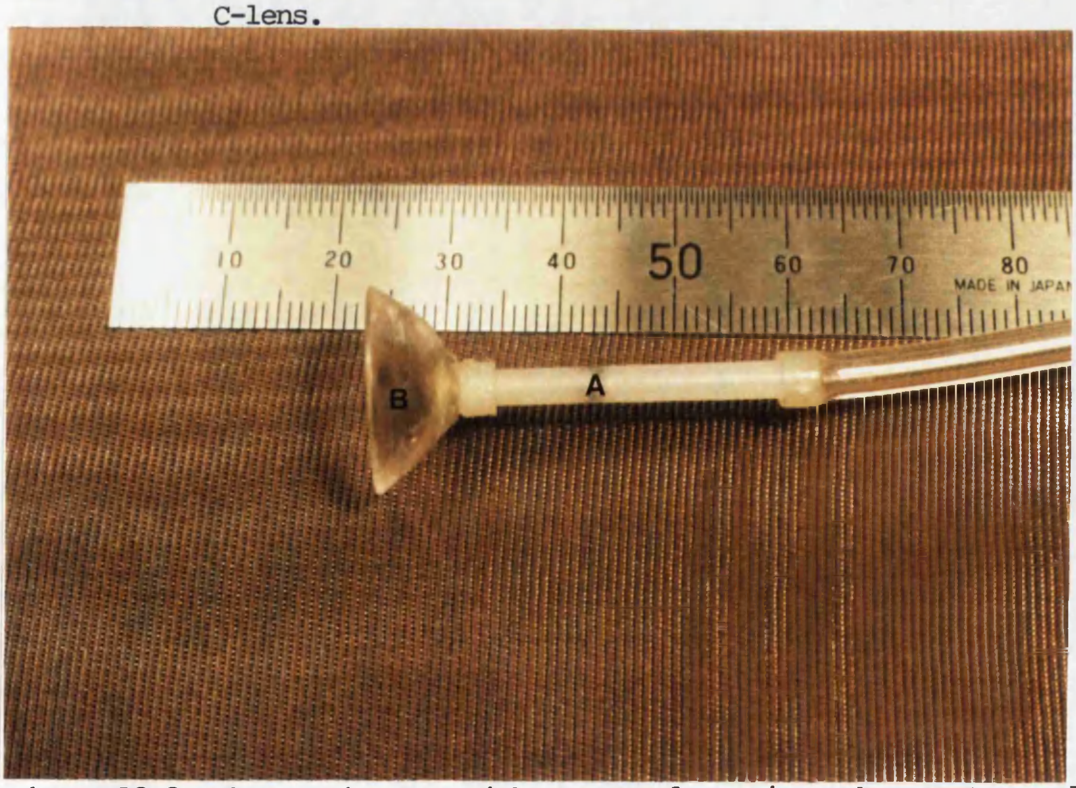


Figure 12.2 The suction cup with a central suction tube: A-stem and suction passage, B-contact lens.

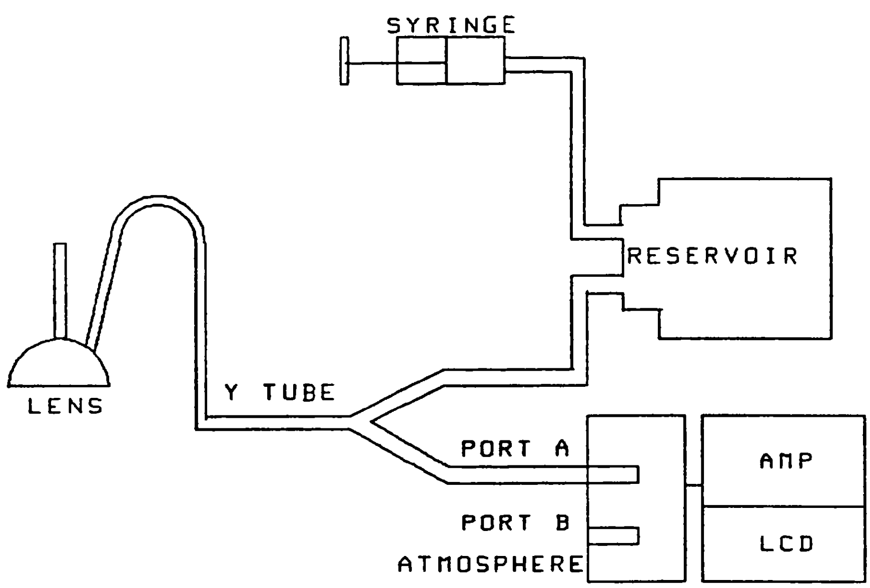


Figure 12.3 The pressure reduction system.

inserting a stent in one of the drilled holes in the plunger. The pressure is monitored by a digital display meter showing the output of the pressure transducer.

To the centre of the convex surface of the suction cup, a solid plastic rod which is 30 mm long and 3 mm in diameter is glued. This serves to attach the eye indirectly to the load measuring device. The length of 30 mm was chosen for the attached rod to provide the potential for upward and medial rotational forces to be applied over the brow and nose respectively.

12.2 PROTOTYPE "A": THE STRAIGHT TRACK DESIGN

12.2.1 Design Description

A force transducer (SMD 0100-2110-10 Suffolk, UK) was mounted on carriage (A) (pinion) travelling on a linear track (rack) (Fig 12.4). The body of the linear potentiometer (B) (LP-20F Midori Precision, Japan) was coupled with the main body of the rack (C), while the leg of this potentiometer (D) was connected to the moving carriage. Turning a knob (E) moves the carriage, and hence the transducer, along the rack, bringing the leg of the potentiometer with it.

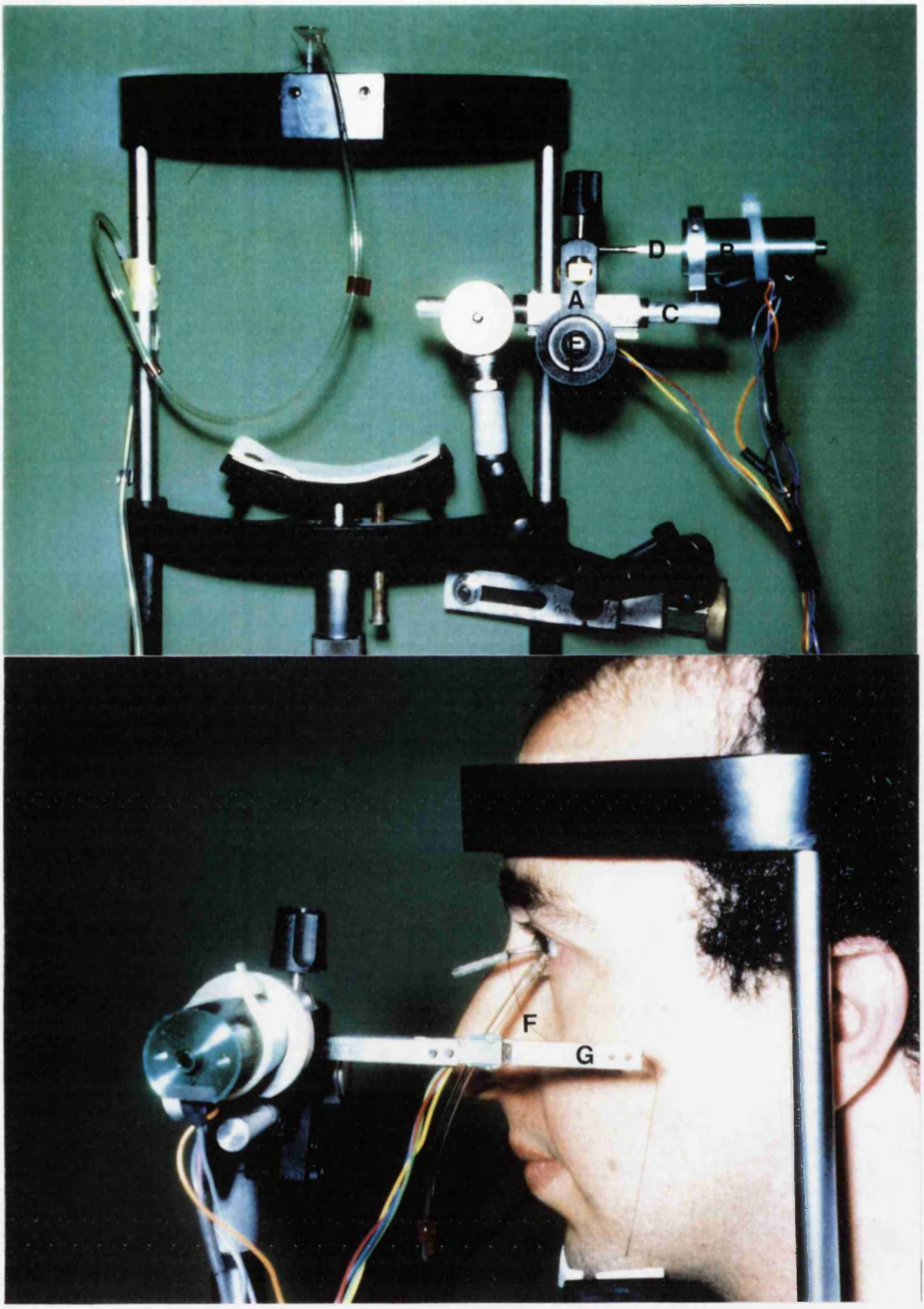


Figure 12.4 The linear track design: A-carriage on a pinion, B-the body of the potentiometer, C-the rack, D-the potentiometer leg, E-knob, F-2/0 surgical silk, G-force transducer.

A 2/0 surgical silk loop (F) was glued to the stem of the contact lens and tightened to the tip of the force transducer (G) at a predetermined point (Fig 12.4). The force transducer measures the tension in the silk which has been created by pulling the eye.

The equipment was mounted upon a flexible metal bar (H) which could be fixed to either of the side bars of the slitlamp microscope.

The outputs of the force transducer, via the amplifier, and of the potentiometer are transmitted to an X-Y plotter (Fig 12.5).

12.2.2 Test Procedure

Following the instillation of a drop of a topical anaesthetic (Minims Benoxinate 0.4%, Smith & Nephew), the subject rests his chin on the chinrest of the slitlamp microscope. The sterile contact lens is then placed on the eye and a vacuum is created in between by depressing the plunger of the syringe until the desired negative pressure of 70-80 mmHg is obtained. The subject is asked to look straight ahead at a fixed target with the fellow eye.

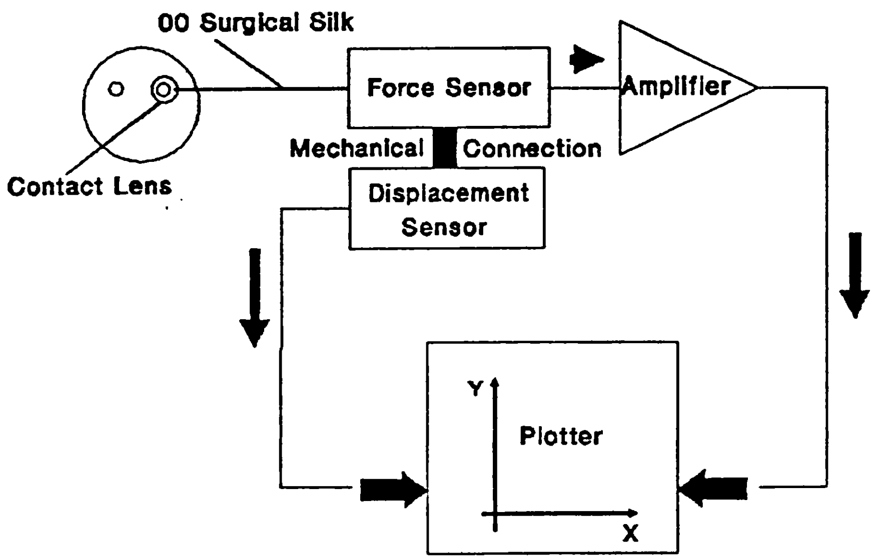


Figure 12.5 Diagram of the system's components for the linear track design.

When the instrument is correctly aligned with the eye, the X-Y plotter is initiated to start recording both components of the test from the zero position. Manual rotation of the knob E (Fig 12.4) moves the eye in the intended direction whilst the plotter records the force-displacement curve. The test is repeated 3 times in each direction.

All investigations are carried out within ten minutes otherwise, the test is discontinued to avoid any potential rise of the intraocular pressure (Knox, 1974; Stephens & Reineck, 1967). The procedure is terminated by releasing the tension on the surgical silk, abolishing the vacuum and removing the lens.

12.2.3 Results

Six subjects with normal eyes volunteered for the test. The test was carried out and recorded 3 times in each direction of investigation and an average value of the three readings was calculated.

The instrument was not successful in rotating the eye beyond 20° of eye rotation due to dislodging of the contact lens. In many subjects the eye could not be rotated more than 4 mm (8-10°). The results were not reproducible for the three attempts in the same direction for the same subject. Forces recorded for rotations through 5° were more consistent than others and ranged between 150 mN (15g) and 250 mN (25g). Figure 12.6 shows the results of three successive tests in the lateral direction for the only three subjects for whom sufficient data could be obtained.

It became clear that the angle between the suture and both the load cell beam and the stem attached to the contact lens could not be maintained at a right angle throughout the procedure. This had major effects on the forces applied to the eye and measured by the load cell. When the suture is not perpendicular to the load cell and contact lens stem, the force (F_c) measured by the load cell is not the force (F) in the suture. There is also an antero-posterior force component (F_s) applied to the eyeball in addition to the desired tangential force (F_t). Figure 12.7 shows a schematic diagram of the forces produced by pulling the eyeball using the linear device.

12.2.4 Criticism of the Design:

The design was unsuccessful in performing adequately the forced duction and force generation tests for the following reasons:

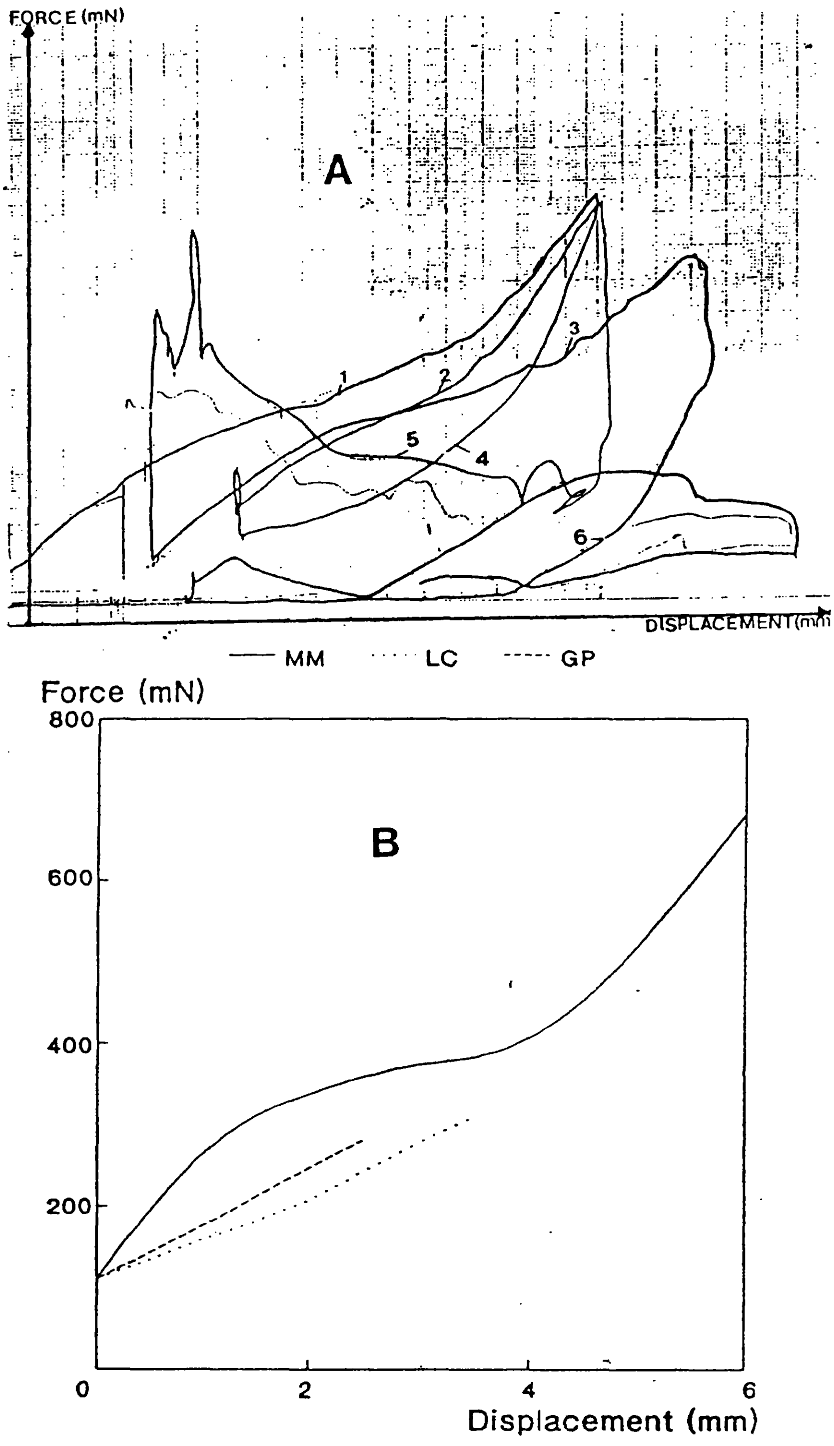


Fig 12.6 Examples of FDT results obtained from control subjects using the linear track design in the lateral direction

A) subject AD -note that the repeated recordings are not reproducible and that the unloading curves 4-6 are very irregular.

B) Averaged recordings from the 3 normal control subjects upon whom sucessful recordings could be made.

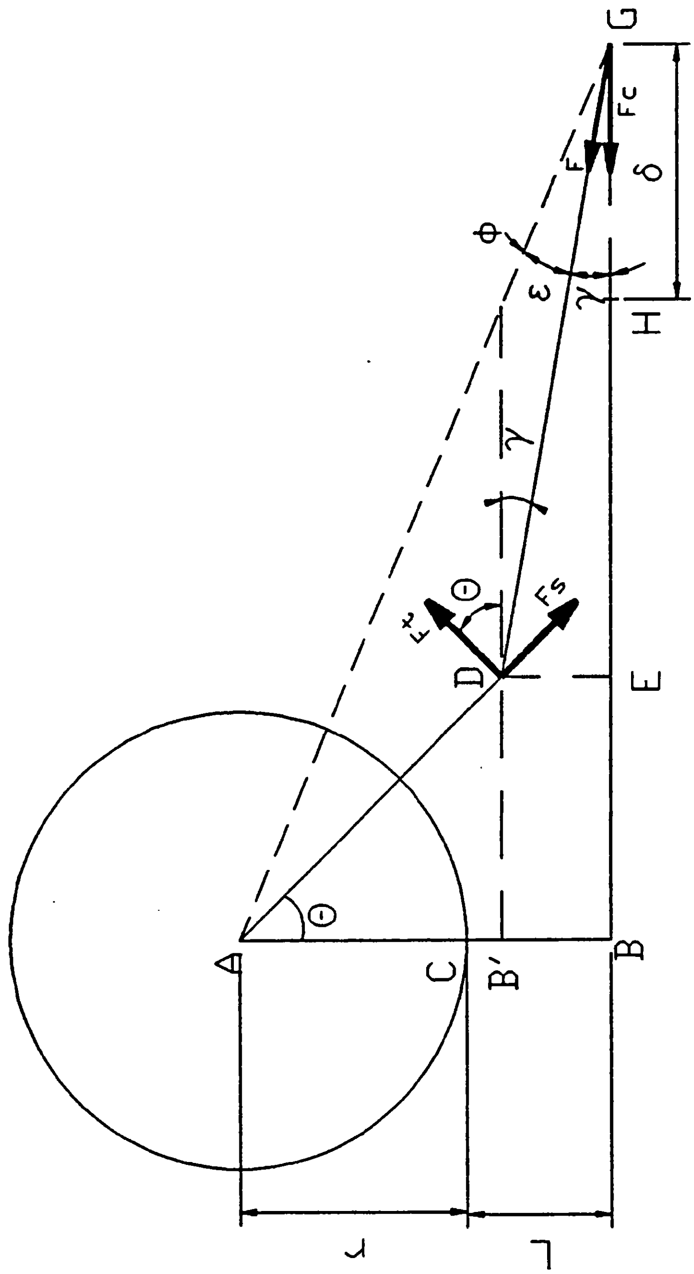


Figure 12.7 Shows a schematic diagram of the forces produced by pulling the eyeball using the linear track device. The system's relevant measurements comprise:

r: The radius of rotation of the eyeball, usually about 13.5 mm.
L: Distance between the surface of the cornea and the silk coupling to the stem. It was 6 mm.
L_2: Length of suture coupling between the stem and force transducer.
δ: The measured linear displacement from the primary position.
θ: The angle of rotation of the eyeball.
F_S: The antero-posterior force component applied to the beam.
F_T: The tangential force component applied to the beam.

1. Slippage of the contact lens beyond 20° of lateral rotation due to the pull of the silk suture. Medial rotation was also extremely limited due to the interference of the nose with the free movement of the suture which also influenced the results obtained.

2. The forced duction test was not possible in the vertical direction because of the bulk of equipment and the inevitable interference of the forehead and cheek with the smooth movements of the silk suture.

3. The track was linear while the eye rotates around its centre of rotation. Hence, the force applied to the eye and measured by the load cell was not the required tangential force. There was an additional axial force component (F_s) which tended to pull the globe out of the orbit, thereby stretching the muscles rather than producing natural eye movements.

4. The relationship between the linear displacement of the potentiometer and the rotational displacement of the eye was not linear.

5. It was impossible to perform the force generation test on the tested muscle since this required re-

orientation of the device, during the procedure, as it became close to the nose or the eyebrow.

12.3 PROTOTYPE "B": THE ROTATIONAL DESIGN

12.3.1 Design Description:

Since eye movements are rotational, the concept of design moved towards building an instrument which produces this displacement.

The rotational design (Fig 12.8) comprised a rectangular metal base (1), 3 x 13 x 1 cm in dimension, which rotated around a shaft axis (2) fitted at the end of this base by means of a roller bearing. In order to measure the rotation of the device, a disc gear (3) was mounted on top of the fixed shaft (2) to transfer the rotation to a second gear (4) which was fixed on the shaft of the potentiometer (5). The force transducer (6) was mounted on the other side of the metal base by means of another shaft (7). Rotation of the shaft (7) causes rotation of the base (1) around shaft (2). This results in rotation of the gear (4) which is coupled with the gear wheel (3). The suction cup was coupled to the force sensor by a piece of 2/0 surgical silk glued to the stem of the suction cup and fixed onto the tip of the force transducer. The outputs of the

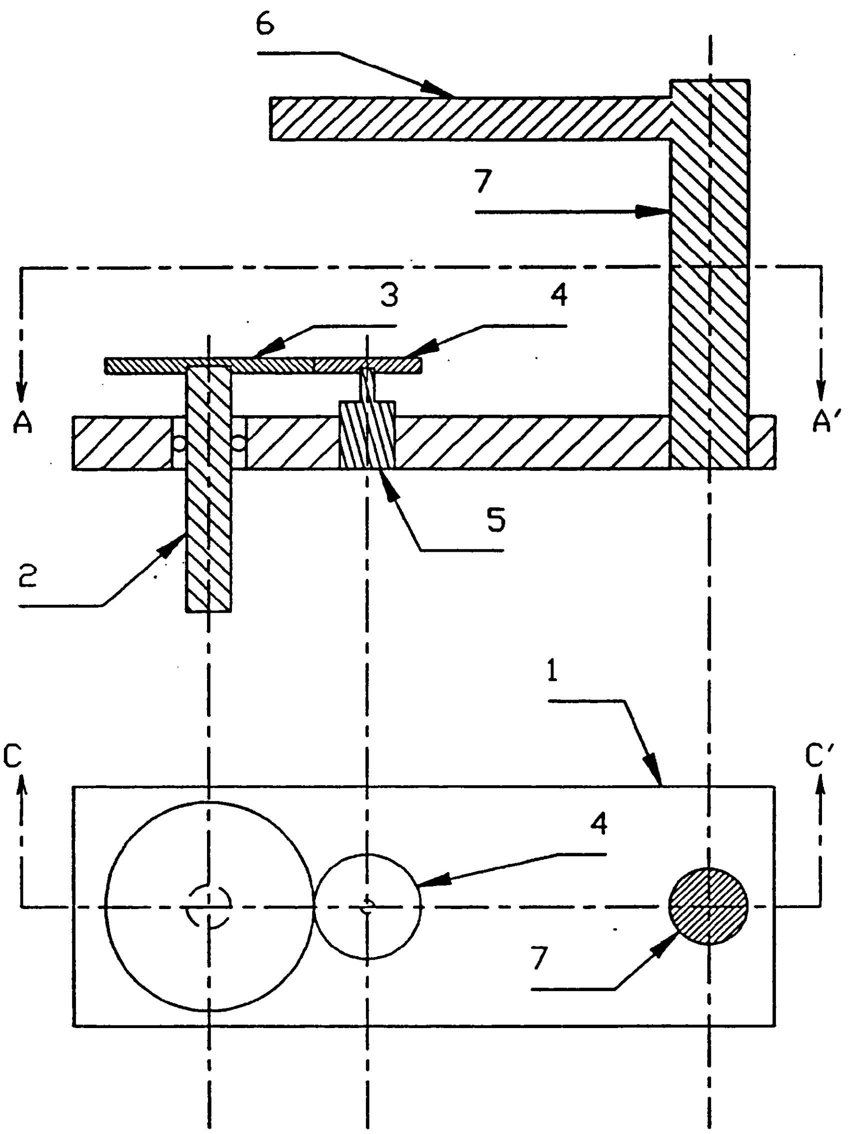


Figure 12.8 The rotational system
1- metal base 2- fixed shaft 3- disc gear 4- gear
5- potentiometer
6- force transducer 7- shaft

force sensor and rotational potentiometer were again recorded on a Y-X plotter (Fig 12.5).

The distance from the tip of the force transducer, where the surgical silk was coupled, to the centre of rotation of the instrument was equal to the radius of rotation of the eye and the length of the stem (Fig 12.9).

BG = BH = AC = AD = r+L where:

r = the distance between the centre of rotation of the eyeball and the cornea.

L = the length of the stem where the surgical silk attached to the load cell is coupled to.

12.3.2 Test Procedure

The test procedure was similar to that of the previous design except for setting up and aligning the test device with the centre of rotation of the eye. The vertical planes passing through the rotational centre of the fixed shaft (2) and the tip of the beam (6) were orthogonal with the one passing through the axis of horizontal rotation of the eye (A) and (B) (Fig 12.9). Both vertical planes passing through BG and AC were parallel.

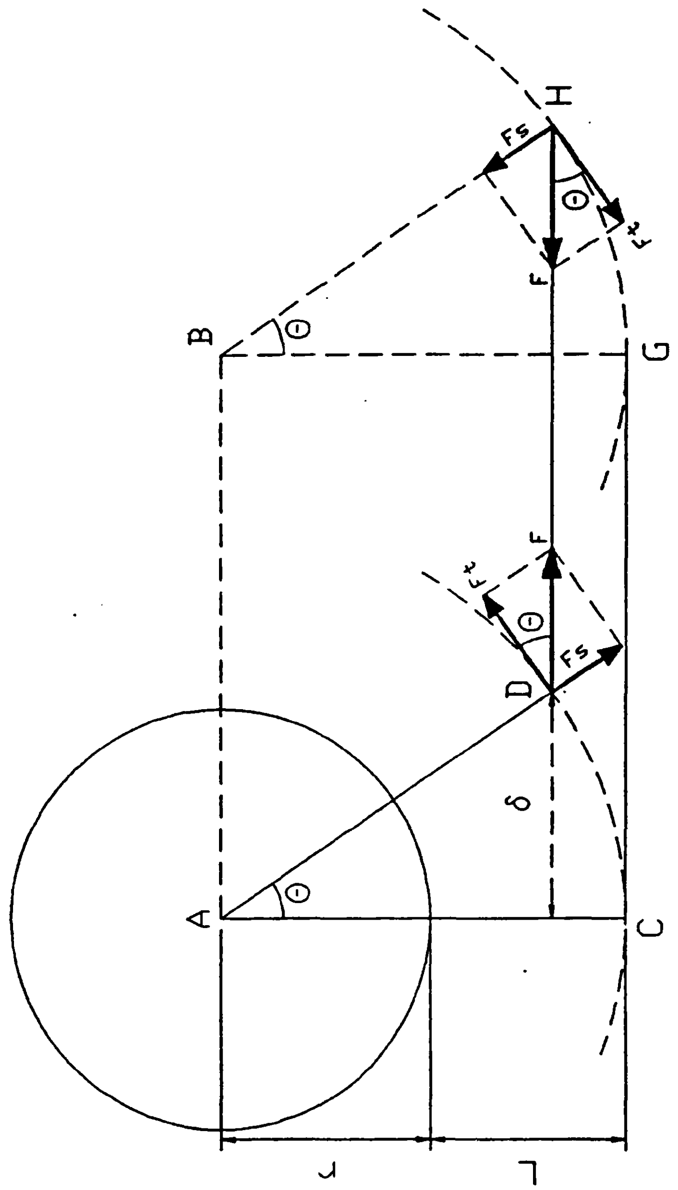


Figure 12.9 Schematic diagram of the forces created by the rotational design.

12.3.3 Results

Nine subjects with normal eyes were tested. It was possible to carry out forced duction tests for 25° of lateral rotation without dislodging the lens. In one case, it was possible to perform the FDT through 45° of lateral rotation. Medial rotation was attempted on five subjects, and could not be achieved beyond 5° because of the interference of the nose with the silk suture coupling the stem and force transducer. The mean value of three readings in each tested direction was calculated (Fig 12.10). Forces necessary to rotate the eye through 5° of rotation ranged between 75 and 150 mN (7.5-15g) and were smaller than those needed in the previous design (250 mN=25g). A degree of reproducibility of data could be obtained from the same subject. Again, the angle between the silk suture and both the load beam and the stem did not remain at right angle.

12.3.4 Criticism of the Design

1. Although this design took into consideration the rotational nature of the eye movement and measured the angle of rotation with the rotational potentiometer, the resultant data continued to represent a combination of two components: the tangential force and an unwanted antero-posterior force. However, in

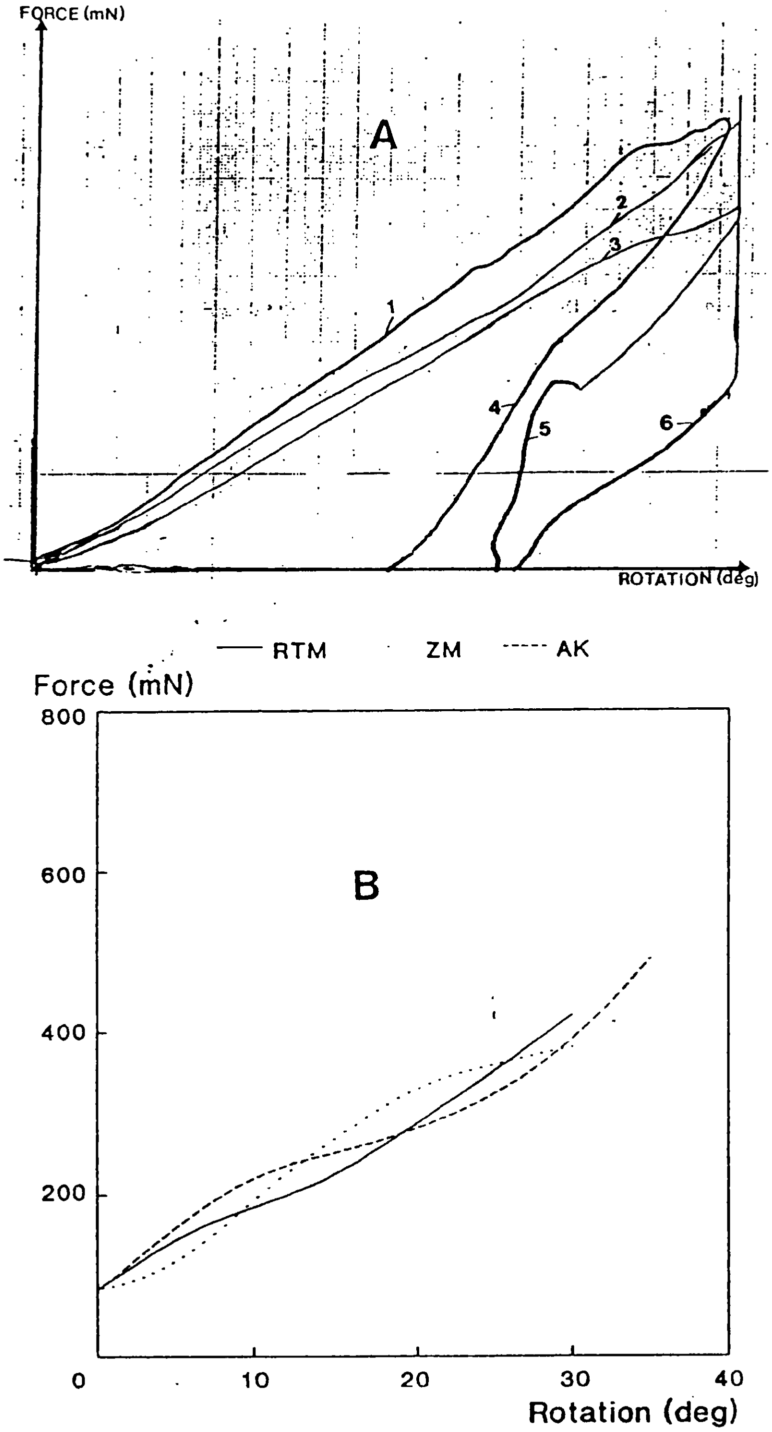


Figure 12.10 Results of normal control subjects obtained by using the rotational design system on lateral direction. A) Subject AK showing reproducibility (note that the unloading curves 4-6 fell to zero due to loss in tension in the silk). B) Three control subjects showing how the results fell within the same range.

this design, the value of the antero-posterior force was reduced by following the natural arc of eye movement.

2. Alignment of the device with the centre of rotation of the eye was difficult and time consuming.

3. It was difficult to measure accurately vertical and medial forces due to the bulk of the instrument which interfered with proper orientation.

4. The force generation test could not be performed in medial rotation due to the limitation imposed by the nose.

12.3.5 Recommendations

The rotational device was clearly an improvement on the linear mode allowing greater angles of rotation and reducing the risk of conjunctival trauma. However, critical evaluation of the results of the force analysis suggests that major defects still existed which could be overcome with design improvement. The first improvement would be the application of a force which would continuously be tangential to the arc of eyeball rotation. In addition, the coupling mechanism required some improvement. The silk suture could only transmit tensile forces, buckling when compressive forces were applied. Moreover, the attachment of the silk to the load cell and coupling rod was a delicate and time consuming operation.

Following the unsatisfactory outcome of the previous designs, the next step towards a new design was to take the following additional important factors into consideration:

1. The rotational movements of the eyeball obtained with the second prototype should be retained.

2. The placement of the force-transducer in the same vertical and transverse planes of eyeball rotation.

3. To enable the previous condition to be fulfilled, the coupling between the eyeball and the force-transducer should not restrain the eye from its natural forward and backward movement during the course of rotation (translation).

4. Align the centre of the measuring device to coincide with the centre of rotation of the eyeball.

12.4 PROTOTYPE "C": THE MANUAL CIRCULAR TRACK DESIGN

Although the modifications required to improve the test system in order to allow the coincidence of the centres of rotation of the test system and the eyeball appear to be modest, their implementation necessitated a complete redesign of the test apparatus.

12.4.1 Design Description

(a) Theoretical Concept

The new design had to solve several technical problems. The theoretical concept, which is illustrated in Figure 12.11, is to design a track for the equipment to move along which is parallel to the eye movement in all directions of gaze. Such a track should essentially be circular. movement of the corneal apex would make an inner circle, the centre of which is the centre of rotation of the eye under examination. The radius of the outer circle (circular track) will equal the sum of the radius of the eye, the contact lens stem and the length of the force-transducer beam. This required the coupling between the load cell tip and the contact lens stem to allow slight antero-posterior movement while keeping the displacement-force applied to the eye tangentially. In addition, it was necessary for the device to be capable of

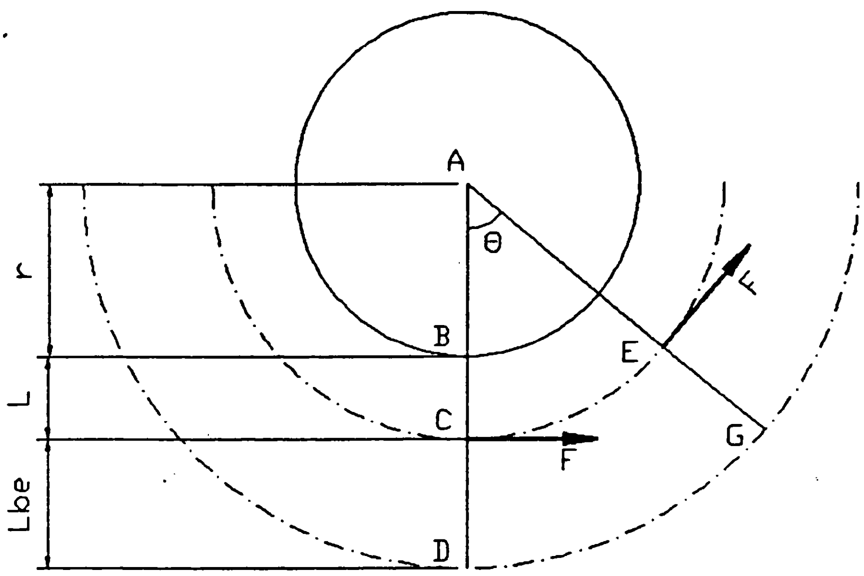


Figure 12.11 The simulation diagram of the circular track device.

carrying out the FDT and FGT in all four main directions of gaze without major manipulations. These requirements could not be met by using a thread coupling.

(b) Mechanics of the Design

The angular displacement of the eye (via the contact lens stem) was produced by moving the "displacement and force-measuring" system, which is mounted on a carriage (B), on the upper section of a circular track (C) (Fig 12.12). The centre of curvature being located at the nominal centre of rotation of the eyeball. This track has a diameter of 25 cm and forms a 130° circular arc which allows it to clear both sides without touching the face. Movement of a carriage mounted on this track was produced by a manual handle (D) which could be easily and freely rotated in both directions. It comprised a 12 mm radius pinion geared to the reciprocal rack machined into the track. Moving the carriage led to the rotation of a 25 mm radius gear coupled with the gear (E) of the pinion and mounted on the shaft of the rotational potentiometer (F) (RS 173-388, UK). Forces applied to the eye were measured by the bilateral force-transducer (G).

The use of a surgical silk loop to couple the force-transducer to the contact lens stem proved to be impractical because of the error it created on the antero-posterior

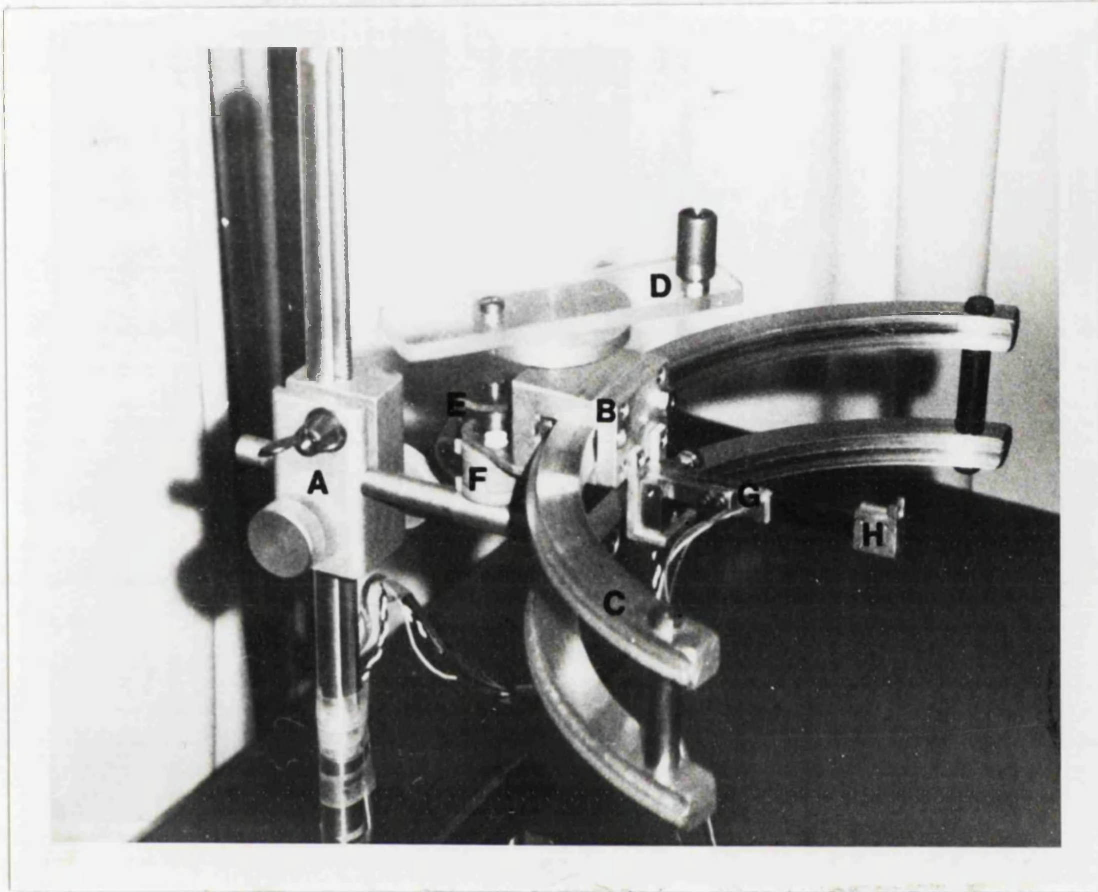


Figure 12.12 The manual circular design. A: metal clamp, B: carriage, C: track, D: manual handle, E: gear, F: potentiometer, G: force transducer, H: fork.

vector and was also time consuming. It was therefore abandoned. Instead, the stem of the contact lens was coupled to the test device by a U-shaped aluminum fork (H) fitted on the tip of the force-transducer (Fig 12.13). The fork was coupled to the stem at a fixed point 25.5 mm in front of the nominal centre of rotation of the eye to align the centres of rotation of the eye and the curved track. The fork was designed with a non-parallel walled slits into which the stem fits. This allowed lateral movement but did not restrain the small antero-posterior movement of the eye which occurs during rotation nor did it produce additional strain on the contact lens which might otherwise dislodged it.

The force at the tip of the force transducer was measured and the rotational force, as on the surface of the eye, was obtained. The strain gauge beam was chosen to be stiff with minimal distortion at the loads measured.

In the case of displacement of the stem within the fork, a correction factor was introduced to the data to take into account the difference in length of the arm of the force.

Both ocular distortion and lateral translation of the contact lens could have contributed an error to the recordings. Eight video recordings were made to look for evidence of lateral translation but none was observed.

A)

B)

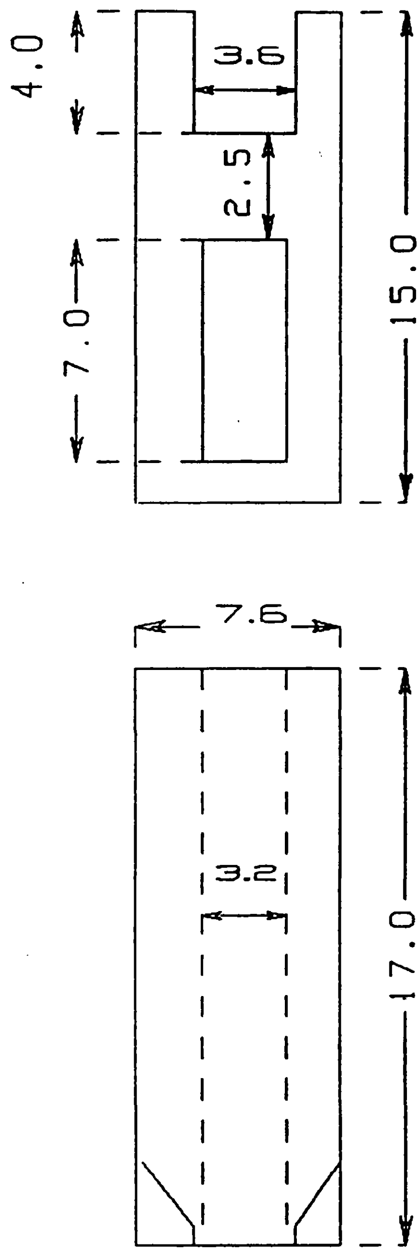


Figure 12.13 The fork design
A)- frontal view B)- plane view.
(all dimension in mm)

Distortion was not sought but should be considered in future studies.

The system was mounted on a metal stand with a metal clamp (A), holding the system through a shaft welded to the bottom track. Vertical or horizontal orientation of the track was brought about by releasing a control screw in the clamp and rotating the device. In addition, the slit-lamp microscope table was fitted with a four-sided rect-angular wooden base which prevented slippage of the instrument from the rimmed table and allowed rapid reorientation of the system for other meridians of investigation.

In addition to the mechanical improvement, the signal recovery and recording were improved by replacing the X-Y plotter with a personal computer containing an analogue to digital conversion interface.

(c) Signal Recovery and Recording

The output from both the force and displacement trans-ducers was digitized through a PC26 12 bit ADC Board (Amplicon Liveline Ltd) and sampled and recorded on a microcomputer (Compaq Computer Corporation) at a frequency of 50 Hz for the forced duction test and 100 Hz for the force generation test. The force-displacement curve was displayed in realtime to give the examiner an immediate indication of the success of the test or the need to repeat it.

(d) Test Procedure

Following instillation of a topical anaesthetic drop, the intraocular pressure is measured, the suction cup is fitted, a 70-80 mmHg pressure reduction is applied, and the subject is instructed to rest his chin on the chin rest as previously described. The instrument is appropriately orientated for the eye to be tested and the software is loaded on the computer for data collection and display. The stem of the contact lens is located in the fork of the strain gauge beam at a fixed point on the stem. The subject is asked to maintain his eyes in the primary position of gaze by fixing a target with the fellow eye.

The test is initiated by activating the data-collection program by a keyboard entry. During the FDT, the subject is instructed not to make any active eye movements. Duction is produced by manual movement of the load cell carrier on the curved track. The carriage is driven to its maximum excursion in the chosen direction; put into reverse and driven back to the resting position. The procedure is repeated three times on each occasion.

The FGT is carried out by locking the carriage in the central position to restrain eye movement while the fellow eye follows a moving target to a designated angle of rotation (35° horizontally, and 30° vertically) in the direction of function of the muscle in question and back to the central position of gaze. An extended Bjerrum screen at 2-metre distance is used to measure the angle of rotation. The test is repeated four times in each direction.

Following successful completion of both tests in the horizontal meridian, the contact lens stem is released from the fork, the track is rotated through a right angle and the test procedure is repeated in the vertical meridian. This time, the subject's lids are drawn apart by the examiner's fingers to allow free and wide range of rotation to be obtained. The contact lens (suction cup) is then removed and the intraocular pressure is measured and recorded. All tests are completed within ten minutes of the application of the contact lens. Only one eye is tested at each experimental session.

12.4.2 Results

Fifteen normal control subjects were evaluated. Figure 12.14 shows the initial collected data which can be compared with those obtained from the rotational design

"Prototype B" (Fig 12.10). The force required to rotate the eye 35° was only 260 mN (26g) compared with 470 mN (47g) recorded by the rotational instrument.

The close agreement of the test results from the normal subjects was encouraging. More consistent results were obtained by the use of the circular system (Fig 12.14) compared to the poorly reproducible results obtained by the two previous devices (Fig 12.6 and Fig 12.10).

12.4.3 Criticism and Recommendations

It was difficult to move the carriage in the curved track at a uniform speed. This resulted in a slightly irregular force/displacement trace and a large standard deviation for the FDT data. A final improvement was made, incorporating a motor drive to replace the manual drive.

12.5 PROTOTYPE "D": THE MOTORISED CIRCULAR TRACK DESIGN

A main objective was to choose the ideal speed of eye rotation that generated a linear force-rotation curve. This choice was made by examining the previous test results, manually selecting the parts of graphs which displayed smoothness and calculating the rotational velocities which had produced these regular and smooth data.

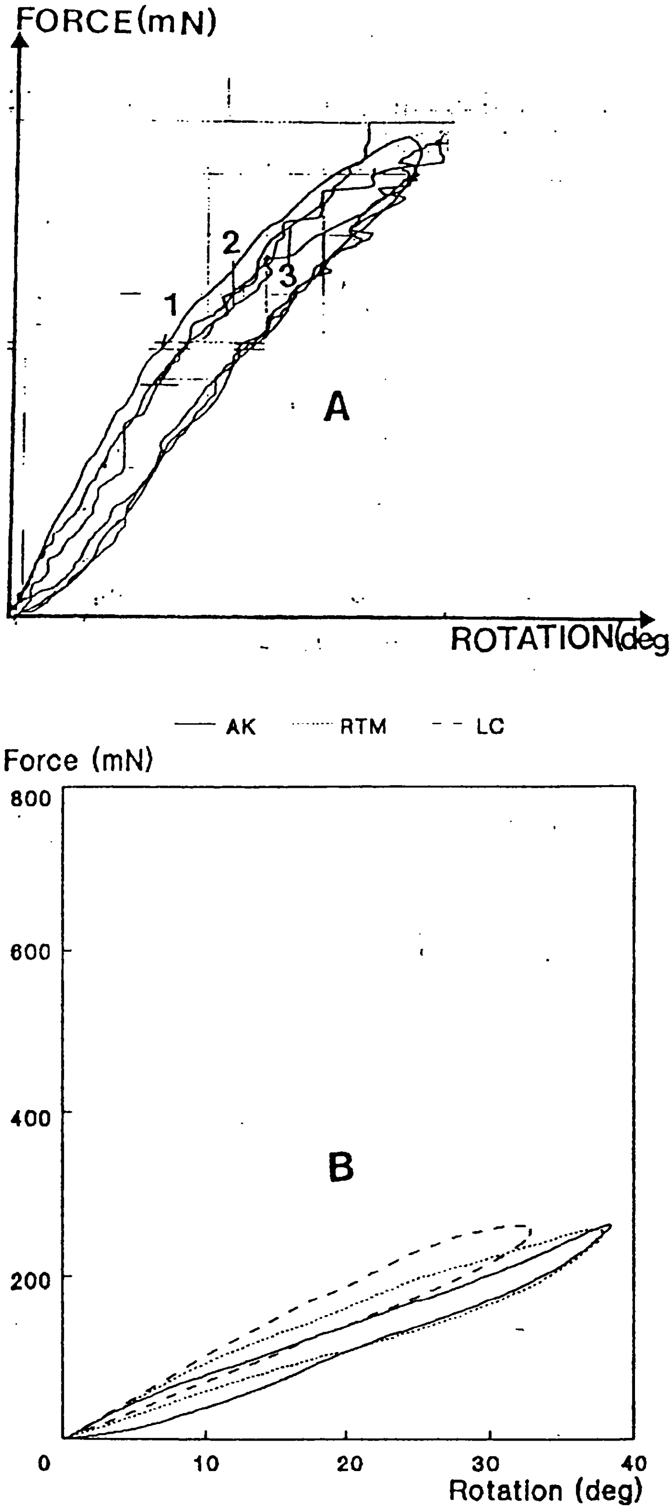


Fig 12.14 Samples of control data obtained using the circular design for the lateral FDT A) subject LC showing the reproducability in both loading and unloading curves B) three subjects.

The selected speed was 10 deg/s. This speed also ensured that the test could be completed within the allotted time.

12.5.1 Choice of Motor

Driving the carriage at a fixed speed required the use of a DC motor of sufficient torque. The moment of the motor needed to drive the carriage (M) was the sum of the moment required to overcome the carriage gearing resistance and the moment needed to rotate the eye in the worst situation (ie against high resistance).

$$M_{total}=M_{carriage}+M_{motor+gearbox}+M_{eye} \quad (12\text{-}1)$$

To rotate the carriage, a force of 981 mN (100g) with a moment arm of 5 cm was required. The maximum force required to rotate the eye was estimated to be 1962 mN (200g) with a moment arm, from the motor shaft, of 9 cm (Fig 12.15).

The DC motor and the gear box weighed 300g (2943 mN) and had a 3 cm arm. Utilising the previous equation:

$$M_{total}=M_{carriage}+F_{mass}\cdot l+F_{eye}\cdot L_1 \quad (12\text{-}2)$$

$$M_{total}=(981x5) + (2943x3) + (1962x9)=31392 \text{ mN.cm}$$

$$=0.314\text{N.M} \quad (12\text{-}3)$$

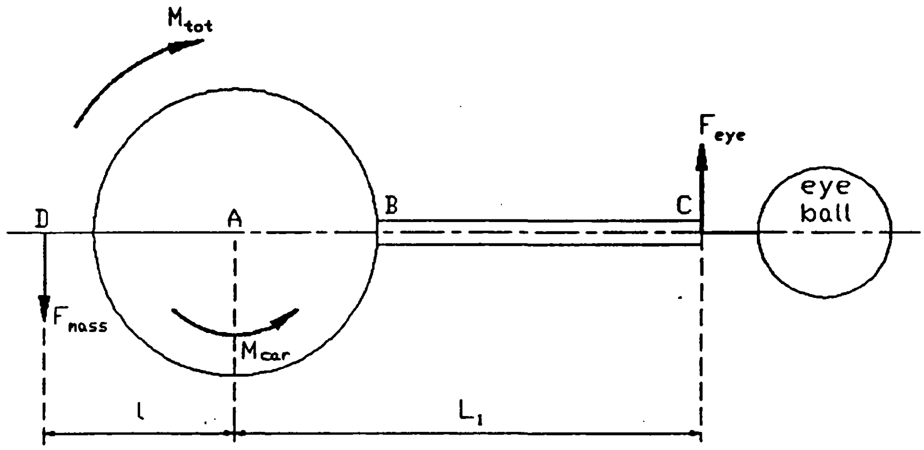


Figure 12.15 Moments analysis diagram.

A 12V DC servo motor (RS 336-292 1990, UK), which had the following specifications, was selected:

No-load speed	7400 rpm or 123.3 rps
Stall torque	8.8 mN.m
Power output	1.7 W

A reduction gearbox was required to bring the motor stall torque to above the total machine torque, the reduction factor Z, being at least:

$$Z=\frac{0.314}{88*10^{-4}}=35.7 \qquad (12\text{-}4)$$

Multiplying the reduction factor Z by a safety factor of 3 gives a value of 107. This means that the DC motor would be able to handle the highest possible weight without reaching its stall torque with a safety factor of 3. Since the available reduction gearbox was 160:1 (RS 336-264 1900, UK), this increases the safety factor to 4.5.

To find out whether the selected 12V DC motor would supply the carriage with the required speed (10 deg/s or higher), the output speed on the carriage shaft after passing the reduction gearbox was calculated:

$$\frac{123.3}{160} = 0.7706 \text{ rps} \qquad (12\text{-}5)$$

In an ideal condition, this will drive the carriage on the circular track relative to the diameter of the gear mounted on this shaft at 58.12 mm/s, which is equivalent to 29 deg/s. However, this speed is theoretical. The expected speed is lower, depending on the friction and backlash between the gears and other causes of resistance.

Two electrical microswitches (H) were mounted on both ends of the carriage and connected to the remote system drive control box of the motor to protect it from being over-loaded as it arrived to either end of the track (Fig 12.16A). When the carriage reaches one end of the track or another, it hits one of the two microswitches which automatically cuts off the power supply to the motor (Fig 12.16B). A small switch on the remote control box reconnects the power supply and initiates the carriage movement in the opposite direction.

12.5.2 Test Protocol

The smoother force/rotation curves showed that repeated extension cycling led to a progressive change in response with repeated rotation cycling (Fig 12.17). Higher forces were required to rotate the eye in the first cycle than

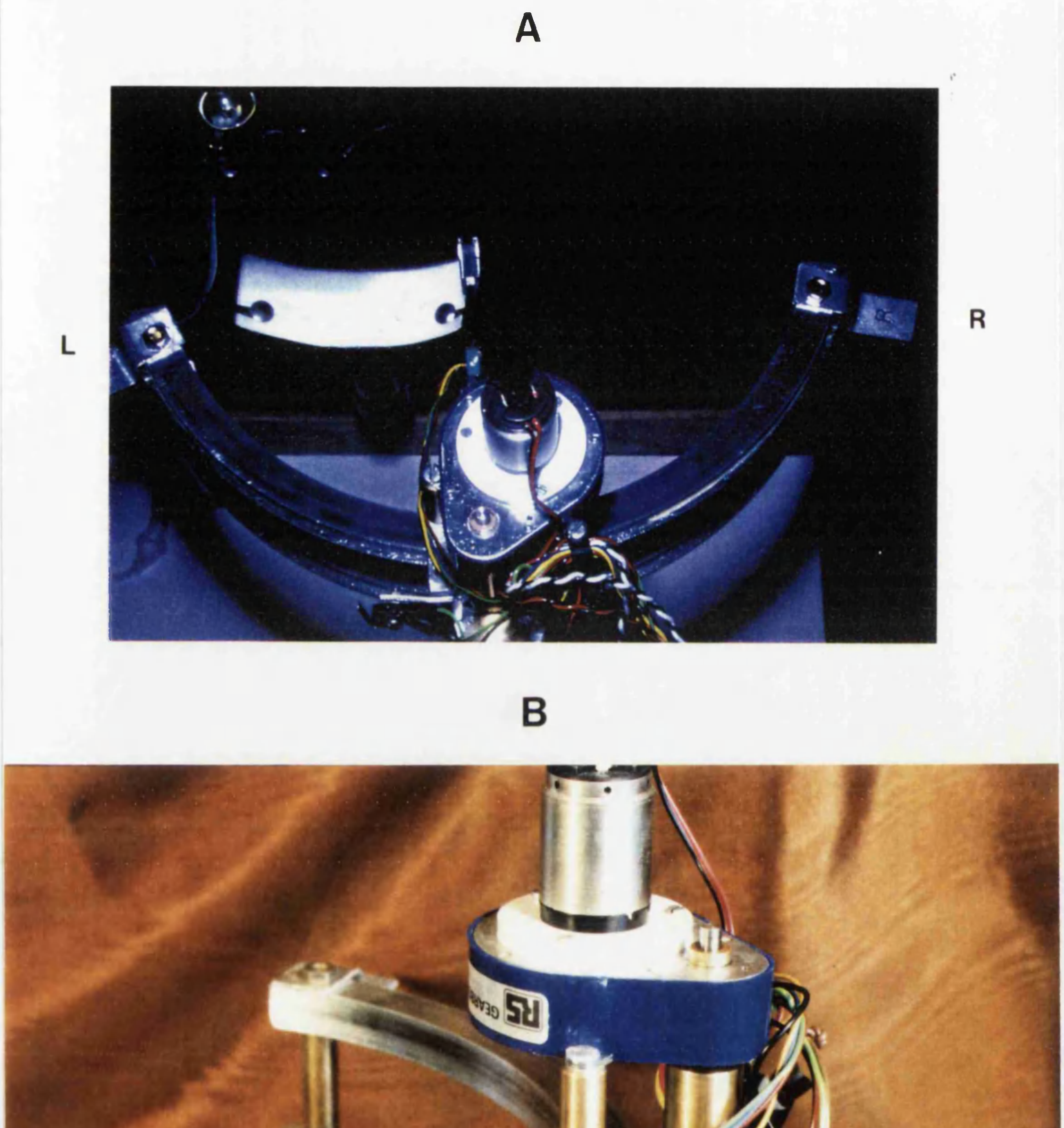


Figure 12.16 The motorised circular system.
A: Close view of the microswitches on the carriage,
B: Plane view of the system.

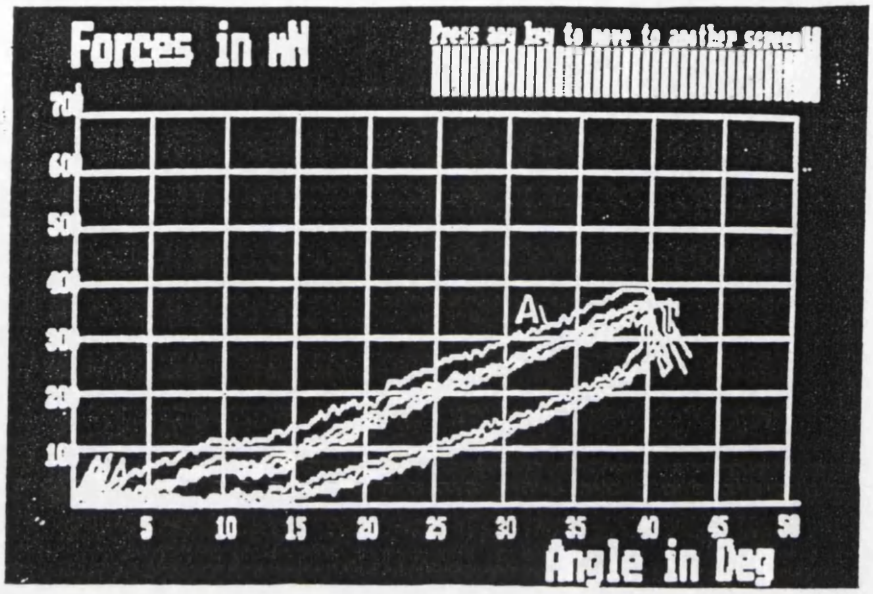


Fig 12.17 FDT result for normal subject on medial direction showing progressive change in response A) First cycle which is different from the following 3 cycles.

the following cycles. This preconditioning is a feature of all soft body tissues (Zajac, 1989). The presence of preconditioning suggested that it would be best if the FDT was carried out by cycling the rotation repeatedly between the primary position and maximum angle of excursion in the direction of choice. This would be followed by the same procedure in the opposite direction on the same meridian and by the FGT thereafter. A standard record form (Appendix 5) was completed for each subject.

12.5.3 Data Handling

The test system (Fig 12.18) is composed of three parts: the mechanical test apparatus, the hardware (computer and electronic components) and the software. A major task was to build up a network of communication between the different components to establish a realtime data collection and display. This required a knowledge of frequency and noise characteristics of the signal, and writing of a suitable software.

12.5.4 Sampling Frequency

A Nyquist criterion is that the sampling frequency should be equal to or greater than twice the highest frequency components of the original signal (Malmstadt et al, 1981). An analysis of the frequency spectrum of the signal was

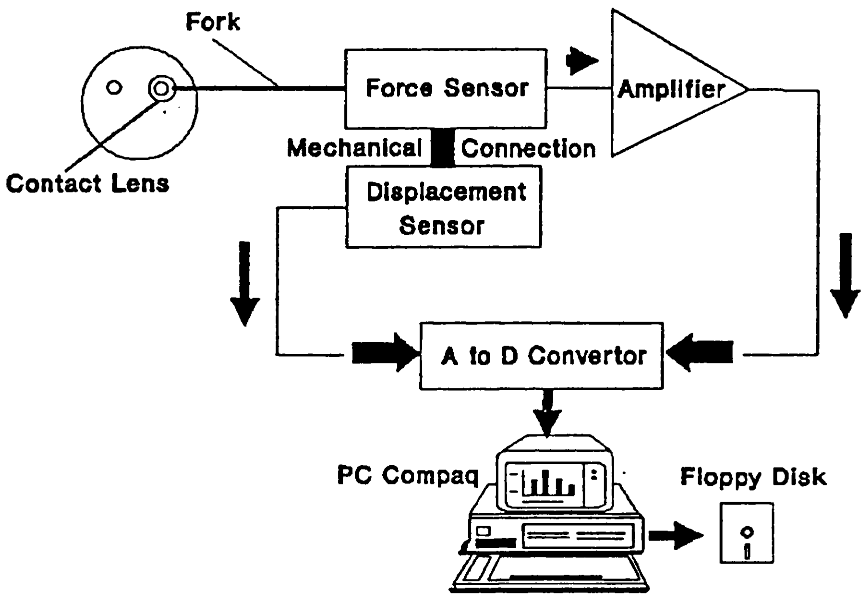


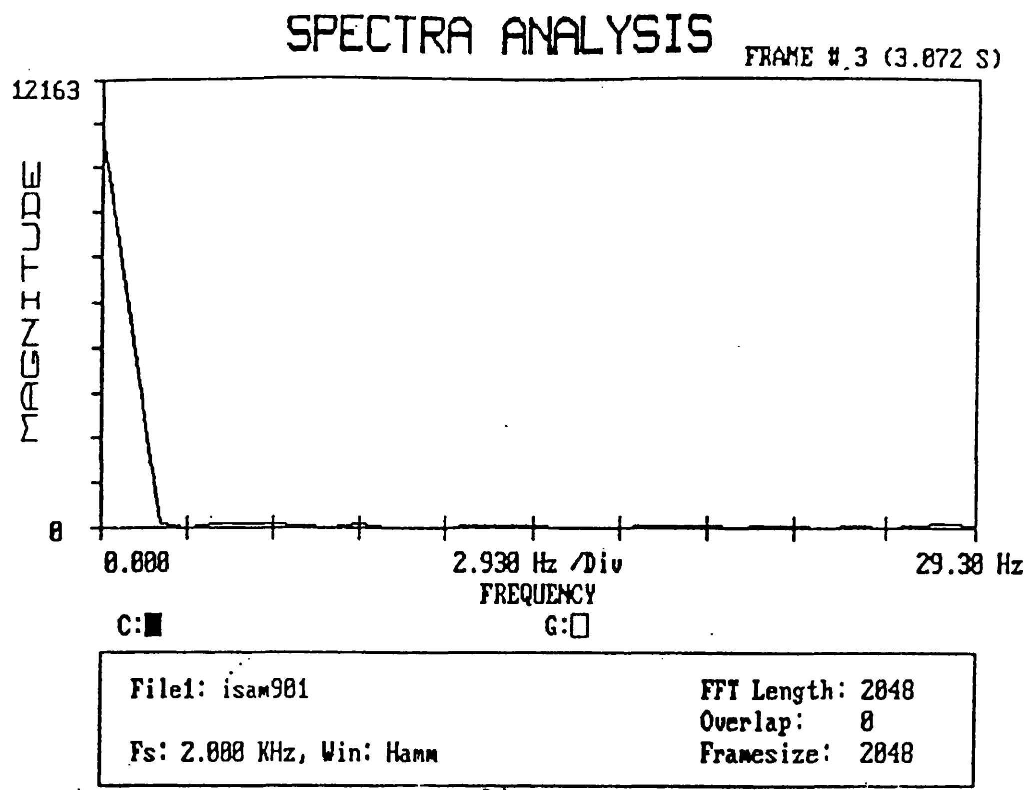
Figure 12.18 Diagram of the different components of the circualr system

carried out to detect the highest frequency component in order to decide the sampling frequency. There were three output signals to be sampled: force and displacement for the FDT, and force alone for the FGT. The output signals for both tests were recorded at the frequency of 2 kHz. The frequency spectrum recorded signals were then obtained using the hypersignal (Hyperception Dallas, USA) package.

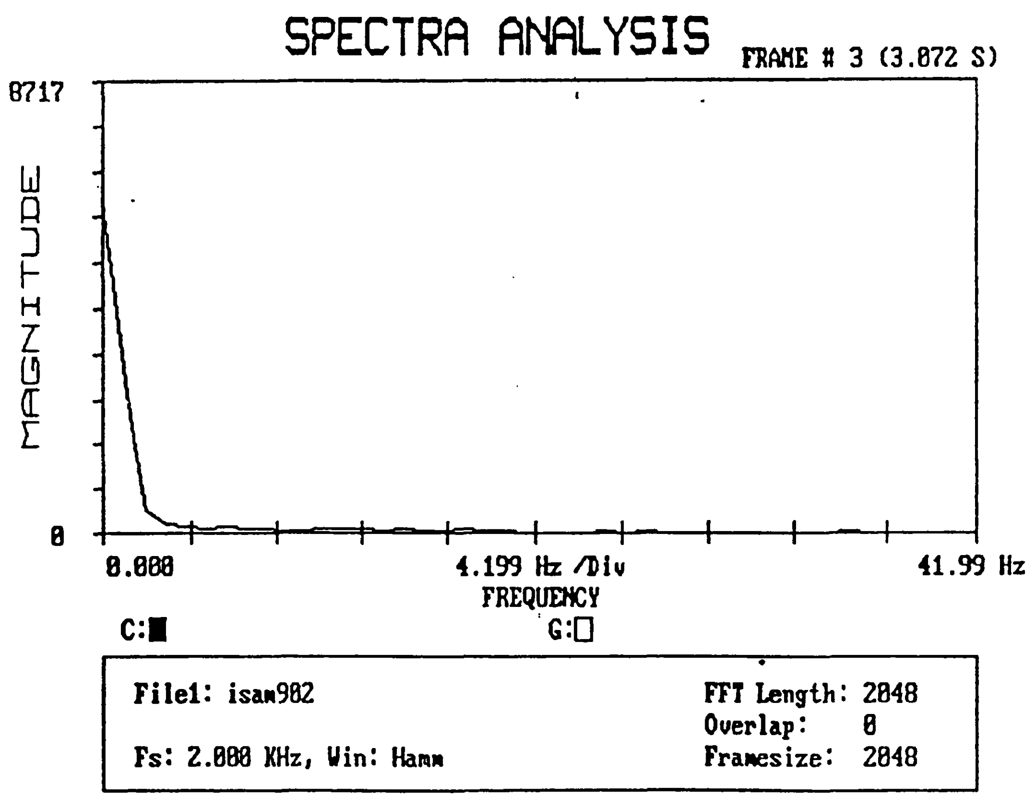
Analysis of the displacement signal for the FDT showed the highest frequency component to be 2 Hz (Fig 12.19A), while for the force transducer components it was up to 12 Hz (Fig 12.19B). Hence, the recommended sampling frequency according to Nyquist should not be less than 24 Hz. In practice, 50 Hz was used. The FGT signal (Fig 12.19C) showed signal frequency components of up to 20 Hz which means, again according to Nyquist, a recommended frequency of at least 40 Hz. In practice, 100 Hz was used.

The sampling frequency of 50 Hz, which was selected to be used in the FDT data collection, was influenced by two factors:

1. The Nyquist theory, previously mentioned.
2. The fact that the highest possible frequency to be used in this process was 50 Hz. This is because of the length of time analogue to digital conversion takes 40 microsecond, multiarithmetic procedures

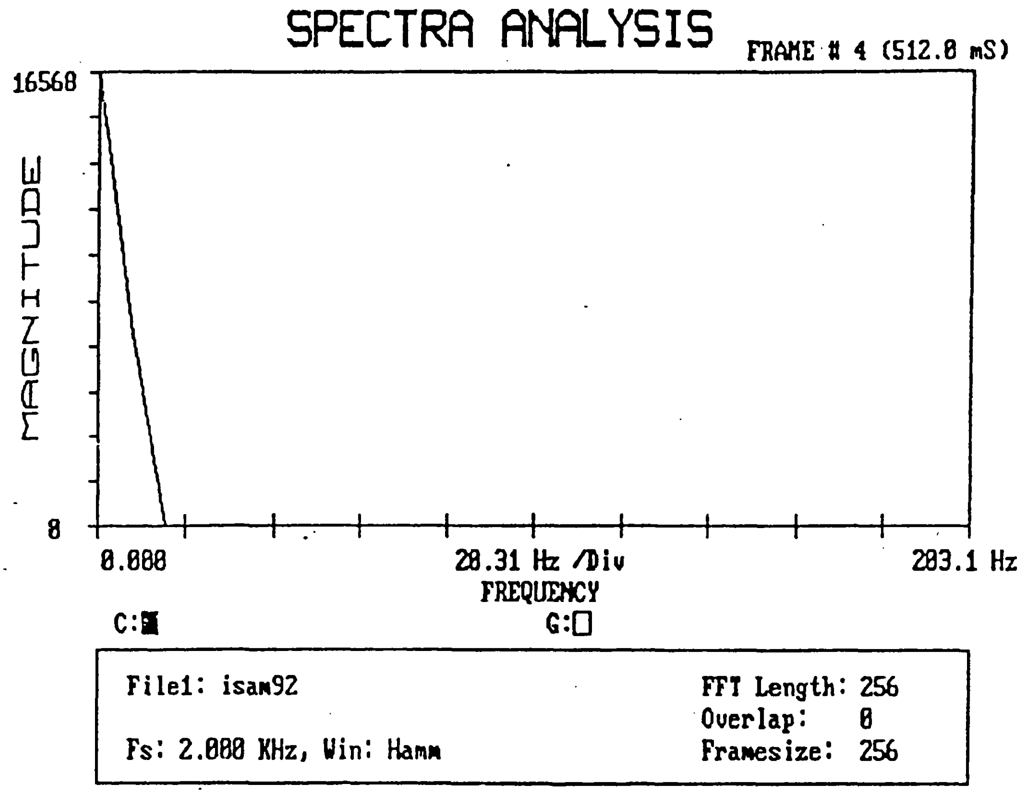


A)



B)

Figure 12.19 Continue



C)

Fig 12.19 Frequency spectrum of : A) rotational transducer in FDT B) force transducer in FDT C) force transducer in FGT.

which convert the collected data to a form of force (mN) and rotation (Deg), and the realtime plotting data after multiplication by the plotting factors.

12.5.5 Handling the Collected Data

Suitable software programs were written in Turbo Pascal version 4 language (Borland, UK). Such a program receives signals from the force-transducer and the potentiometer through the analogue to digital conversion card to the Compaq computer.

The data collection software is composed of two parts:

1. The main program body (Sunday.pas).
2. An interaction unit saved in another file (Sunday1.inc) composed of multi-procedures.

Running the main program will load the unit Sunday1.inc which is already compiled as a low-level language to facilitate quicker performance. This arrangement is useful for saving a large amount of RAM (Random Access Memory) of the computer which will be available for data storage.

The program runs through different stages (Fig 12.20) and the menu offers six choices:

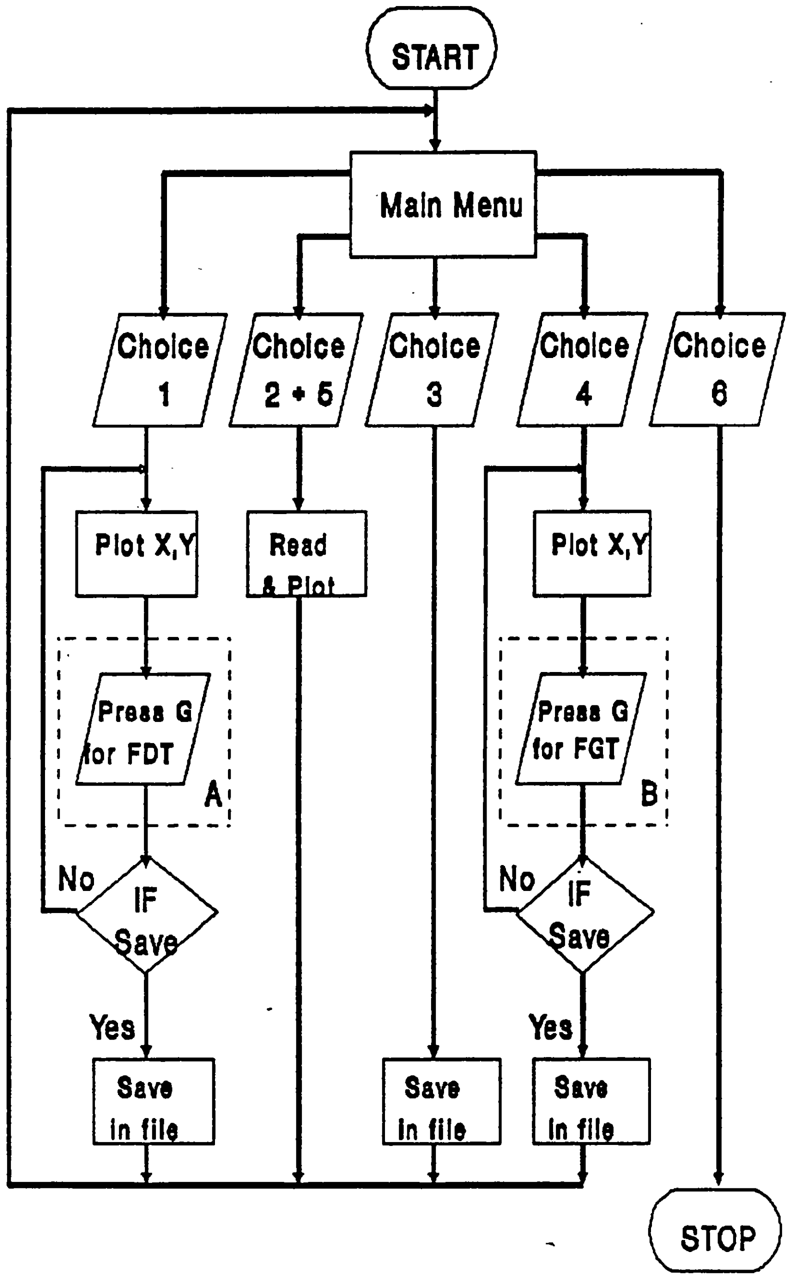


Figure 12.20 Flow chart of the data collection program Sunday1.pas

1. Collect data for the FDT.
2. Plot existing data for the FDT.
3. Collect personal details about the subject.
4. Collect data for the FGT.
5. Plot existing data for the FGT.
6. Stop running the program.

The choices are selected from the menu by typing the required number which will replace the displayed screen with another appropriate to the option selected.

Selecting choice (1) will prompt a second screen menu to fill in with the basic data on the file-code: eye to be tested and the direction of rotation. It will also set up the sampling rate to 50 Hz. Following that, the user will receive a third screen displaying plotted axes of force versus angle and a prompt to press "G" to start collecting test data.

Pressing "G" starts data collection and plotting, in real time, the force-angle curve and assigning the data to a two dimensional array. Depending on the quality and reproducibility of curve obtained, the user may chose to save the data on file under the subject's name and extension .FDT or to re-collect data if there is evidence of active resistance or assistance from the patient.

Selecting to restart collecting data will take the program one stage back while saving the data to file will take the program to the intermediate stage where the menu of six choices is re-displayed.

Choice number (2) gives the examiner the opportunity to view previously collected data for the FDT following which he will be taken back to the main menu.

Choice number (3) is about collecting personal details. Having filled all these details, the file is saved under the patient's name and extension .PER.

Choice number (4) is about collecting data for the FGT and is similar to choice (1). However, the sampling rate is set to 100 Hz rather than 50 Hz.

Similarly, Choice number (5) is the FGT analogue to the FDT choice number (2) while choice number (6) switches off the program (software) and takes the computer back to the main operating system (MS-DOS).

Collecting and displaying data for either test goes through three stages (Fig 12.21):

Stage 1: Data are collected for the first 20 samples before averaging the values obtained, thus

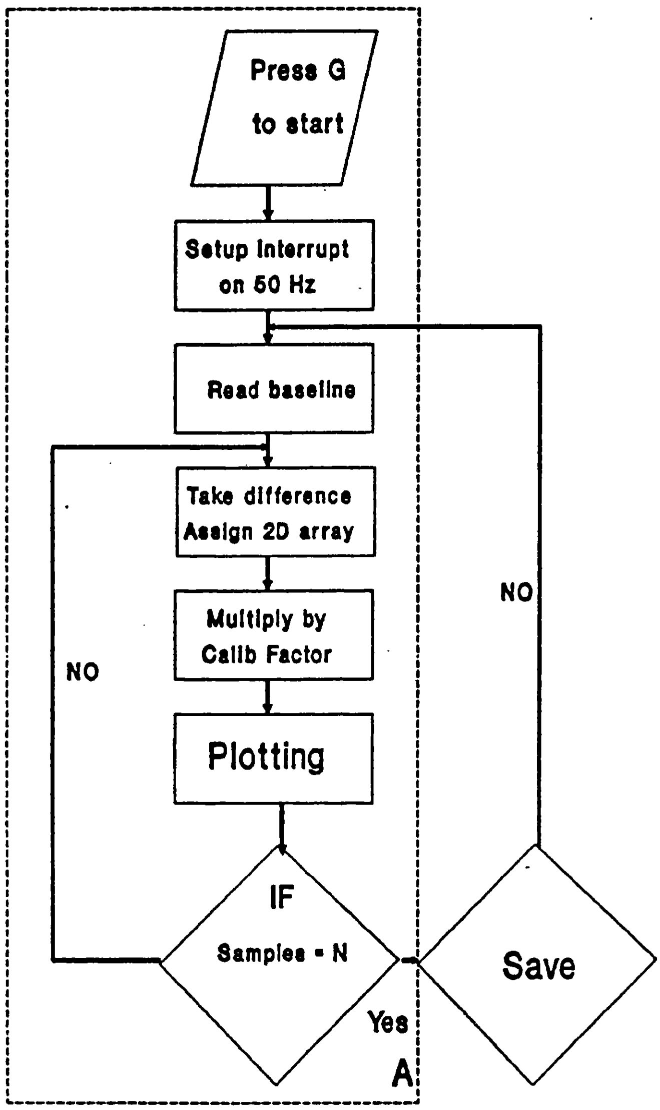


Figure 12.21 Flow chart of the stage A in figure 12.20.

providing a baseline for both force and displacement.

Stage 2: Actual test starts. The differences between the test and baseline values are computed and then multiplied by the calibration factor. This converts the voltage values into displacement and force which are assigned to the two dimensional array of displacement and force.

Stage 3: Values for the force and displacement are multiplied by the drawing scale factor for screen plotting. For the FGT, only the force array is required.

12.5.6 Filtering the Data

Output signals commonly contain "noise", which can often be reduced by filtering.

It is essential to know the magnitude of noise compared to the original signal in order to decide if filtering is required and to determine the choice of digital or analogue filtering.

To evaluate the magnitude of noise, the baseline output of the two channels, force and displacement, were connected to an oscilloscope and displayed on the screen. The displayed 50 Hz noise had a magnitude of 5 mV. The maximum output signal during tests was 560 mV. Hence, the signal to noise ratio (S/N) was:

$$\frac{S}{N}=20\log_{10}\frac{560}{5}=41 \text{ dB (Malmstadt et al, 1981)} \qquad (12\text{-}6)$$

Starting the motor drive increased the noise to 15 mV, giving a signal noise ratio of:

$$\frac{S}{N}=20\log_{10}\frac{560}{15}=31.44 \text{ dB} \qquad (12\text{-}7)$$

The best and only solution in this case was to introduce an analogue filter (first order active - low pass filter) with 20 Hz cutoff frequency. Figure 12.22 shows the circuit design for this analogue filter which eliminated the undesirable signal above 20 Hz.

Further measurements of noise following the introduction of the filter showed that noise level was 1 mV under baseline conditions giving a value of:

$$\frac{S}{N}=20\log_{10}\frac{560}{1}=55 \text{ dB} \qquad (12\text{-}8)$$

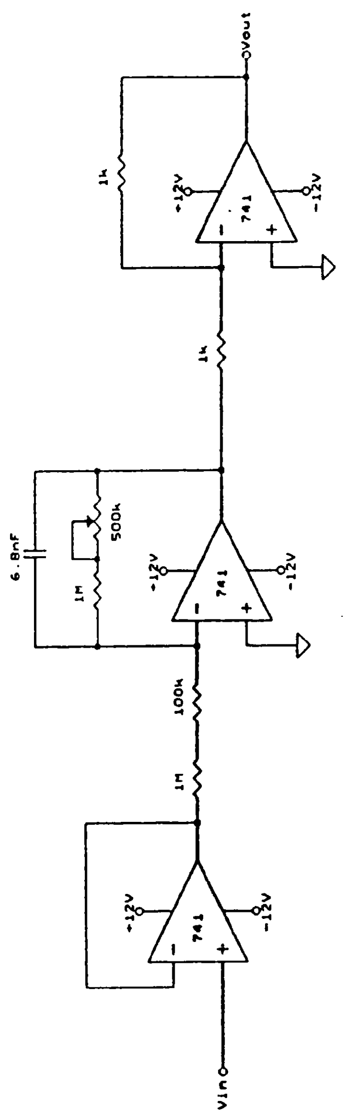


Figure 12.22 Circuit design for the first order active-low pass analogue filter with 20 Hz cutoff frequency which eliminated undesirable signals above 20 Hz.

A useful improvement on the previous value of 41 dB (Eq 12-6). A similar improvement was obtained with the FGT signal.

With the active motor drive, the new noise level was 3 mV, bringing the signal to noise ratio to a value of 45 dB compared to the previous value of 31.44 dB.

12.6 SIMULATION AND VALIDATION

The function of the test apparatus was the measurement of the load-extension behaviour of the extraocular muscles. Although reliable experimental results were obtained, the validation of the instrument required it to be used on a test system with known properties. This could only be done by simulation which required the production of a suitable analogue which had a fixed centre of rotation and contained an element of known mechanical properties.

12.6.1 Simulation Prototype

The simulation design (Fig 12.23)) consisted of a 25 mm diameter plastic ball (D) mounted on top of a shaft (A). Roller bearing (B) defined the centre of the rotation of the ball. The device was fixed on an aluminum L-shaped base (C). Two metal pins were fixed to the ball, at a right angle, in the horizontal mid transverse plane. A

Figure 12.23 Lateral and frontal views of the simulation system which comprises of: A: shaft, B: roller bearing, C: base, D: plastic ball, E: hook-carrier plate and F: spring.

carrier plate (E) supported one end of a linear extensible helical spring, the other end of which engaged one of the pins on the ball at a right angle. Rotation of the ball by the other pin extends the spring. The spring produces a force which results from the rotation of the ball, and both the force and rotation could be measured by the circular system. Figure 12.24 shows a schematic diagram of the forces and geometry of the simulation prototype. Applying slow speeds to rotate the ball made it possible to ignore inertia and consider moments only.

12.6.2 Testing Procedure

The load-extension characteristic of the helical spring used in the analogue was evaluated using an Instron 4505 tensile test machine.

A 10 N load cell was mounted on the machine. The spring was clamped between an upper grip connected to the load cell via a universal joint, and a lower grip connected to the moving cross bar. The spring was extended at a constant rate of 5.7 mm/s (equivalent to 16.7 deg/s).

The test starts by initiating the computer which drives the Instron machine to extend the spring for a maximum extension of 10 mm and back to the initial position. This

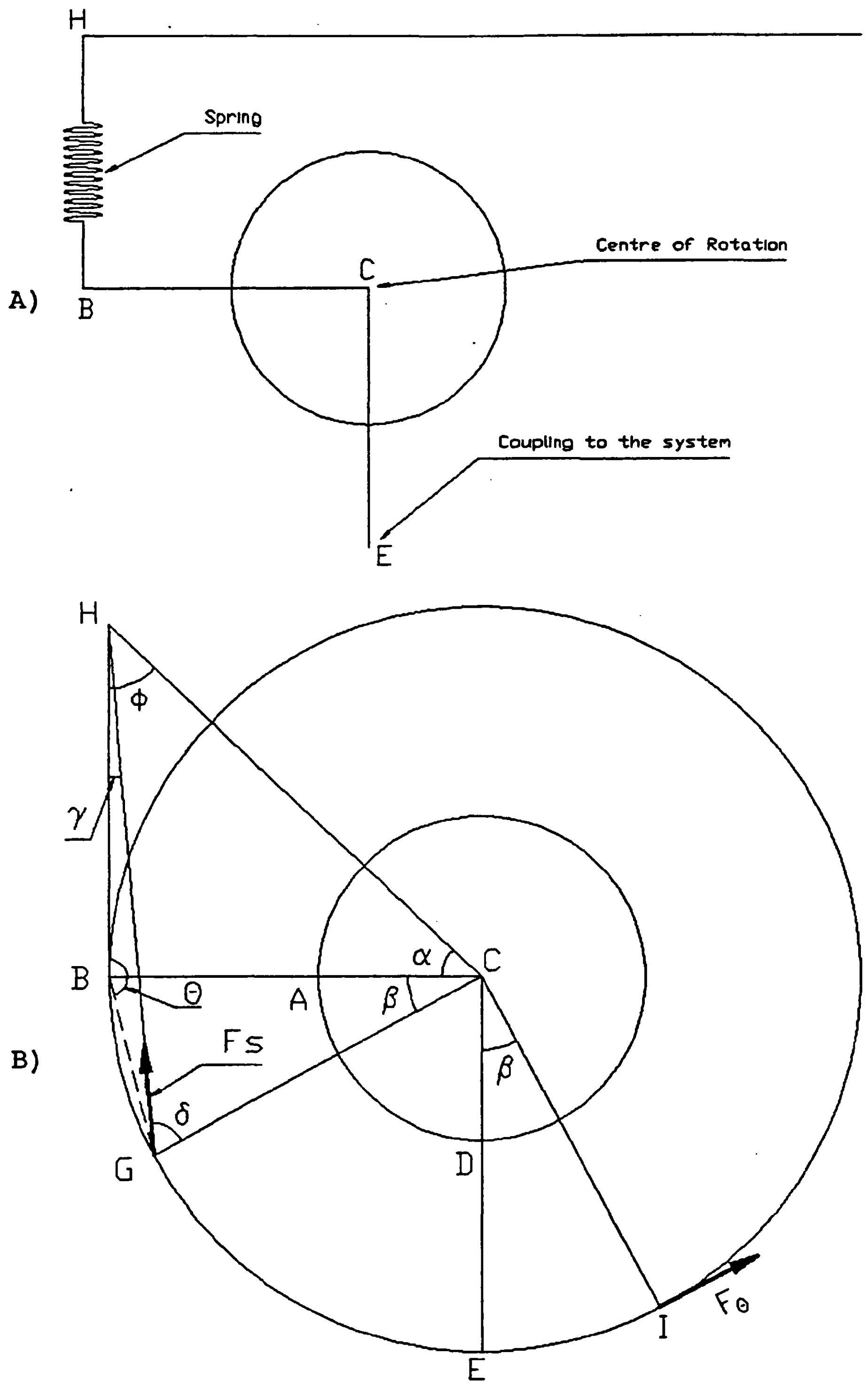


Figure 12.24 A: Plane diagram of the simulation components.
B: Force diagram of the simulation system.

$CA=CD=R_b$ Radius of the plastic ball.
AB & DE Lengths of the two metal pins.
H Fixation point of spring on base.
HB Initial length of the spring.

Applying slow speeds to rotate the ball makes it possible to ignore inertia effects and consider moments only.

procedure was repeated three times. An extension-load chart was obtained for each cycle from an HP plotter.

The friction in the roller bearing of the analogue was then evaluated. The axes of rotation of the eye test device and the analogue were made coincident, and the rod ED located in the fork at the end of the load cell of the test device. The carriage motor was then started and the load-rotation behaviour of the spring free analogue determined. The ball was rotated to maximum excursion and back to the zero position. This procedure was repeated three times.

The spring was then coupled to the rod AB and the carriage motor started, rotating the ball to the maximum excursion short of the spring contacting the ball, and back to the initial position. Again, this procedure was repeated three times.

The average of the three trials was taken and frictional forces were subtracted from the total forces required to rotate the ball while the spring was coupled. The output was processed by a Turbo Pascal Programme (Simulation.Pas) which was especially written to refer the measured forces to the real internal spring forces.

12.6.3 Results and Analysis

The data obtained from both tests were plotted and the force-angle (of rotation) curves were obtained (Fig 12.25). The curve regression equation for the result of testing the spring by the Instron machine was of the shape of:

$$\text{Force (mN)} = 4.31 + 7.2 * \text{Extension (mm)} \qquad (12\text{-}9)$$

While the results of the three trials were combined together in one file and the regression equation obtained which was:

$$\text{Force (mN)} = 4.37 + 7.28 * \text{Extension (mm)} \qquad (12\text{-}10)$$

The differences between the regression equations of the Instron measurements and those of the tested machine were studied and the mean difference was found to be 0.4g force (4 mN). when both data obtained were regressed against each other the resulting regression equation was:

$$\text{Instron} = 0.095 + 0.987 * \text{Design} \qquad (12\text{-}11)$$

As noticed, the slope is 0.987 indicating that the data is almost identical.

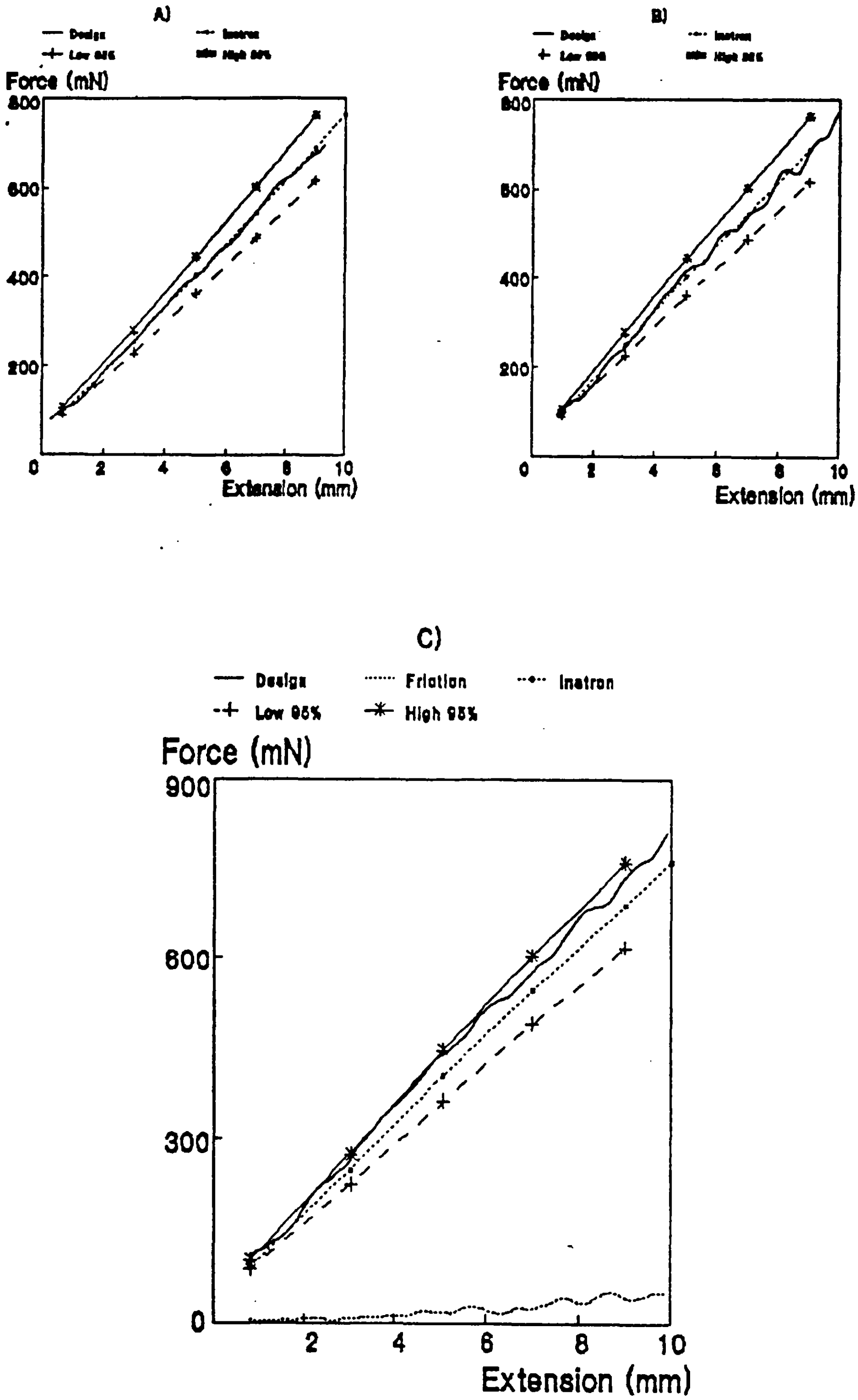


Figure 12.25 Results of the spring measurement by the Instron machine with their 95% range compared to the ones obtained by the use of the simulation prototype and the circular system. A) and B) show the resultant measured force after accounting for friction, while C) shows all force components including the friction forces (note that these forces are still within the 95% confidence limits).

CHAPTER 13

RESULTS

13. RESULTS

The design of the force(d) duction/generation apparatus went through a number of stages which took into consideration the different anatomical and physiological features of the eye and its adnexae.

The objective of the project was to design and construct a system which is accurate and practical for clinical use. The design progressed from a simple manual system on a linear track to a motorised arc-based system on a circular track (Chapter 12). This improvement in the design has led to a greater reproducibility and acceptability of the measurements.

This chapter gives details of the results of the studies of normal subjects for both the manual and motor driven version of the circular track design. The data presented has been corrected for changes in the length of the coupling system, which arose from minor mismatches between the centres of rotation of the track and of the eye.

Examples are then given of the results obtained, using the motorised system, from patients with different motility abnormalities.

The patients with orbital bone fracture investigated were classified into three different groups according to the main cause of their motility disorder:

1. Muscle or other soft tissue entrapment.
2. Muscle bruising/haematoma.
3. Muscle palsy consequent to damage to the nerve supply.

13.1 RESULTS FROM NORMAL SUBJECTS

13.1.1 The Manual System

Fifteen normal subjects, 10 male and 5 female, who had neither undergone eye surgery nor had defects in ocular motility, volunteered for the test. They were aged between 16 and 65 years. Either the right or the left eye was investigated according to the subject's preference. For those subjects who did not express a preference, the choice of the eye was made in such a way as to yield the same number of right or left eyes.

Hysteresis was noticed in all recordings.

Smoothness of the force-rotation curve was dependent on the speed at which the carriage was driven, the higher the speed the smoother the curve obtained. Figure 13.1 shows

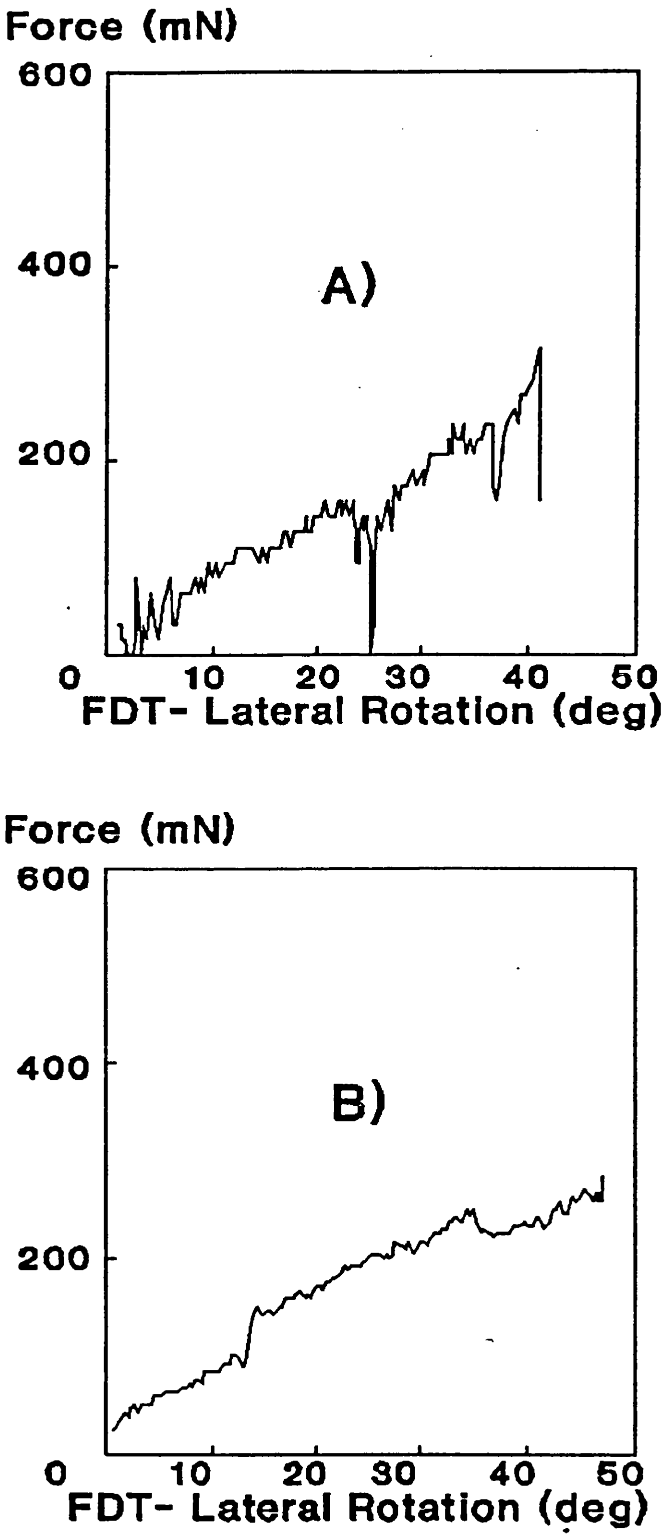


Figure 13.1 . Lateral FDT results for control subject by the use:
A)- Manual system B)- Motorised system

the test results obtained manually (13.1A) and using the motor driven system (13.1B) for the same subject.

The irregularity of the recording shown in figure 13.1A was due to the variation in the speed of rotation using a manual technique, perhaps combined with the phenomenon of damping, which can be expected to be strongly influenced by the speed of rotation. In contrast, figure 13.1B shows the force-rotation curve obtained by pulling the eye laterally at a constant speed of 16.7°/s (section 12.5). The curve is much smoother because of the uniform speed which was constantly in excess of the resistance engendered by damping.

The force-rotation characteristics obtained for each subject depended on the direction of rotation. In the lateral direction, reproducible results were obtained for up to 30°, but only up to 25° in the other test directions. For large rotations, the forces were variable and not reproducible.

A database of normal-subjects test-results was established to use for comparison with patient's test results. The mean value of force (x) and the 95% confidence limits were calculated for increments of rotation of 5 degrees. The confidence limits were calculated from the following equation (Eason et al, 1980):

$$\text{Confidence limits} = x \pm \frac{t_v(0.025)s}{\sqrt{n}} \qquad (13\text{-}1)$$

where $t_v(0.025)$ is the t distribution at 95% for v degrees of freedom, (s) the standard deviation and (n) the number of data points. The resulting mean values and confidence limits are tabulated for increasing rotation for all principal directions in Table 13.1 and shown in figure 13.2. The relationship between angular displacement and the force produced was almost linear.

13.1.2 The Motorised System

Data was obtained from 18 normal subjects, 14 male and 4 female. The same rules for eye selection were applied as previously described (section 13.1.1). The mean values and confidence limits are listed in Table 13.2 and shown in figure 13.3. The relationship between angular displacement and the force produced was, once again almost linear.

The responses in all control subjects showed evidence of preconditioning in that the first displacement always required a greater force for a specific angular displacement than did the next three recordings. Hysteresis was a feature of all recordings (Fig 13.4), in that the forces obtained during the unloading phase of each recording were

Direction	Angle (Deg)	Mean force x (mN)	Upper Confidence Limit $x+t_v(0.025)\frac{s}{\sqrt{n}}$ (mN)	Lower Confidence Limit $x-t_v(0.025)\frac{s}{\sqrt{n}}$ (mN)
	5	61	70	52
	10	116	137	95
Lateral	15	153	177	130
	20	192	210	174
	25	232	251	214
	30	275	297	253
	5	53	63	44
	10	117	142	93
Medial	15	165	192	138
	20	197	228	166
	25	260	296	223
	5	67	78	55
	10	137	155	118
Superior	15	179	193	166
	20	247	270	225
	25	304	335	273
	5	66	85	48
	10	126	149	102
Inferior	15	187	210	164
	20	250	275	224
	25	292	319	265

Table 13.1 Mean values and 95% confidence interval envelopes for forced duction results in normal test subjects using the manual design.

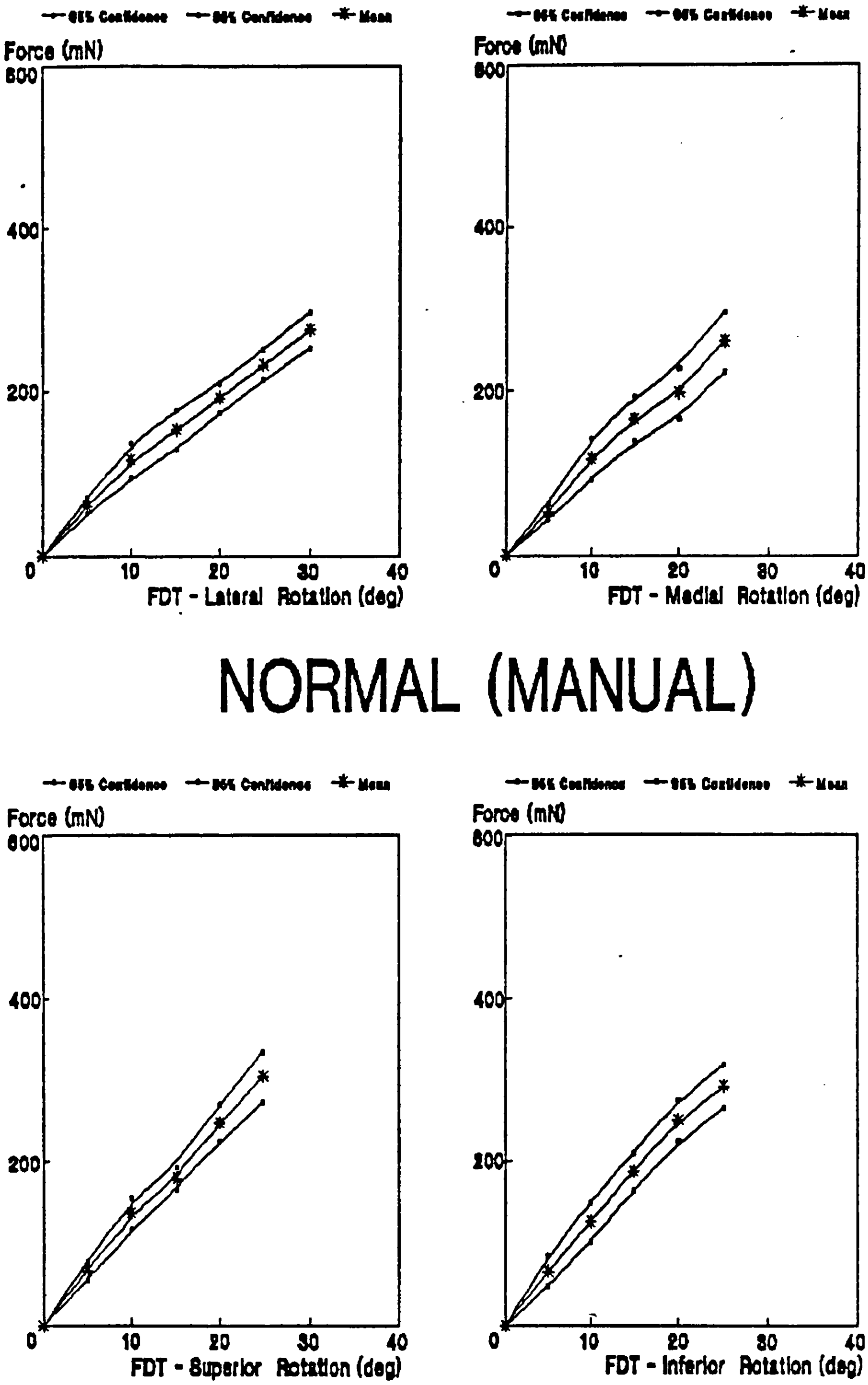


Figure 13.2 Normal data for FDT in the four principal directions for manual system (mean and 95% confidence interval envelopes).

Direction	Angle (Degree)	Mean Force x (mN)	Upper Confidence Limit $x+t_v(0.025)\frac{s}{\sqrt{n}}$ (mN)	Lower Confidence Limit $x-t_v(0.025)\frac{s}{\sqrt{n}}$ (mN)
Lateral	5	46	54	38
	10	76	86	67
	15	113	127	98
	20	152	170	134
	25	186	207	166
	30	219	241	198
	35	238	260	216
	40	274	297	251
Medial	5	51	62	40
	10	96	113	78
	15	138	153	123
	20	168	185	152
	25	196	218	174
	30	221	255	187
Superior	5	53	62	45
	10	108	125	91
	15	157	180	134
	20	204	223	184
	25	254	275	231
	30	306	341	271
Inferior	5	66	81	51
	10	119	135	103
	15	166	183	151
	20	218	235	200
	25	265	289	241
	30	314	353	275

Table 13.2 Mean values and 95% confidence interval envelopes for forced duction in normal test subjects by using the motorized design.

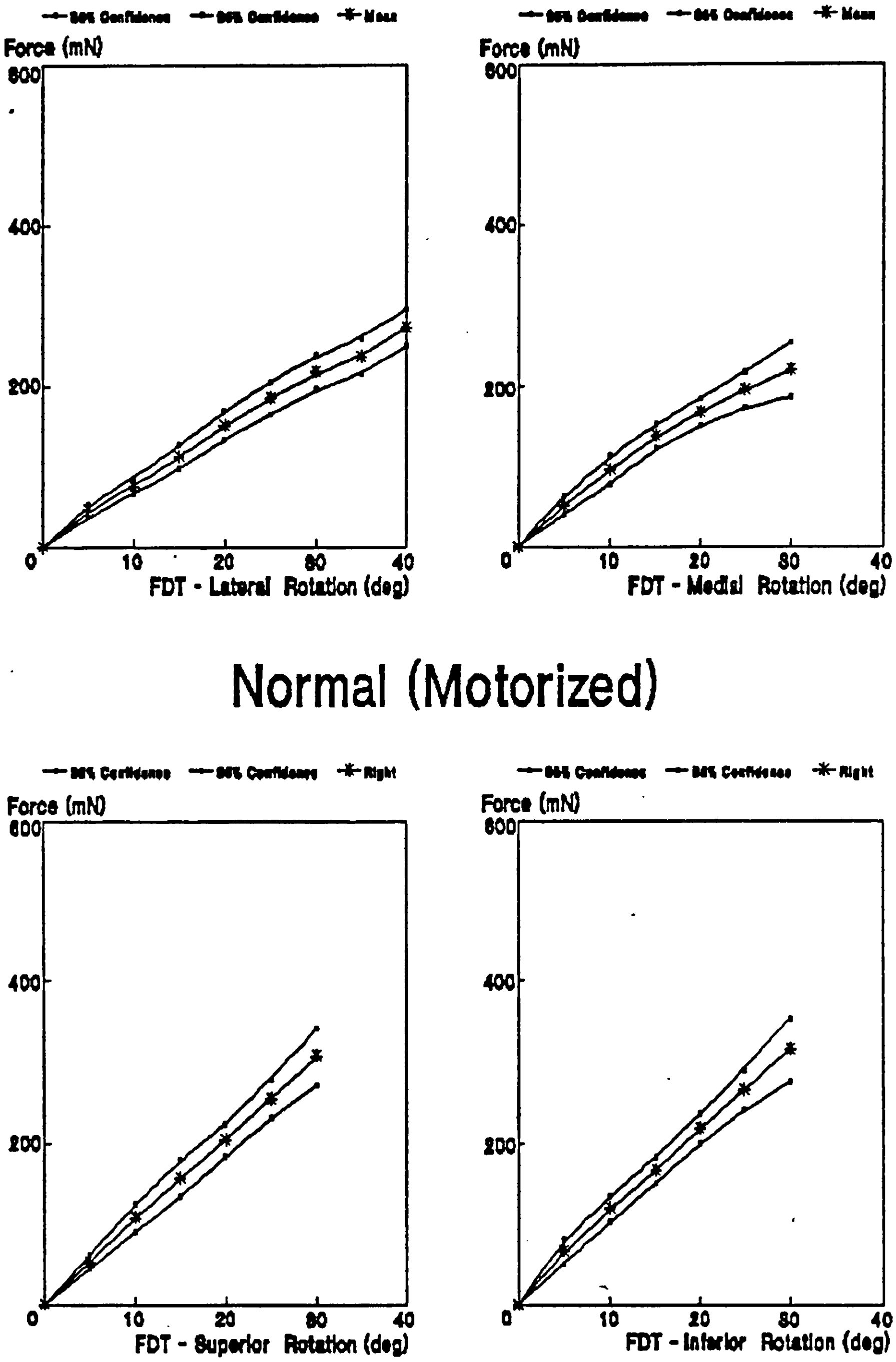
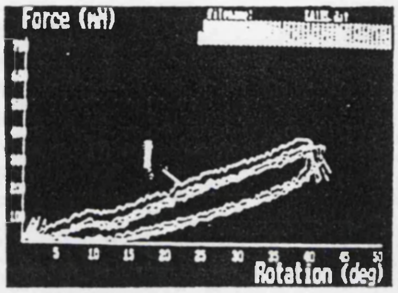
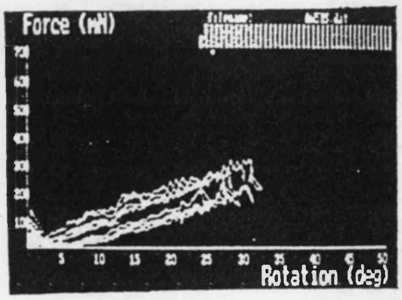


Figure 13.3 Mean values and 95% confidence envelopes for forced ductions in normal test subjects (motorised design).



A)

The Motorised Design



B)

Fig 13.4 Normal data sequence recordings for FDT

A) control LA - right eye- lateral direction FDT I- shows the preconditioning curve. B) control AW - left eye- superior direction FDT.

consistently lower than those measured during the loading phase.

The data presented in these studies was obtained during increasing rotation of the preconditioned cycles.

Data comparison between the manual and motorised systems showed that greater rotation values were obtainable with the motorised system. This was due to the stability and uniform speed afforded by the motor for the system, and the ability to reverse rapidly the movement of the carriage after achieving the desired angle of rotation.

The higher values of means (and the 95% confidence range obtained) by the manual system were due to the damping effect which was due to the reconditioning of the surrounding tissue as the speed of rotation of the eye decreased within a certain range. Moreover, the non-linearity of the force/velocity applied has also led to artefactual variations.

The mean values of the force required to pull the eye medially or laterally were not significantly different one from another (t-test of the means: $p=0.06$), nor were those forces required to pull the eye superiorly or inferiorly (t-test: $p=0.11$).

13.1.3 The Force Generation Test

Forces measured in the force generation tests were produced by the activity of the ocular muscle under test.

Data for the force generation test was collected from one eye of each of 30 normal subjects, 22 males and 8 females whose ages ranged between 16 and 65 years.

In this test, the carriage was locked in the primary position, while the contact lens rod was coupled to the fork. The patient was instructed to follow a target, moving along an extended Bjerrum screen at 2-metre distance, with his contralateral eye. Because the test was conducted in this way, there was no difference between the results collected using the manual or the motorised system.

The recorded forces were often more irregular than than those with the forced duction test, with rapid transient spikes presumably produced by the synchronous activity of motor units, being apparent.

As the contralateral eye followed the moving target, the measured force increased to a maximum value at the greatest excursion and fell to zero upon returning to the central rest position (CRP). Figure 13.5 shows a typical

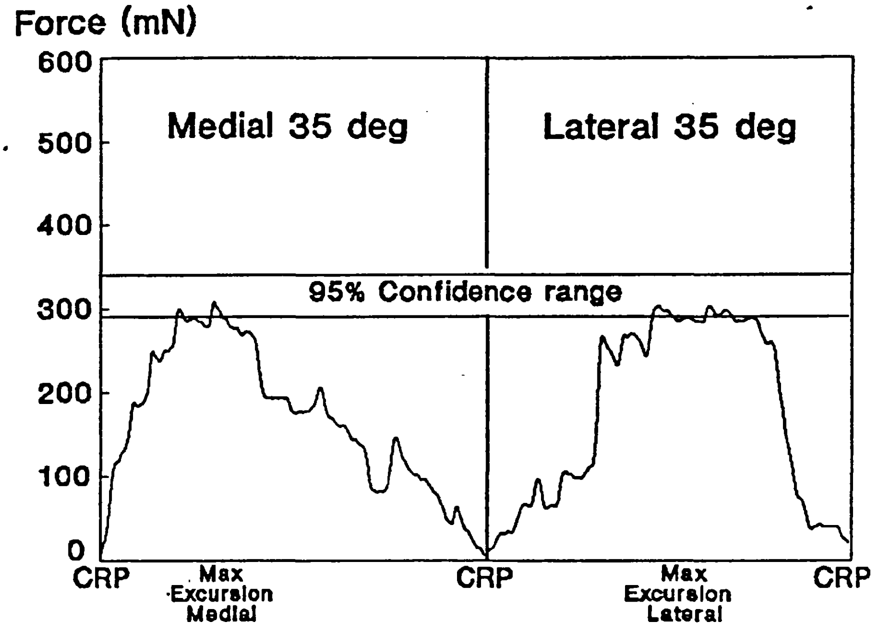


Figure 13.5 Normal control subject results for FGT in the horizontal plane. CRP: Central Resting Point

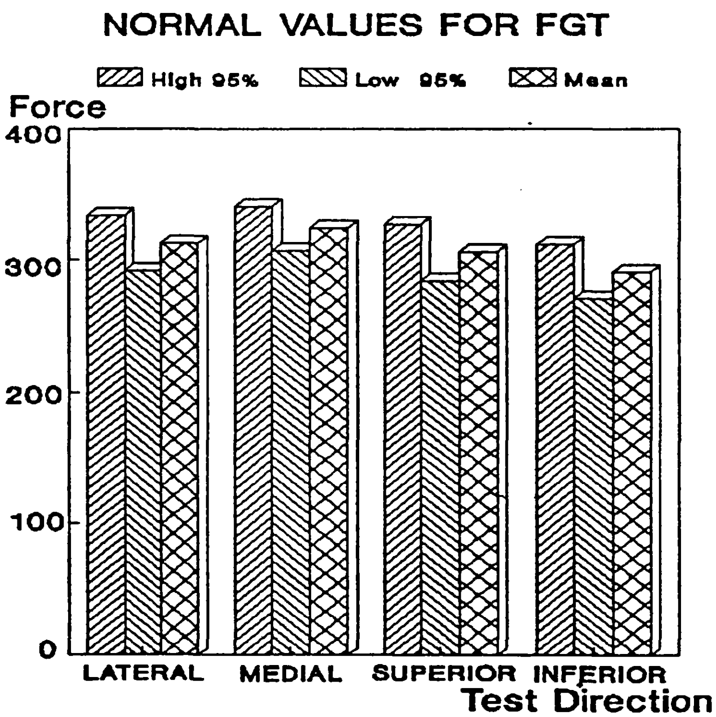


Figure 13.6 Mean values and 95% confidence intervals for forces generated at 35^0 Hor and 30^0 Ver rotation.

normal test result for horizontal eye movement where it is apparent that the maximum forces generated were similar for both lateral and medial excursions.

The maximum rotation of the contralateral eye for horizontal movement was 35° and for vertical movement 30° from centre. Table 13.3 and figure 13.6 show the mean values and the 95% confidence limits of the force at these maximum excursions. The t-test showed that the forces generated in upgaze were not significantly different from those generated in downgaze ($p=0.15$), neither were the forces generated by the lateral and medial rectus muscles ($p=0.29$).

Force values obtained from the FGT were compared with those obtained from the FDT for the same direction and angle of rotation. The t-test results suggested that the forces obtained from the FGT were significantly higher than those obtained from the FDT for both horizontal and vertical eye movement. For an angle of rotation of 35°, the mean difference was 25 mN ($p=0.05$).

13.2 PATIENTS WITH ORBITAL FRACTURES

Twenty-four patients with different types of orbital fracture, who attended the Oral and Maxillofacial Surgery Unit at Canniesburn Hospital have been examined. The data

FGT Test Direction	Mean Force x (mN)	Upper Confidence Limit $x+t_v(0.025)\frac{s}{\sqrt{n}}$ (mN)	Lower Confidence Limit $x-t_v(0.025)\frac{s}{\sqrt{n}}$ (mN)
Lateral (35^0)	313	334	292
Medial (35^0)	324	341	307
Superior (30^0)	306	327	284
Inferior (30^0)	291	312	271

Table 13.3 Mean values and 95% confidence limits for forces generated at maximum rotation (in mN).

collected from the normal subjects was used to elicit any abnormalities in the results obtained for each case. It was deemed inappropriate to group the patient data in view of the heterogeneity of the conditions investigated.

The results were organised into three different groups according to the injury sustained:

1. Orbital floor fracture in which the inferior rectus muscle was involved either by entrapment or trauma (bruise and/or haemorrhage).
2. Medial wall fracture with medial rectus involvement.
3. Lateral rectus paralysis.

It was expected that increased FDT values would be obtained for the first and second groups, while lateral rectus palsy would be accompanied by a reduced FGT value on attempted abduction.

The results from each patient were compared with the data obtained from normal test subjects.

13.2.1 Orbital Floor Fracture

Seven patients with orbital floor fracture were selected to be presented in this section. Table 13.4 summarises their details.

Patient	Age	Sex	Injury	Ophthalmic Diagnosis
1	36	F	Involved in RTA(road traffic accident) 25/8/90, she initially had soft tissue repair only at another hospital, but the bones of the face had not been repaired.	On 8/10/90 she developed slight enophthalmos (1-2 mm) of the right eye which was displaced down by 4 mm. She suffered from limitation of elevation in abduction resulting in double vision on upgaze. She was managed conservatively. On 12/3/91 she attended the ophthalmic clinic. An orbital floor blow-out fracture with entrapment of the adjacent tissue of the inferior rectus was confirmed by CT.
2	24	F	Slipped on a wet surface on 9/5/91, struck the left side of her face.	Diplopia of both up and downgaze. X-ray confirmed blow-out fracture of orbital floor with entrapment.
3	25	M	Allegedly assaulted and kicked in the face on 7th April 1991.	Suffered from double vision on upgaze. X-ray showed blow-out fracture of left orbital floor.
4	37	M	Allegedly assaulted and kicked on the face on 5th Jan 1990. He sustained a blow-out fracture of the left orbital floor as evidenced by plain X-rays and CT scan.	Suffered from intra-orbital anaesthesia and diplopia on upward gaze and downward of the left eye.
5	17	M	Involved in RTA resulted in a blow-out fracture of the lateral wall and floor of the right orbit.	CT scan showed no evidence of muscle entrapment, but ophthalmic examination showed mild diplopia on up and down ward gaze.
6	43	M	Fell in a railway station and hit the left side of his face on 26/2/90. He sustained a blow-out fracture to the floor of his left orbit.	Examination showed 1-2 mm enophthalmos and retraction of globe on upgaze. The orbital floor was surgically explored and a moderate size defect (10 mm) was repaired with silastic sheet implant. Postoperatively assessment revealed a persistent diplopia on upgaze.
7	23	M	Sustained blow-out fracture of the left orbital floor following a fight on 23/3/90.	Ophthalmic examination revealed slight restriction of the left eye on downgaze with diplopia.

Table 13.4 Orbital floor fracture patients details.

(A) With entrapment

Case 1: A 36-year old female who sustained her injuries following a road traffic accident (Fig 13.7: CT scan). The FDT was carried out for the affected eye in the superior direction. The forces required to rotate the eye in upgaze by more than 10° were significantly higher than the 95% confidence range (Fig 13.8) and started rising markedly after 25° of rotation.

A peculiar non-repetitive curve pattern full of sudden sharp increases of forces at most angles was observed in the first three cycles of rotation in the superior direction (Fig 13.9A), which can, probably be explained by the intermittent release of some of the entrapped tissues which were stretched during upward rotation.

The downward notch (x) observed on the graph during the relaxation phase of the inferiorly directed FDT cycle implies the presence of tethering which transiently released the stem of the contact lens from coupling with the fork, but which was then overcome with a return to the original configuration of the curve.

The clinical and radiographic evidence confirmed inferior rectus muscle entrapment.

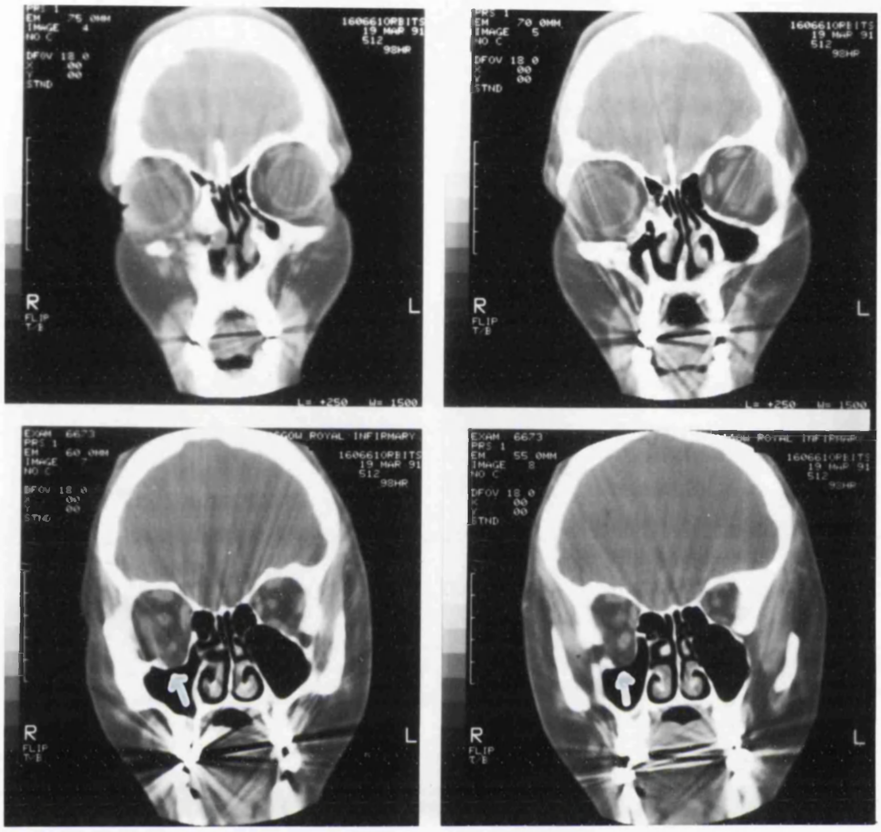


Figure 13.7 Coronal CT scan for case 1.
The arrow indicates the site of the blow-out fracture.

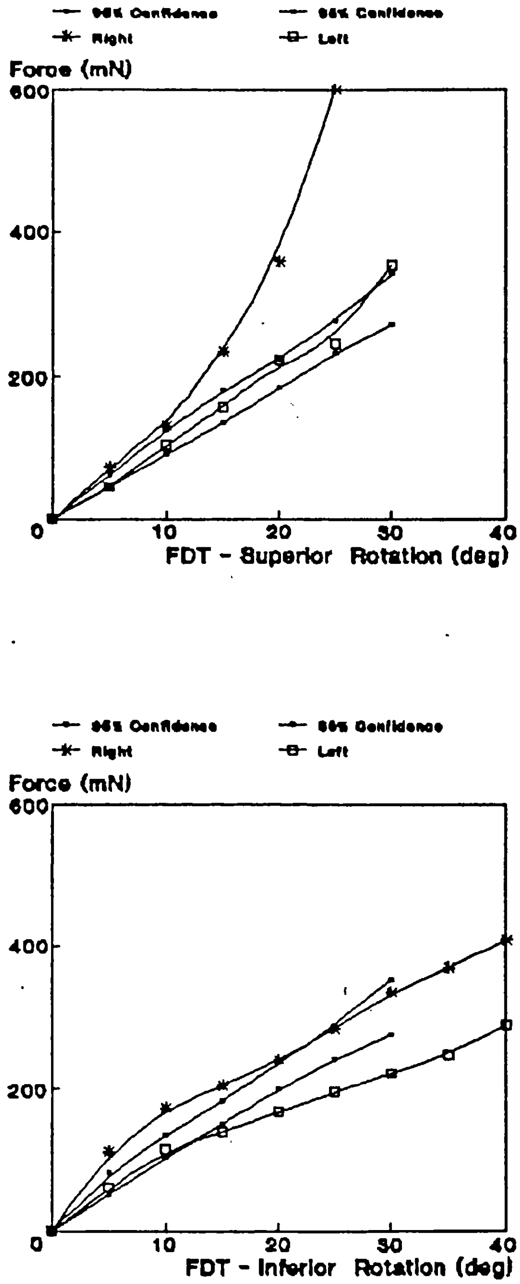
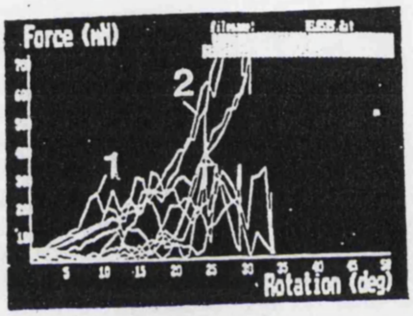
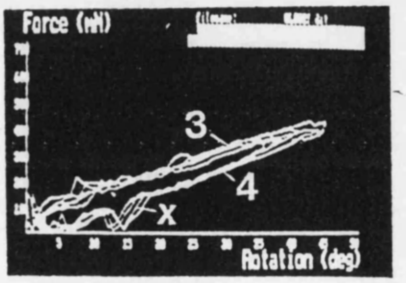


Fig 13.8 FDT results in both superior and inferior directions for subject 1



A)



B)

Fig 13.9 Sequence recordings for FDT case 1

A) superior direction FDT: 1- first recording 2-last recording.

B) inferior direction FDT: 3- loading curve 4- unloading curve

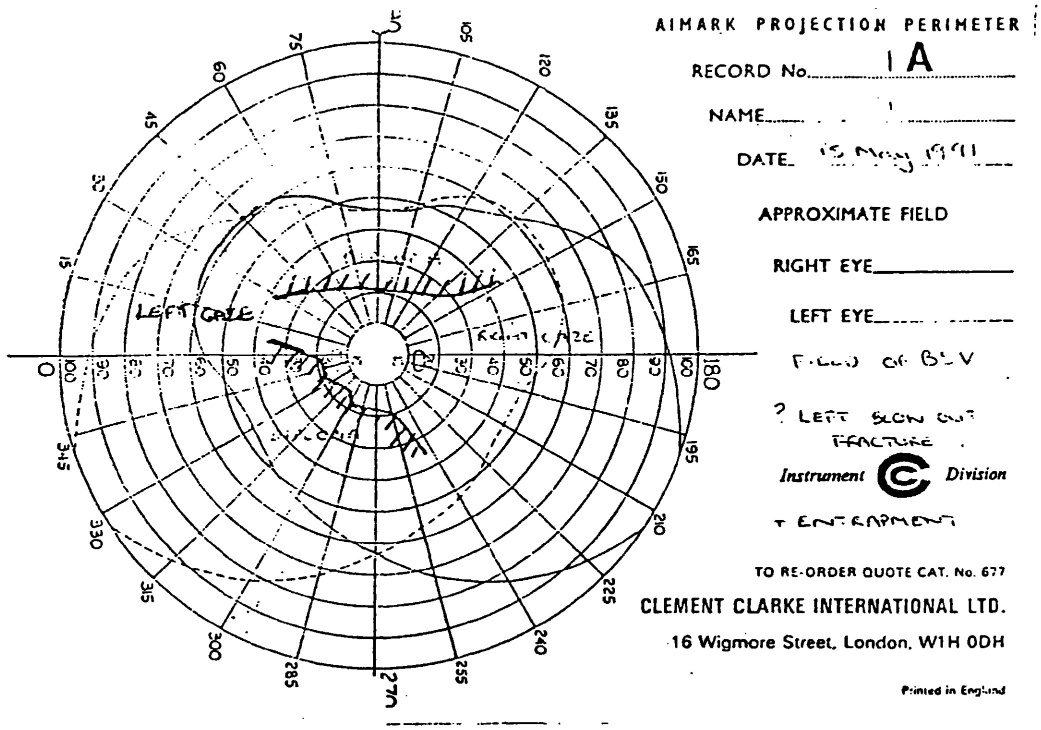
Case 2: A 24-year old female who slipped on a wet surface and struck the left side of her face. She was initially managed conservatively but her fields of binocular single vision (Fig 13.10) showed only slight improvement between two visits, one week apart, while she continued to suffer diplopia.

The FDT in the upward direction (Fig 13.11) at the first visit showed force values falling within the normal 95% confidence range until 25° of rotation. The force then rose to values above the upper normal confidence limit. FDT in the inferior direction remained within the upper normal 95% confidence limit throughout the test.

At the second visit, the forces for upward rotation were within the normal range until 35° of rotation then rose rapidly. The FDT in the inferior direction was greater than the normal range for all angles of rotation.

The FGT was normal at 300 mN for both directions.

The results in the superior direction on both visits corresponded to the field of binocular single vision in which there was improvement in the field of vision on upgaze. The patient was referred for surgery due to her continuing symptomatology. It is of interest that the increasing force required to depress the eye was not accompanied by



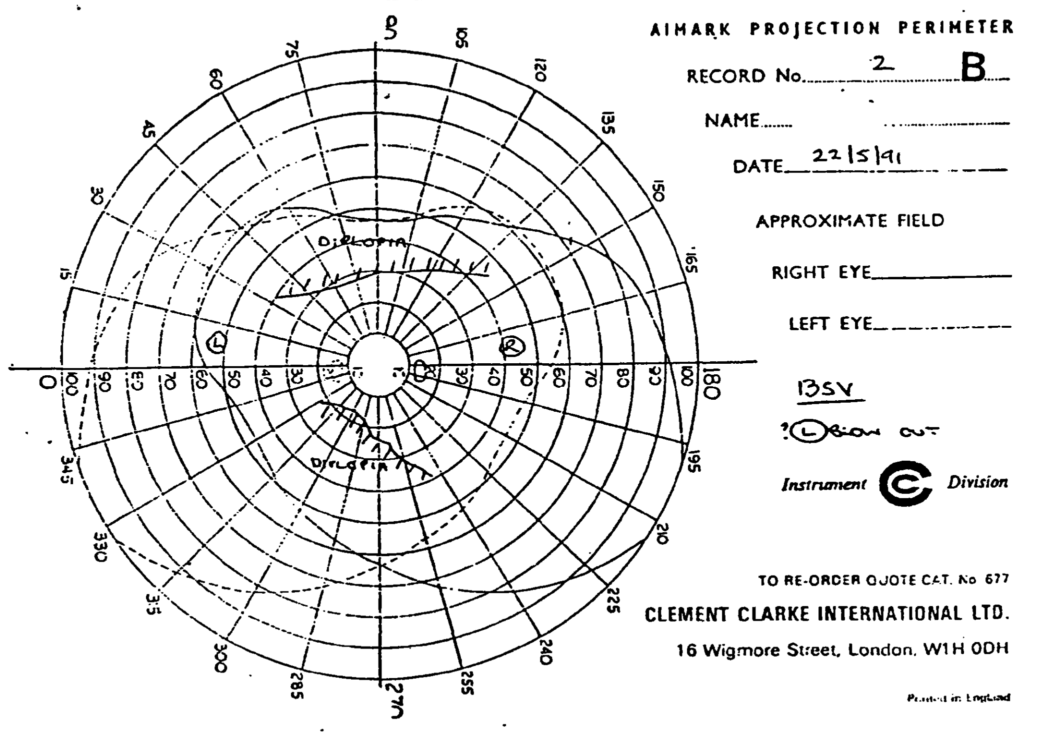


Figure 13.10 Continue

C

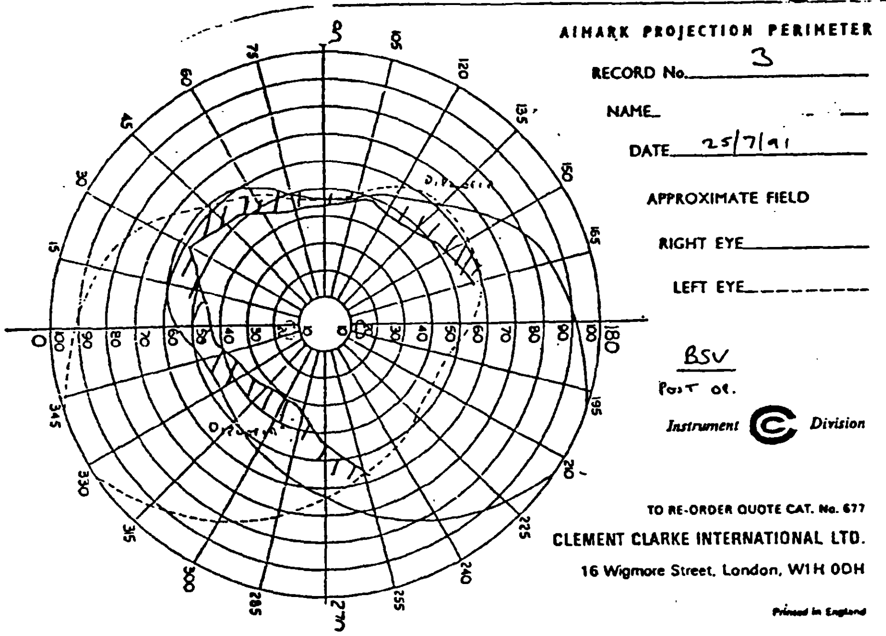


Fig 13.10 Binocular single vision recording for subject 2 at three different visits A and B before surgery C after surgery.

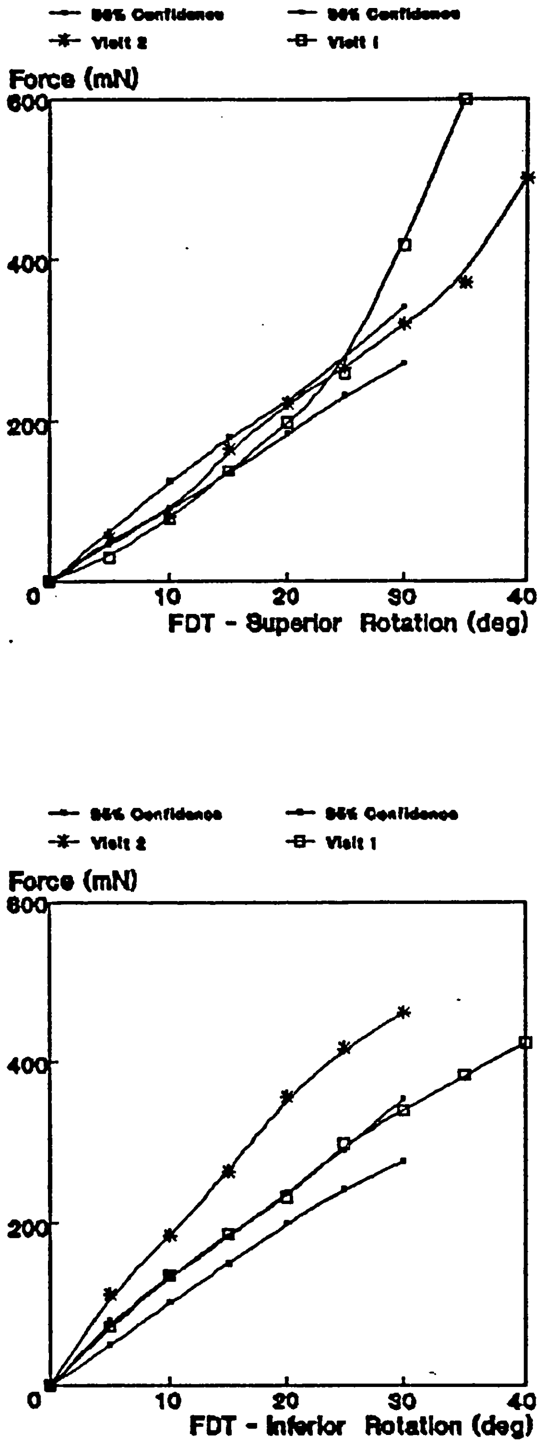


Figure 13. 11 FDT results in both superior and inferior direction for subject 2

any concomitant diminution in the field of BSV on down-gaze.

Post-operatively, the superior FDT showed force-values within the 95% confidence limits until 30° of rotation, and higher at 35°, while it was within the 95% range for the inferior FDT. This coincided with the BSV where diplopia was witnessed at the extremes of vertical gaze.

Table 13.5 lists the results for both the FGT and the FDT at the same angle (30°). It is clear that FDT values on both visits were higher than the corresponding FGT values, while, on the third visit, the FGT was higher than the FDT in both directions especially on elevation.

Case 3: A 25-year old male who was allegedly assaulted and kicked in the face. He sustained a blow-out fracture of the left orbital floor which was evident on plain x-ray (Fig 13.12).

The FDT value in upward gaze (Fig 13.13) on the first visit was greater than the normal range at all angles, with a very steep curve starting at 25° angle.

At the second visit, 4 weeks later, the forces were still significantly greater than the normal range but the steep part of the curve displayed on the first visit was no

	Forces At 30 deg (mN)			
	Superior		Inferior	
	FGT	FDT	FGT	FDT
1st Visit	300	418	300	339
2nd Visit	300	320	300	461
3rd Visit	540	293	340	290
normal 95% confidence limits	284-327	271-341	271-312	275-353

Table 13.5 FGT and FDT results at the same angle for patient 2. First and 2nd visits are pre-op, while 3rd visit is post-op.

Figure 13.12 Plain X ray for subject 3 showing blow out fracture. The arrow indicate the prolapse of orbital soft tissue into the maxillary sinus.

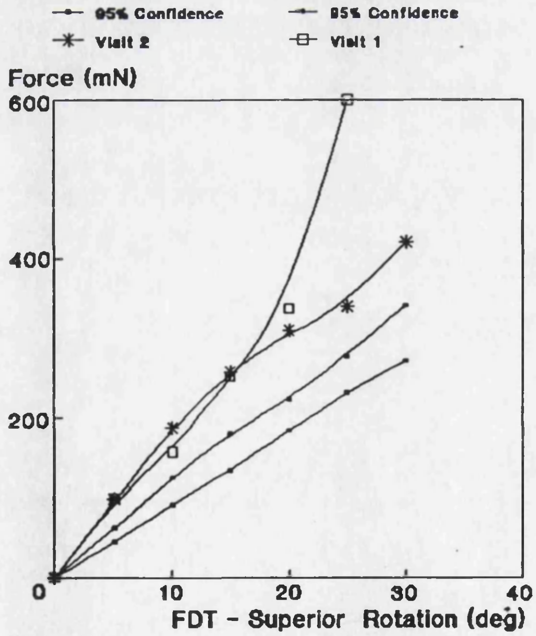


Figure 13.13 FDT results for subject 3 in the superior direction.

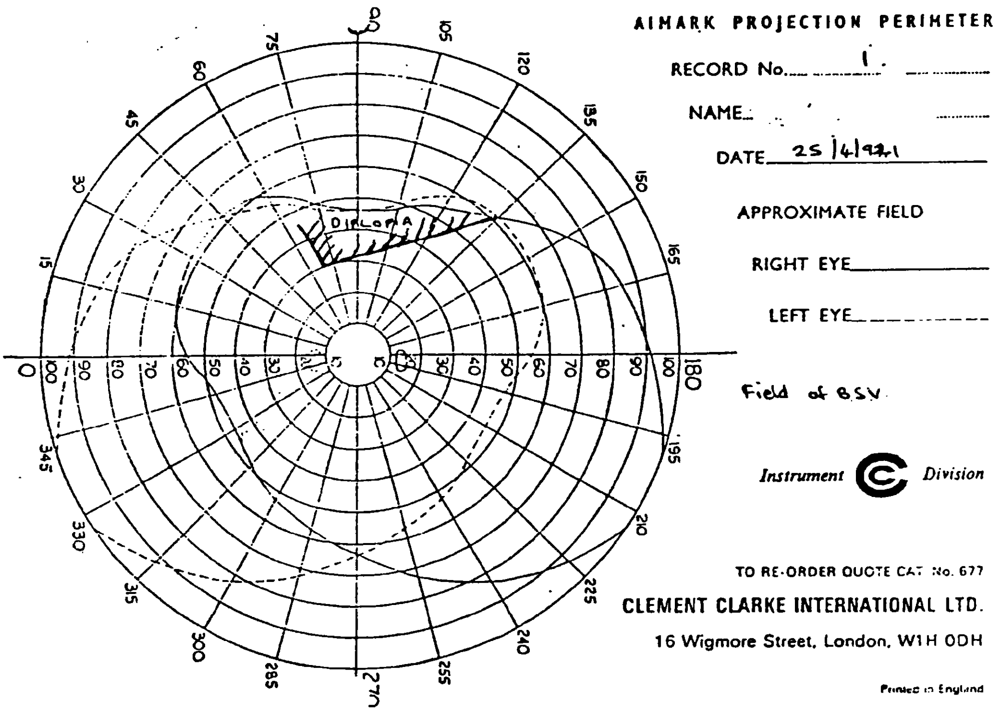
longer present. The fields of binocular single vision plotted on these two consecutive visits (Fig 13.14) show that diplopia occurred initially at 30° of upgaze but this value became 36° on the subsequent visit hence surgical intervention was not deemed to be necessary.

These results demonstrate that improvement in function was accompanied by diminution in resistance to eye movement. Table 13.6 presents the results of both the FDT and FGT at 30° on both visits, and shows how both values for the FDT were higher than the corresponding FGT values.

Direction	FGT (mN)		FDT (mN)	
	1st Visit	2nd Visit	1st Visit	2nd Visit
Superior	230	300	600	339

Table 13.6 FGT and FDT results at 30° for patient 3.

It was of interest to note that one day after the initial FDT the patient had no double vision on upgaze but this symptom returned two days later. It is possible that the FDT itself was partially responsible for the continuing recovery of eye movement function.



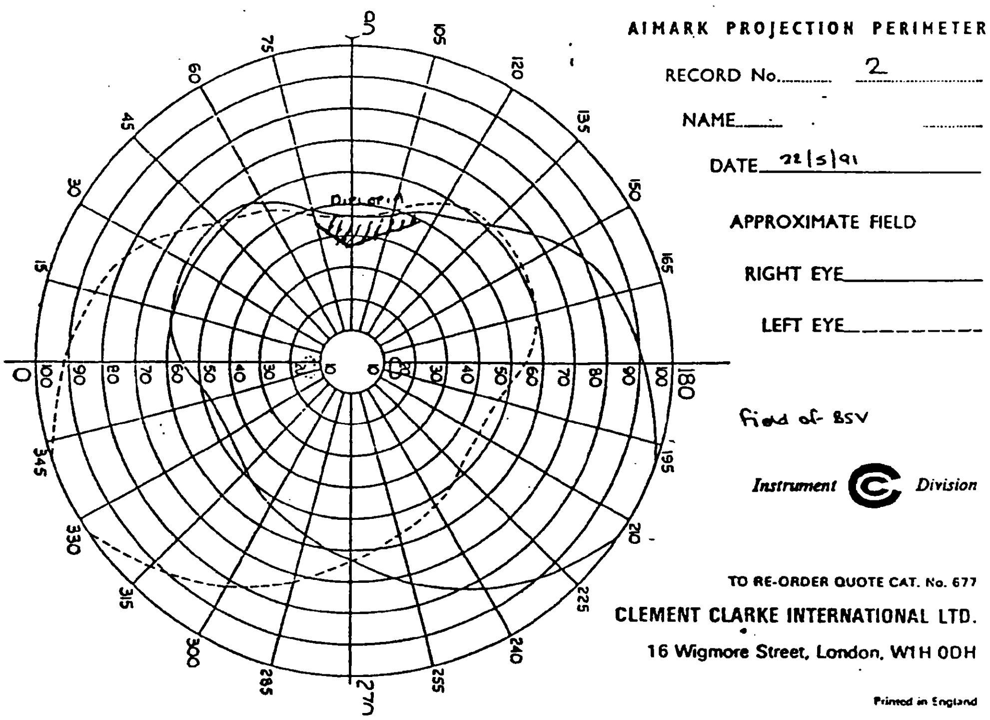


Figure 13.14 Binocular single vision results for subject 3 at two different visits.

Case 4: A 37-year old man who sustained a blow-out fracture of the left orbital floor following alleged assault and a kick on the face.

The forced duction test was performed. The results for the inferior direction were within the normal range (Fig 13.15), while the forces required to rotate the eye upwards were above the normal range right from the start suggesting an entrapment of the inferior rectus which was confirmed by the CT scan. Again, an irregular pattern was noticed in the recordings (Fig 13.16 A & B).

Exploration of the left orbital floor confirmed the diagnosis of a floor blow-out fracture and muscle entrapment. Soft tissue was freed and the bone defect was bridged by silastic sheets.

Case 5: A 17-year old male who sustained a blow-out fracture of the right orbital floor and the lateral wall following a road traffic accident.

It was not possible to decide clinically whether the superior rectus muscle was weak or there was entrapment of the inferior rectus.

Forced duction and force generation tests were performed on both eyes. The results of the FDT in the superior

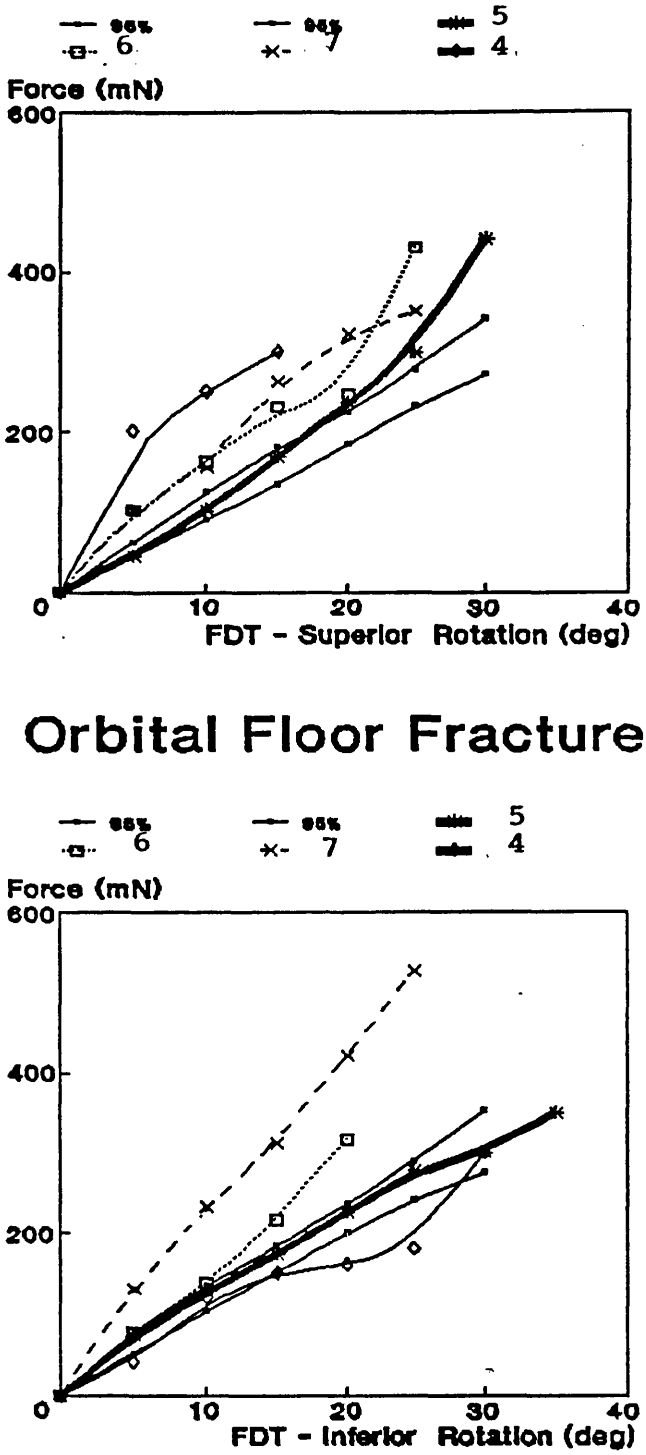
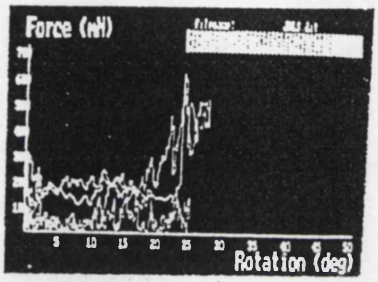
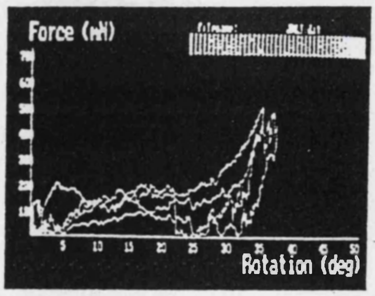


Figure 13.15 FDT results for patients 4, 5, 6, and 7 in both superior and inferior directions.



A)



B)

Figure 13.16 FDT sequence recordings for subject 4.
A) superior direction, B) inferior direction.

direction (Fig 13.15) showed normal forces for up to 17° of rotation but, for greater rotations, the forces were greater than the upper 95% confidence limit particularly for rotations between 20° and 30° for which the force was 440 mN (44g).

The force generation tests (Table 13.7) showed that there was no significant difference between the two eyes and that the superior rectus of the affected eye produced force-values comparable with those from the normal eye, making it unlikely that the diplopia was due to superior rectus weakness.

These results suggested that there was an entrapment of the inferior rectus muscle. The entrapment was confirmed by subsequent surgical exploration of the orbit. FDT results at 35° (Table 13.7) were higher than those obtained by FGT.

(B) With soft tissue trauma

Case 6: A 43-year old man fell in a railway station and hit the left side of his face leading to a blow-out fracture of his left orbital floor. The orbital floor was surgically explored and a moderate size defect (10 mm) was repaired with a silastic implant.

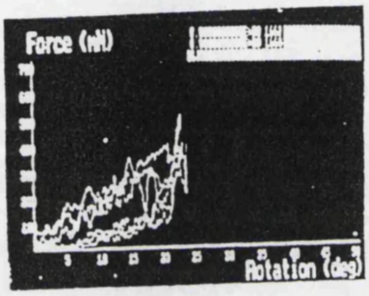
Patient	Test	Forces at 30 deg	
		Superior	Inferior
5	FGT	300	280
	FDT	440	300
6	FGT	200	200
	FDT	430	400
7	FGT	350	200
	FDT	350	526
Normal 95% confidence range	FGT	284-327	271-312
	FDT	271-341	275-353

Table 13.7 Force generation test results for orbital floor fracture patients (all forces in mN).

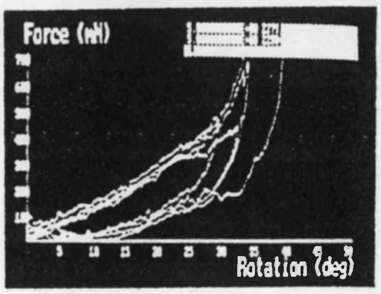
Post-operative assessment revealed persistent diplopia on upgaze. The forced duction and force generation tests were carried out on both eyes. The force values for the FDT for the normal eye for both vertical meridians fell within the 95% confidence range. Forces for the affected eye in the same directions were above the 95% limits of normal range (Fig 13.15). FDT curves in the superior direction comprised a pattern with irregular spikes (Fig 13.17) which were reproducibly present and presumably resulted from damaged muscle tissue acting against the surrounding soft tissue and orbital floor. The FDT in the inferior direction was also high suggesting that the swollen muscle resisted the rotational compression. It is of interest that the FDT results were higher than the FGT (Table 13.7), which indicated that the mechanical resistance was higher than the actual forces exerted by muscle innervation, causing the diplopia in that specific direction. The next ophthalmic examination, three weeks later, confirmed a complete recovery in eye motility.

Case 7: A 23-year old man sustained a blow-out fracture of the left orbit following a fist fight.

The FDT and the FGT were carried out in both vertical directions. The FDT showed forces greater than the normal 95% confidence range in both directions. These could be explained by the trauma and swelling affecting the muscle,



A)



B)

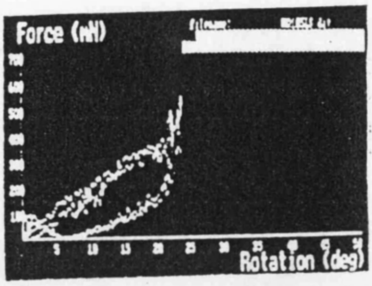
Fig 13.17 FDT sequence recordings for subject 6 A)- superior direction B)- inferior direction.

which influenced the forces produced by passively stretching and compressing the muscles (Figs 13.15 and 13.18 A & B). The FGT results were within the normal range for attempted elevation while it was lower than the 95% normal range for attempted depression providing an indication of weakness in the inferior muscle concerned. The FDT in downgaze was again higher than the FGT, which points out the fact that the mechanical load was higher than the innervation power of the muscle concerned. This corresponds to the field of binocular single vision which has been carried out a week after the FDT.

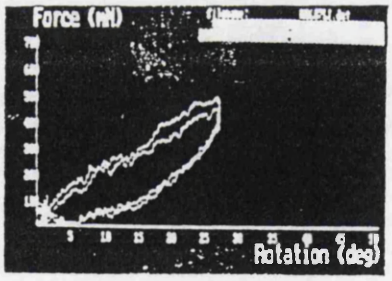
As a result of this test, from which a bruised inferior rectus was inferred, conservative management was recommended and no surgery was carried out. Progressive resolution took place. The fields of binocular single vision (Fig 13.19) demonstrate the follow up results on three successive dates after which the patient recovered completely.

The observation which was consistently made was the higher FDT results than the FGT.

In both cases with bruised muscles, the relaxation phase dropped markedly making the hysteresis loop very wide, implying muscle swelling due to haematoma and oedema,



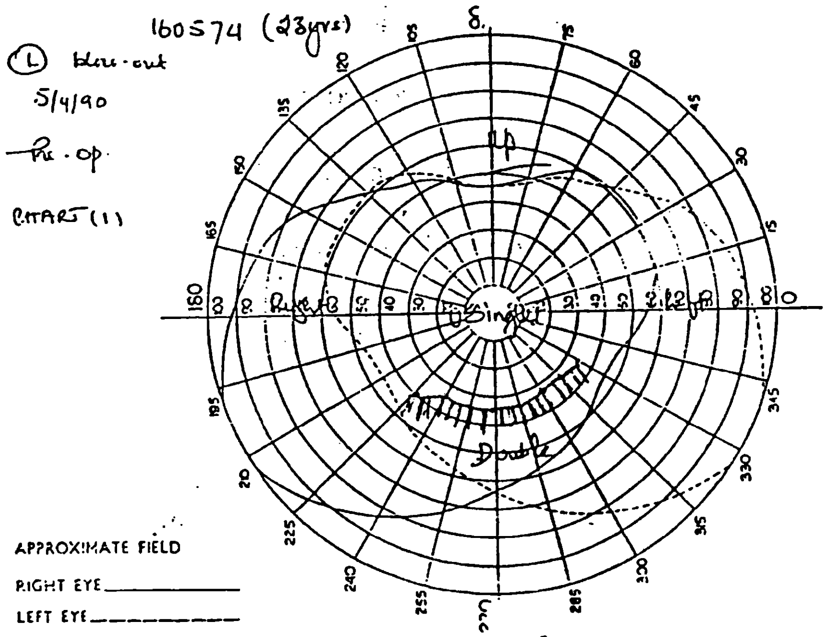
A)



B)

Fig 13.18 FDT sequence recordings for subject 7 A)- superior direction B)- inferior direction.

A)



B)

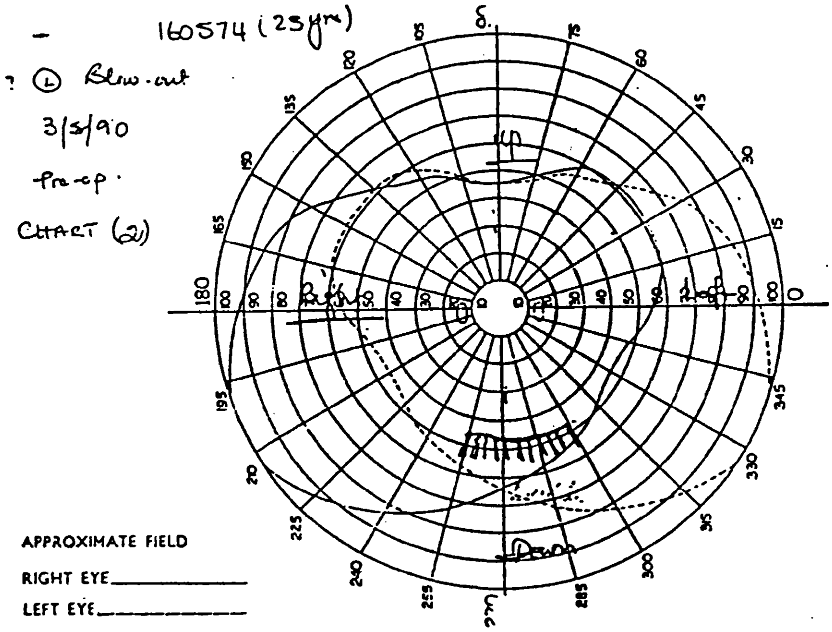


Figure 13.19 Continue

C)

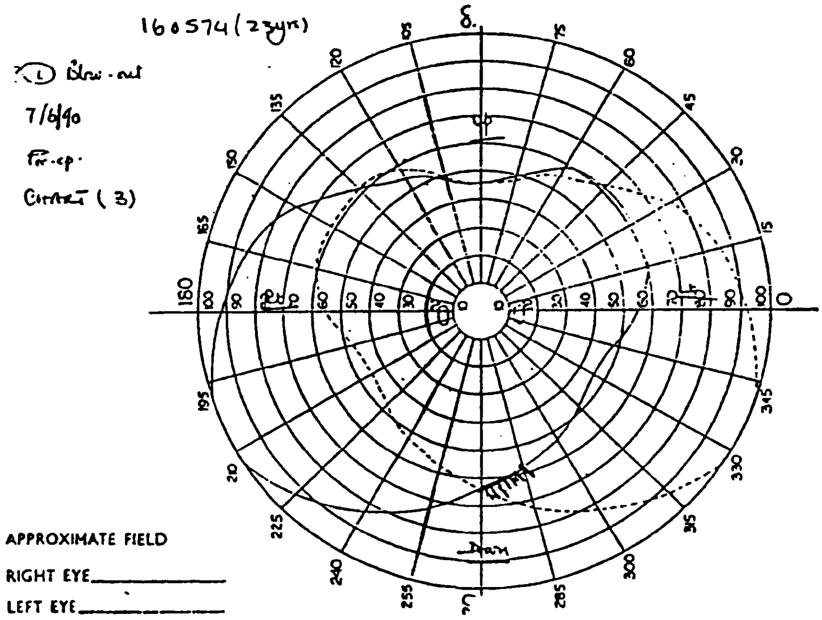


Fig 13.19 Fields of binocular single vision showing the follow up for subject 7 A)- on 5/4/90 B)- on 3/5/90 C)- on 7/6/90

which caused a delay in the response for both stretching and compression.

13.2.2 Medial Wall Fracture

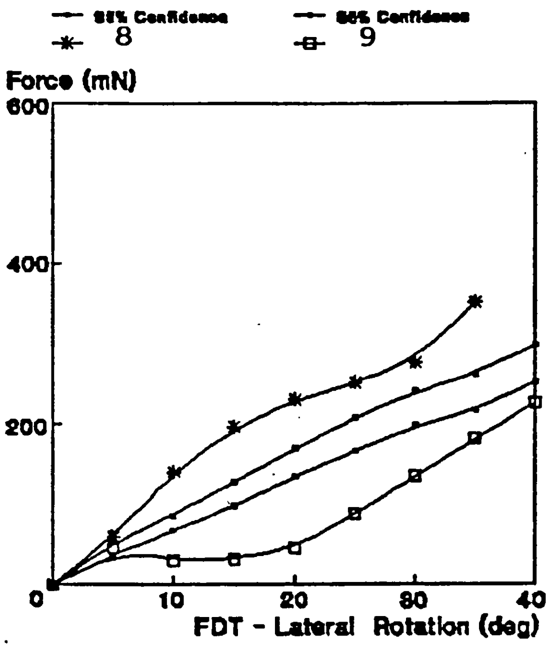
Case 8: A 36-year old man who was allegedly assaulted. He sustained blow-out fractures of the right orbital floor and medial wall which were evident on CT. Ophthalmic examination revealed normal vision but there was a slight impairment of abduction.

The FDT in both medial and lateral directions showed forces which were significantly higher than normal (Figure 13.20). The results of the FGT (Table 13.8) revealed a drop of forces to less than the 95% confidence range, which supports the opinion of a bruised medial rectus.

Patient	Lateral		Medial	
	FGT (mN)	FDT (mN)	FGT (mN)	FDT (mN)
8	200	350	200	-
9	220	181	380	270

Table 13.8 FGT results for medial wall fracture patients.

Case 9: A 38-year old man sustained a fracture of the base of the skull and severe midfacial injuries. Ophthalmic



Medial Wall Fracture

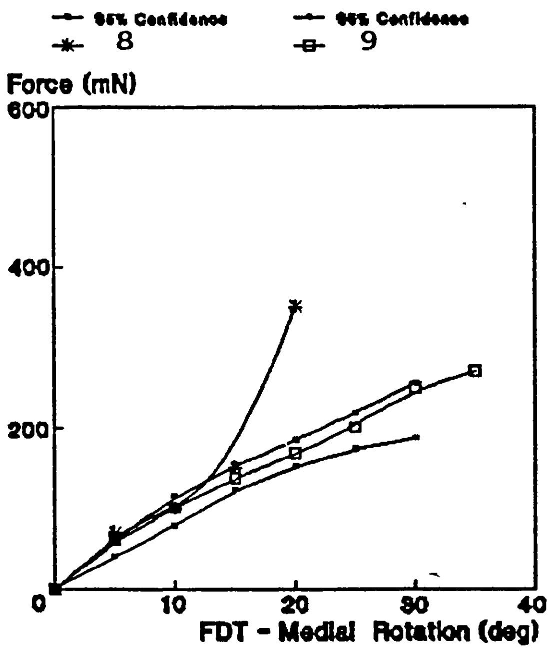


Figure 13.20 FDT results for medial wall involvement patients.

examination revealed failure of right abduction with diplopia in all directions of gaze but especially on attempted abduction. There was 3 mm of enophthalmos.

The FGT was carried out which revealed a force value of only 220 mN (22g) for the lateral rectus, confirming paresis, as compared to 380 mN (38g) for the healthy medial rectus muscle. The low FDT value for lateral rotation (Fig 13.20) can be explained on the basis of enophthalmos. The recession of the globe into the orbit shortens all the rectus muscles. However, the medial rectus is shortened by a greater proportion than the lateral rectus as its overall length is less. The reduced forced duction on lateral rotation can thus be explained by shortening of the medial rectus (Fig 13.21). The forced duction result for adduction was, however, normal. This may be attributed to the concomitant greater area of contact between the lateral rectus and the eye during adduction with an accompanying increased resistance to rotation. Additional cases would be required to verify this finding.

13.2.3 Lateral Rectus Palsy

Four patients with lateral rectus palsy attended the clinic and underwent forced duction and force generation testing. Table 13.9 summarises the patients' details and Table 13.10 lists the FGT and FDT results for the corres-

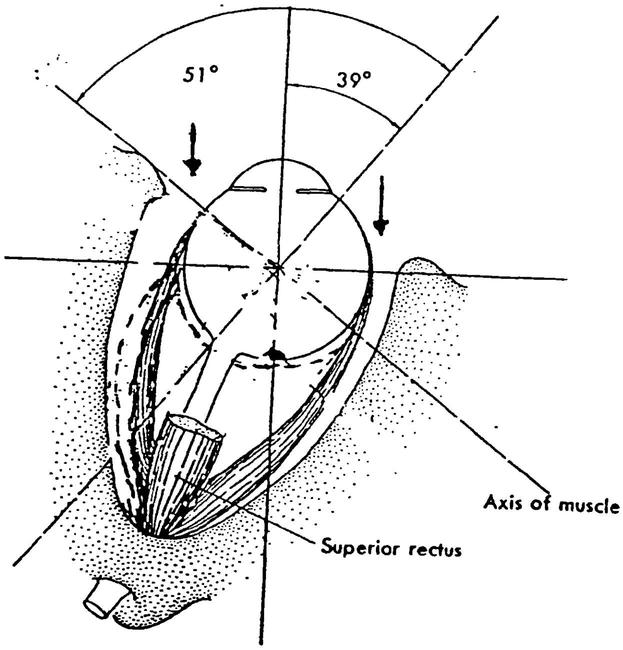


Figure 13.21 Illustrates how enophthalmos could increase the arc of contact between the lateral rectus and the globe.

Patient	Age	Sex	Injury	Ophthalmic diagnosis
10	22	M	Le Fort II maxillary and bilateral malar fractures on 13/8/88.	6th nerve palsy examined 30/1/89.
11	32	M	Sustained head injury CT shows large extradural haematoma on the right side on 28/5/90.	6th nerve palsy examined on 14/6/90.
12	22	M	Allegedly assaulted. Right periorbital bruising, CT. show fracture of the right lateral wall 14/6/90.	Ptosis and complete 6th nerve palsy. Examined on 28/6/90.
13	17	F	Involved in RTA Pre & Post traumatic amnesia with intra-cranial haemorrhage. Blow-out fracture was suspected in the plain X-rays but was not confirmed by the CT scan 21/6/90.	Impaired left abduction proptosis diplopia. Examined on 31/7/90.

Table 13.9 Case summary for lateral rectus palsy patients.

Patient		Forces At 35 deg (mN)	
		Lateral	Medial
10	FGT	100	370
	FDT	250	210
11	FGT	40	350
	FDT	295	80
12	FGT	100	300
	FDT	200	125
13	FGT	40	280
	FDT	195	285
Normal 95% confidence range	FGT	292-334	307-341
	FDT	216-260	-

Table 13.10 FGT and FDT results for lateral rectus palsy patients.

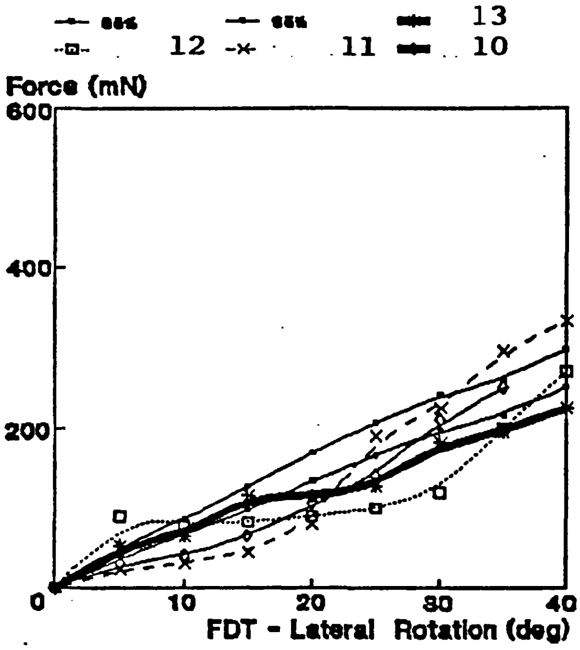
ponding angle of rotation of 35°. The FDT results are also shown in figure 13.22.

The force-values obtained were lower than the 95% normal confidence range in the lateral direction. The ratio of the FGT value for the lateral rectus to that of the medial rectus ranged from 11% to 33%.

These cases have the following features in common. FDT in the lateral direction displays a non-linear curve which exceeded the 95% normal confidence range at certain stages while, for the medial direction, the curve fell below the normal range for most of the patients. The FGT was low for the affected muscle, while it was within the normal range for the medial rectus. Again FDT values were higher than the generated forces (FGT) in the lateral direction.

Generally, there were small hysteresis loops for the lateral direction with a loose lateral rectus and a contracted medial rectus. However, in the medial direction, larger hysteresis loops were noted due to the absence of the lateral rectus tendency to retract to its original position after being extended.

Figure 13.23 shows FGT results for the affected eye of case 11 where the forces generated by attempted adduction had peak values in the normal range of about 300 mN (30g)



Lateral Rectus Palsy

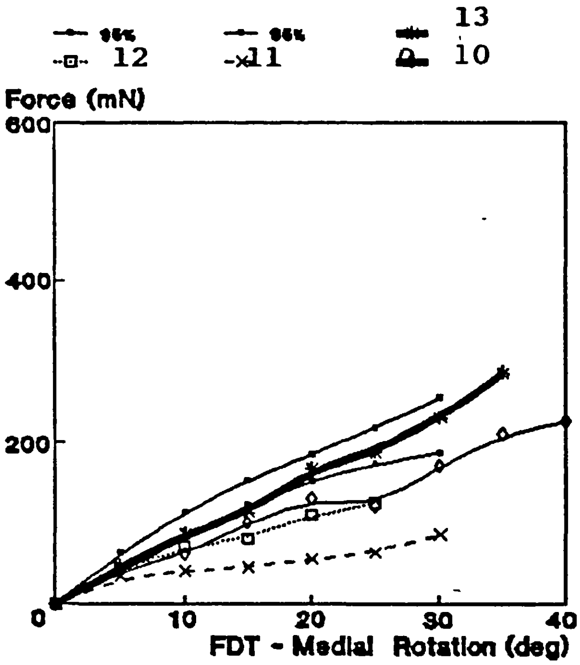
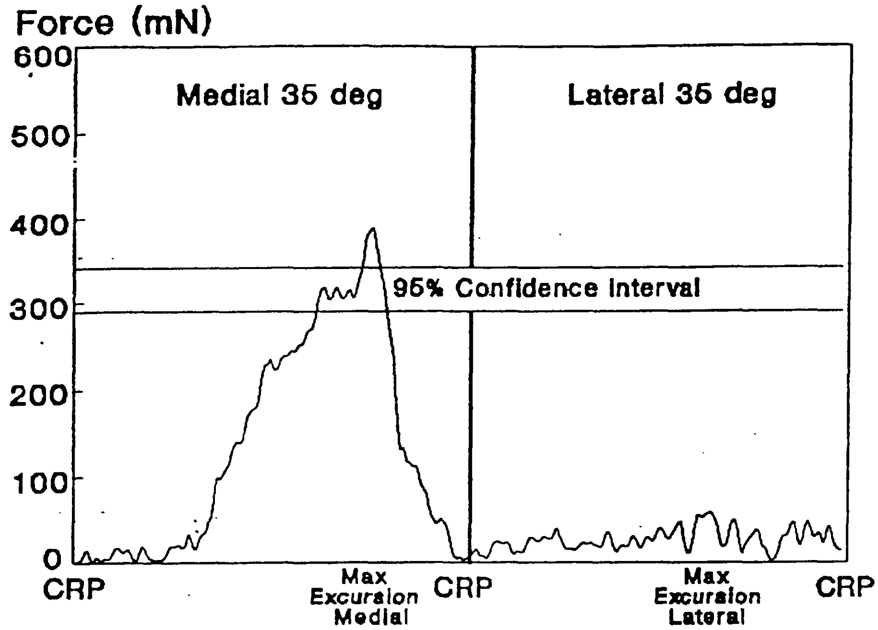
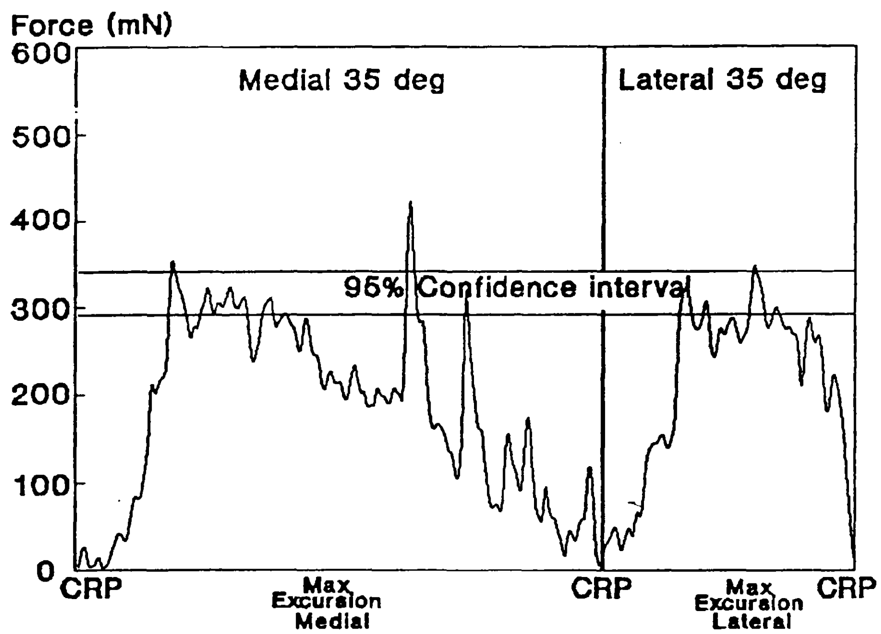


Figure 13.22 FDT results for patients with lateral rectus palsy.



A)



B)

Fig 13.23 FGT in the horizontal plane for patient 11 A) affected (right eye) B) left eye CRP: central resting position.

but only 40 mN (4g) for attempted abduction. The unaffected eye showed results within the normal force range for adduction movement but there was a considerable force overshoot.

The FDT for the same eye in the medial direction produced forces lower than the 95% confidence limits indicating lowered resistance of the lateral rectus. However, in the lateral direction, the forces were largely variable, initially being lower than the 95% confidence limits.

13.3 SUMMARY

This chapter described individual cases. The main purpose of these studies was to establish criteria for possible future use of the system and to aid diagnosis. The data obtained also allows interpretation of the different patterns of results which may be seen clinically.

The common features of each group and their general significance will be discussed in the following chapter.

CHAPTER 14

DISCUSSION AND CONCLUSIONS

14. DISCUSSION AND CONCLUSIONS

The technology used to build the system took into consideration the factors required to facilitate the performance of the forced duction and force generation tests in less than 10 minutes. The use of the Compaq computer equipped with an analogue to digital convertor, in addition to the different electronic components, provided the facility to acquire the data both accurately and quickly. It has also given the examiner an immediate indication of the successful completion of the test. The data obtained from the normal subjects and patients has established the basis for the future development and potential marketing of the device.

14.1 TEST SYSTEM

The test apparatus is robust and reliable and has proved sensitive enough to obtain useful measurements of force and displacement in all directions. The tests described are simple to perform as long as the correct steps of the protocol are followed. Patient cooperation is required to ensure the success of the test since it is performed on the awake patient. This contrasts with previous devices which have been designed for use under general anaesthesia (Metz & Cohen, 1984; Rosenbaum & Myer, 1980).

The measuring system was evaluated by using the simulation prototype and has proved sensitive enough to measure forces in the range of +/- 4 mN (0.4g).

14.1.1 Coupling

The use of the contact lens greatly reduces the trauma often associated with these tests for which alternative methods of coupling have been used, such as a pair of forceps (Collins et al, 1981; Rosenbaum & Myer, 1980; Scott et al, 1972; Jaensch, 1929) or a silk suture sewn to the sclera or to the detached muscle tendon (Metz & Cohen, 1984; Madroszliewicz, 1970; Robinson et al, 1969).

The reduction of pressure beneath the contact lens provides an effective coupling between the eyeball and the system. Slippage was rarely observed and when it occurred it was detected by the concomitant changes in the pressure readings. This allowed immediate action to be taken to re-establish the pressure reduction. This was an advancement on the previous suction cups described by Miller and Kalina (1984), Knox (1974), Robinson et al (1969), and Stephen and Reinecke (1967).

The pressure reduction under the lens may compress the aqueous veins, disrupt the normal drainage of aqueous humour and raise the intraocular pressure. Previous studies (Collins & van Derwerff, 1980; Knox, 1974; Stephen & Reinecke, 1967) have shown that a pressure of 10.7 kPa (80 mmHg) can be tolerated for at least 10 minutes without ill effects. These parameters were observed in the present study, and in no case was there a significant increase in the intraocular pressure which was measured both before and after the test.

The development of the contact lens from one with an oblique separate passage, to one with a central suction passage through the coupling rod (stem) reduces artifacts which may influence the measurements. Such artifacts include the interference imparted by the suction system to the recording by striking the upper or lower lid, or the nose. A central suction duct, thus, allows the test to be performed in all meridians with greater facility.

14.1.2 The Track

The force analysis for the different prototypes developed highlighted the fact that two force components might be applied to the eye, axial forces and tangential forces. In addition, the force measured by the load cell did not always accurately reflect either of these two forces but might be strongly influenced by the angles between the coupling thread and the contact lens stem and load cell (Fig 12.7).

The linear track: The force and displacement analysis of the linear track showed that the magnitude of both the tangential and the antero-posterior forces, as well as the relationship between these forces and the force measured by the cell load, depended on the geometry of the test system. Both tangential and antero-posterior components in the straight track design were dependent on three elements: the displacement, the eye's radius of rotation, and the length of both the stem and the silk thread.

The rotational prototype: This was an improvement on the linear track in that the force measured by the cell load was equal to the tangential force applied to the eyeball, but the presence of an antero-posterior force component influenced the force measurement, altered the condition of the test and tended to dislodge the contact lens.

The circular track device: With its coupling capability eliminated the antero-posterior component and ensured that the measured force was equal to the tangential force applied to the eye.

The results obtained from the three different prototypes showed a steady improvement in quality and reliability of the results.

Data collection with real-time display of force-rotation curves on the computer screen gave the user the opportunity to immediately repeat the test in the case of unsatisfactory results or artifacts. On-line recording of the data in a coded file for each patient allowed easy reference to the data when required.

Collins et al (1981) described the use of an X-Y oscilloscope and data saving on a multitrack FM tape recorder. This method is not as efficient as saving the data on a computer as the tapes had to be changed for individual patients making comparison between different patients' recordings difficult.

Metz and Cohen (1984) employed a better system for recording; however, they did not have the option of real-time continuous display.

The use of intraocular pressure measurement (Zappia et al, 1971) as an alternative to the forced duction test was limited to an angle of 15° up and down only and could not be applied continuously. Moreover, it was not suitable for the force generation test.

Compared to the alternative methods, the current system provided accurate measurements for both the FDT and the

FGT and allowed both tests to be carried out at the same sitting.

14.1.3 The Drive Mechanism

Analysis of the results obtained using the curved track device with manual rotation revealed that a rate of displacement less than 10°/s produced irregular results probably caused by the irregular movement of the load cell carriage produced manually.

Using a DC motor to drive the carriage, which carried the force and displacement transducers, at a speed greater than 10°/s (16.7°/s), improved the force rotation curves by increasing their smoothness and reducing the variation of the recordings as compared with those obtained with the manual driven system. Collins et al (1981) claimed to have employed a rate of displacement of 10°/s. However, these authors used manual techniques only and provided no evidence that this regular rate was actually achieved.

14.1.4 Measuring the Angle of Rotation

To allow an accurate measurement of the angle of rotation, a ten-turn rotational potentiometer was employed. This is probably more accurate and reproducible than other methods such as the millimetre rule (Metz & Cohen, 1984; Scott et al, 1972).

14.2 FORCE-DISPLACEMENT RESPONSE OF NORMAL EXTRAOCULAR MUSCLE

The linear force/rotation relationship of the muscles, obtained from the relaxed extraocular muscles in the forced duction tests, are unlike the non-linear behaviour shown by stretching relaxed muscles by simple tension (Zajac, 1989).

Length-tension tests on the horizontal rectus muscles which have been detached from the eyeball under local anaesthetic (Robinson, 1975) have shown that these muscles demonstrate non-linear behaviour typical of soft tissues, with a high initial compliance and a terminal linear stiffness, but the extent of the compliant phase depends on the resting length and isometric tension in the muscle. In the central rest position, the isometric tension obliterates the initial compliant phase which resulted in the muscle displaying a linear length-tension or rotation-tension response. This was confirmed by Collins and associates (1981) when they measured the stiffness associated with innervated muscles.

Previous forced duction tests have produced a range of values for the force/rotation stiffness of the extraocular muscles. The stiffness was found by Robinson (1975) to be 7.5 mN/deg (0.76g/deg), while Collins et al (1981) found the stiffness in the horizontal plane to be 7.8-16.7 mN/deg (0.8-1.7g/deg) medially and 7.6-11.8 mN/deg (0.78-1.2g/deg) laterally. These values are comparable with those obtained for the normal subjects in the present study (Table 14.1) which demonstrated smaller range of values.

Direction of pull	Stiffness (mN/deg)	
	Mean	Range
Lateral	7.8	7.2-8.4
Medial	8.8	7.9-9.7
Superior	10.6	10.2-11.0
Inferior	11.0	10.5-12.2

Table 14.1 Stiffness values obtained for forced duction in the 4 test directions for normal subjects using the motor driven device.

There are two previous reports on the forms of the force/rotation response obtained by forced lateral and medial duction tests on conscious subjects. Scott and associates

(1972) calculated the eye rotation by an unspecified means from the linear displacement which they measured using their test apparatus. The results showed that the force was a linear function of the rotation for small rotations with an effective stiffness of 5-17.5 mN (0.5-1.8g/deg). At greater rotations, the response became non-linear but was the inverse of that shown by soft tissues, ie the compliance fell with increasing rotation.

Although Scott and co-workers could calculate the rotation from the linear displacement, the load measuring system could only measure the lateral component of the tangential force applied to the eyeball. Compensating for this by replotting the rotation/load data by replacing load with load/(cos angle of rotation) produces a linear force/rotation characteristic for the muscles.

Collins and associates (1981) held their load sensing forceps perpendicular to the globe to ensure that they measured the tangential force. Their results showed a linear force-rotation response and a mean stiffness of 9.4 mN/deg (0.96g/deg) for lateral duction, and 10.5 mN/deg (1.1g/deg) for medial duction, very similar values to those obtained in the current study.

14.2.1 Forces Generated by Normal Extraocular Muscles

In the forced duction test both the force and rotation were measured on the same eye. These could be investigated independently on the contralateral eye. The force generation test was more complex, with the measured force being generated by the muscle under test but the rotation occurring in the contralateral eye.

The magnitude of the forces generated by 50° force generation test were reported by Collins et al (1981) as being 748 mN (76g) for lateral movement and 591 mN (60g) for medial movement, being approximately twice the values found in the current investigation for a 30° rotation.

The similarity of the rotational forces required to rotate the eye either medially or laterally in the current study is in accordance with the principle of Hering's law which states that reciprocal and equal innervation is received by synergistic muscles.

Results obtained from forced duction and force generation tests, performed on the same subject's eye, direction and angle of rotation, show that FGT reflects the eye's potential capability to achieve the mechanical task of rotation. It is this stored energy that generates the required force to overcome the mechanical load as the eye rotated.

14.2.2 Stress-Strain Relationship

Most of the studies concerned with biological tissues have been presented in the form of stress-strain relationships. The task in this section is to calculate the strain and the corresponding stress.

It will be assumed that the strain in muscle is uniform and the strain in tendon is zero (ie it is much stiffer than muscle). Note that one of the coordinates of each insertion is small (Table 14.2; Scott, 1986) and the determination of rotation/strain relations can therefore be treated as a two-dimensional problem for each muscle, and that the lengths of all the muscles are greater than the tendons' lengths.

To calculate the strain, several points should be taken into account (Fig 14.1): I=insertion, T=tangent point at which the muscle first touches the globe, O=origin of muscles, T'=point where tendon joins muscle, and R=radius of eye at I.

On rotation ϕ (radian) the position of T remains constant relative to the origin of the muscle and:

$$\text{Strain}=\frac{(\text{Musclelength})_{L-O}+R.\phi}{(\text{Musclelength})_{L-O}} - 1 \qquad (14\text{-}1)$$

Muscle	Origin: Anatomic	Origin: Coordinates† (mm)		Insertion: Anatomic	Insertion: Coordinates (mm)	
Lateral rectus	Orbit apex, annulus zinnii	X	−13.00	7 mm from lateral limbus	X	+10.08
		Y	−34.00		Y	+6.50
		Z	+0.60		Z	0
Medial rectus	Orbit apex, annulus zinnii	X	−17.00	5 mm from medial limbus	X	−9.65
		Y	−30.00		Y	+8.84
		Z	+0.60		Z	0
Inferior rectus	Orbit apex, annulus zinnii	X	−16.00	6.5 mm from inferior limbus	X	0
		Y	−31.76		Y	+8.02
		Z	−2.40		Z	−10.24
Superior rectus	Orbit apex, annulus zinnii	X	−16.00	7.6 mm from superior limbus	X	0
		Y	−31.76		Y	+7.63
		Z	+3.60		Z	+10.48
Inferior oblique	Behind lacrimal fossa	X	−11.10	Posteriorly in temporal-inferior quadrant	X	+8.70
		Y	+11.34		Y	−7.18
		Z	−15.46		Z	0
Superior oblique	Functional origin at trochlea	X	−15.27	Posteriorly in temporal-superior quadrant	X	+2.90
		Y	+8.24		Y	−4.41
		Z	+12.25		Z	+11.05

Table 14.2 Coordinates of origin and insertion of the extraocular muscles. (After Scott, 1986).

$$\text{Young's Modulus} = \frac{\text{Stress}}{\text{Strain}}$$

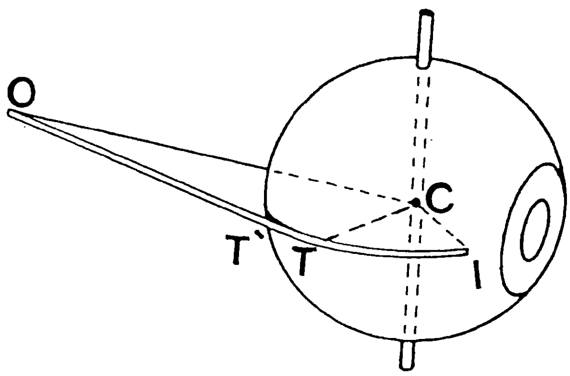


Figure 14.1 Each muscle can be considered to run in a straight line from its origin (O), to a point of tangency (T), where it first touches the globe, and thence to the anatomic insertion (I). The centre of rotation of the globe is (C) and these points define the muscle plane.

$$\text{Stress} = \frac{\text{Load or Force}}{\text{Cross-sectional area}}$$

$$\text{Strain} = \frac{\text{Extension in length}}{\text{Original length}}$$

The radii of the eye can be obtained from the coordinates of the points of insertion of each muscle (Table 14.2; Scott, 1986). At the insertion of the lateral rectus, the radius was 11.7 mm, at the insertion of the medial rectus 13.1 mm and both superior and inferior recti were 13 mm. The strain for the lateral rectus was:

$$\epsilon=\frac{40+11.7\phi}{40} - 1=0.29\phi \qquad (14\text{-}2)$$

$$\epsilon=0.29\text{x}0.017=5\text{x}10^{-3}/\text{degree}=0.5\%/\text{degree} \qquad (14\text{-}3)$$

while for the medial rectus:

$$\epsilon=\frac{39+13.1\phi}{39} - 1=0.34\phi \qquad (14\text{-}4)$$

$$\epsilon=0.34\text{x}0.017\text{x}10^{-3}/\text{degree}=0.57\%/\text{degree} \qquad (14\text{-}5)$$

while for the superior and inferior recti the strain was:

$$\epsilon=0.3250=5.5\text{x}10^{-3}/\text{degree}=0.55\%/\text{degree} \qquad (14\text{-}6)$$

Table 14.3 lists the strains and corresponding stresses taking the cross-sectional areas for the horizontal recti as 16.9 mm^2, and vertical recti 13.1 mm^2 (Scott, 1986).

Rotation (deg)		Strain ε %	Stess σ (mN/mm^2)	Young's modulus (kPa)
Lateral rectus	5	2.5	3	120
	10	5	5.7	114
	15	7.5	8.2	109
	20	10	9.9	99
	25	12.5	11.6	92
	30	15	13	87
Medial rectus	5	2.9	2.7	95
	10	5.7	4.5	78.9
	15	8.6	6.7	78.4
	20	11.4	8.9	78.1
	25	14.3	11	77.2
	30	17.1	12.6	73.7
	35	20	14.1	70.6
	40	22.8	16.2	71.1
Superior rectus	5	2.8	5	183
	10	5.5	9.1	165
	15	8.3	12.7	153.6
	20	11	16.6	151.3
	25	13.8	20.2	147
	30	16.5	24	145.3
Inferior rectus	5	2.8	4.1	147.3
	10	5.5	8.2	149.8
	15	8.3	11.9	144
	20	11	15.6	141.6
	25	13.8	19.3	140
	30	16.5	23.4	141.5

Table 14.3 Calculated strain, stress and Young's modulus for the recti muscles.

The values obtained in this study for Young's modulus were 103 kPa +/- 12.94 kPa for the lateral rectus, 77.8 kPa +/- 7.7 kPa for the medial rectus, 158 kPa +/- 14 kPa for the superior rectus, and 144 kPa +/- 3.7 kPa for the inferior rectus and showed decreasing values by increasing the strain. Fung (1981) reported similar results in testing smooth muscle in vitro, and obtained values of 150 kPa and 77 kPa for two different strain rates.

14.2.3 Influence of Trauma and Disease on Ocular Motility

The detection of motility disorder of the eye using bio-mechanical/tissue characterisation principles has been reported by a number of investigators. However, these investigators have generated limited data, and consequently the interpretation of the results is of limited clinical relevance. The system developed for this study and the data obtained has enabled a more detailed analysis.

The present study has confirmed the importance of both tests being performed alongside each other.

14.2.4 FDT and FGT Values

Blow-out fracture cases

(1) With soft tissue entrapment (13 patients):

The general characteristic of the force-rotation is that the values initially fall within the 95% confidence limits of normal subjects, but rise abruptly to higher values at larger rotations at an angle which corresponds to the onset of diplopia when testing the field of binocular single vision (cases 2 & 3). This can be accounted for by an increase in the frictional forces due to entrapment of soft tissue adjacent to the musculature, or entrapment of the soft tissue adjacent to the point of extensions requiring a greater force. At this point, the actual muscle forces supplied by innervation are not sufficiently large to overcome the mechanical resistance to rotation of the eyeball. FGT values for the muscles involved were usually within the normal confidence range (2, 3 & 5).

(2) Probable bruised muscle (3 patients):

Values for the FDT were higher than the normal 95% confidence limits in both directions of muscle pull at all angles for cases 6 and 7. This was accompanied by low FGT values corresponding to injury sustained by the muscle.

(3) Lateral rectus palsy (4 patients):

The FDT values in the medial direction were lower than the 95% confidence range due to the loss of tone in the lateral rectus muscle. The FGT values for the lateral rectus were considerably lower than the 95% normal confidence limits in accordance with the degree of paralysis.

14.2.5 Qualitative Effects

In addition to the quantitative analysis of the curves in chapter 7, qualitative analysis of the rotation curves was also performed. This took into consideration the following features: (a) roughness of the loading and unloading curves (b) linearity and the nature of departures from linearity, and (c) hysteresis (area between loading and unloading curves). The analysis presented in the following sections revealed differences between the different patient groups.

(a) Roughness (recording irregularities)

As illustrated in figure 13.4, small random irregularities were superimposed on both loading and unloading curves. These were observed in normal subjects. A similar pattern was observed for the patients but the irregularities were more pronounced, perhaps because the patients were apprehensive and less relaxed than the normal volunteers. Irregular non-reproducible curves with marked peaks in the superior direction (Figs 13.9 & 13.16) were observed in 2 (1 & 4) of 13 patients who had a blow-out fracture with entrapment. The curves also showed sudden spikes of increasing load, possibly due to stepwise release of entrapped tissue while stretching the muscle. Such a "therapeutic effect" of the forced duction test has been reported previously (Delplace & Poletti, 1976).

However, patient (1) also exhibited highly reproducible irregularities during the unloading phase of the test in the inferior direction, which could be due to tethering, generating a resisting force which prevented the eye from progressing into the primary position, thus loosening the stem in the fork and hence dropping the measured forces to zero.

Subject (6), one of two who probably had a bruised inferior rectus muscle, showed a markedly irregular curve behaviour during both loading and unloading phases of the tests in the superior direction. This might have been caused by the enlarged muscle size as a sequence of oedema and haematoma.

Patients in the other groups did not, in general, show pronounced irregularities.

(b) Departure from linearity

The force-rotation results obtained from tests on normal subjects were linear. Those obtained from patients were commonly non-linear. They could be classified as showing either a monotonically increasing gradient with rotation (case 2, Fig 13.11, superior direction), or irregular curves (Fig 13.22).

Generally, patients with blow-out fracture with entrapment showed monotonically increasing curves (case 1 and 2) when testing the affected muscle, which may be caused by the increased forces required to stretch the muscle with the entrapped tissue. Usually, the curve for the affected muscle following entrapment comprises two distinct segments (eg case 2). The segment corresponding to that observed with normal subjects is followed by an increase in slope change which appears to correspond with the angle of onset of diplopia.

Of those with trauma, one case (6) showed a slight monotonically increasing force (Fig 13.17), which might be due to enlargement of the muscle produced by oedema.

All four patients with lateral rectus palsy showed irregular curves in the lateral direction (Fig 13.22), an indication of the reduction in the tone of the lateral rectus muscle and hence slackness.

(c) Hysteresis loop

The size of the hysteresis loop will depend on the elastic nature of the muscle (Scott, 1986) and any frictional effects. In general, it can be expected to increase with the presence of fluids eg haematoma or oedema, and decrease with fibrosis. Backlash due to the gear head of the motor may have contributed to the hysteresis, but this is a consistent and reproducible phenomenon. By contrast, the hysteresis observed was variable in its characteristics and related to the patient group being studied. Thus, backlash is unlikely to have made a significant contribution.

The results of blow-out fracture cases with soft tissue entrapment showed that normal hysteresis loops were present apart from the two patients with irregular curve patterns which represent a secondary cause for a larger hysteresis loop (case 1 and 4).

Both patients with a bruised muscle (case 6 & 7) exhibited a large hysteresis loop. This was presumably due to the fluid filling of the muscle (oedema/haematoma) causing an increased viscous component.

These patients also exhibit a greater reproducibility of successive recordings but the initial recordings are not significantly different from the subsequent ones. In both cases there is a greater irregularity in the recordings for FDT in both directions, which is consistent with greater damage to the inferior rectus in each case.

Small hysteresis loops in both lateral and medial directions were observed in all four cases of lateral rectus palsy, which may be due to loss of muscle tissue and relatively more fibrous tissue in the lateral rectus, accompanied by diminished compliance of the medial rectus.

14.3 THE RELATIONSHIP BETWEEN FGT, FDT AND DIPLOPIA

The FDT results show that significant forces are required to rotate the eye even when the muscle being stretched is relaxed. The force required to overcome this passive resistance and move the eyeball is produced by the antagonistic muscle, and its value has been measured at specific rotations of either 30° (superior and inferior rectus) or 35° (lateral and medial rectus) by the FGT.

In the normal subjects, the value obtained from the FGT was invariably greater than the FDT value required to stretch the same muscle by rotating the eye to the angle used for the FGT. This, presumably, reflects the ability of the normal eye muscles to produce the eye movements required to maintain binocular vision.

All patients in the blow-out fracture group showed a marked reduction in the ratio of the values of FGT of the muscle to FDT for the antagonist muscle at the same eye rotation (Table 14.4).

	Mean	Range
Normal Subjects	1.15	1.10-1.22
Trauma	0.59	0.47-0.79
Palsy	0.31	0.14-0.50

Table 14.4 FGT/FDT ratio for normal subjects and for the different patients' groups.

There is a functional association between the FGT/FDT ratios and the occurrence of diplopia, with all patients showing diplopia having a ratio of less than 1. However, the FGT/FDT ratio may not be very helpful for low values in cases with muscle bruising or paresis, in which case, electromyography or ultrasound could be used to differentiate between the two conditions.

The results of the BSV test obtained for patient 2, who had muscle entrapment, were quantified using the method of Woodruff et al (1987). The results obtained during the 3 sequential visits by the patient are displayed in figure 14.2, and for this patient there is a strong positive correlation between the two variables. No general conclusion can be drawn for a single patient and more data is required. It does appear, however, that unless the FGT is greater than the FDT in the antagonist muscle, measured at the same angle, there will be limitation of eye rotation and resultant diplopia.

Table 14.5 summarises the results presented in this section of thesis.

14.4 COMPARISON WITH ANTAGONIST MUSCLES

FDT values were plotted against each other for the same angle (at 5° increments of rotation) for the same meridians both horizontally (Fig 14.3A) and vertically (Fig 14.3B).

Normal control results (Fig 14.3) showed an almost linear relationship very close to the unit slope of equal force at equal rotation.

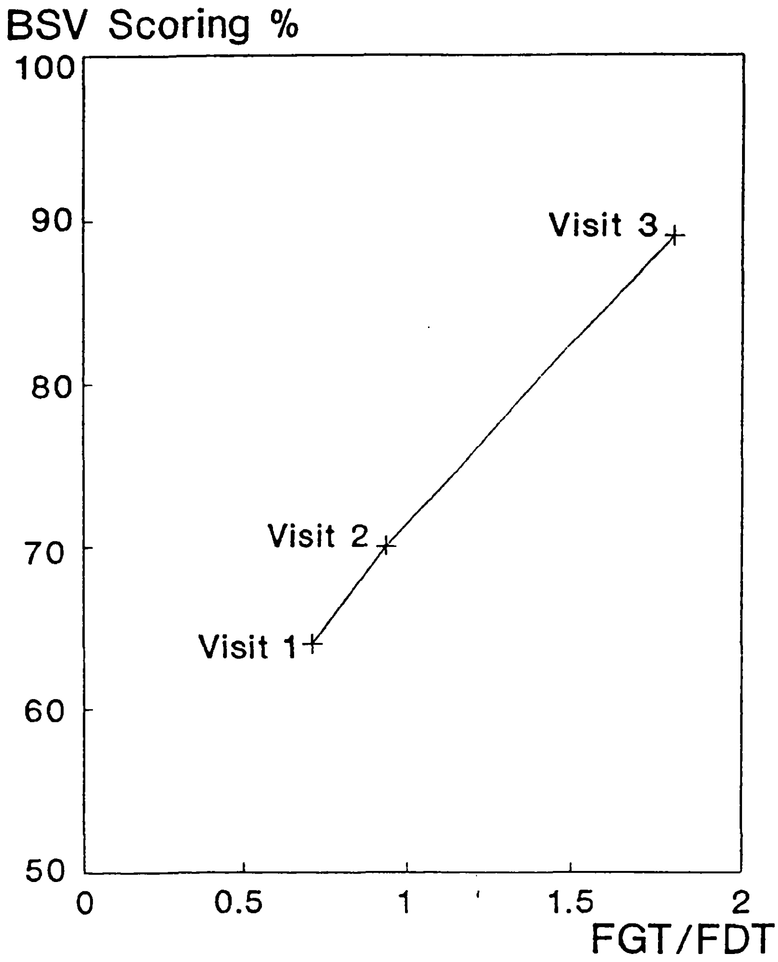


Figure 14.2 Illustrates the relationship between B.S.V. scoring and FGT/FDT ratio for patient 2. First and second visits before surgery. Third visit after surgery.

Subject	Diagnosis	Direction of pull	Diplopia direction	FDT				FGT			FGT / FDT
				Linear	= 95 %	> 95 %	< 95 %	= 95 %	> 95 %	< 95 %	
1	Inferior entrapment	Superior Inferior	Y -	N Y	- -	Y Y	- -	- -	- -	- -	- -
2	Inferior entrapment	Superior Inferior	Y Y	N Y	- -	Y Y	- -	Y Y	- -	- -	0.7 0.88
5	=	Superior Inferior	Y Y	N Y	- Y	Y -	- -	Y Y	- -	- -	0.68 0.93
4	=	Superior Inferior	Y Y	N N	- -	Y -	- Y	- -	- -	- -	- -
3	Inferior prolapse	Superior	Y	N	-	Y	-	-	-	Y	0.38
6	Inferior bruising	Superior Inferior	Y N	Y N	- -	Y Y	- -	- -	- -	Y Y	0.46 0.5
7	=	Superior Inferior	N Y	Y Y	- -	Y Y	- -	Y -	- -	- Y	1 0.38
8	Medial bruising	Lateral Medial	Y N	N N	- -	Y Y	- -	- -	- -	Y Y	0.57 -
10	Lateral palsy	Lateral Medial	Y N	N N	- -	- -	Y Y	- -	- Y	Y -	0.4 1.76
11	=	Lateral Medial	Y N	N N	- -	- -	Y Y	- Y	- -	Y -	0.13 4.4
12	=	Lateral Medial	Y N	N N	- -	- -	Y Y	- Y	- -	Y -	0.5 2.4
13	=	Lateral Medial	Y N	N N	- Y	Y -	Y -	- Y	- -	Y -	0.2 0.98

Table 14.5 Summary of the abnormalities detected in the different patients' groups. Although there are too few patients to test the sensitivity or selectivity of the group of interest, some conclusions can be made.

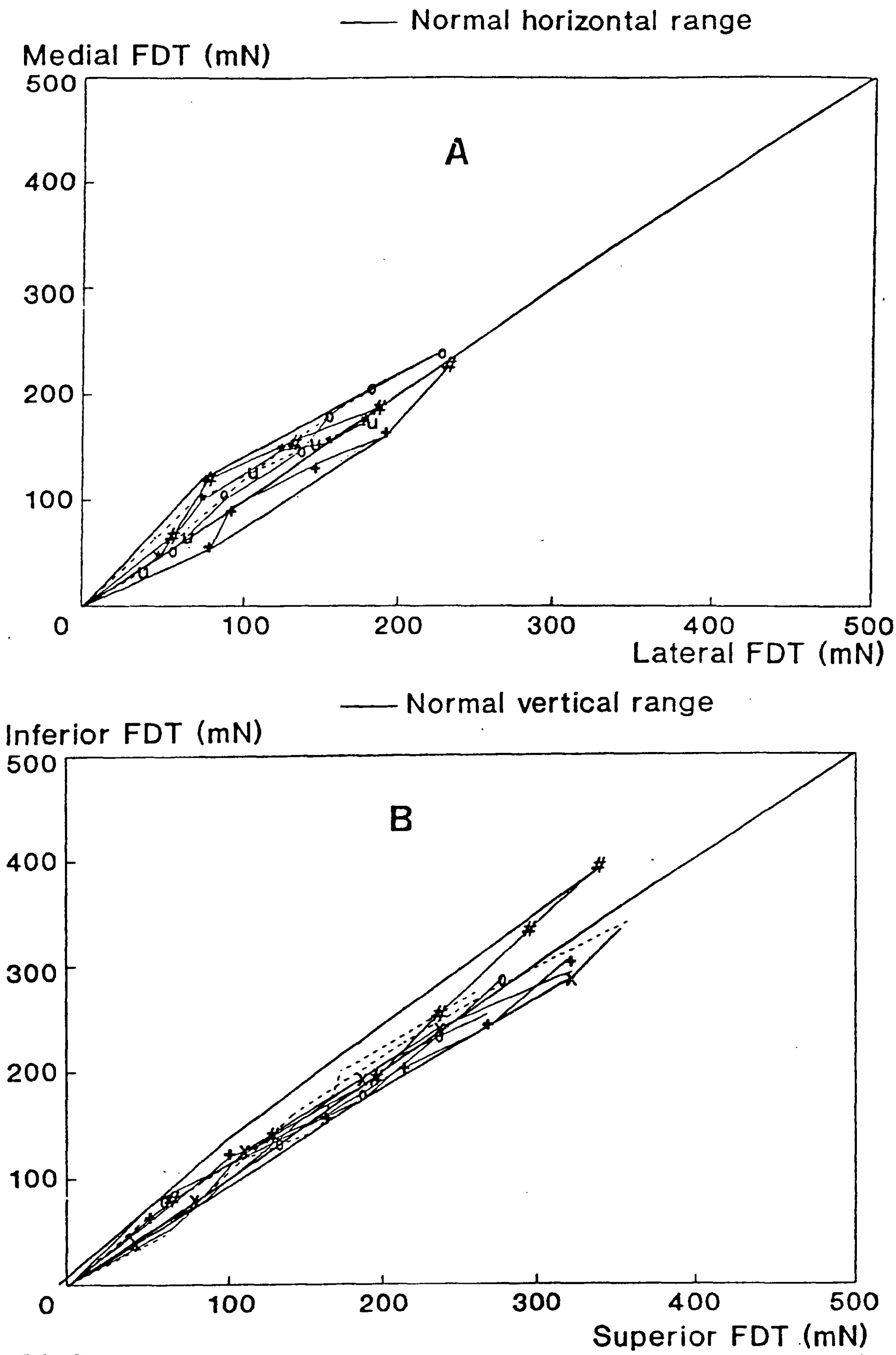


Fig 14. 3 FDT results relationship in the same meridian at intervals of 5^{0} for 10 normal controls. The boundaries for the normal range were drawn to show the narrow band in which the normal data fall.

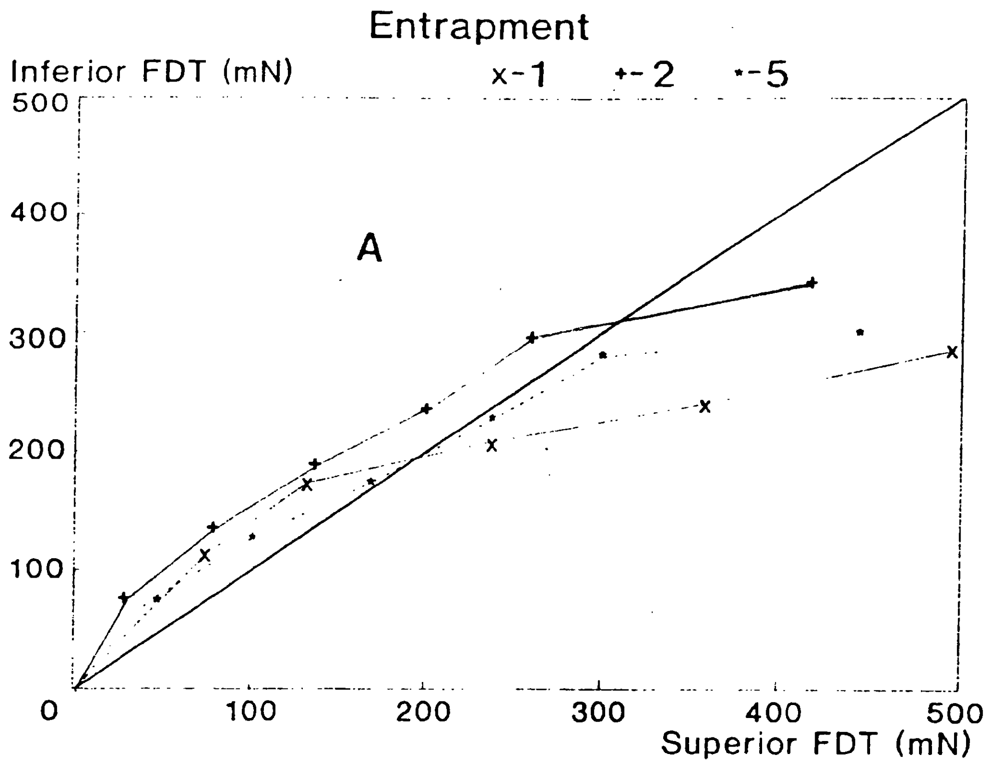
A) Horizontal meridian B) Vertical meridian

This method was applied to the different patient groups and showed different patterns of response due to their abnormalities.

Patients with inferior entrapment showed a monotonically decreasing pattern (Fig 14.4A), with the FDT in the inferior direction initially being higher than the FDT in the superior direction. However, in the later part of these curves the situation is reversed with the curves crossing the unitary slope. The cross over point coincided with the angle for which the BSV test detected the onset of diplopia.

Patients with trauma (bruises/haematoma) showed a monotonically increasing curve pattern (Fig 14.4B), with most of the points being above the unit gradient, particularly at large rotations. This is due to the increased forces required to compress the bruised and enlarged inferior rectus compared to those required to stretch the same muscle.

Non-linear irregular curve patterns were observed in the group of patients with lateral rectus palsy (Fig 14.4C) as a sequence to the paralysed lateral rectus muscle and loss of tone. The points were also compressed into a smaller area than normal.



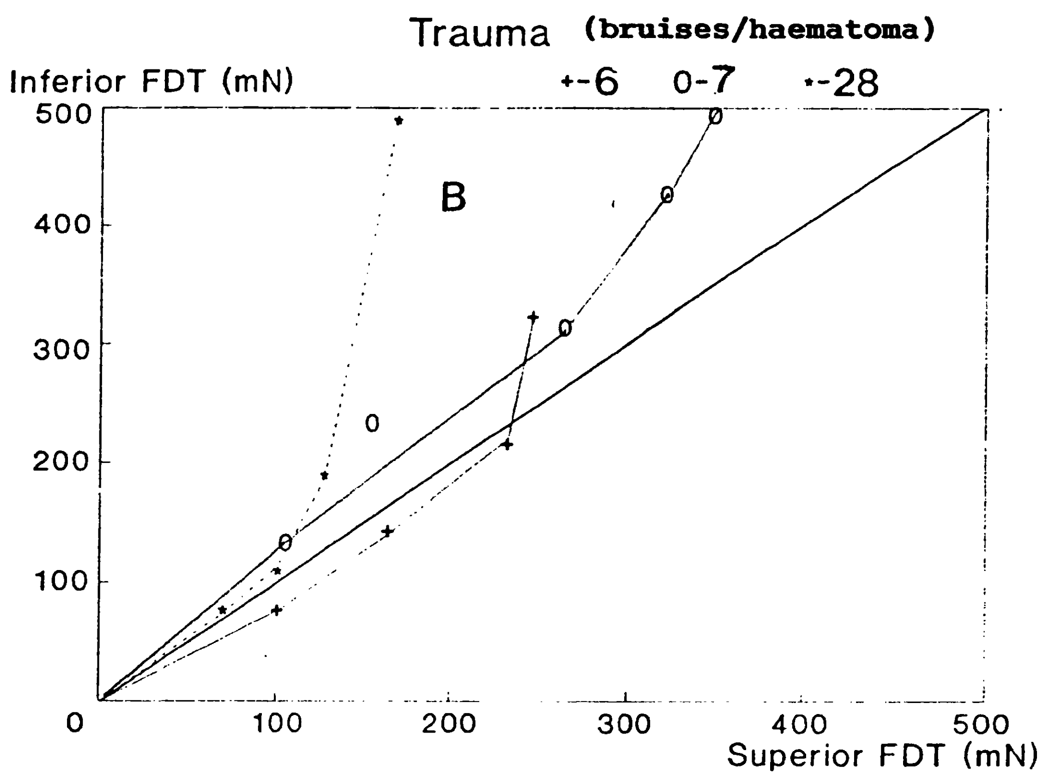


Figure 14.4 Continue

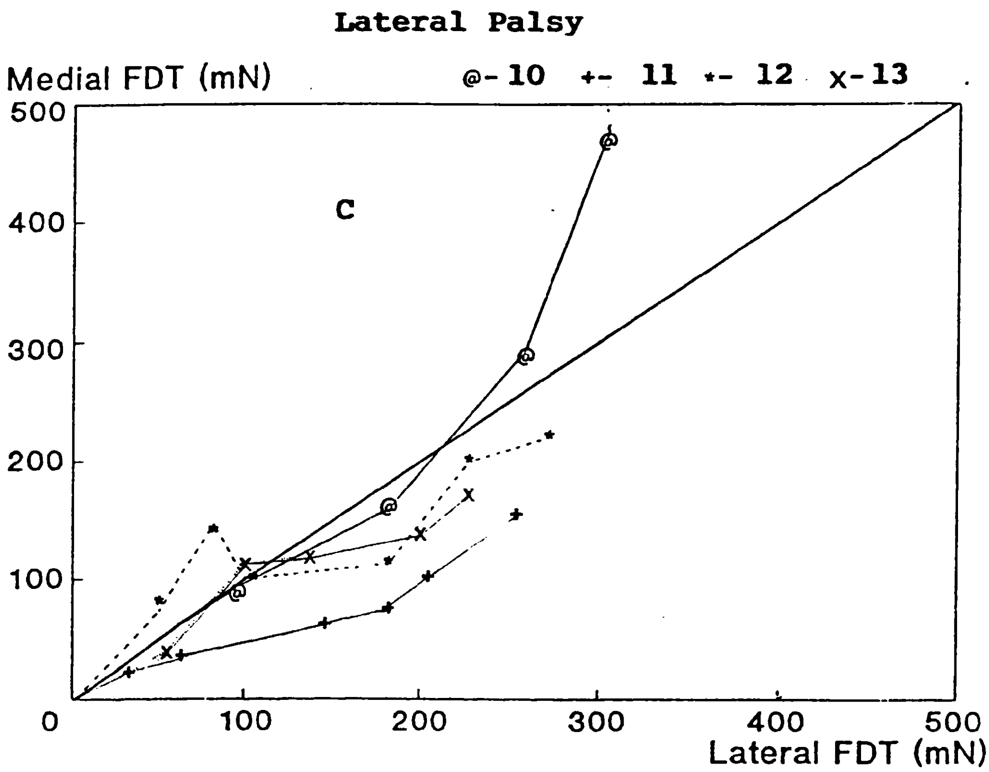


Figure 14.4 FDT results plotted against each other in the same meridian. A: 3 patients with inferior muscle entrapment, B: 3 patients with bruised muscles, C: 4 patients with lateral palsy.

14.5 CONCLUSIONS

The test system described provided a reliable and acceptable means for carrying out quantitative forced duction and force generation tests. The developmental steps improved the system and highlighted the disadvantages of previous designs.

The normal results fall within a relatively narrow range and the patient tests show that specific eye muscle deficits produce different patterns of results falling outside the normal range, and these are specific to the underlying pathology.

14.6 SUGGESTIONS FOR FUTURE WORK

The present system is designed for use in the out-patient department. A further step would be to modify the design to allow it to be used on patients under general anaesthesia before, during and after surgery.

The FGT provides valuable additional information to supplement the FDT results. The design should be modified to improve the FGT by activating a target to travel at a fixed speed of 16.7 deg/sec, the same as that of the FDT, thus producing a force generation/angle output.

Studying the pattern of the force-rotation curve for the FGT may yield valuable information about the innervation, nature and extent of disturbances in strabismus. Accurate FG and FD testing may prove useful in planning surgical management.

High FGT values were observed in muscles functioning against restriction of the antagonist muscles in patients with dysthyroid-eye disease (Metz, 1985). High FDT values were also obtained for the restricted fibrosed muscles in both horizontal and vertical directions. The developed equipment would be useful in studying ocular motility disorders in thyroid-eye patients and in planning future management.

Since the equipment can be oriented in all meridians, establishing a normal data base for the 45° meridian could help in the diagnosis of Brown's syndrome by determining the forces required to cause elevation in adduction.

More data and patients with different abnormalities are needed for the development of an expert system and a systematic study is required to detect features correlating with disease/abnormalities combined with automatic detection of abnormalities in output eg roughness and non-linearity of the FDT.

The current design is based on the assumption that the average diameter of the eye is 25.5 mm (Tortora & Anagnostakos, 1984), and the average distance from the cornea to the centre of rotation of the eye is 13.5 mm (Scott, 1986). These figures, although applicable to the present study, may not be representative of the entire population of subjects studied. The assumption that the eye is an ideal ball, and the fact that the centre of rotation is not fixed but travels on a centrode (Carpenter, 1988; Fry & Hill, 1963 & 1962), may be an additional factor contributing to the introduction of alterations in the length of the stem coupled to the fork; consequently the stem and fork do not remain aligned at all times.

The alterations become more pronounced in trauma/disease, where additional factors like oedema, fibrosis etc could be introduced. This problem has to be looked at carefully in future investigations.

A system of recording would initially be required in order to provide the basis for appropriate mathematical modelling for the eye movement, and using that model to correct the data acquired from the present system.

The apparatus described has been designed and built simply as a prototype. The results obtained indicate that there may well be an international market for a purpose built piece of equipment, built to the specifications described in this thesis. The test system incorporated a computer and the clinical usefulness of the device would be augmented by the development of an expert system to characterise the data.

REFERENCES

REFERENCES

Al-Qurainy, I A, Dutton, G N, Moos, K F, Reynolds, S T & McMillan, N (1988). Orbital injury complicated by entrapment of the superior oblique tendon: a case report. **British Journal of Oral and Maxillo-facial Surgery, 26,** 336-340.

Altonen, M, Kohonen, A & Dickhoff, K (1976). Treatment of zygomatic fractures: internal wiring-antral packing-reposition without fixation. A comparative study. **Journal of Maxillofacial Surgery, 4,** 107-115.

Anderson, M (1961). Loss of accommodation and convergence following whiplash injury to the cervical spine. **British Orthoptic Journal, 18,** 117-120.

Anderson, R L, Panie, W R & Gross, C E (1982). Optic nerve blindness following blunt forehead trauma. **Ophthalmology, 89,** 445-455.

Andree, G & Klein, A G (1983). Ophthalmological findings after head and brain injuries. **Fortschritte der Ophthalmologie, 80,** 179-181.

Archer, D B (1986). Traumatic retinal vasculopathy. **Transactions of the Ophthalmological Societies of the United Kingdom, 105,** 361-384.

Arden, R L & Moore, G K (1989). Complete post-traumatic ptosis: A mechanism for recovery? **Laryngoscope, 99,** 1175-1179.

Armitage, P (1971). Statistical Methods in Medical Research, 4.8, 7.5. Oxford: Blackwell.

Atiyeh, W A, May, M & Gildersleeve, G (1966). Acute true blow-out fracture of the orbit. **Southern Medical Journal, 59,** 332-336.

Babajews, A & Williams, J Ll (1986). Blindness after trauma insufficient to cause bony injury: Case report and review. **British Journal of Oral and Maxillofacial Surgery, 24,** 7-11.

Barclay, T L (1958). Diplopia in association with fractures involving the zygomatic bone. **British Journal of Plastic Surgery, 11,** 147-157.

Barr, D R (1970). Ocular muscle complications of orbital fractures. **International Ophthalmology Clinics, 10,** 153- 160.

Bastek, J V, Foos, R Y & Heckenlively, J (1981). Traumatic pigmentary retinopathy. **American Journal of Ophthalmology, 92,** 621-624.

Beirne, O R, Schwartz, H C & Leake, D L (1981). Unusual ocular complications in fractures involving the orbit. **International Journal of Oral Surgery, 10,** 12-16.

Benedict, W L (1936). Diseases of the orbit. In: The Eye and Its Diseases, ed. Berens, C, Ch. 33, p. 330. Philadelphia: W. B. Saunders Company.

Benson, W E, Shakin, J & Sarin, L K (1991). Blunt trauma. In: Duane's Clinical Ophthalmology, ed. Tasman, W & Jaeger, E A, Vol 3, Ch. 31. Philadelphia: J B. Lippincott Company.

Berkowitz, R A, Putterman, A M & Patel, D B (1981). Prolapse of the globe into the maxillary sinus after orbital floor fracture. **American Journal of Ophthalmology, 91,** 253-257.

Berlin, R (1873). Zur sogenannten commotio retinae. **Klinische Monatsblatter fur Augenheilkunde, 11,** 42-78

Binder, P S (1978). Evaluation of the eye following periorbital trauma. **Head and Neck Surgery, 1,** 139 -147.

Bleeker, G M, Oei, T H, Gillissen, J P A, Verkerk, H H & Peeters, H J (1975). Fractures of the orbital roof. **Ophthalmologica, 171,** 360-360.

Blight, R & Hart, J C D (1977). Structural changes in the outer retinal layers following blunt mechanical non-perforating trauma to the globe: an experimental study. **British Journal of Ophthalmology, 61,** 573-587.

Bloch, F, Hansen, W W & Packard, M (1946). The nuclear induction experiment. **Physiological Reviews, 70,** 474-485.

Bourne, W M, McCarey, B E & Kaufman, H E (1976). Clinical specular microscopy. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 81,** 743-753.

Brinkley, J R & Walonker, F (1983). Convergence amplitude insufficiency. **Annals of Ophthalmology, 15,** 826-831.

Britten, M J A (1965). Follow-up of 54 cases of ocular contusion with hyphaema with special reference to the appearance and function of the filteration angle. **British Journal of Ophthalmology, 49,** 120-127.

Burger, L J, Kalvin, N H & Smith, J L (1970). Acquired lesions of the fourth cranial nerve. **Brain, 93,** 567-574.

Butt, W D (1979). Sudden blindness following reduction of a malar fracture. **Annals of Plastic Surgery, 2,** 522-524.

Candler, R G (1945). Ocular sequelae of head injuries: The response of personnel affected by head injury to orthoptic treatment. **Transactions of the Ophthalmological Society of the United Kingdom, 65,** 57-67.

Carpenter, R H S (1988). Static and dynamic of the eye. In: Movements of the Human Eye, ed. Barker, M & Schubert, J. 2nd Ed. Ch. 7, pp 139-181. London: Pion Ltd.

Catone, G A, Morrissette, M P & Carlson, E R (1988). A retrospective study of untreated orbital blow-out fractures. **Journal of Oral and Maxillofacial Surgery, 46,** 1033-1038.

Cherry, P M H (1978). Indirect traumatic rupture of the globe. **Archives of Ophthalmology, 96,** 252-256.

Cherry, P M H (1972). Rupture of the globe. Archives of **Ophthalmology, 88,** 498-507.

Cibis, G W, Weingeist, T A & Krachmer, J H (1978). Traumatic corneal endothelial rings. **Archives of Ophthalmology, 96,** 485-488.

Cogan, D G (1969). Pseudoretinitis pigmentosa. Report of two traumatic cases of recent origin. **Archives of Ophthalmology, 81,** 45-53.

Cole, H G & Smith, B (1963). Eye muscle imbalance complicating orbital floor fractures. **American Journal of Ophthalmology, 55,** 930-935.

Collins, C C, Carlson, M R, Scott, A B & Jampolsky, A (1981). Extraocular muscle forces in normal human subjects. **Investigative Ophthalmology and Visual Science, 20,** 652-664.

Collins, C C, Scott, A B & O'Meara, D (1969). Elements of the peripheral oculomotor apparatus. **American Journal of Optometry, 46,** 510-515.

Collins, R & Van Derwerff, T J (1980). Measurement techniques. In: Mathematical Models of the Dynamics of the Human Eye. Ch. 4, pp 62-63. Berlin: Springer-Verlag.

Converse, J M (1977). The blow-out fracture of the orbit: Some common sense. In: Controversy in Ophthalmology, ed. Bruckhurst, B T, Hutchinson & Lesell, S, Philadelphia: Saunders.

Converse, J M, Cole, G & Smith, B (1961). Late treatment of blow-out fracture of the floor of the orbit. **Plastic and Reconstructive Surgery, 28,** 183-191.

Converse, J M & Smith, B (1963). Naso-orbital fractures. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 67,** 622-634.

Converse, J M & Smith, B (1956). Enophthalmos and diplopia in fractures of the orbital floor. **British Journal of Plastic Surgery, 9,** 264-265.

Converse, J M, Smith, B, Obear, M F & Wood-Smith, D (1967). Orbital blow-out fractures: A ten-year survey. **Plastic and Reconstructive Surgery, 39,** 20-36.

Cooper, J & Duckman, R (1978). Convergence insufficiency: Incidence, diagnosis and treatment. **Journal of the American Optometric Association, 49,** 673-680.

Coppeto, J R (1987). Delayed post-traumatic eyelid emphysema. **Annals of Emergency Medicine, 16,** 1190-1191.

Courville, C B (1962). Forensic neuropathology. **Journal of Forensic Sciences, 7,** 1-28.

Courville, C B (1942). Coup-contrecoup mechanism of cranio-cerebral injuries. **Archives of Surgery, 45,** 19-43.

Cox, M S & Freeman, H M (1979). Ocular Trauma, pp 285-293. New York: Appleton-Century-Crofts.

Cox, M S, Schepens, C L & Freeman, H M (1966). Retinal detachment due to ocular contusion. **Archives of Ophthalmology, 76,** 678-685.

Cross, A,G, (1945). Ocular sequelae of head injuries: Neuromuscular aspects. **Transactions of the Ophthalmological Society of the United Kingdom, 65,** 20-34.

Crouch, E R Jr & Apple, D J (1974). Post-traumatic migration of retinal pigment epithelial melanin. **American Journal of Ophthalmology, 78,** 251-254.

Crumley, R L & Leibsohn, J (1976). Enophthalmos and diplopia in orbital floor fractures. **Transactions of the Pacific Coast Oto-ophthalmologic Society, 57,** 105-109.

Crumley, R L, Leibsohn, J, Krause, C J & Burton, T C (1977). Fractures of the orbital floor. **Laryngoscope, 87,** 934-947.

Culbertson, W W (1983). Diagnosis and management of ocular injuries. **Otolaryngologic Clinics of North America, 16,** 563-573.

Curtin, H D, Wolfe, P & Schramm, V (1982). Orbital roof blow-out fractures. **American Journal of Roentgenology, 139,** 969-972.

Dastoor, H D (1975). Severe oculo-orbital injuries. **International Surgery, 60,** 217-219.

Davidson, T M, Olesen, R M & Nahum, A M (1975). Medial orbital wall fracture with rectus entrapment. **Archives of Otolaryngology, 101,** 33-35.

Davis, C E (1956). Etiology and management of convergence insufficiency. **American Orthoptic Journal, 6,** 124-127.

Davis, P K B & Shaheen, (1985). Soft tissue injuries of the face and scalp: fractures of the larynx. In: Maxillofacial Injuries, ed. Rowe, N L & Williams, J Ll, Vol 1, Ch. 6. Edinburgh: Churchill Livingstone.

DeHaven, C R & Harle, T S (1966). Orbital blow-out fracture; a "black eye". **Texas Medicine, 62,** 71-74.

Delori, F, Pomerantzeff, O & Cox, M S (1969). Deformation of the globe under high speed impact: Its relation to contusion injury. **Investigative Ophthalmology, 8,** 290-301.

Delplace, M P & Poletti, J (1976). Therapeutic effect of the forced duction test. **Bulletin des Societes d Ophthalmologie de France, 76,** 967.

de Visscher, J G A M & van der Wal, K G H (1988). Medial orbital wall fracture with enophthalmos. **Journal of Cranio-Maxillo-Facial Surgery, 16,** 55-59.

Dingman, R O & Natvig, P (1964). Surgery of Facial Fractures. Philadelphia: W B. Saunders Company.

Doden, W & Bunge, H (1965). Fusionsstorungen nach schadeltraumen. **Klinische Monatsblatter fur Augenheilkunde, 146,** 845-853.

Dodick, J M, Berrett, A & Galin, M A (1969). Hypocycloidal tomography and orbital blow-out fracture. **American Journal of Ophthalmology, 68,** 483-486.

Dodick, J M, Galin, M A & Kwitko, M L (1970). Concomitant blow-out fracture of the orbit and rupture of the globe. **Archives of Ophthalmology, 84,** 707-709.

Duane, A (1922). Studies in monocular and binocular accommodation with their clinical applications. **American Journal of Ophthalmology, 5,** 865-877.

Duguid, I M (1985). Ophthalmic injuries. In: Maxillofacial Injuries, ed. Rowe, N L & Williams, J Ll, Vol 2, Ch. 16. Edinburgh: Churchill Livingstone.

Duke-Elder, S (1972). System of Ophthalmology, Vol 14, Part I, Ch. 6, p 710. London: Henry Kimpton.

Dulley B & Fells, P (1974). Orbital blow-out fractures: To operate or not to operate, that is the question. **British Orthoptic Journal, 31,** 47-54.

Dunnington, J H & Berke, R N (1943). Exophthalmos due to chronic orbital myositis. **Archives of Ophthalmology, 30,** 446-466.

Eason, G, Cole, C W & Gettinby, G (1980). Mathematics and Statistics for the Bio-sciences. pp 453-454. Chichester: Ellis Herwood.

Editorial (1975). Black eyes and blow-out fractures. **British Medical Journal, 1,** 50.

Edmund, J & Godtfredsen, E (1963). Unilateral optic atrophy following head injury. **Acta Ophthalmologica, 41,** 693-697.

Edwards, W C & Ridley, R W (1968). Blow-out fracture of the medial orbital wall. **American Journal of Ophthalmology, 65,** 248-249.

Ellis, E III, El-Attar, A & Moos, K F (1985). An analysis of 2067 cases of zygomatico-orbital fracture. **Journal of Oral and Maxillofacial Surgery, 43,** 417-428.

Emery, J M, von Noorden, G K & Schernitzauer, D A (1971). Orbital floor fractures. Long-term follow-up of cases with and without surgical repair. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 75,** 802-812.

Fischbein, F I & Leski, W S (1969). Blow-out fracture of the medial wall of the orbit. **Archives of Ophthalmology, 81,** 162-163.

Fonseca, R J & Walker, R V (1991). Oral and Maxillofacial Trauma. Philadelphia: W S. Saunders Company.

Fradkin, A H (1971). Orbital floor fractures and ocular complications. **American Journal of Ophthalmology, 72,** 699-700.

Franceschetti, A & Klinger, M (1943). Die posttraumatische encephalopathic (Ophthalmologischer Teil). **Schweizer Archiv fur Neurologie, 50,** 267-344.

Francois, J & Derouck, A (1955). Etude electro-oculographique des paralysies oculaires. **Acta Ophthalmologica, 53,** 523-550.

Friberg, T R (1979). Traumatic retinal pigment epithelial oedema. **American Journal of Ophthalmology, 88,** 18-21.

Fried, L A (1980). Anatomy of the Head, Neck, Face and Jaws. 2nd ed. Philadelphia: Lea and Febiger.

Friedburg, D (1980). "Blow-out fractures"-Diagnosis and indications for therapy from the ophthalmologic view. **Ber Zusammenkunft Dutsch Ophthalmol, 78,** 797-804.

Frost, D E & Kendell, B D (1991). Applied surgical anatomy of the head and neck. In: Oral and Maxillofacial Trauma, ed. Fonseca, R J & Walker, R V, Vol I, Ch. 12, pp 223-278. Philadelphia: W B. Saunders Company.

Fry, G A & Hill, W W (1963). The mechanics of elevating the eye. **American Journal of Optometry and Archives of American Academy of Optometry, 40,** 707-716.

Fry, G A & Hill, W W (1962). The centre of rotation of the eye. **American Journal of Optometry and Archives of American Academy of Optometry, 39,** 581-595.

Fujino, T (1974). Experimental "blow-out" fracture of the orbit. **Plastic and Reconstructive Surgery, 54,** 81-82.

Fujino, T & Makino, K (1980). Entrapment mechanism and ocular injury in orbital blow-out fracture. **Plastic and Reconstructive Surgery, 65,** 571-576.

Fung, Y C (1981). Smooth muscle. In: Biomechanical Properties of Living Tissues, ed. Fung, Y C. Ch. 11, pp 355-382. New York: Springer-Verlag.

Gerlock, A J, Sinn, D P & McBride, K L (1981). Clinical and Radiographic Interpretation of Facial Fractures. Boston: Little, Brown, and Company.

Gillison, T P A & Oei, T H (1975). Follow-up of orbital fractures treated surgically. In: Modern Problems of Ophthalmology: Orbital Disorders, ed. Bleeker, G M, pp 471-473. Basel: Karger.

Gjerris, F (1976). Traumatic lesions of the visual pathways. In: Handbook of Clinical Neurology, ed. Vinken, O J & Bruyn, G W. Vol 24, pp 27-57. Amsterdam: North Holland Publishing Company.

Glaser, J S & Goodwin, J A (1991). Neuro-ophthalmologic examination: The visual sensory system. In: Duane's Clinical Ophthalmology, ed. Tasman, W & Jaeger, E A, Vol 2, Ch. 2, p 9. Philadelphia: J B. Lippincott Company.

Goldberg, M F & Paton, D (1976). Management of Ocular Injuries, pp 214-235. Philadelphia: W S. Saunders.

Goldstein, J H (1964). The intraoperative forced duction test. **Archives of Ophthalmology, 72,** 647-649.

Gordon, S & Macrae, H (1950). Monocular blindness as a complication of the treatment of a malar fracture. **Plastic and Reconstructive Surgery, 6,** 228-232.

Gould, H R & Titus, C O (1966). Internal orbital fractures: The value of laminagraphy in diagnosis. **The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, 97,** 618-623.

Greenwald, H S, Keeney, A H & Shannon, G M (1974). A review of 128 patients with orbital fractures. **American Journal of Ophthalmology, 78,** 655-664.

Gruss, J S, Hurwitz, J J, Nik, N A & Kassel, E E (1985). The pattern and incidence of nasolacrimal injury in naso-orbital-ethmoidal fractures: the role of delayed assessment and dacryocystorhinostomy. **British Journal of Plastic Surgery, 38,** 116-121.

Gwyn, P P, Carraway, J H, Horton, C E, Adamson, J E & Miladick, R A (1971). Facial fractures: associated injuries and complications. **Plastic and Reconstructive Surgery, 47,** 225-230.

Hagler, W S & North, A W (1968). Retinal dialysis and retinal detachment. **Archives of Ophthalmology, 79,** 376-388.

Hakelius, L & Ponten, B (1973). Results of immediate and delayed surgical treatment of facial fractures with diplopia. **Journal of Maxillofacial Surgery, 1,** 150-154.

Harley, R D (1975). Surgical management of persistent diplopia in blow-out fractures of the orbit. **Annals of Ophthalmology, 7,** 1621-1626.

Hart, C T (1969). Disturbances of fusion following head injuries. **Proceedings of the Royal Society of Medicine, 62,** 704-706.

Hart, J C D & Frank, H J (1975). Retinal opacification after blunt non-perforating concussional injuries to the globe. A clinical and retinal fluorescein angiographic study. **Transactions of the Ophthalmological Societies of the United Kingdom, 95,** 94-100.

Haskell, R (1985). Applied surgical anatomy. In: Maxillofacial Injuries, ed. Rowe, N L & Williams, J Ll, Vol I, Ch. 1, pp 1-42. Edinburgh: Churchill Livingstone.

Heinze, J (1969). Cranial nerve avulsion and other neural injuries in road accidents. **Medical Journal of Australia, 2,** 1246-1258.

Heller, R M (1970). The clinical and radiological presentation of blow-out fracture of the orbit in children. **Experience and Reason-Briefly Recorded, 46,** 796-799.

Helveston, E M (1977). The relationship of extraocular muscle problems to orbital floor fractures: early and late management. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 83,** 660-662.

Helveston, E M, Bick, S E & Ellis, F D (1980). Differential intraocular pressure as an indirect measure of generated muscle force. **Ophthalmic Surgery, 11,** 386-391.

Henderson, D (1973). Classification of malar fractures. Unpublished.

Hitchin, A D & Shuker, S T (1973). Some observations on zygomatic fractures in the eastern region of Scotland. **British Journal of Oral Surgery, 2,** 114-117.

Hollinshead, W H (1968). Anatomy for Surgeons: The Head and Neck. Vol I. 2nd Edition. New York: Harper and Row.

Holt, G R & Holt, J E (1985). Nasoethmoid complex injuries. **Otolaryngologic Clinics of North America, 18,** 87-98.

Holt, G R & Holt, J E (1983). Incidence of eye injuries in facial fractures: an analysis of 727 cases. **Otolaryngology-Head and Neck Surgery, 91,** 276-279.

Holt, J E, Holt, G R & Blodgett, J M (1983). Ocular injuries sustained during blunt facial trauma. **Ophthalmology, 90,** 14-18.

Hughes, B (1964). The results of injury to special parts of the brain and skull. In: Acute Injuries of the Head: Their Diagnosis, Treatment, Complications and Sequels, ed. Rowbotham, G F, Ch. 12, p 419. Edinburgh & London: E & S Livingstone Ltd.

Ioannides, C, Treffers, W, Rutten, M & Noverraz, P (1988). Ocular injuries associated with fractures involving the orbit. **Journal of Cranio-maxillofacial Surgery, 16,** 157-159.

Jabaley, M E, Lerman, M & Sanders, H J (1975). Ocular injuries in orbital fractures. A review of 119 cases. **Plastic and Reconstructive Surgery, 56,** 410-418.

Jackson, I T, Schiele, U U & Adham, M (1987). The conjunctival approach to the orbital floor and maxilla - advantages and dis-advantages. **Annals of Plastic Surgery, 19,** 46-48.

Jaensch, P A (1929). Paresen der schragen heber. **V. Graefe's Arch. Ophthal, 121,** 113-118.

Jampolsky, A (1971). Ocular divergence mechanisms. **Transactions of the American Ophthalmic Society, 68,** 730-822.

Jennett, B & Teasdale, G (1981). Management of Head Injuries, Ch. 4, pp 89-90. Philadelphia: F A. Davis Company.

Jones, D E P & Evans, J N D (1967). Blow-out fractures of the orbit: An investigation into the anatomical basis. **Journal of Laryngology and Otology, 81,** 1109-1120.

Jordan, D R, White, G L Jr, Anderson, R L & Thiese, S M (1988). Orbital Emphysema: A potentially blinding complication following orbital fractures. **Annals of Emergency Medicine, 17,** 853-855.

Kaneko, H, Koga, A & Adachi, K (1971). Quantitative designation of the forced duction test. 1. Difference between normal and pathological extraocular muscles. **Nippon Ganka Gakkai Zasshi, 75,** 1515-1523.

Katz, B, Herschler, J & Brick, D C (1983). Orbital haemorrhage and prolonged blindness: a treatable posterior optic neuropathy. **British Journal of Ophthalmology, 67,** 549-553.

Kaufman, J H & Tolpin, D W (1974). Glaucoma after traumatic angle recession. **American Journal of Ophthalmology, 78,** 648-654.

Ketchum, L D, Ferris, B & Masters, F W (1976). Blindness without direct injury to the globe: A complication of facial fractures. **Plastic and Reconstructive Surgery, 58,** 187-191.

King, E F & Samuel, E (1944). Fractures of the orbit. **Transactions of the Ophthalmological Society of the United Kingdom, 64,** 134-153.

Knox, D L (1974). Suction cup forced duction. **American Journal of Ophthalmology, 77,** 762-763.

Koornneef, L (1992). Orbital Connective Tissue. In: Duane's Foundations of Clinical Ophthalmology, ed. Tasman, W & Jaeger, E A, Vol 1, Ch. 32. Philadelphia: J B Lippincott Company.

Koornneef, L (1978). The connective tissue apparatus of the human orbit. What about it? **Proceedings of the Third International Symposium on Orbital Disorders.** The Hague, W Junk BV.

Koornneef, L (1976). The development of the connective tissue in the human orbit. **Acta Morphologica Neerlando-Scandinavica, 14,** 263-290.

Koornneef, L & Los, J A (1975). A new anatomical approach to the human orbit. **Modern Problems of Ophthalmology, 14,** 49-56.

Koutroupas, S & Meyerhoff, W L (1982). Surgical treatment of orbital floor fractures. **Archives of Otolaryngology, 108,** 184-186.

Kreiger, A E (1971). Orbital blow-out fractures. **American Orthoptic Journal, 21,** 64-76.

Krohel, G B, Kristan, R W, Simon, J W & Barrows, N A (1986). Post-traumatic convergence insufficiency. **Annals of Ophthalmology, 18,** 101-104.

Lam, B L & Weingeist, T A (1990). Corticosteroid-responsive traumatic optic neuropathy. **American Journal of Ophthalmology, 109,** 99-101.

Langnickel, R & Munich, H (1978). A contribution to the operative treatment of diplopia in patients with long-standing orbital floor fractures. A combined operative technique by an ophthalmologist and an otolaryngologist is described. **HNO, 26,** 301-303.

Lang, W (1889). Traumatic enophthalmos with retention of perfect acuity of vision. **Transactions of the Ophthalmological Society of the United Kingdom, 9,** 41-46.

Last, R J (1984). Anatomy: Regional and Applied. Sec. 6, Edinburgh: Churchill Livingstone.

Lee, J (1983). Ocular motility consequences of trauma and their management. **British Orthoptic Journal, 40,** 26-33.

Le Fort, R (1900). Fractures de la machoire superieure. **Congres International de Medecine,** Paris, 275-278.

Leib, M L (1986). Computed tomography of the orbit. In: Advanced Imaging Techniques in Ophthalmology, ed. Halk, B, **International Ophthalmology Clinics, 26,** 103-121.

Leibsohn, J, Burton, T C & Scott, W E (1976). Orbital floor fractures: A retrospective study. **Annals of Ophthalmology, 8,** 1057-1062.

Lew, D & Sinn, D P (1991). Diagnosis and treatment of midface fractures. In: Oral and Maxillofacial Trauma, ed. Fonseca, R J & Walker, R V, Vol I, Ch. 19, pp 515-542. Philadelphia: W S. Saunders Company.

Lichtenstein, S B (1991). Management of penetrating and perforating injuries to the anterior segment. In: Duane's Clinical Ophthalmology, ed. Tasman, W & Jaeger, E A, Vol 5, Ch. 13. Philadelphia: J B Lippincott Company.

Linhart, W O (1943). Emphysema of the orbit: A study of 7 cases. **Journal of the American Medical Association, 23,** 89-90.

Los, J A (1970). A new method of three-dimensional reconstruction of approach to the human orbit following a clinical enquiry. **Acta Morphologica Neerlando-Scandinavica, 8,** 273-283.

Lubin, J R, Albert, D M & Weinstein (1980). Sixty-five years of sympathetic ophthalmia: a clinicopathologic review of 105 cases (1913-1978). **Ophthalmology, 87,** 109-121.

Luce, E A, Tubb, T D & Moore, A M (1979). Review of 1000 major facial fractures and associated injuries. **Plastic and Reconstructive Surgery, 63,** 26-29.

Lundin, K, Ridell, A, Sandberg, N & Ohman, A (1973). One thousand maxillofacial and related fractures at the ENT-Clinic in Gothenburg. A two-year prospective study. **Acta Otolaryngologica, 75,** 359-361.

Mackensen, G (1966). Okulographie. In: Augenmuskellaehmungen, ed. Hamburger, F A & Hollwich, F, Vol 46.

McClurg, F L & Swanson, P J (1976). An orbital roof fracture causing diplopia. **Archives of Otolaryngology, 102,** 497-498.

McCoy, F J (1958). Complications of middle third facial fractures. **Journal of the International College of Surgeons, 30,** 207-219.

McCoy, F J, Chandler, R A, Magan, C G Jr, Moore, J R & Siemsen, G (1962). An analysis of facial fractures and their complications. **Plastic and Reconstructive Surgery, 29,** 381-391.

McVay, C B (1984). Surgical Anatomy. 6th ed. Philadelphia: W B. Saunders Company.

Madroszkiewicz, M (1970). Oculomyodynamometry: The strength and work of extraocular muscles in squint. **Ophthalmologica, 161,** 491-498.

Magnus, W W (1971). A conjunctival approach to repair of fracture of medial wall of orbit: Report of a case. **Journal of Oral Surgery, 29,** 664-667.

Malmstadt, H V, Enke, C G & Crouch, S R (1981). Electronics and instrumentation for scientists. pp 406-407 & 411. California: The Benjamin/Cummings.

Mandelcorn, M S & Hill, J C (1973). Orbital blast injury. A case report. **Canadian Journal of Ophthalmology, 8,** 597-600.

Marsh, J L, Vannier, M W & Gado, M H (1988). Advanced radiographic evaluation of facial fractures. In: Facial Injuries, ed. Schultz, R C, 3rd ed, Ch. 5, pp 72-107. Chicago: Year Book Medical Publications, Inc.

Martin, R T (1988). Ophthalmologic considerations in head and facial trauma. **Indiana Medicine, 81,** 944-948.

Matsuzaki, H (1964). Studies on ocular changes occuring in head injuries as posttraumatic entities. Report III. Accommodative disturbance and its loading fluctuation. **Nippon Gankaa Gakkai Zasshi, 68,** 569-574.

Mauriello, J A, DeLuca, J, Krieger, A, Schulder, M & Frohman, L (1992). Management of traumatic optic neuropathy - a study of 23 patients. **British Journal of Ophthalmology, 76,** 349-352.

Mausner, J S & Bahn, A K (1974). Epidemiology: An Introductory Text, p. 242. Philadelphia: Saunders.

Metz, H S (1985). Quantitative evaluation of the strabismus patient. **International Ophthalmology Clinics, 25,** 13-36.

Metz, H S (1983). Restrictive factors in strabismus. **Survey of Ophthalmology, 28,** 71-83.

Metz, H S (1980). Saccades with limited downward gaze. **Archives of Ophthalmology, 98,** 2204-2205.

Metz, H S (1979). Double elevator palsy. **Archives of Ophthalmology, 97,** 901-903.

Metz, H S (1977). Saccadic velocity studies in patients with endocrine ocular disease. **American Journal of Ophthalmology, 84,** 695-699.

Metz, H S (1976). Forced duction, active force generation, and saccadic velocity tests. **International Ophthalmology Clinics, 16,** 47-73.

Metz, H S & Cohen, G H (1984). Quantitative forced traction measurements in strabismus. In Strabismus II,ed. Reineke, R D, pp 755-766. New York: Grune and Stratton.

Metz, H S, Scott, W E, Madston, E & Scott, A B (1974). Saccadic velocity and active forced studies in blow-out fractures of the orbits. **American Journal of Ophthalmology, 78,** 665-670.

Mewe, L (1980). Assessment of injuries to the lids and lacrimal apparatus: catamnestic investigations. **Klinische Monatsblatter fur Augenheilkunde, 176,** 254-257.

Milauskas, A T & Fueger, G F (1966). Serious ocular complications associated with blow-out fractures of the orbit. **American Journal of Ophthalmology, 62,** 670-672.

Milauskas, A T, Fueger, G F & Shulze, R R (1966). Clinical experiences with orbitography in the diagnosis of orbital floor fractures. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 70,** 25-39.

Miller, G R & Glaser, J S (1966). The retraction syndrome and trauma. **Archives of Ophthalmology, 76,** 662-663.

Miller, G R & Tenzel, R R (1967). Ocular complications of midfacial fractures. **Plastic and Reconstructive Surgery, 39,** 37-42.

Miller III, F S & Kalina, R E (1984). Suction cup for forced duction testing. **American Journal of Ophthalmology, 97,** 390-391.

Morgan, B G D, Madan, D K & Bergerot, J P C (1972). Fractures of the middle third of the face: a review of 300 cases. **British Journal of Plastic Surgery, 25,** 147-151.

Muller, E J & Schoeman, H S (1977). Zygomatico-maxillary fractures: a statistical analysis of 1,233 cases. **Journal of the Dental Association of South Africa, 32,** 585-588.

Mustarde, J C & Rycroft, B (1971). Repair and Reconstruction in the Orbital Region. Ch. 11, p 200; Ch. 16, p 292. Edinburgh and London: Churchill Livingstone.

Neely, J C (1945). The ocular sequelae of head injuries. **Transactions of the Ophthalmological Society of the United Kingdom, 65,** 47-56.

Newell, F W (1986). Ophthalmology: Principles and Concepts. Ch. 7, p 185 & Ch. 14, p 278. St Louis: The C V. Mosby Company.

Nicholson, D H & Guzak, S U Jr (1971). Visual loss complicating repair of orbital floor fractures. **Archives of Ophthalmology, 86,** 369-375.

O'Connor, R (1943). Contracture in ocular muscle paralysis. **American Journal of Ophthalmology, 26,** 69-71.

Ord, R A, Awty, M D & Pour, S (1986). Bilateral retrobulbar haemorrhage: A short case report. **British Journal of Oral and Maxillofacial Surgery, 24,** 1-6.

Ord, R A, Le May, M, Duncan, J G & Moos, K F (1981). Computerised tomography and B-scan ultrasonography in the diagnosis of fractures of the medial orbital wall. **Plastic and Reconstructive Surgery, 67,** 281-288.

Paterson, A (1944). Discussion on disorders of personality after head injury. **Proceedings of the Royal Society of Medicine, 37,** 556-561.

Paterson, A (1942). Emotional and cognitive changes in the post-traumatic confusional state. **The Lancet, 243,** 717-720.

Paton, D & Emery, J M (1973). Injuries of the eye, the lids, and the orbit. In: Management of Trauma, ed. Ballinger, W F, 2nd ed, pp 219-254. Philadelphia: W. B. Saunders.

Patterson, Jr R W & Depue, Jr R V (1962). Blow-out fracture of the orbit. **American Journal of Ophthalmology, 53,** 841-845.

Pearl, R M & Vistnes, L M (1978). Orbital blow-out fractures: An approach to management. **Annals of Plastic Surgery, 1,** 267-270.

Petro, J, Tooze, F M, Bales, C R & Baker, G (1979). Ocular injuries associated with periorbital fractures. **Journal of Trauma, 19,** 730-733.

Pfeiffer, R L (1943). Traumatic enophthalmos. **Archives of Ophthalmology, 30,** 718-726.

Phalen, J J, Baumel, J J & Kaplan, P A (1990). Orbital floor fractures: a reassessment of pathogenesis. **Nebraska Medical Journal, 75,** 100-103.

Piddle, W J (1968). Blow-out fractures of the orbit. **Manitoba Medical Review, 48,** 322-323.

Purtscher, O (1910). Nochunbekannte Befunde nach Schaedel-trauma. **Ber ud versamml d deutsch Ophth Gesellsch, 36,** 294-301.

Putterman, A H (1975). Temporary blindness after blepharoplasty. **American Journal of Ophthalmology, 80,** 1081-1083.

Putterman, A M, Stevens, T & Urist, M J (1974). Non surgical management of blow-out fractures of the orbital floor. **American Journal of Ophthalmology, 77,** 232-239.

Raflo, G T (1984). Blow-in and blow-out fractures of the orbits: clinical correlations and proposed mechanisms. **Ophthalmic Surgery, 15,** 114-119.

Rao, N A, Robin, J, Hartmann, D, Sweeney, J A & Marak, G E Jr (1983). The role of the penetrating wound in the development of sympathetic ophthalmia: experimental observations. **Archives of Ophthalmology, 101,** 102-104.

Rao, N A & Wong, V G (1981). Aetiology of sympathetic ophthalmitis. **Transactions of the Ophthalmological Societies of the United Kingdom, 101,** 357-360.

Raskind, R (1976). Problems at the reading distance. **American Journal of Orthoptics, 26,** 53-59.

Rauch, S D (1985). Medial orbital blow-out fracture with entrapment. **Archives of Otolaryngology, 111,** 53-55.

Read, J E & Crouch, E R Jr (1991). Trauma: Ruptures and bleeding. In: Duane's Clinical Ophthalmology, ed. Tasman, W. & Jaeger, E A, Vol 4, Ch. 61. Philadelphia: J B Lippincott Company.

Read, J & Goldberg, M F (1974). Comparison of medical treatment for traumatic hyphaema. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 78,** 799-815.

Records, R E (1979). Primary care of ocular emergencies. 1. Traumatic injuries. **Postgraduate Medicine, 65,** 143-147.

Reeh, M J & Tsujimura, J K (1966). Early detection and treatment of blow-out fracture of the orbit. **American Journal of Ophthalmology, 62,** 79-82.

Risco, J M, Stratas, B A, & Knott, R H (1984). Prolapse of the globe into the ethmoid sinus. **American Journal of Ophthalmology, 97,** 659-660.

Robertson, D M, Douglas Hines, J & Rucker, C W (1970). Acquired sixth nerve paresis in children. **Archives of Ophthalmology, 83,** 574-579.

Robinson, D A (1975). A quantitative analysis of extraocular muscle cooperation and squint. **Investigative Ophthalmology, 14,** 801-825.

Robinson, D A, O'Meara, D M, Scott, A B & Collin, C C (1969). Mechanical components of human eye movements. **Journal of Applied Physiology, 26,** 548-553.

Rosenbaum, A L & Myer, J H (1980). New instrument for the quantitative determination of passive forced traction. **Ophthalmology, 87,** 158-163.

Ross, W H (1981). Traumatic retinal dialysis. **Archives of Ophthalmology, 99,** 1371-1374.

Rowbotham, G F (1964). Acute Injuries of the Head: Their Diagnosis, Treatment, Complications and Sequels. pp 408-433. Edinburgh: E & S Livingstone.

Rowe, N L & Williams, J Ll (1985). Maxillofacial Injuries. Edinburgh: Churchill Livingstone.

Rucker, C W (1966). The causes of paralysis of the third, fourth and sixth cranial nerves. **American Journal of Ophthalmology, 61,** 1293-1298.

Rucker, C W (1958). Paralysis of the third, fourth and sixth cranial nerves. **American Journal of Ophthalmology, 46,** 787-794.

Rumlet, M B & Ernest, J T (1972). Isolated blow-out fracture of the medial orbital wall with medial rectus muscle entrapment. **American Journal of Ophthalmology, 73,** 451-453.

Russell, W R (1960). Injury to cranial nerves and optic chiasm. In: Injuries of the Brain and Spinal Cord and their Coverings, ed. Brock, S, Ch. 5, p 121. London: Cassell.

Ryan, B F, Joiner, B L & Ryan, T A (1985). Minitab Handbook, 2nd Ed. Boston: Duxbury Press.

Schillinger, R J (1966). The prevention of over-correction and under-correction in horizontal strabismus surgery. **Journal of Pediatric Ophthalmology, 3,** 38-41.

Schultz, R C (1988). Facial Injuries. Ch. 1 & 4. Chicago: Year Book Medical Publishers, Inc.

Schultz, R C (1970). Supraorbital and glabellar fractures. **Plastic and Reconstructive Surgery, 45,** 227-233.

Schultz, R C (1967). Facial injuries from automobile accidents: A study of 400 consecutive cases. **Plastic and Reconstructive Surgery, 40,** 415-425.

Scobee, R G (1948). Anatomic factors in the etiology of Heterotropia. **American Journal of Ophthalmology, 31,** 781-795.

Scott, A B (1986). Ocular motility. In: Biomedical Foundations of Ophthalmology, ed. Duane, T D & Jaeger, E A, Vol 2, Ch. 23, pp 1-60. Philadelphia: Harper & Row.

Scott, A B (1975). Strabismus muscle forces and innervation. In: Basic Mechanics of Ocular Motility and their Clinical Implications, ed. Lennerstrand, G & Bach-y-Rita, P, pp 181-191. Oxford: Pergamon.

Scott, A B, Collin, C C & O'Meara, D M (1972). A forceps to measure strabismus forces. **Archives of Ophthalmology, 88,** 330-333.

Sellors, P J & Mooney, D (1973). Fundus changes after traumatic hyphaema. **British Journal of Ophthalmology, 57,** 600-607.

Shammas, H & Matta, C S (1974). Optic atrophy following eyelid laceration. Report of a case. **Lebanese Medical Journal, 27,** 191-194.

Silva, M (1973). Fracture of the orbital floor. **Medical Journal of Australia, 1,** 865.

Sinn, D P & Karas, N D (1991). Radiographic evaluation of facial injuries. In: Oral and Maxillofacial Trauma, ed. Fonseca, R J & Walker, R V, Vol I, Ch. 14, pp 301-322. Philadelphia: W S. Saunders Company.

Slingsby, J G & Forstot, S L (1981). Effect of blunt trauma on the corneal endothelium. **Archives of Ophthalmology, 99,** 1041-1043.

Slomovits, T L & Glaser, J S (1991). The pupils and accommodation. In: Duane's Clinical Ophthalmology, ed. Tasman, W & Jaeger, E A, Vol 2, Ch. 15. Philadelphia: J B. Lippincott Company.

Smith, B C, Barr, D R & Langham, E J (1971). Complications of orbital fractures. **New York State Journal of Medicine, Oct,** 2407-2411.

Smith, B & Converse, J M (1957). Early treatment of orbital floor fractures. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 61,** 602-608.

Smith, B & Regan, W F Jr (1957). Blow-out fracture of the orbit. Mechanism and correction of internal orbital fracture. **American Journal of Ophthalmology, 44,** 733-739.

Smith, R R & Blount, R L (1971). Blow-out fracture of the orbital roof with pulsating exophthalmos, blepharoptosis, and superior gaze paresis. **American Journal of Ophthalmology, 71,** 1052-1054.

Spoor, T C, Hartel, W C, Lensink, D B & Wilkinson, M J (1990). Treatment of traumatic optic neuropathy with corticosteroids. **American Journal of Ophthalmology, 110,** 665-669.

Stanworth, A (1974). Defects of ocular movement and fusion after head injury. **British Journal of Ophthalmology, 58,** 266-271.

Steidler, N E, Cook, R M & Reade, P C (1980). Residual complications in patients with major middle third facial fractures. **International Journal of Oral Surgery, 9,** 259-266.

Stephens, K F & Reinecke, R D (1967). Quantitative forced duction. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 71,** 324-329.

Strachan, I M, Brown, B H, Johnson, S G & Robinson, P (1975). An apparatus for measuring forces in strabismus. Transactions of the Third International Orthoptic Congress, Boston, pp 123-128. Chicago: Symposia Specialists Medical Books.

Stranc, M F (1970[a]). Primary treatment of naso-ethmoid injuries with increased intercanthal distance. **British Journal of Plastic Surgery, 23,** 8-25.

Stranc, M F (1970[b]). The pattern of lacrimal injuries in naso-ethmoid fractures. **British Journal of Plastic Surgery, 23,** 339-346.

Tajima, S, Sujimoto, C, Tanino, R, Oshiro, T & Harashina, T (1974). Surgical treatment of malunited fracture of zygoma with diplopia and with comments on blow-out fracture. **Journal of Maxillofacial Surgery, 2,** 201-210.

Tasman, W I (1972). Peripheral retinal changes following blunt trauma. **Transactions of the American Ophthalmological Society, 70,** 190-198.

Teasdale, G & Jennett, B (1974). Assessment of coma and impaired consciousness. **The Lancet, 2,** 81-84.

Thaller-Antlanger, H (1980). Is there a typical motility disturbance in orbital wall fractures. **Klinische Monatsblatter fur Augenheilkunde, 177,** 845-854.

Thering, H R & Bogart, J N (1979). Blow-out fracture of the medial orbital wall, with entrapment of the medial rectus muscle. **Plastic and Reconstructive Surgery, 63,** 848-852.

Tokel, S L & Potter, G D (1969). Radiographic diagnosis of fracture of the medial wall of the orbit. **American Journal of Ophthalmology, 67,** 772-773.

Tonjum, A (1966). Gonioscopy in traumatic hyphaema. **Acta Ophthalmologica, 44,** 650-664.

Tortora, G J & Anagnostakos, N P (1984). The special senses. In: Principles of Anatomy and Physiology. 4th Ed, Ch. 17, pp 374-387. London: Harper & Row Publications.

Troutman, R (1974). Microsurgery of the Anterior Segment of the Eye. Vol 1, Introduction and basic techniques, pp 220, 274-279. St Louis: C V. Mosby.

Turvey, T A (1977). Midfacial fractures: a retrospective analysis of 593 cases. **Journal of Oral Surgery, 35,** 887-891.

Underwood, R & Firmin, D (1987). Introduction to Magnetic Resonance of the Cardiovascular System, p 5. London: Current Medical Literature Ltd.

Verdaguer, T J, Rojas, B & Lechuga, M (1975). Genetical studies in non-traumatic retinal dialyses. **Modern Problems of Ophthalmology, 15,** 34-39.

von Noorden, G K (1985). Binocular Vision and Ocular Motility. 3rd Ed, p 380. St. Louis, Toronto, Princeton: CV Mosby Company.

von Noorden, G K (1979). Management of trauma of the muscles and optic pathways. In Ocular Trauma, pp 323-326 New York: Appleton-Century-Crofts.

von Noorden, G K, Brown, D J & Parks, M (1973). Associated convergence and accommodation insufficiency. **Documenta Ophthalmologica, 34,** 393-403.

Waddell, E (1982). Brown's syndrome revisited. **British Orthoptic Journal, 39,** 17-21.

Walsh, F B & Hoyt, W F (1969). Clinical Neuro-ophthalmology. 3rd ed, Vol 3. Baltimore: The Williams and Wilkins Company.

Weidenthal, D T & Schepens, C L (1966). Peripheral fundus changes associated with ocular contusion. **American Journal of Ophthalmology, 62,** 465-477.

Wesley, R E & McCord, C D (1982). Tension pneumocephalus from orbital roof fracture. **Annals of Ophthalmology, 14,** 184-190.

Winslow, R L & Tasman, W (1978). Juvenile rhegmatogenous retinal detachment. **Transactions of the American Academy of Ophthalmology and Otolaryngology, 85,** 607-618.

Wolff, E (1968). Anatomy of the Eye and Orbit. 6th ed, Ch. 1, pp 1-29. London: H K. Lewis & Co. Ltd.

Wolff, J (1900). The occurence of traction movements of the eyeball together with congenital defects in the external ocular muscles. **Archives of Ophthalmology, 29,** 297-309.

Wolin, M J & Lavin, P J M (1990). Spontaneous visual recovery from traumatic optic neuropathy after blunt head injury. **American Journal of Ophthalmology, 109,** 430-435.

Wolter, J R (1979). Subperiosteal haematoma of the orbit in young males: a serious complication of trauma or surgery in the eye region. **Journal of Paediatric Ophthalmology and Strabismus, 16,** 291-296.

Wood, C M & Richardsen, J (1990[a]). Chorioretinal neovascular membranes complicating contusional eye injuries with indirect choroidal ruptures. **British Journal of Ophthalmology, 74,** 93-96.

Wood, C M & Richardsen, J (1990[b]). Indirect choroidal ruptures: aetiological factors, patterns of ocular damage, and final visual outcome. **British Journal of Ophthalmology, 74,** 208-211.

Wood, G D (1986). Blindness following fracture of the zygomatic bone. **British Journal of Oral and Maxillofacial Surgery, 24,** 12-16.

Woodruff, G, O'Reilly, C & Kraft, S P (1987). Functional scoring of the field of binocular single vision in patients with diplopia. **Ophthalmology, 94,** 1554-1561.

Yamamoto, Y, Sakurai, M & Asari, S (1983). Towne (half-axial) and semisagittal computed tomography in the evaluation of blow-out fractures of the orbits. **Journal of Computer Assisted Tomography, 7,** 306-309.

Yanoff, M & Fine, B (1975). Ocular Pathology: A Textbook and Atlas. pp 145-148. Hagerstown: Harper & Row.

Yee, K F, Cravens, J & Kotler, R (1975). Traumatic ophthalmoplegia: a complication of fractures of the zygomaticomaxillary complex. Review of aetiology with report of an unusual case. **Laryngoscope, 85,** 570-575.

Yee, R D (1983). Eye movement recording as a clinical tool. **Ophthalmology, 90,** 211-222.

Zajac, F E (1989). Muscle and tendon. **CRC Review of Biomedical Engineering, 17,** 359-411.

Zappia, R J, Winkelman, J Z & Gay, A J (1971). Intraocular pressure changes in normal subjects and the adhesive muscle syndrome. **American Journal of Ophthalmology, 71,** 880-883.

APPENDICES

APPENDIX 1

THE CODEBOOK

THE CODEBOOK

Name of Patient

Reference Number

01 Sex of Patient

0	Male	1	Female

02 Age of Patient

0	0 - 9		
1	10 - 19	5	50 - 59
2	20 - 29	6	60 - 69
3	30 - 39	7	70 - 79
4	40 - 49	8	80 & over

03 Geographical Distribution

0	Outside Glasgow		
1	G1--------------------	85	G85

04 Month of Incident

1	January	7	July
2	February	8	August
3	March	9	September
4	April	10	October
5	May	11	November
6	June	12	December

05 Cause of Injury

0	Car (no seat belt)	7	Fall
1	Car (seat belt on)	8	Fall + Alcohol
2	Car (back seat)	9	Sport
3	Motorcycle	10	Industrial
4	Other RTA	11	Black-out
5	Alleged assault	12	Epilepsy
6	Assault + Alcohol	13	Other

06 Amnesia

0	Absent	2	Post-traumatic
1	Retrograde	3	Both

07 Severity of Amnesia

0	No amnesia	4	Severe (1-7 days)
1	Very mild (<5 minutes)	5	Very severe (1-4 weeks)
2	Mild (5-60 minutes)	6	Extremely severe (>4 weeks)
3	Moderate (1-24 hours)		

08 Pre-operative Ophthalmic Assessment

0	No	1	Yes

09 Previous Eye History

0	Nil		
1	Glasses	4	Surgery
2	Amblyopia	5	Old Eye Injury
3	Squint	6	Other

10 Affected Eye/Side

0	Neither	2	Left
1	Right	3	Both

11 Malar Fracture Sites

0	No malar fracture		
1	Type I	5	Type V
2	Type II	6	Type VI
3	Type III	7	Type VII
4	Type IV	8	Undefined/other

12 Maxillary Fracture Sites

0	No maxillary fracture		
1	Le Fort I	3	Le Fort III
2	Le Fort II	4	Combination

13 Other Fracture Sites

0	None	3	Skull (occipital)
1	N.O.E.	4	Skull (temporal)
2	Nasal	5	Skull (frontal/ supra-orbital)

14 Treatment

0	Nil		
1	Elevation	9	En.+ Pins + Pack
2	Elevation + Pins	10	En.+ K wire + Pack
3	Elevation + K wire	11	En.+ Wires + Pins + Pack
4	Elevation + Wires	12	Orb. floor exploration
5	En.+ Pins + Wires	13	O.F.E.+ Silastic
6	En.+ Wire + K wire	14	O.F.E.+ Pack
7	En.+ Pack	15	O.F.E.+ Other
8	En.+ Pack + Wires	16	Combination & Others

15 Motility Abnormality

0	Absent	1	Present

16 Manifest Deviation in the Primary Position

0	Absent	1	Present

17 Symptoms of Diplopia

0	None		
1	Data not available	3	Moderate
2	Mild	4	Severe

18 Diplopia on Examination

0	Absent	2	In extreme gaze
1	Close to midline	3	After Surgery

19 Pain on Eye Movement

0 Absent
1 On elev/depression
2 On add/abduction
3 Combination/all move.

20 Convergence Insufficiency

0 Absent
1 Unknown degree
2 Mild
3 Moderate
4 Severe

21 Extra-ocular Movement Restriction

0 None
1 On elevation
2 On depression
3 On elev/depression
5 On adduction
6 On add/abduction
7 Combination
8 Fixed eye

22 Entrapment of Soft Tissue

0 None
1 Floor entrapment
2 Medial wall entrapment
3 Roof entrapment
4 Combination

23 Nerve Palsies

0 None
1 III, superior branch
2 III, inferior branch
3 IV nerve
4 VI nerve
5 VII nerve
6 Combination

24 Injury to Trochlea

0 No
1 Yes

25 Muscle Exercises

0 No
1 Yes

26 Squint Surgery

0 No
1 Yes

27 Recovery of Diplopia

0 No diplopia
1 < 1 Day
2 1 Day - 1 Week
3 1 Week - 1 Month
4 1 - 6 Months
5 7 - 12 Months
6 > 12 Months

28 Head Posture

0 No
1 Yes

29 Eye Abnormality

0 No
1 Yes

30 Post-traumatic Neuralgia/anaesthesia

0 None
1 Supra-trochlear
2 Supra-orbital
3 Zygomatico-temporal
4 Zygomatico-facial
5 Infra-orbital

31 Eye Displacement

0 None
1 Up displacement
2 Down displacement
3 Medial displacement
4 Lateral displacement

32 Duration of Displacement

0 Temporary
1 Permanent

33 Proptosis

0 Absent
1 Present

34 Enophthalmos

0 Absent
1 Present

35 Retrobulbar Haemorrhage

0 Absent
1 Present

36 Orbital Emphysema

0 Absent
1 Present

37 Eyelid Injury

0 Absent
1 Swelling/bruising
2 Laceration

38 Conjunctival Injury

0 Absent
1 Chemosis
2 Subconj. haemorrhage
3 Laceration

39 Corneal Damage

0 None
1 Abrasion
2 Puncture wound
3 Laceration

40 Scleral Injury

0 None
1 Partial thickness
2 Full thickness

41 Traumatic Pupillary Changes

0 None
1 Traumatic mydriasis
2 Sphincter rupture
3 Traumatic miosis
4 Hippus

42 Iris Damage

0 None
1 Iridodialysis
2 Hyphaema

43 Angle Recession

0 None
1 < 90°
2 90° - 180°
3 180° - 270°
4 270° - 360°

44 Lens Damage

0	None		
1	Vossius' ring	3	Traumatic cataract
2	Dislocation/subluxation	4	Ruptured capsule

45 Photo-stress Test

0	Negative		
1	Positive	2	Not done

46 Changes in Accommodation

0	Normal for age	2	Moderate failure
1	Mild failure	3	Severe failure

47 Changes in Visual Acuity

0	No change		
1	Mild reduction	3	Severe reduction
2	Moderate reduction	4	Loss of Vision

48 Visual Field Loss

0	No loss		
1	Central loss	3	> One direction
2	Peripheral/1 direction	4	All directions

49 Commotio Retinae

0	Absent	2	Moderate
1	Mild/resolving	3	Severe

50 Choroidal Damage

0	None	2	> One tear
1	One tear	3	Macula involved

51 Retinal/Vitreous Damage

0	None	4	Flat retinal hole/tear
1	Vitreous floater	5	Detachment, macula on
2	Vitreous haemorrhage	6	Detachment, macula off
3	Pigmentary retinopathy	7	Total detachment

52 Optic Nerve Damage

0	None	2	Partial Injury
1	Contusion	3	Total damage/avulsion

53 Naso-lacrimal Damage

0	No	1	Yes

54 Malingering

0	No		
1	Yes	2	Not known/suspected

APPENDIX 2

DATA COLLECTION SHEET FOR THE OCULAR AND MOTILITY DISORDERS

(Prospective Study)

11	Malar Fracture Site											
10	Affected Eye/Side											
09	Previous Eye History											
08	Pre-op Ophth Assessment											
07	Severity of Amnesia											
06	Post-traumatic Amnesia											
05	Cause of Injury											
04	Month of Incident											
03	Geographical Distribution											
02	Age of Patient											
01	Sex of Patient											
	Hosp. No.											
	Name of Patient											
	Serial Number	01	02	03	04	05	06	07	08	09	10	11

44	Lens Damage											
43	Angle Recession											
42	Iris Damage											
41	Traumatic Pupillary Changes											
40	Scleral Injury											
39	Corneal Damage											
38	Conjunctival Injury											
37	Eyelid Injury											
36	Orbital Emphysema											
35	Retrobulbar Haemorrhage											
34	Enophthalmos											
33	Ptosis											
32	Duration of Displacement											
31	Eye Displacement											
30	P-T Neuralgia/anaesthesia											
29	Eye Abnormality											
28	Abnormal Head Posture											
27	Recovery of Diplopia											
26	Squint Surgery											
25	Muscle Exercises											
24	Injury to Trochlea											
23	Nerve Palsies											
22	Entrapment of Soft Tissue											
21	Extraocular Movements											
20	Convergence											
19	Pain on Eye Movement											
18	Diplopia on Examination											
17	Symptoms of diplopia											
16	Manifest Dev. in 1° Position											
15	Motility Abnormality											
14	Treatment											
13	Other Fracture Sites											
12	Maxillary Fractures											

55	Patient Discharged											
54	Malingering											
53	Naso-lacrimal Damage											
52	Optic Nerve Injury											
51	Retinal/vitreous Damage											
50	Choroidal Damage											
49	Commotio Retinae											
48	Visual Field Loss											
47	Changes in Visual Acuity											
46	Changes in Accommodation											
45	Photo-Stress Test											
44	Lens Damage											

APPENDIX 3

COMPUTER LISTING OF VARIABLES

FOR THE POPULATION OF STUDY

DATA LIST FILE

```
DATA LIST FILE = 'C:\QURAINY\ALQDATA.DAT' /
SERIALNO 1-3
ID 5-10
SEX 12
AGE 13
GEODIST 14-15
MONTH 16-17
CAUSEINJ 18-19
AMNESIA 20
SEVAM 21
PREOPASS 22
EYEHIST 23
AFFEYE 24
MALSITE 25
MAXSITE 26
OTHSITE 27
TREAT 28-29
MOTAB 30
EYEDEV 31
SYMDIP 32
EXADIP 33
EYEPAIN 34
CONV 35
EOM 36
ENTRAP 37
NERVPAL 38
TROCHINJ 39
EXERCISE 40
SQUINT 41
DIPREC 42
HEADPOST 43
EYEAB 44
NEURAN 45
EYEDISP 46
TIMEDISP 47
PROPT 48
ENOTHALM 49
BULBHAEM 50
ORBITAIR 51
```

```
CONINJ 53
CORINJ 54
SCLERINJ 55
PUPILINJ 56
IRISINJ 57
ANGREC 58
LENSINJ 59
PHOTEST 60
ACC 61
VA 62
VF 63
COMRET 64
CHORINJ 65
RETINJ 66
OPTINJ 67
NASLAINJ 68
MALINGER 69
PATDIS 70.

VARIABLE LABELS
/SERIALNO 'SERIAL NUMBER'
/ID 'HOSPITAL NUMBER'
/SEX 'SEX'
/AGE 'AGE'
/GEODIST 'GEOGRAPHICAL DISTRIBUTION'
/MONTH 'MONTH OF INCIDENT'
/CAUSEINJ 'CAUSE OF INJURY'
/AMNESIA 'AMNESIA'
/SEVAM 'SEVERITY OF P-T AMNESIA'
/PREOPASS 'PRE-OPERATIVE OPHTHALMIC ASSESSMENT'
/EYEHIST 'PREVIOUS EYE HISTORY'
/AFFEYE 'AFFECTED EYE'
/MALSITE 'MALAR FRACTURE SITES'
/MAXSITE 'MAXILLARY FRACTURE SITES'
/OTHSITE 'OTHER FRACTURE SITES'
/TREAT 'TREATMENT'
/MOTAB 'MOTILITY ABNORMALITY'
/EYEDEV 'MANIFEST DEVIATION IN PRIMARY POSITION'
/SYMDIP 'SYMPTOMS OF DIPLOPIA'
/EXADIP 'DIPLOPIA ON EXAMINATION'
/EYEPAIN 'PAIN ON EYE MOVEMENT'
/CONV 'CONVERGENCE'
/EOM 'EXTRA-OCULAR MOVEMENTS'
/ENTRAP 'ENTRAPMENT OF SOFT TISSUE'
/NERVPAL 'NERVE PALSIES'
/TROCHINJ 'INJURY TO TROCHLEA'
/EXERCISE 'MUSCLE EXERCISE'
/SQUINT 'SQUINT SURGERY'
/DIPREC 'RECOVERY OF DIPLOPIA'
/HEADPOST 'HEAD POSTURE'
/EYEAB 'EYE ABNORMALITY'
/NEURAN 'POST-TRAUMATIC NEURALGIA/ANAESTHESIA'
/EYEDISP 'EYE DISPLACEMENT'
/TIMEDISP 'DURATION OF DISPLACEMENT'
/PROPT 'PROPTOSIS'
/ENOTHALM 'ENOPHTHALMOS'
/BULBHAEM 'RETROBULBER HAEMORRHAGE'
/ORBITAIR 'ORBITAL EMPHYSEMA'
/LIDINJ 'EYELID INJURY'
/CONINJ 'CONJUNCTIVAL INJURY'
/CORINJ 'CORNEAL DAMAGE'
/SCLERINJ 'SCLERAL INJURY'
/PUPILINJ 'TRAUMATIC PUPILLARY CHANGES'
/IRISINJ 'IRIS DAMAGE'
/ANGREC 'ANGLE RECESSION'
/LENSINJ 'LENS DAMAGE'
/PHOTEST 'PHOTO-STRESS TEST'
/ACC 'CHANGES IN ACCOMMODATION'
/VA 'CHANGES IN VISUAL ACUITY'
```

```
/COMRET 'COMMOTIO RETINAE'
/CHORINJ 'CHOROIDAL DAMAGE'
/RETINJ 'RETINAL/VITREOUS DAMAGE'
/OPTINJ 'OPTIC NERVE INJURY'
/NASLAINJ 'NASO-LACRIMAL DAMAGE'
/MALINGER 'MALINGERING'
/PATDIS 'PATIENT DISCHARGED'.


VALUE LABELS
/SEX 0 'Male' 1 'Female'
/AGE 0 '0-9' 1 '10-19' 2 '20-29' 3 '30-39' 4 '40-49' 5 '50-59' 6 '
     7 '70-79' 8 '80 & OVER'
/MONTH 1 'JANUARY' 2 'FEBRUARY' 3 'MARCH' 4'APRIL' 5 'MAY' 6 'JUNE
       8 'AUGUST' 9 'SEPTEMBER' 10 'OCTOBER' 11 'NOVEMBER' 12 'DEC
/CAUSEINJ 0 'CAR (NO SEAT BELT)' 1 'CAR (SEAT BELT ON)' 2 'CAR (BA
          3 'MOTORCYCLE' 4 'OTHER RTA' 5 'ALLEGED ASSAULT' 6 'ASSA
          7 'FALL' 8 'FALL+ALCOHOL' 9 'SPORT' 10 'INDUSTRIAL' 11 '
          12 'EPILEPSY' 13 'OTHER'
/AMNESIA 0 'ABSENT' 1 'RETROGRADE' 2 'POST-TRAUMATIC' 3 'BOTH'
/SEVAM 0 'NO P-T AMNESIA' 1 'VERY MILD' 2 'MILD' 3 'MODERATE' 4 'S
       5 'VERY SEVERE' 6 'EXTREMELY SEVERE' 7 'DATA N/A'
/PREOPASS 0 'NO' 1 'YES' 2 'NO ASSESSMENT AT ALL'
/EYEHIST 0 'NO' 1 'GLASSES' 2 'AMBLYOPIA' 3 'SQUINT' 4 'SURGERY'
         5 'OLD EYE INJURY' 6 'COMBINATION & OTHER'
/AFFEYE 0'NEITHER' 1 'RIGHT' 2 'LEFT' 3 'BOTH'
/MALSITE 0 'NO FRACTURE' 1 'TYPE I' 2 'TYPE II' 3 'TYPE III' 4 'TY
         5 'TYPE V' 6 'TYPE VI' 7 'TYPE VII' 8 'UNDEFINED/OTHER'
/MAXSITE 0 'NO FRACTURE' 1 'LE FORT I' 2 'LE FORT II' 3 'LE FORT I
         4 'COMBINATION/OTHER'
/OTHSITE 0 'NONE' 1 'N.O.E.' 2 'NASAL' 3 'SKULL (OCCIPITAL)'
         4 'SKULL (TEMPORAL)' 5 'SKULL (FRONTAL)' 6 'COMBINATION/O
/TREAT 0 'NIL' 1 'ELEVATION ONLY' 2 'E+PINS' 3 'E+K WIRE' 4 'E+WIR
       5 'E+WIRES+PINS' 6 'E+WIRES+K WIRE' 7 'E+PACK' 8 'E+PACK+WI
       9 'E+PACK+PINS' 10 'E+PACK+K WIRE' 11 'E+WIRES+PINS+PACK'
       12 'FLOOR EXPLORATION' 13 'O.F.E.+SILASTIC' 14 'O.F.E.+PACK
       15 'O.F.E.+OTHER' 16 'COMBINATION'
/MOTAB 0 'NO' 1 'YES'
/EYEDEV 0 'NO' 1 'YES'
/SYMDIP 0 'NONE' 1 'DATA N/A' 2 'MILD' 3 'MODERATE' 4 'SEVERE'
/EXADIP 0 'NONE' 1 'CLOSE TO MIDLINE' 2 'IN EXTREME GAZE' 3 'AFTER
/EYEPAIN 0 'NONE' 1 'ON ELEV/DEPR' 2 'ON ADD/ABD' 3 'COMBINATION'
/CONV 0 'NORMAL' 1 'U/K INSUFFICIENCY' 2 'MILD INSUFFICIENCY'
      3 'MOD INSUFFICIENCY' 4 'SEVERE INSUFFICIENCY'
/EOM 0 'FREE' 1 'RESTRICTED UP' 2 'RESTRICTED DOWN' 3 'R. ON ADDUC
     4 'R. ON ABDUCTION' 5 'R. ON ADD & ABD' 6 'COMBINATION' 8 'FI
/ENTRAP 0 'NONE' 1 'FLOOR ENTRAPMENT' 2 'MEDIAL WALL ENTRAP'
        3 'ROOF ENTRAPMENT' 4 'COMBINATION'
/NERVPAL 0 'NONE' 1 'IIIrd, SUPERIOR' 2 'IIIrd, INFERIOR' 3 'IVth 
         4 'VIth NERVE' 5 'VIIth NERVE' 6 'COMBINATION'
/TROCHINJ 0 'NO' 1 'YES'
/EXERCISE 0 'NO' 1 'YES'
/SQUINT 0 'NO' 1 'YES'
/DIPREC 0 'NO DIPLOPIA' 1 'SECONDS/MINUTES' 2 'HOURS/DAYS' 3 'DAYS/
        4 '1/12-6/12' 5 '7/12-12' 6 '> ONE YEAR'
/HEADPOST 0 'NO' 1 'YES'
/EYEAB 0 'NO' 1 'YES'
/NEURAN 0 'NONE' 1 'SUPRA-TROCHLEAR' 2 'SUPRA-ORBITAL' 3 'ZYGOMATI
        4 'ZYGOMATICO-FACIAL' 5 'INFRA-ORBITAL'
/EYEDISP 0 'NONE' 1 'UP DISPLACEMENT' 2 'DOWN DISPLACEMENT'
         3 'MED DISPLACEMENT' 4 'LAT DISPLACEMENT'
/TIMEDISP 0 'TEMPORARY' 1 'PERMANENT'
/PROPT 0 'NO' 1 'YES'
/ENOTHALM 0 'NO' 1 'YES'
/BULBHAEM 0 'NO' 1 'YES'
/ORBITAIR 0 'NO' 1 'YES'
/LIDINJ 0 'NONE' 1 'SWELLING/BRUISE' 2 'LACERATION'
/CONINJ 0 'NONE' 1 'CHEMOSIS' 2 'S.C.H.' 3 'LACERATION'
/CORINJ 0 'NONE' 1 'ABRASION' 2 'PUNCTURE WOUND' 3 'LACERATION'
```

```
/PUPILINJ 0 'NONE' 1 'TRAUMATIC MYDRIASIS' 2 'SPHINCTER PUNCTURE'
          3 'TRAUMATIC MIOSIS' 4 'HIPPUS' 5 'COMBINATION'
/IRISINJ 0 'NONE' 1 'IRIDODIALYSIS' 2 'HYPHAEMA' 3 'PROLAPSE'
/ANGREC 0 'NONE' 1 '< 90 DEGREES' 2 '90-180 DEGREES' 3 '180-270 DEG
        4 '270-360 DEGREES'
/LENSINJ 0 'NONE' 1 "VOSSIUS' RING" 2 'DISLOC/SUBLUX' 3 'TRAUMATIC
         4 'RUPTURED CAPSULE'
/PHOTEST 0 'NEGATIVE' 1 'POSITIVE' 2 'NOT DONE'
/VA 0 'NONE' 1 'MILD REDUCTION' 2 'MODERATE REDUCTION' 3 'SEVERE RE
    4 'LOSS OF VISION'
/VF 0 'NONE' 1 'CENTRAL LOSS' 2 'PERIPHERAL, 1 DIRECT' 3 '> ONE DIR
    3 'ALL DIRECTIONS'
/ACC 0 'NONE' 1 'MILD FAILURE' 2 'MODERATE FAILURE' 3 'SEVERE FAILU
/COMRET 0 'NONE' 1 'MILD/RESOLVING' 2 'MODERATE' 3 'SEVERE'
/CHORINJ 0 'NONE' 1 'ONE TEAR' 2 '> ONE TEAR' 3 'MACULA INVOLVED'
/RETINJ 0 'NONE' 1 'VITREOUS FLOAT/DET' 2 'VITREOUS HAEM' 3 'RPE DE
        4 'FLAT HOLE/TEAR' 5 'DETACH/MACULA ON' 6 'DETACH/MACULA OF
        7 'TOTAL RETINAL DETACH'
/OPTINJ 0 'NONE' 1 'CONTUSION' 2 'PARTIAL INJURY' 3 'TOTAL DAMAGE'
/NASLAINJ 0 'NO' 1 'YES'
/MALINGER 0 'NO' 1 'YES'
/PATDIS 0 'NO' 1 'YES'.
```

Serial Number	Hospital Number		Coded Clinical Data
001	135974	03	0509000013000300000000000000000000000000000000000000001
002	114366	16	0708000123000200000000000000150000000200000000000000001
003	131602	05	0705000013000100000000000000100000000200000000000000001
004	087482	02	0605220023400100000000000000100000001200000000000000001
005	135965	02	0305101023000000000000000000100000001200000000000000001
007	049455	04	0506001014020510020060000030102000001200000000000010001
008	137789	03	0503000014000500000000000000100000000200000000000000001
009	133766	03	1206000014000510220000000040100000000200000000000000001
010	137295	03	0506001014000200000000000000100000001200000000000000001
011	136784	14	0407000023000100000000000000150000001000000000000000001
012	130609	08	0507000624200400000000000000100000001000000000000000001
013	135912	13	0904370113050100000000000000100000000000100000000000001
014	131097	02	0609001123000100000000000000100000000200000000000000001
017	137770	02	0609001013000100000000000000100000001200000000000000001
018	136442	03	0206001024000200000000000000100000000000000000100000011
019	132166	03	0806101013000300000000000000152000001200000000000000001
020	136366	02	0405001023000300000000000000100000001000000000000000001
021	131486	01	0706001025001300121001000040150000001200000000101000001
022	130415	02	0506000223060100000000000000100000001200000000000000001
023	097620	02	1000330013000100000000000000100000000000600000040000001
024	138292	07	0708001001000000000000000000000000000000000000000000001
025	137595	03	0608220013000300000000000000100000001200000000000000001
026	135135	13	0107001114060000000000000000150000001000000000000000001
029	132890	03	0907000125000710031071001040150001001200000000000000000
030	137570	06	0610000023060610100000000010000000000000000000000000001
031	130219	02	1109001017000710100000000000150000001200000000000000001
032	072012	13	0605001121000000000000000000150000000200000000000000001
033	135317	03	0205001014000110100000000000100000001200000000000000001
034	135136	03	0106002021000000000000000000100000001200000000000000001
035	130947	04	0606001021000000000000000000100000001200000000000000001
044	129817	01	0613001015000010110011000040152000111000000000000000001
047	137800	12	0605000727000110000050000000150001001200000000000000001
048	136453	02	0206101024001611222050001040150000000200020000101000101
049	130130	12	0307000003000200000000000000500000000000000000000000001
050	135370	02	0705000024000200000000000000100000002200000000000000001
051	137954	03	0606000023020211020200001040100000001000000000000000001
053	131012	04	0606370034221600000000000000100000001200000000000000001
054	135934	05	0308001014000500000000000000100000001200000000000000001
055	137215	04	0606271023000100000000000000000000000000000000000000001
056	135964	02	0305000024000200000000000000100000000200000000000000001
057	131196	02	0600371031011610110011000040152001000000000002000000101
058	132136	02	0805000027001610020070000030150000101200000000100000001
059	137912	01	0606001011000010110000000030100000001000000000000000001
060	135500	00	0213001025001311311071000061150011002200000000000000000
061	135300	02	0208100236000000000000000000150000001000000000000000001
072	130251	13	0406001021000000000000000000100000001200000000100000001
075	125072	02	0505000014000511120000000040151110001000000000000000001
076	130971	02	0609000013000300000000000000100000001000000000000000001
077	133477	03	1001271013000300000000000000100000001000000000000000001

079	131377	03	06080000140005101200000000401500000010000000000000000001
080	135978	03	05050010230001000000000000001000000010000000000000000001
081	132214	02	08060000250013101200700000401500010010000000000000000001
083	132112	02	08062710250013101200110010301000010012000000000000000001
086	132767	02	09050000230001000000000000001000000012000000000000000001
087	131458	12	06050010210000000000000000001000000012000000000000000001
088	137078	02	05050010130003022107000000301000000012000000001000000001
089	137201	02	06090000110000000000000000000000000000000000000000000001
090	137769	12	06050000110000000000000000001000000002000000000000000001
091	136976	04	05070000010000000000000000000000000000000000000000000001
092	134146	14	11070000240003000000000000001500000012000000000000000001
093	136626	05	04100010240202000000000000001000000022000000001000000001
094	135975	02	03062710240001000000000000001000000010000000000000000001
095	131578	01	07032710250213103200110000201500000012000000000000000001
096	136585	05	02080012170001000000000000001000000010000000000000000001
097	132491	01	09070000150013102200300000300500000000000000000000000001
098	136435	02	03050003110000000000000000001000000010000000000000000001
100	077452	01	02071007330001000000000000001000000002000000000000000001
101	135213	01	01070011230003000000000000001000000010000000000000000001
102	133922	02	11052710140006100001000000001000000010000000000000000001
103	133510	02	10090010230001000000000000001000000010000000000000000001
104	136218	02	03121002406040000000000000001000000002000000000000000001
105	138254	02	07063710130001000000000000001500000002000000000000000001
108	136039	02	03050010252213102203300010401520010000000000000000000001
110	135290	16	02070010140000000000000000001000000012000000000000000001
111	133476	02	10090020230003000000000000000000000000000000000000000001
112	129825	12	04050002230005000000000000001000000012000000000000000001
113	131712	03	06062700130516000000000000001500000012000000000202000001
115	137445	04	06051011240206102200100000301000000010000010000000000001
117	131476	03	07090000150013101210110000301520000110000000102010000001
119	136499	07	02070011130000000000000000001000000012000000000000000001
120	135798	12	03031001300103000000000000001000000012000000000000000001
121	114042	11	04060001272116113110310000601520100012000000000200000100
122	135899	02	04090010250000000000000000001500000002000000000000000001
123	135849	02	02050003130003000000000000001000000012000000000000000001
124	130417	05	05110000230005000000000000001000000012000000001010000001
125	131029	12	06050010210000102200000000301000000000000000001010000001
126	137589	03	06082711140016000000000000001020100012003000101010000001
127	135871	03	04062710210000000000000000001000000012000200001010000001
128	132863	06	09063705342605000000000000001000000012000000003000020001
129	137913	05	07082710110000000000000000001000000012000000000000000001
131	131452	03	06060012210000000000000000001500100100000000000000000001
132	132779	05	08052700240504102200205010401000010000000000001000000001
133	137560	02	06060000120001000000000000001000000010000000000000000001
134	140463	02	12052210150000100200100000501000000012000000000000000001
135		13	05070010110000000000000000000000000000000000000000000001
136	130878	02	02050000230001000000000000001500000002000000000000000001
137	136410	14	02060010110001100200000000001000100012000000000000000001
138	116291	04	10051010330002000000000000001020000012000000001010000001
139	135876	05	04050010202604000000000000001000000012000000000000000001
140	137272	03	05063700342016000000000000001021010012000000000000000001
141	135991	03	03052712270006000000000000001000000012000000102010000001
146	133913	03	11060005130001000000000000001000000012000000000000000001
147	136199	11	03041010170004100232200000301000100012000000101010000001
148	137784	04	06070000130003000000000000001000000010000000000000000001
149	130891	03	05100016210000000000000000001500000012000000000000000001
152	137588	03	06050001130005112000000000101000000000000000001000000001
154	130024	03	07061001303016101200100000201000000012001000000000000001
155	098369	03	04061015210000101000000000200000000000000000000000000001
156	133466	18	10070010130000000000000000001000000010000000000000000001
157	131286	06	06062701240005000000000000001521000012000000102010000001
158	136426	13	02050010130003000000000000001000000012000000001000000001
160	132127	02	08090020130002000000000000001000000012000000000000000001
162	135206	02	01091010340016101200110000601000000012000000000000000001
163	116053	02	05060000140006101200000000201000000002000000000000000001
164	133408	04	09022702240416110000505000001000000000000000001044000001
165	137108	11	05040000250013104100710000501520010012000000001010000001
166	136059	02	03081000001602000000000000000000000000000000000000000001
173	136627	03	04050003130001101200200000201000000012000000000000000001

175	135132	14	0105001024021610120001000050102001001200000000000000000
176	131451	12	0605000024000310420011000020100000001200000000000000001
177	137202	03	0506271015001320120030000050100001001200000000000000001
178	130954	03	0509001013060031210020000020100000001200000000100000001
179	137185	13	0508000010010400000000000000050000000000000000000000001
180	133718	02	1006000314001300000000000000150000001200000000000000001
181	133532	02	1005001012020410220000000030120000001200000000000000001
182	137561	02	0609000024000110220000000020100000001200000000000000001
183	044731	04	1205000013000600000000000000150000001200000000000000001
184	134660	05	1107370037051610000020000000152001001200000000440003001
185	124879	13	0205370115000000000000000000150000001200000000101000001
186	131446	03	0610000023000110020000000030100000001200000000000000001
187	129679	02	0305000002000100000000000000000000000000000000000000001
188	137698	14	0607100117201611320030000030152000001200000010101000001
189	130569	02	0406000014000100000000000000100000001200000000000000001
190	137294	02	0506101023000100000000000000100000001200000000000000001
191	136748	01	0405270113001600000000000000100000001200000000000000001
192	138281	13	0705001236000010001000000000150000001200000000201000001
193	136331	02	0503370014000500000000000000100010001000200000000020001
194	131742	05	0610001024000411120010000020152000001200000000000010001
195	136038	12	0602001030020000000000000000100000001200000000000000101
196	120873	02	0609001224000311320010000020100001001200000000000000001
197	135308	05	0107001313000110020000000020100000001200000000000000001
198	134743	02	1208001124001600000000000000000000000000000000000000001
199	132775	02	0906000024000400000000000000000000000000000000000000001
200	130683	06	0506170011000100000000000000100000001200021000000000001
201	130362	04	0408001021000000000000000000100000001200000000000000001
202	133092	03	0901101024001611310070000050100000001200000000100100001
203	132498	03	0801001024001600000000000000100000002200000000000000001
204	131478	03	0705101011000010020020001030100000001200000000000000001
205	133063	01	0909000523000300000000000000100000001200000000000000001
206	131962	05	0604170034201600000000000000000000000000000000000000001
207	137809	02	0609000011000100000000000000100000001000000000000000001
208	132814	03	0901000023020410002120000020100000001200000000000000001
209	137598	03	0605001714000410000000500000122000001200000000000000001
210	134856	03	0204371337251600000000000000100000001200000000400003001
211	137297	13	0505271113000000000000000000100000001200000000000000001
212	132535	15	0807000114000410320000000020100010001200000000000000001
213	137180	02	0501270000360500000000000000000000000000000000000000001
214	130338	02	0409000026001300000000000000150000001200000000000000001
215	119310	03	0705000023000000000000000000100000000200000000000000001
216	131669	04	0507000114000110000200000000100000001200000001000000001
217	207882	03	0606000020200410413000500030150000000000000000301000001
219	133924	02	1106001112000100000000000000100000001200000000000000001
220	133938	02	1109000024060610020000000030100000001200000000000040001
221	056312	02	0205000013000300000000000000100000001200000000000000001
222	133407	02	0903101024201610311011000030122000001200100010201020001
223	136449	02	0305000114000400000000000000100000001200000000000000001
224	114973	17	0907001115000000000000000000100000001200000000400003001
229	115697	02	0909001024000510020000000030100000001200000000201000001
245	132470	04	0805001012000000000000000000100000001200000000000000001
249	129651	02	0306000030200410000100000000100000001200000000000000001
250	131030	02	0603270030100200000000000000100000001000000000000000001
251	136513	13	0206000023200200000000000000100000001000000000000000001
252	133093	14	0906001021000100000000000000100000001200000000000000001
254	132859	05	0908000314100500000000000000100000001200000000000000001
255	133414	03	1005001031000000000000000000100000001200000000000000001
256	133440	02	1006000024001211411342001160100000002200000000000000001
257	134358	05	1205000013000100000000000000100000001200000000000000001
258	137597	02	0607001023000300000000000000100000001200000000000000001
259	134408	04	0105000017060300000000000000100000001200000000000000001
260	129454	11	0205000015001310311210000030150000001200000000000000001
261	130223	02	0506000023000100000000000000100000001200000000000000001
262	133445	15	1007000022000100000000000000100000001000000000000000001
263	130947	04	0605001023000000000000000000100000001200000000000000001
264	124673	03	0305270514060500000000000000100000001200020000000000001
265	135970	03	0309000014000410120000000020100000001200000000201000001
266	135976	02	0305000024000200000000000000100000001200000001000000001
267	134658	02	1205001021000000000000000000100000001000000000000000001

270	132084	02	0706001017060510120010000050150001001200000000000000001
271	131964	05	1107000034221610120000000020100000001200000000000000001
272	135946	03	0305001031000000000000000000100000001200000000000000001
273	130025	12	0602370020020000000000000000000000000000000000000000001
274	138299	01	0702000230010400000000000000100000001200000000000000001
275	135079	04	0105270013050710000000500000100000001200000000400003001
276	131857	03	0506270134200500000000000000102001001200000100000000001
277	131096	02	0606101023000000000000000000100000001200000000000000001
278	135692	03	0201270017000611310120500040151000002200000000100000101
279	136635	03	0406371017000000000000000000100000001200000000000000001
280	130230	02	0209000012060100000000000000100000001200000000000000001
284	137201	02	0509000012000100000000000000000000000000000000000000001
285	134815	01	0106001011000100200000000030100000001200000000000000001
289	065197	02	1106100014001610020230000030150010001200000000201000001
290	130456	04	0405000014221610020000000030100000002200000000000000001
291	135309	03	0109000013000100000000000000100000001000000000000000001
293	135289	04	0112000013000200000000000000100000001200000000000000001
294	137469	06	0606101134100000000000000000100000000000000100000000001
295	135478	04	0203000013000210000010000000000000000000000000000000001
296	132144	02	0103270527000400000000000000150000001000000000000000001
297	133932	01	1206370423000100000000000000100000001200000000000000001
298	132120	03	0807230023000000000000000000100000001200000000000000001
299	138502	02	0706000013000100000000000000150000002000000000000000001
300	132468	04	0306001121000000000000000000100000002000000000101000001
301	131626	02	0309000003000100000000000000000000000000000000000000001
302	130028	11	0102000023000100000000000000100000001200000000000000001
305	137827	02	0602371017060210121030000030100000001200000101000040001
314	135877	01	0307000014001310020000000030100000001200000000000000001
315	130317	13	0406101035001310413071000040150001001200000010301000001
316	137338	02	0505001000020000000000000000000000000000000000000000001
317	135425	03	0409001111000000000000000000100000002000000000000000001
318	137588	02	0610000114000210000010000000100000000000000100000000001
319	132764	14	0906000025001300000000000000150000001200000000000000001
320	132114	13	0807001013000100000000000000150000001200000000000000001
321	114361	14	0506100423000500000000000000100000001200000000101000001
323	132161	06	0708231014000500000000000000100000001200000000000000001
326	135362	02	0301000030010400000000000000100000001200000000000000001
327	133910	01	1106001027000500000000000000150000001200000000000000001
328	135793	04	0305271017001600000000000000150000001200000000000040001
329	135967	13	0205001225000510220130000060150000001200000000000000001
330	137731	06	0601371010010011100000000030102000001200000000000000001
331	134759	01	1205001215001310021031000040150001001200000000000000001
332	137936	03	0705000013000000000000000000150000001200000000000000001
333	131631	02	0405000023000510000050000000100000001200000000000000001
334	135205	16	0107271224000310020010000030100000001200000000000000001
335	127718	03	1106270017001610020050000050150000001200000000100040001
336	131743	11	0601270013061300000000000000100000001200000000200000001
337	135986	04	0507000114000400000000000000100000001200000000000000001
338	114583	14	0707000013000100000000000000100000001200000000000000001
339	135321	13	0104270014050400000000000000120000001200000000000000001
346	131133	02	0605001015001310021011000020100001001200000000000000001
347	136977	03	0508001023000110020000000030100000001200000000000000001
348	133701	12	1005002031000000000000000000100000001200000000000000001
352	142200	01	0306001027001310220010000020150000001000000000000000001
353	142033	04	0310000127000600000000000000100000001200000000000000001
354	134762	12	0906000023000710221030000020100010001200000010100000001
355	138971	02	0910001020020010001000000000000000000000000000000000001
356	142440	11	0305001026010400000000000000150000001200000000000000001
357	138811	02	0801240024000300000000000000150000001200000000000000001
358	141087	02	0105230214001310000160000000151000001200000013323020000
359	139354	11	0909001027001600000000000000100000000000102000200100001
360	139190	03	0903230030200210221000000030150000001200000000000000001
361	139369	12	1007000023000000000000000000150000002000000000000000001
362	138960	00	0807001014000010020020000030100000001000000000000000001
363	142402	02	0307000024000610200000000020100000001200000000000000001
364	134647	02	1009001023000210220040000000150000001200000000000000001
365	142153	02	0306000023000300000000000000100000002000000000000000001
366	139436	04	0106270023100300000000000000100000001200300000000000001
367	140485	12	1205001015001310221010000000100001001200000000000000001

369	14[illegible]188	02	0309000024000100000000000000150000001200000000000000001
370	141020	13	0107270603000400000000000000150000001200000000000000001
371		02	0113230034200100000000000000100000000200000000000000001
372	140304	02	1009000023000100000000000000150000001200000000000000001
373	138265	02	0705000000402000000000000000000000000000000000000000001
374	138958	03	0910001021000000000000000000150000001000000000000000001
375	140374	03	0308001215001310221011000040152000001200000000000000001
376	140244	02	1106000013000110002000000000100000001200000000000000001
377	138429	04	0805000013000100000000000000150000000000000000000000001
378	140393	05	1206271034000510020070000000100000001200000000000000001
379	138808	07	0807000013000300000000000000100000000200000000000000001
380	138904	02	0806331011100000000000000000100000002200000000000000001
381	139297	14	0907000113000310000100000000150000001000000000000000001
382	142424	03	0305001023000300000000000000150000001200000000000000001
383	141895	02	0306001014000310020010000030150000000000000000000000001
384	139897	01	1013000015001300000001000000150001011200000000000000001
385	140462	04	0110241015001310000070001000152001002200001000200000001
386	139093	03	0907001003000100000000000000150000000000000000000000001
387	138977	02	0909000317000100000000000000150000001200000000000000001
388	135371	02	0805000023000300000000000000150000000200000000000000001
389	140843	03	0205000024000400000000000000150000001200000000000000001
390	141820	03	0110001013020100000000000000150000001200000000000000001
392		13	1013000013000400000000000000100000001230020000440023001
393	138972	14	0308000003000100000000000000000000000000000000000000001
394	139986	02	1106000014000400000000000000100000001000000000000000001
395	141823	02	0213001123000300000000000000100000001200000000000000001
396	124768	04	0501360023221300000000000000100000000000000000120002101
397	140301	05	1105000013000100000000000000100000001200000300100000001
398	140225	03	1005000033060400000000000000150000001200000000000000001
399	141850	02	0209001101000000000000000000000000000000000000000000001
401	053373	14	0308001023000300000000000000150000001200000000000000001
402	142059	04	0305000014000400000000000000150000001200000000000000001
403	139336	03	0906000202000100000000000000000000000000000000000000001
404	140431	12	1208000124000400000000000000150000001200000000000000001
405	134373	02	0900270004260411410050400040100000001200000000000000001
406	141028	11	1206001015000000000000000000150000001200000000000000001
407	138966	09	0909001023000300000000000000150000001000000000000000001
408	139610	05	1001230113001611310070001040152000001200000010201000001
409	138947	16	0802000023000310220000000030100000001200000000000000001
410	140486	02	1206000021000110220050000030100000001200000002000000001
411	139889	04	1005000123000310220070001030100000001200000000000000001
412	140390	04	1206320524000200000010000000150000000200000000000000001
413	142403	04	0308001123000310000050000000100000000200000000000000001
414	139891	02	1005000013000100000000000000150000001200000001000000001
415	141042	04	0113000013000110000200000000150000001200000002000000001
416	140342	02	1106000015001310321071001040150001001200000002000000000
417	142152	04	0305001023100300000000000000100000001200000000000000001
418	138259	04	0709000023000300000000000000150000000000000000000000001
419	140351	03	1207000003020300000000000000000000000000000000000000001
420	120788	05	1106250114000300000000000000150000001200000000000000001
421		12	0105001021000000000000000000100000000000000003000000001
422	139267	02	0909000013000100000000000000150000001200000000000000001
423	140624	05	0308000013000100000000000000150000001200000000000000001
424	138968	01	0905001313000100000000000000100000001200000000000000001
425	139888	01	1009000013000300000000000000100000001200000000000000001
426	140479	01	1205220014000200000000000000150000001200000000000000001
427	137788	02	0609000423000100000000000000100000000200000000000000001
428	139335	02	0905270013000310020010000030100000001200000000000000001
429	138445	02	0809001003000300000000000000000000000000000000000000001
430	138443	02	0805001025001300000000000000102000000000000000000000001
431	130344	02	1005000016001300000000000000150000001200000000000000001
432	138530	02	0809000023000200000000000000150000001000000000000000001
433	141080	04	0107270114001300000000000000100000000000000000000000101
434	142384	12	0306000026001300000000000000150000001200000000000000001
435	138924	01	0805001025001310220010001050150001000000000000000000001
436	141824	14	0207001025001300000000000000152010001200000000000000001
437	140364	02	1106001021000000000000000000100000001200000002000000001
438	141062	03	0106000004000300000000000000150000000000000000000000001
439	140877	02	1209000003000100000000000000000000000000000000000000001

441	139353	02	0906001031000000000000000000100000001200000000000000001
442	135377	02	1009000121020110021010001030100000000000000000000000001
443	137639	05	0810000018461600000000000000100000000000000000440003001
444	140226	05	1107000300010000000000000000000000000000000000000000001
445		01	0905000013000100000000000000150000000000000000000000001
446	140302	02	1109000223000100000000000000150000000000000000000000001
447	139308	02	0905000013000100000000000000100000001200000000000000001
448	141816	03	0206000013000410200000000000150000001200000000000000001
449	141858	01	0205000026000400000000000000100000000000000000000120001
450	140201	03	1101270017000310000050000000100000001200000000000000001
451	091516	02	1204270423000100000000000000100000000000000000000010001
452	140334	02	1109000114000100000000000000150000001200000000000000001
453	141022	06	0104230104000400000000000000100000000000000000000000001
454	140923	02	1206231011000000000000000000150000001200000000000000001
455	140783	12	1205000116021300000000000000150000001200000000000000001
456	140324	02	1101231124000000000000000000100000001200000000440003001
457	141483	03	1206210034000400000000000000150000000000000000000000001
458	139617	02	1009000022000100000000000000100000001200000000000000001
459	137903	03	0608001011000010020010000030100000001200000000000000001
460	141039	01	0306000024000100000000000000100000001200000000000000001
461	140338	02	1109000007000200000000000000000000000000000000000000001
462	140447	11	1101000025061611311271400050150001001200100000200100001
463	138509	12	0802220214000100000000000000100000001200000000000000001
464	141001	06	1206001020010100000000000000100000001200000000000000001
465	141863	16	0205000117000200000000000000100000001200000000000000001
466	139625	03	1010000137160010311073410050152001001200000000000000001
467	142401	11	0306001001000000000000000000150000000000000001000000001
468	141049	02	0107000025021310021030000030100000000000000000000000001
469	142116	02	0305000002000100000000000000000000000000000000000000001
470	139872	02	1000250020020010200400001000150000000000000000000000001
471	142144	01	0107000015001310221011000030100001000000000000000000001
472		01	1109000125001310222050001030100000001200100000000000001
473	142134	04	0307000024000300000000000000150000001200000000000000001
474	139984	02	1005001023000100000000000000150000001200000001000000001
475	139650	01	1001271221000000000000000000100000001200000000000000001
476	140206	04	1106001115001310021011000030100000001200000000000000001
477	140460	03	1205000103000100000200000000100000000000000001000000001

APPENDIX 4

CANNIESBURN HOSPITAL APPENDIX SHEET C

MAXILLOFACIAL INJURIES

Card No. A 1

CANNIESBURN HOSPITAL
REGIONAL PLASTIC AND
ORAL SURGERY UNIT
APPENDIX SHEET C (1)
MAXILLO-FACIAL INJURIES

HOSPITAL NUMBER 2-8

SURVEY NUMBER 9-14

NAME.. Age.......................

Sex...................... 15

GENERAL DATA not recorded elsewhere

TREATMENT PRIOR TO ARRIVAL

Dressings...

Sutures...

Operations...

....

Tetanus Toxoid...

Antibiotics...

Started (time)...

Other Drugs...

....

I.V. Fluids...

....

CAUSE OF INJURY

0. car (no seat belt)
1. car (seat belt)
2. motor-cycle
3. other RTA
4. sport
5. industrial
6. alleged assault
7. dental surgery
8. fall
9. other

16

DATE OF ACCIDENT (day / month / year) 17-22

TIME OF ACCIDENT (24 hour clock) 23-26

REFERRING HOSPITAL AREA BOARD 27

1. Greater Glasgow
2. Argyll and Clyde
3. Lanarkshire
4. Forth Valley
5. Ayrshire & Arran
6. Dumfries & Galloway
7. Other ....

PLACE OF ACCIDENT

1. home
2. work
3. public
4. other

28

SOURCE OF REFERRAL...

....

DATE FIRST EXAMINED (day / month / year) 29-34

TRANSPORT

1. stretcher
2. chair
3. walking

35

PRELIMINARY ASSESSMENT

CEREBRAL STATE

0 conscious 1 unconscious [ ] 36

If unconscious, specify depth [ ] 37
1 responds to command
2 responds to pain
3 no response

Pulse rate per minute [ ][ ][ ] 38-40

AIRWAY

0 clear 1 obstructed [ ] 47

SHOCK

0 absent 1 present [ ] 48
(i.e. I.V. fluids needed)

EPISTAXIS

0 absent 2 left [ ] 49
1 right 3 both

BLEEDING FROM EAR

0 absent 2 left [ ] 50
1 right 3 both

PUPIL REACTION

0 Normal both [ ] 41
1 Abnormal either

If abnormal specify by appropriate ringing.

	Right	Left
	dilated constricted	dilated constricted
Light reflex	brisk slow nil	brisk slow nil
Accommodation	brisk slow nil	brisk slow nil
Consensual Reflex	present absent	present absent

C.S.F. LEAKAGE

0 nil 2 R. otorrhoea 4 combined [ ] 42
1 rhinorrhoea 3 L. otorrhoea

AMNESIA

0 absent 1 present [ ] 43

If present RA/ PTA/ BOTH
delete as appropriate.

DURATION

1 hours [ ] 44 Number [ ][ ] 45-46
2 days
3 weeks

ASSOCIATED INJURIES

(non maxillo-facial)
0 none 1 present [ ] 51

If present specify [ ] 52
1 skull 4 other fractures
2 thoracic cage 5 other
3 abdominal 6 multiple

....

....

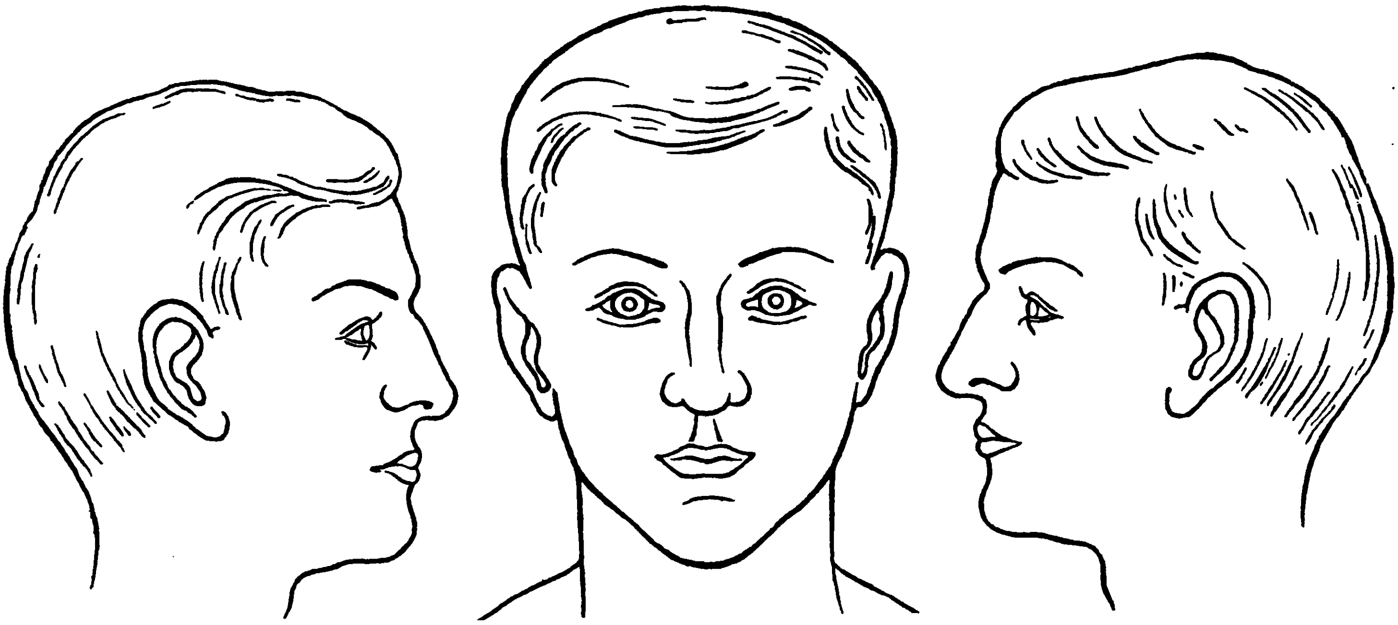
MAXILLO-FACIAL EXAMINATION: Extra-Oral

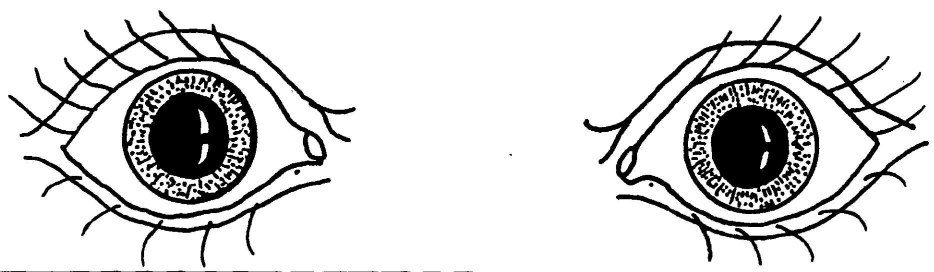
INSPECTION

Chart injuries below: extent in cms.
special features — penetrating nerve injury, etc.

SOFT TISSUE INJURIES

0 none 4 lacerations [ ] 53
1 abrasions 5 bruising [ ] 54
2 puncture wounds 6 swelling
3 tissue loss 7 multiple





PALPATION

Orbital margins ....

Nasal bones ....

Malar ....

Condyles ....

....

....

Mandibular borders ....

....

Maxillary stability ....

....

....

....

OTHER OBSERVATIONS

MAXILLO-FACIAL EXAMINATION: Intra-Oral

Lacerations 0 none 1 present [ ] 55

Ecchymosis 0 none 1 present [ ] 56

Occlusion 0 normal 1 deranged [ ] 57

If deranged, specify ....

....

Mandibular midline
0 normal 1 displaced [ ] 58

If displaced specify ....

....

Maxillary midline
0 normal 1 displaced [ ] 59

If displaced, specify ....

....

Dentures 0 none 1 intact 2 broken 3 lost 4 at home upper [ ] 60 lower [ ] 61

Teeth (count only teeth usable for splint fixation) [ ] 62

0 edentulous
1 lower anteriors only
2 less than 7 teeth
3 7-16 teeth
4 17-full dentition for age

Dental Injury

broken	0 no	1 yes	[ ]	63
chipped	0 no	1 yes	[ ]	64
Subluxed	0 no	1 yes	[ ]	65
Avulsed	0 no	1 yes	[ ]	66

Specify details ....

....

....

Oral Hygiene
1 good 2 fair 3 poor [ ] 67

Chart as follows: —

missing teeth -8-|

teeth for extraction (write reason adjacent) ⑧|

roots X|

unerupted teeth 8|

			e	d	c	b	a	a	b	c	d	e			
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
			e	d	c	b	a	a	b	c	d	e			
RIGHT															LEFT

Card No. [B] 1

Repunch Columns 2-14 from Card A 2-14

MIDDLE THIRD FRACTURES

FRACTURES: ZYGOMATICO-MAXILLARY or ORBITAL
(definitive assessment; if necessary, postoperative)
0 intact
1 unilateral
2 bilateral
[ ] 28

FRACTURES: MAXILLA
(definitive assessment; if necessary, postoperative)
0 intact
1 fractured
[ ] 29

Only complete this section if these regions fractured

MALAR FRACTURE SITES [R | L] 15-16

0 intact
1 undisplaced (any site)
2 zygomatic arch only
3 Tripod F.Z. suture undistracted
4 Tripod F.Z. suture distracted
5 pure blow-out
6 orbital rim only
7 comminuted-other than above

CLINICAL FEATURES

Facial flattening
0 no
1 yes
[R | L] 17-18

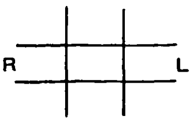
Infraorbital anaesthesia
0 no
1 yes
[R | L] 19-20

Trismus
0 no
1 yes
[ ] 21

Recent epistaxis
0 no
1 yes
[R | L] 22-23

Subconjunctival ecchymosis
0 no
1 yes
[R | L] 24-25

Diplopia 0 no
1 yes
[ ] 26

chart distribution
thus 1 or 11: — or =
R  L

Eye movements
0 normal
1 abnormal
[ ] 27

describe abnormality if present:—..
....
....

ORBITAL INVOLVEMENT [R | L] 30-31

0 not involved 3 roof
1 floor only 4 lat. wall plus/minus floor
2 medial wall 5 multiple

PUPIL LEVEL
0 normal 1 abnormal
specify if abnormal
[R | L] 32-33
....

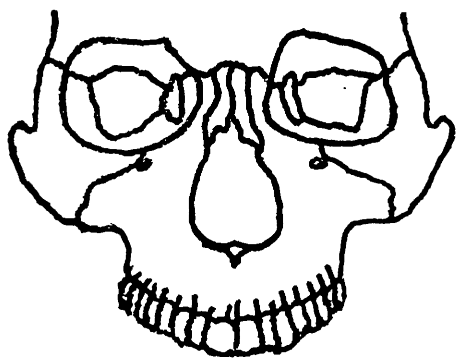
ENOPHTHALMOS
0 normal 1 yes
[R | L] 34-35

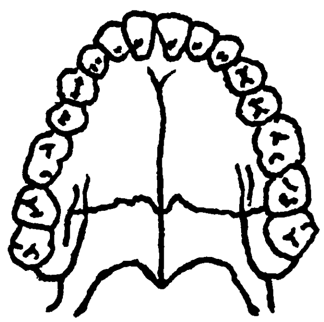
INJURY TO GLOBE
0 no 1 yes
specify if injured
[R | L] 36-37
....
....

INJURY TO LACRIMAL APPARATUS
0 no 1 yes
[R | L] 38-39
specify if injured
....
....

VISUAL ACUITY / R / L

PREOPERATIVE OPHTHALMIC ASSESSMENT
0 no 1 yes
[ ] 40





MAXILLARY FRACTURE SITES
(classify MAJOR fracture on each side)

0 nil 4 combined I, II or III
1 Le Fort I 5 tuberosity
2 Le Fort II 6 alveolar (except 5)
3 Le Fort III 7 other
[R | L] 41-42

PALATAL SPLIT
0 nil 1 present
[ ] 43

INFECTION
0 no 1 yes
if present specify
[ ] 44
....
....

DESCRIPTION

A 1 simple 3 compound extra-orally
2 compound intra-orally 4 compound both
[ ] 45

B 0 undisplaced 2 displaced
[ ] 46

RACTURES: NASAL BONES (definitive assessment; if necessary, postoperative)
0 intact 1 fractured [ ] 56

.ASSIFICATION

1 undisplaced 3 deviation
2 depressed bridge 4 extensive naso-ethmoid [ ] 47

SEPTUM

haematoma 0 no 1 yes [ ] 57

fracture detected 0 no 1 yes [ ] 58

SUBPERIOSTEAL HAEMATOMA
0 no 1 yes [ ] 59

RACTURES: CRANIO-FACIAL

ontal/Temporal 0 Intact
Sphenoidal 1 Fractured [ ] 48

ontal sinus Fractured
0 no
1 yes [ ] 49

MANDIBULAR FRACTURES

FRACTURES: MANDIBLE (definitive assessment; if necessary, postoperative)
0 intact 1 fractured [ ] 60

Inly complete these sections if MANDIBLE FRACTURED

RACTURED SITE [ ] 50

1 condyle 6 symphysis
2 coronoid 7 ramus
3 angle 8 alveolus [ ] 51
4 body 9 other
5 canine 0 more than 2 sites

LASSIFICATION (if single or double)

1 Class I — teeth on both fragments
2 Class II — teeth on only 1 fragment [ ] 52
3 Class III — edentulous [ ] 53

NGLE/BODY FRACTURES

1 HFVF 4 HUVU
2 HFVU 5 not classifiable [ ] 54
3 HUVF [ ] 53

INFECTION 0 no 1 yes
if present specify [ ] 61

....

....

DESCRIPTION (main site/s)

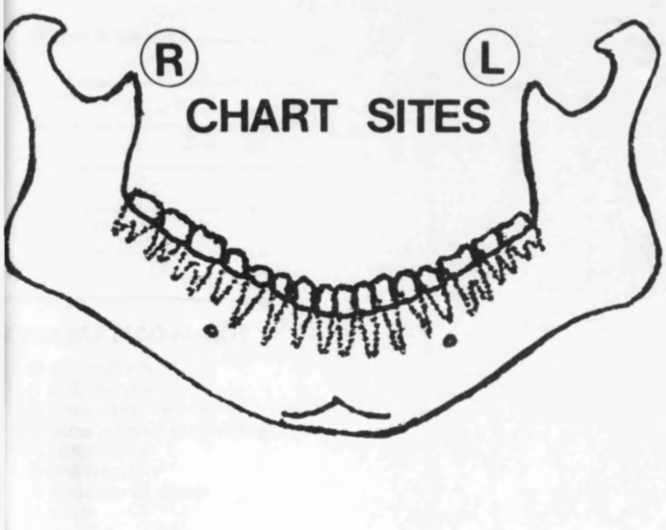
A 1 simple 3 compound extra-orally
2 compound intra-orally 4 compound both [ ] 62

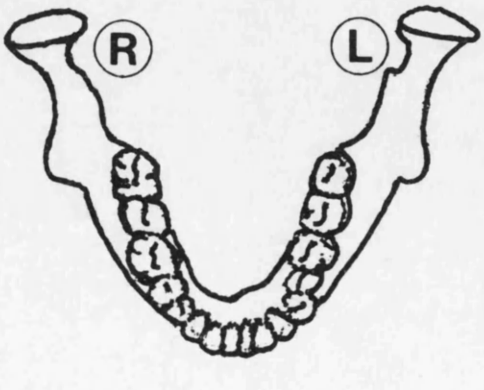
B 0 undisplaced 1 displaced [ ] 63

C 0 non-comminuted 2 comminuted
1 Greenstick [ ] 64

D 1 bone loss 0 no bone loss [ ] 65

E 1 mental Dysaesthesia 0 no change [ ] 66





Card No. C 1

Repunch Columns 2-14 from Card A

TREATMENT

Note: where the same fixation contributes to the stability of fractures in more than one region complete for each region independently.

PRELIMINARY TREATMENT

(debridement, removal of fragments, antibiotics, etc.)

....

....

....

....

....

....

....

IMPRESSIONS

0 none 1 taken [ ] 15

DEFINITIVE TREATMENT

0 nil 2 conservative treatment only [ ] 24
1 undertaken

Time elapsed since injury until definitive treatment [ ] 25

0 not known 3 3-4 days 5 > 7 days
1 < 24 hours 4 5-7 days
2 1-2 days

Treated as: [ ] 26

1 out-patient 3 in-patient other hospital
2 in-patient on visit 4 other

Other specify ....

Anaesthesia [ ] 27

0 none 2 local analgesia
1 general 3 sedation + L.A.

Reduction [ ] 28

0 none 3 traction (including elastics)
1 closed 4 late refracture
2 open 5 other

SUMMARY OF DEFINITIVE TREATMENT

FIXATION-MANDIBLE

IMF [ ] 16
- 0 none
- 1 IMF — eyelet wiring
- 2 IMF — arch bars on either jaw
- 3 IMF — cast cap splints
- 4 IMF — Gunning-type splints
- 5 IMF — other

Connecting bar used [ ] 17
0 no 1 yes

OTHER FIXATION

[ ] 18 [ ] 19 [ ] 20

0 none 5 bone plate
1 LBW via incision 6 pins
2 LBW via wound 7 tranosseous circum. wire
3 UBW 8 other internal wiring
4 cortical wire 9 other

If 8 or 9 specify ....

....

....

....

....

....

FIXATION-MAXILLA

IMF [ ] 29
- 0 none
- 1 IMF — eyelet wiring
- 2 IMF — arch bars on either jaw
- 3 IMF — cast cap splints
- 4 IMF — Gunning-type splints
- 5 IMF — other

Connecting bar used [ ] 30
0 no 1 yes

OTHER FIXATION

0 none 2 craniomandibular [ ] 31
1 craniomaxillary 3 other

Specify [ ] 32 [ ] 33 [ ] 34
- 1 headcap
- 2 halo
- 3 supraorbital pins
- 4 4-pin box frame
- 5 other extraskeletal
- 6 internal suspension (circumzygomatic)
- 7 internal suspension (frontal suspension)
- 8 other internal suspension (including combined 7 & 8)
- 9 other

specify ....

....

ZYGOMATICO-MAXILLARY

[ ] [ ] 21-22 (R L)

- 0 no fixation
- 1 F.Z. wiring
- 2 infraorbital wiring
- 3 other orbital rim wiring
- 4 antral pack
- 5 pin fixation
- 6 multiple of above
- 7 other

specify ....

....

NASAL

[ ] 23
- 0 no fixation
- 1 direct wiring
- 2 lead plates (or similar)
- 3 POP splint (or similar) only
- 4 other

ORBITAL FLOOR

Explored [ ] [ ] 35-36 (R L)
0 no 1 yes

if explored [ ] [ ] 37-38 (R L)
- 0 no graft inserted
- 1 silastic sheet
- 2 bone graft
- 3 cartilage graft
- 4 other implant

OTHER OPERATIVE TREATMENT

....

....

....

NOTES

DEFINITIVE REVIEW

DURATION OF FIXATION 39-40
(to nearest week)

If not possible to determine
enter I in box 41 and state why below 41

....

....

CESSATION OF C.S.F. LEAK (if relevant) 42-43

Number of days following injury
0 0 = not known

SCARS specify 44

0 none
1 present
2 present and may require revision

....

....

....

COMPLICATIONS 45

0 none 1 present

Delayed union 46
0 no 1 yes
(i.e. > 4/52 for maxilla > 6/52 for mandible)

Non-union 47
0 no 1 yes
or
Malunion (Malar Flattening) 48
0 no 1 yes
specify..

....

....

Persistant diplopia 49
0 no 1 yes

Malocclusion 50
0 no 1 yes

Prolonged trismus 51
0 no 1 yes

Prolonged paræsthesia 52
0 no 1 yes

Anosmia 53
0 no 1 yes

Epiphora 54
0 no 1 yes

Other 55
0 no 1 yes
specify..

....

OTHER COMPLICATIONS.....................................

....

....

....

....

....

....

....

....

....

....

....

....

....

CONTINUATION NOTES

APPENDIX 5

DATA COLLECTION SHEET FOR THE FORCED DUCTION AND FORCE GENERATION TESTS

DATA COLLECTION SHEET

Date of Examination:

Patient's Name:

Code:

Age:

Sex: M [ ] F [ ]

Eye: R [ ] L [ ]

Provisional Diagnosis:

Intraocular Pressure: Before the Test [ ] mmHg

After the Test [ ] mmHg

1	Lat (FDT)
2	Lat (FDT)
3	Med (FDT)
4	Med (FDT)
5	Sup (FDT)
6	Sup (FDT)
7	Inf (FDT)
8	Inf (FDT)
9	Lat (FGT)
10	Med (FGT)
11	Sup (FGT)
12	Inf (FGT)

Comments:

GLASGOW
Y