# ORBITAL BLOW-OUT FRACTURES: CORRELATION OF PREOPERATIVE COMPUTED TOMOGRAPHY AND POSTOPERATIVE OCULAR MOTILITY*

BY *Gerald J. Harris,* MD, FACS, *George H. Garcia,* MD (BY INVITATION), *Sangeeta C. Logani,* MD (BY INVITATION), *Michael L. Murphy,* MD (BY INVITATION), *Bhavna P. Sheth,* MD (BY INVITATION), AND *Ajay K. Seth,* MD (BY INVITATION)

**ABSTRACT**

*Background/Purpose:* Although the management of orbital blow-out fractures was controversial for many years, refined imaging with computed tomography (CT) helped to narrow the poles of the debate. Many orbital surgeons currently recommend repair if fracture size portends late enophthalmos, or if diplopia has not substantially resolved within 2 weeks of the injury.

While volumetric considerations have been generally well-served by this approach, ocular motility outcomes have been less than ideal. In one series, almost 50% of patients had residual diplopia 6 months after surgery.

A fine network of fibrous septa that functionally unites the periosteum of the orbital floor, the inferior fibrofatty tissues, and the sheaths of the inferior rectus and oblique muscles was demonstrated by Koornneef. Entrapment between bone fragments of any of the components of this anatomic unit can limit ocular motility.

Based on the pathogenesis of blow-out fractures, in which the fibrofatty-muscular complex is driven to varying degrees between bone fragments, some measure of soft tissue damage might be anticipated. Subsequent intrinsic fibrosis and contraction can tether globe movement, despite complete reduction of herniated orbital tissue from the fracture site. We postulated that the extent of this soft tissue damage might be estimated from preoperative imaging studies.

*Methods:* Study criteria included: retrievable coronal CT scans; fractures of the orbital floor without rim involvement, with or without extension into

*From the Section of Orbital and Ophthalmic Plastic Surgery, Department of Ophthalmology, Medical College of Wisconsin, Milwaukee. Supported in part by an unrestricted grant from Research to Prevent Blindness, Inc, New York, NIH Core Grant EY01931, and the Heed Ophthalmic Foundation (Dr. Garcia).

the medial wall; preoperative diplopia; surgical repair by a single surgeon; complete release of entrapped tissues; and postoperative ocular motility outcomes documented with binocular visual fields (BVFs). Thirty patients met all criteria. The CT scans and BVFs were assessed by different examiners among the authors.

Fractures were classified into 3 general categories and 2 subtypes to reflect the severity of soft tissue damage within each category. "Trap-door" injuries, in which bone fragments appeared to have almost perfectly realigned, were classified as type *I* fractures. In the *I-A* subtype, no orbital tissue was visible on the sinus side of the fracture line. In the *I-B* subtype, soft tissue with the radiodensity of orbital fat was visible within the maxillary sinus. In type *II* fractures, bone fragments were distracted and soft tissue was displaced between them. In the *II-A* subtype, soft tissue displacement was less than, or proportional to, bone fragment distraction. In the *II-B* subtype, soft tissue displacement was greater than bone fragment distraction. In type *III* fractures, displaced bone fragments surrounded displaced soft tissue in all areas. In the *III-A* subtype, soft tissue and bone were moderately displaced. In the *III-B* subtype, both were markedly displaced.

Motility outcomes were quantified by measuring the vertical excursion in BVFs. The interval between trauma and surgical repair was also determined.

*Results:* Among the 15 patients with a motility outcome in BVFs which was poorer than the median (86° or less of single binocular vertical excursion), 4 patients (27%) had type *A* fractures; 11 patients (73%) had type *B* fractures. Among the 15 patients with a better outcome than the median (88° or more), 10 patients (67%) had type *A* fractures; 5 patients (33%) had type *B* fractures. These differences became more defined as analysis moved away from the median.

Among 5 patients with type *B* fractures and better than the median result in BVFs, 3 patients (60%) had surgical repair during the first week after injury. Among the 11 patients with type *B* fractures and less than the median result, 1 patient (9%) had repair during the first week.

*Conclusions:* When the CT-depicted relationship between bone fragments and soft tissues is considered, a wide spectrum of injuries is subsumed under the rubric of blow-out fractures. In general, greater degrees of soft tissue incarceration or displacement, with presumably greater intrinsic damage and subsequent fibrosis, appear to result in poorer motility outcomes. Although this retrospective study does not conclusively prove its benefit, an urgent surgical approach to selected injuries should be considered.

## INTRODUCTION

Earlier investigators[1-6] described internal fractures of the orbital floor and implicated hydraulic forces in their pathogenesis, but Smith and Regan's classic cadaver experiments and coinage of the term "blow-out fracture" in 1957[7] were milestones in the history of this condition. Smith and Regan[7] produced an impact on the orbital soft tissues that was transmitted equally throughout the orbit. The orbital walls fractured at their weakest points, while the orbital rims remained intact. The soft tissues were displaced or incarcerated, correlating with the clinical findings of enophthalmos and restricted motility.

During the next 2 decades, much of the discourse concerning blow-out fractures centered on the criteria and timing for surgical intervention. Consensus was elusive for several reasons. Enophthalmos could be repaired more effectively with early surgery, yet it was often not clinically manifest until several weeks after the injury. Diplopia might result from nerve damage or from muscle edema, hematoma, or incarceration, and forced duction testing was often equivocal and nondiagnostic. Even in cases with unquestionable incarceration, in which early release of entrapped tissues appeared to be indicated, traumatic diplopia sometimes resolved spontaneously.[8] Furthermore, a substantial number of patients had persistent diplopia despite surgical intervention.[8-11] Finally, the potential complications of surgery, including vision loss,[12] were to be considered. Treatment recommendations covered a wide spectrum, which varied from early intervention for all fractures[7,9,13,14] to expectant observation of all fractures for 4 to 6 months, with delayed repair of late enophthalmos and/or strabismus surgery for persistent diplopia.[15]

The advent of computed tomography (CT) in the late 1970s and early 1980s was a major advance in the depiction of orbital fractures.[16-18] The ability to differentiate fracture size and extent narrowed the broadly divergent therapeutic approaches to a fairly uniform protocol, which remains in widespread use.[19-23] If CT scans demonstrate a large blow-out fracture, enophthalmos is anticipated (even if it is initially masked by orbital edema or hematoma), and surgery is usually performed within 2 weeks of the injury. If CT scans demonstrate a small fracture that is unlikely to alter orbital volume, surgical decisions are based on ocular motility. In the absence of diplopia, small fractures are rarely repaired. In the presence of diplopia, examinations are repeated for up to 2 weeks, and surgery is generally advised for patients whose symptoms remain clinically significant during this period.

Unfortunately, adherence to this widely accepted therapeutic protocol has not assured a successful result. Although volumetric considerations have been generally well-served, ocular motility outcomes have been less

than ideal.[8-11,16,24,25] In an important study, Emery and associates[8] described 49 patients who underwent surgical repair of blow-out fractures, 43 within the first 2 weeks of the injury. Twenty-four of the 49 patients (49%) had residual diplopia at least 6 months following surgery.

The pathophysiology of orbital fractures was further clarified by Koornneef's demonstration of a complex network of fibrous septa that functionally unites the periosteum of the orbital floor, the inferior fibrofatty tissues, and the sheaths of the inferior rectus and oblique muscles.[26-28] Ocular motility may be limited by displacement of this functional unit or by incarceration of any of its components between fracture fragments.[29-31]

While the relative merits of early versus delayed intervention for *all* blow-out fractures have been widely debated, few studies have identified specific preoperative criteria that might predict the postoperative course, and perhaps suggest a more individualized therapeutic approach. On the basis of current pathogenetic concepts, we reasoned that the inferior fibrofatty-muscular complex is damaged to varying degrees as it is driven between bone fragments. Subsequent intrinsic fibrosis and contraction can tether globe movement, despite complete reduction of this herniated tissue from the fracture site. We postulated that the extent of soft-tissue damage might be estimated from high-quality orbital images. In this study, we analyzed the preoperative CT findings of patients with orbital blow-out fractures, classifying the injuries according to relationships between fracture fragments and soft tissue. Postoperative ocular motility was determined with binocular visual fields (BVFs), and an effort was made to correlate the two findings.

## METHODS

We reviewed the records of patients with blow-out fractures of the orbital floor who underwent surgical repair by a single surgeon (G.J.H.) at the Eye Institute of the Medical College of Wisconsin. Only patients who met the following criteria were included: retrievable coronal CT scans; fractures of the orbital floor, with or without medial wall extension, with intact orbital rims; preoperative diplopia; complete surgical release of entrapped soft tissue; and postoperative ocular motility outcomes documented with Goldmann binocular visual fields (BVFs), usually 4 to 10 weeks after surgery. Hospital policy of CT scan disposal after 5 years and variable compliance of the trauma patient population with follow-up visits were limiting factors. Thirty patients met all criteria. Review of the CT scans and assessment of the BVFs were each performed by different examiners among the authors.

On the basis of the bone fragment/soft-tissue relationships in coronal CT scans, fractures were classified into three general types, and two sub-

types to reflect the severity of soft-tissue damage within each general category. "Trap-door" injuries, in which bone fragments appeared to have almost perfectly realigned, were designated type I fractures. In the I-A subtype, orbital soft tissue could not be definitely identified on the sinus side of the fracture line (Fig 1). In the I-B subtype, soft-tissue with the radiodensity of orbital fat was visible within the maxillary sinus, beyond the trap-door fracture (Figs 2 and 3).

In type II fractures, bone fragments were distracted, and soft tissue was displaced between them. In the II-A subtype, soft-tissue displacement was less than, or proportional to, bone-fragment distraction (Fig 4). In the II-B subtype, soft-tissue displacement was greater than, or disproportional to, bone-fragment distraction. Both width and depth of soft-tissue displacement were considered in making these judgments (Figs 5 and 6).

In type III fractures, displaced bone fragments surrounded displaced soft tissue in virtually all areas. In the III-A subtype, soft tissue and bone fragments were moderately displaced (Fig 7). In the III-B subtype, soft-tissue and bone fragments were markedly displaced (Fig 8).

Motility outcomes were quantified by measuring the vertical excursion in degrees in BVFs (Figs 9 and 10). We were interested in the relative rapidity, as well as the amount of movement recovery. Although the timing of BVFs varied, we sought some comparability by using fields that had

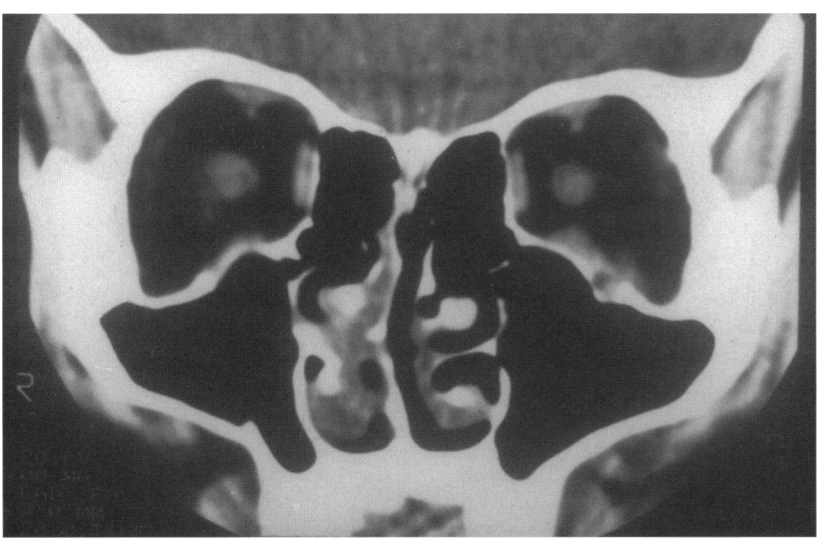

FIGURE 1

Type I-A fracture. Except for some discontinuity in infraorbital canal, left orbital floor appears to be aligned. Patient had severe restrictive diplopia, which was confirmed with forced duction testing and surgical finding of soft tissue pinched between impacted bone fragments.

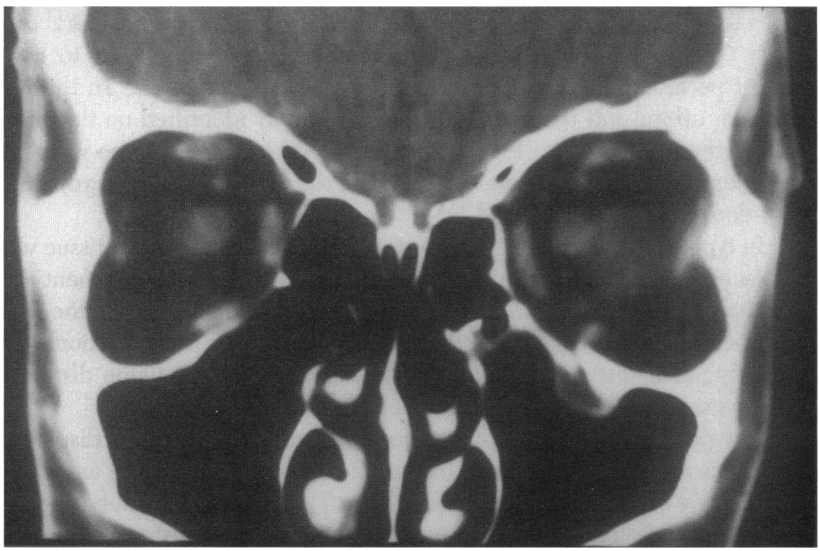

FIGURE 2
Type I-B fracture. Orbital fibrofatty tissue extends into the sinus through what appears to be a single fracture line separating otherwise rigid fragments. Note distortion of inferior rectus profile.

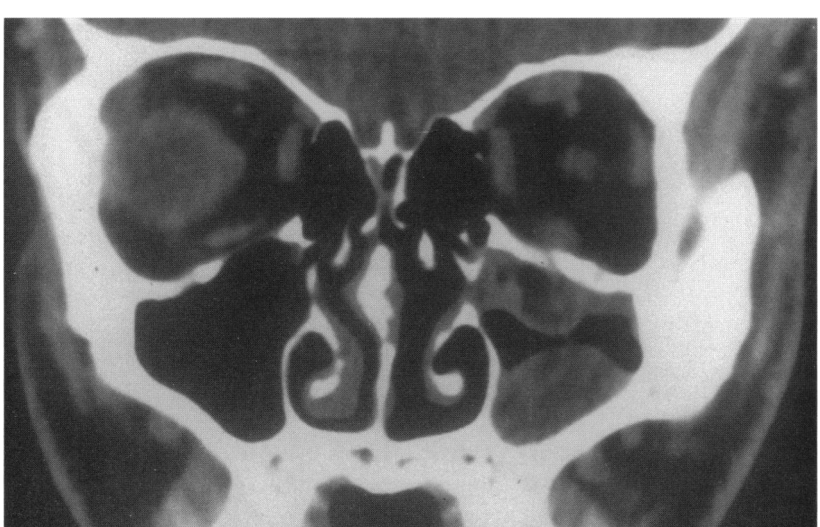

FIGURE 3
Type I-B fracture. Because displacement of bone fragments in trap-door fracture is transient, and therefore not evident in CT scans, radiologic interpretation can underrate its clinical importance. In this case, more anterior and posterior coronal sections showed a similar amount of soft-tissue herniation, suggesting significant compression-ischemia.

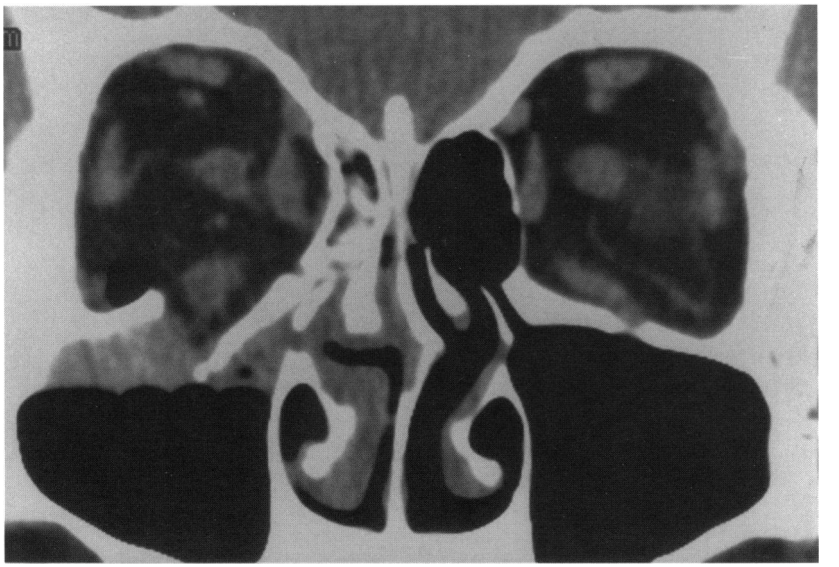

FIGURE 4
Type II-A fracture. Although floor fragments are widely separated, soft tissue is not displaced beyond fracture edges.

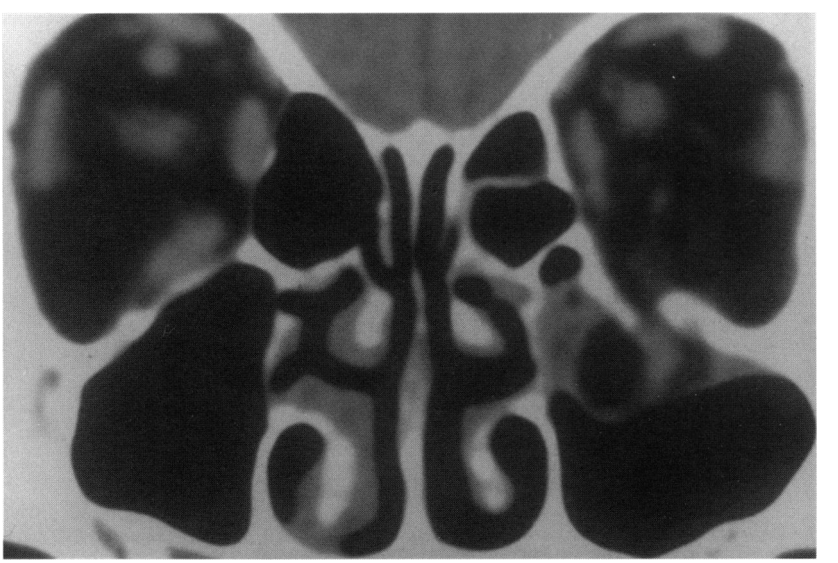

FIGURE 5
Type II-B fracture. Orbital soft-tissue herniation is broader than separation between bone fragments.

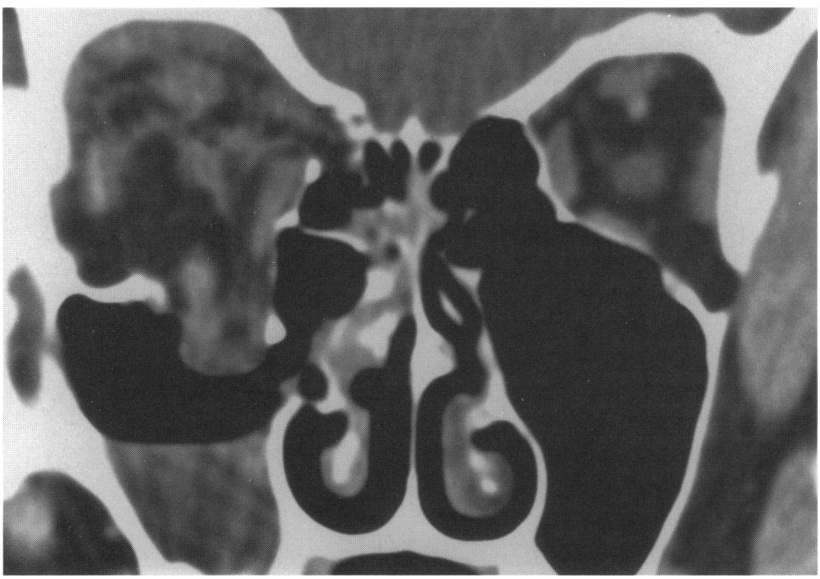

FIGURE 6
Type II-B fracture. Orbital soft tissue extends more deeply than lowest fracture fragments. With enough stretch, attenuation–ischemia may contribute to tissue trauma and subsequent intrinsic fibrosis.

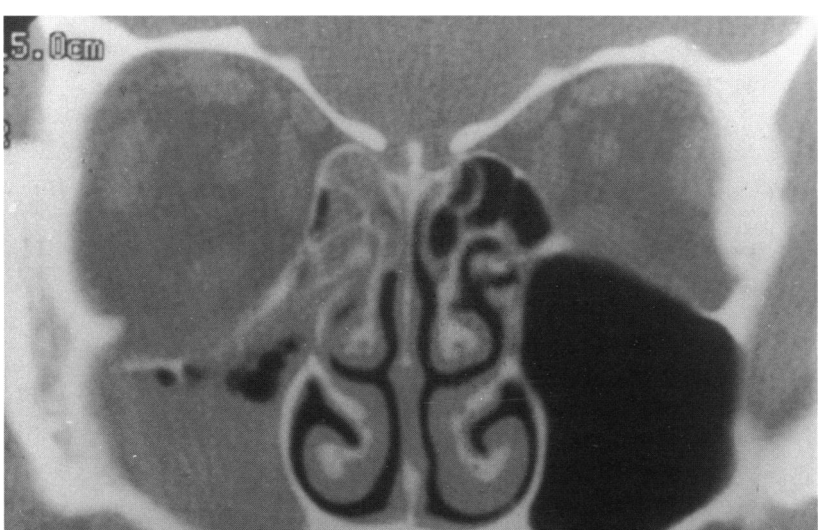


FIGURE 7
Type III-A fracture. Fibrofatty muscular complex does not extend between bone fragments, but is displaced enough to restrict globe movement. In this scan, head rotation exaggerates orbital floor asymmetry.

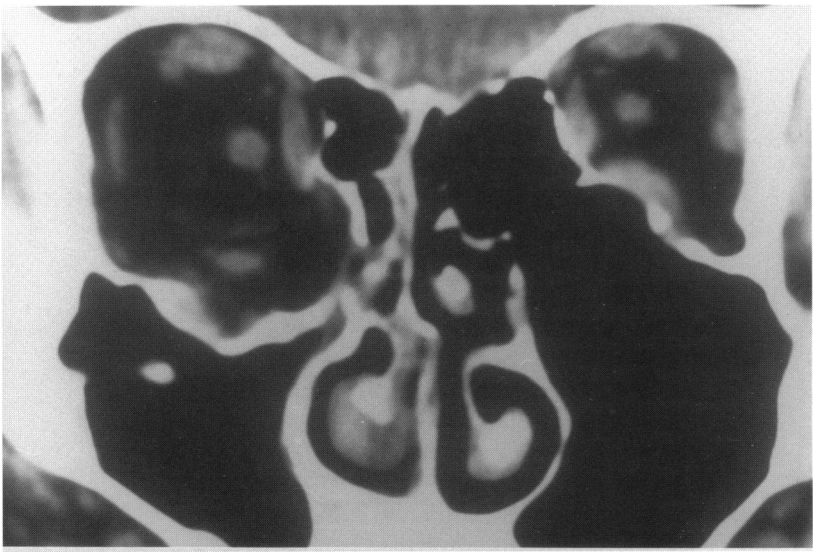

FIGURE 8
Type III-B fracture. This injury includes bony strut between maxillary and ethmoid sinuses, with considerable displacement of bone fragments and distortion of contiguous soft tissue.

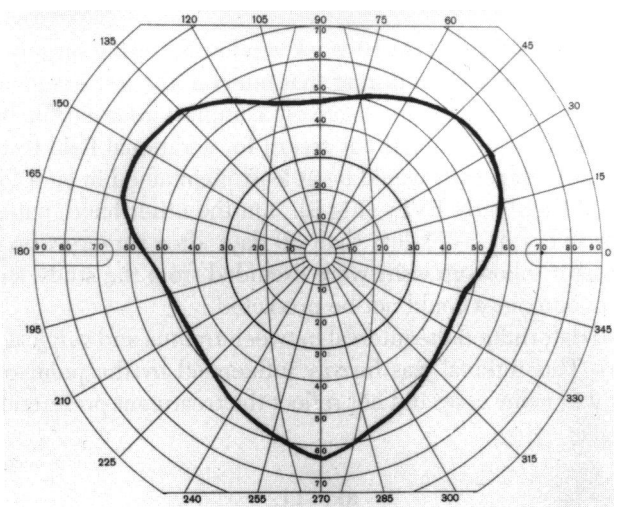


FIGURE 9
Single binocular visual field on postoperative day 20 of patient 28, whose CT scan is shown in Fig 4. Using the format of Feibel and Roper-Hall,[32] this represents a full single BVF, with a vertical excursion of 110°.

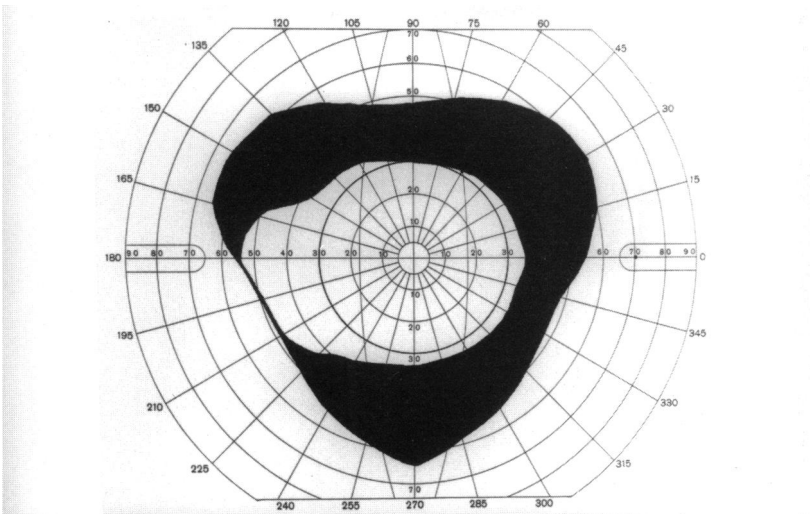

FIGURE 10
Binocular visual field on postoperative day 66 of patient 8 (Fig 5). Shaded area represents double vision. Vertical excursion in single BVF measures 64°.

been performed within a 4- to 10-week window following surgery. If more than 1 BVF was performed during that interval, the last examination was selected. Some patients with relatively complete recovery in BVFs less than 30 days after surgery did not return for additional field testing. We assumed that their results would be at least maintained in later follow-up, and we used their early BVFs (Fig 9). On the other hand, patients with minimal recovery in BVFs less than 30 days after surgery who failed to return for later follow-up visits were excluded from the study, since their longer-term outcomes could not be predicted.

We also determined the interval between trauma and surgical repair in each case. This interval was heavily influenced by the point of patient referral and in many cases did not reflect the treatment preferences of the authors.

## RESULTS

Patients are listed in the Table in order of degrees of vertical single BVF recovered after surgery. With the following exceptions, these figures relate to BVFs performed during a 30- to 69-day postoperative window. Patients 18, 23, 25, 26, 28, 29 and 30, with recovery of at least 94° of sin-

TABLE: POSTOPERATIVE OCULAR MOTILITY IN PATIENTS WITH ORBITAL BLOW-OUT FRACTURES

| PATIENT | VERTICAL EXCURSION WITHIN SINGLE BVF (°)* | SURGERY – BVF INTERVAL (d) | FRACTURE TYPE | TRAUMA-SURGERY INTERVAL (d) |
|---|---|---|---|---|
| 1 | 13 | 62 | II-B | 13 |
| 2 | 36 | 48 | II-B | 8 |
| 3 | 48 | 30 | I-A | 63 |
| 4 | 50 | 48 | II-B | 8 |
| 5 | 50 | 58 | II-B | 2920 |
| 6 | 55 | 39 | III-B | 180 |
| 7 | 60 | 41 | II-B | 8 |
| 8 | 64 | 66 | II-B | 14 |
| 9 | 65 | 57 | II-B | 1 |
| 10 | 77 | 69 | I-B | 53 |
| 11 | 77 | 180 | I-B | 9 |
| 12 | 80 | 60 | II-A | 53 |
| 13 | 85 | 48 | III-B | 44 |
| 14 | 85 | 40 | III-A | 67 |
| 15 | 86 | 4 | I-A | 19 |
| 16 | 88 | 54 | I-B | 11 |
| 17 | 92 | 48 | II-A | 11 |
| 18 | 94 | 21 | III-A | 26 |
| 19 | 98 | 48 | III-A | 9 |
| 20 | 100 | 59 | II-B | 2 |
| 21 | 105 | 68 | II-B | 4 |
| 22 | 110 | 34 | III-A | 11 |
| 23 | 110 | 20 | III-B | 45 |
| 24 | > 110 | 275 | III-A | 33 |
| 25 | > 110 | 8 | III-A | 6 |
| 26 | > 110 | 6 | II-A | 4 |
| 27 | > 110 | 47 | I-B | 4 |
| 28 | > 110 | 20 | II-A | 1 |
| 29 | > 110 | 7 | III-A | 6 |
| 30 | > 110 | 8 | II-A | 33 |

*Maximal vertical excursion of 110° conforms with the format of Feibel and Roper-Hall.[32] This figure was as high as 130° for some patients, possibly reflecting variations in nasal, eyelid, and eyebrow anatomy.

gle binocular vertical excursion less than 30 days after surgery, did not return for later examination. Patient 15 had recovered only 86° in a BVF 4 days after surgery. However, a follow-up BVF more than 2 years later showed no change. BVFs performed beyond the 30- to 69-day interval were utilized in 2 cases. Patient 24 had no diplopia and full ocular motility to clinical examination 36 days after surgery, but a BVF was not performed at that time. Patient 11 had postoperative BVFs performed at 16 days (18°) and 180 days (77°). At what point the excursion improved to 77°

is unknown, but it is clear that the results remained suboptimal even in late follow-up.

The Table lists fracture subtype, which was assigned to each patient by authors who analyzed coronal CT scans, but did not review the BVFs. Among the 30 patients, there were two I-A, four I-B, five II-A, nine II-B, seven III-A, and three III-B fractures. Subtotals were six I, 14 II, and ten III fractures; or 14 A and 16 B fractures. By dividing patients at the median of vertical excursion (between patients 15 and 16), a preponderance of type B fractures was noted among patients with a poorer motility outcome, and a preponderance of type A fractures was noted among patients with a more favorable motility outcome. Among the 15 patients with an outcome poorer than the median (86° or less), 4 patients (27%) had type A fractures; 11 patients (73%) had type B fractures. Among the 15 patients with an outcome better than the median (88° or more), 10 patients (67%) had type A fractures; 5 patients (33%) had type B fractures. These differences became more defined as analysis moved away from the median. For example, among 11 patients with less than 80° of single binocular vertical excursion, 10 (91%) had type B fractures; 1 (9%) had a type A fracture. Among 9 patients with more than 105° of single binocular vertical excursion, 7 (78%) had type A fractures; 2 (22%) had type B fractures.

The intervals between trauma and surgical repair of the fractures are listed in the Table. While type B fractures were generally associated with a poorer prognosis, and type A fractures with a better one, the timing of repair within each group is of interest. Among the 11 patients with type B fractures and less than the median result on BVFs, only 1 patient (9%) had surgical repair during the first week after injury. On the other hand, among the 5 patients with type B fractures and better than the median result on BVFs, 3 patients (60%) had surgical repair during the first week. Among the 10 patients with type A fractures and better than the median result, none had surgery more than 5 weeks after their injury. In contrast, among the 4 patients with type A fractures and less than the median motility outcome, 3 (75%) had surgery more than 7 weeks after trauma.

## DISCUSSION

A few studies have identified preoperative factors that affect the postoperative motility outcome of blow-out fractures. Hawes and Dortzbach[23] examined the interval between trauma and surgery in broad terms. Thirty-eight percent of patients who underwent surgery more than 2 months after the injury had residual diplopia, compared with only 7% who had surgery less than 2 months following trauma. Biesman and associates[25] noted the effect of fracture location. Fractures involving both the floor and medial wall were associated with almost twice the frequency of late

postoperative diplopia, compared with fractures limited to the orbital floor. The investigators suspected that the increased force necessary to create the more extensive fractures might be associated with greater soft-tissue damage. In that study, fracture location was determined from operative reports, and CT scans were not correlated with the postoperative course.

Other investigators have evaluated the prognostic significance of CT findings, but only during the preoperative interval.[33,34] Gilbard and colleagues[33] classified fractures according to the relationship between fracture edges and the inferior rectus profile on coronal CT scans. Muscles without direct contact were defined as "free"; muscles whose medial or lateral edge abutted bone were defined as "hooked"; and muscles whose medial and lateral edges were both in direct contact with bone were defined as "entrapped." One month following trauma, diplopia had resolved in all patients with free or hooked muscles, and diplopia had persisted in all patients with entrapped muscles. The last group then underwent surgery, but the postoperative motility findings were not described.

The proximity of muscle edges to bone fragments may well be a measure of anatomic disruption, and the markedly different radiodensities of muscle and fat facilitate this assessment. However, the inferior fibrofatty tissue, while less conspicuous than muscle in CT scans, seems to play a major role in both preoperative and postoperative ocular motility. The network of fascial septa described by Koornneef[26-28] provides anatomic continuity between the muscle sheaths, the extraconal orbital fat, and the periosteum adherent to the orbital floor. Following trauma, ocular motility may be impaired to varying degrees if this fibrofatty-muscular complex is displaced (Figs 4, 7, and 8), or if any of its components are pinched or incarcerated between bone fragments (Figs 1, 2, 3, 5, and 6).

If entrapment and displacement were the only determinants of late ocular motility, then surgical release and reduction of herniated orbital tissues should restore full movement. However, a poor motility outcome in many of our cases and in those of others[8-11,16,24,25] indicates other contributing factors.

Fujino and Sato[35] suggested that a forcible impact against the inferior orbital rim can cause the orbital floor to buckle and fracture without disrupting the rim. If a blow-out fracture results from this mechanism, then orbital tissue passively shifts with the separation of floor fragments, as it does in trimalar fractures. On the other hand, if the impact is sustained by the globe, as in the Smith and Regan[7] model, then increased orbital hydrostatic pressure forces the soft tissues through the floor, creating the fracture. As the inferior fibrofatty complex is driven between bone fragments, tissues are contused, sheared, and lacerated. If the rearrangement of bone fragments leaves the soft tissues compressed (Figs 2 and 3) or attenuated

(Figs 6 and 8), ischemia may then compound the insult. The predictable pathologic sequela of this combined tissue trauma is intrinsic fibrosis. Contracture of the inferior fibrofatty-muscular complex can then tether globe movement, despite its successful surgical release from the fracture site.

While upgaze may be most affected, the same process may limit full infraduction as well. Metz and colleagues[36] attributed downgaze limitation in this setting to neurogenic or myogenic paresis of the inferior rectus muscle. However, Saunders[37] demonstrated that mechanical restriction may limit both elevation and depression.

Many surgeons, including early investigators, recognized empirically the poor prognostic implications of tight soft-tissue incarceration and advocated early release of entrapped tissues.[6,7,9,13,29,30,38] Without high-quality orbital images, however, such injuries could not be reliably differentiated before surgery from others within the fracture spectrum. The CT scans in this series demonstrate the broad range of injuries subsumed by blow-out fractures. To relate the late clinical findings to the original injury, we have classified blow-out fractures in general terms (I, II, III), and suggested the degree of intrinsic soft-tissue damage within each general category (A, B). Although our system serves to emphasize the differences between extremes (eg, Figs 3 and 7), any classification is admittedly artificial and subjective. Fractures may be intermediate between subtypes, and individual cases may not be easily categorized.

Likewise, the motility outcomes constitute a continuum. In the very simplest cases, patients with CT-documented floor fractures may have no diplopia following the trauma (Fig 11). Whether these injuries result from a direct blow to the globe of only moderate intensity, or from an impact to the orbital rim, is unknown. However, this clinical picture is often seen with minimally displaced zygoma fractures that follow a blow to the malar eminence. Farther along the scale are patients with posttraumatic diplopia that resolves spontaneously. Before the advent of orbital imaging, these cases were all attributed to transient nerve damage or muscle hematoma and edema. Incarceration was viewed as an all-or-nothing phenomenon, which might be implied by forced duction testing, but was ultimately confirmed if motility did not normalize within several weeks of the injury. CT scans now suggest that some patients with spontaneous improvement may actually have lesser degrees of displacement or incarceration; the separation of bone fragments relative to the volume of herniated soft tissue may afford the fibrofatty-muscular complex enough stretch or resilience to restore full fusional amplitudes (Fig 12). The remaining patients in the soft-tissue damage continuum are those with posttraumatic diplopia that does not resolve spontaneously, but responds in varying degrees to the surgical release of herniated tissues. The broad and continuous spectrum

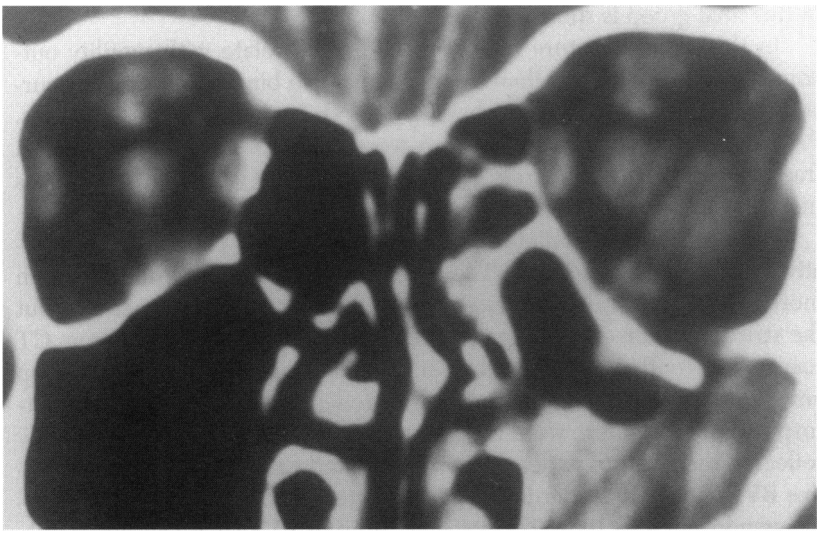

FIGURE 11
Blow-out fracture (II-A) in patient without posttraumatic diplopia. Rotation of patient's head exaggerates floor asymmetry, and displacement was actually minimal. Surgery was not performed.

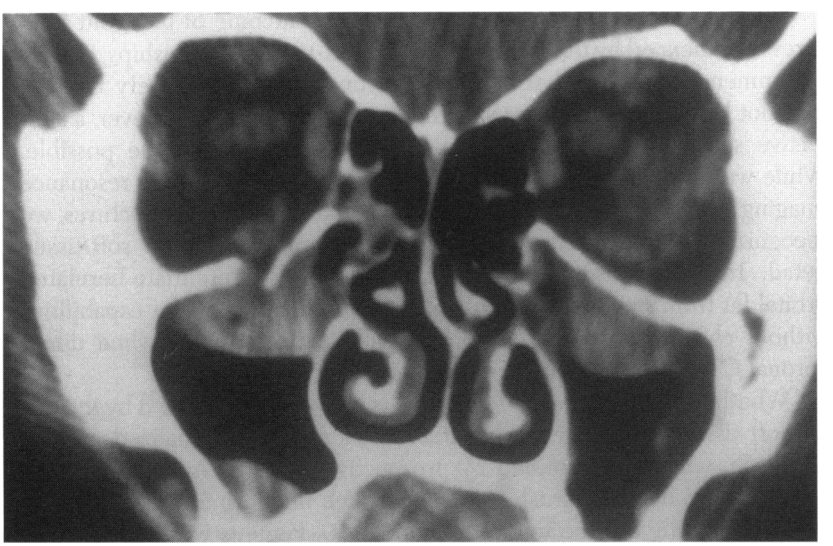

FIGURE 12
Blow-out fracture (II-A) in patient whose posttraumatic diplopia resolved within 2 weeks of injury. Although orbital fat extends through fracture site, amount of soft tissue and distraction of bone fragments are commensurate. Surgery was not performed.

of this final group is shown in the Table.

In this series, fracture type appeared to correlate with motility outcome. Patients with less than the median single binocular vertical excursion were 3 times as likely to have type B fractures as type A fractures. Patients with more than the median were twice as likely to have type A fractures as type B fractures. These differences increased as analysis moved away from the median in either direction.

We recognize the limitations of the present study. These include a relatively small patient cohort. The cases reported span a period in which more than 100 patients underwent surgical repair of orbital fractures, but the strict inclusion criteria limited the sample size. At our institution, CT scans were routinely discarded after 5 years. Compliance and follow-up among the trauma patient population were less than ideal. As in any retrospective series, there were variations in surgical timing, which primarily reflected the point of patient referral. Similarly, the timing of postoperative BVFs was not standardized. We did attempt to compare results within a window of 4 to 10 weeks, however. Earlier BVFs were accepted only if motility had substantially recovered by that point.

Our point of reference for postoperative diplopia differed from that of other investigators,[8,25] who chose a 6-month interval. Our goal was to compare results within a window that we considered a desirable target for the resolution of symptoms.

We believe that the postoperative motility outcome of blow-out fractures is influenced by their bone fragment/soft-tissue relationships, and we recommend careful analysis of direct coronal CT scans. Every fracture may not be easily categorized as either type I, II, or III. However, a subjective sense of soft-tissue distortion and damage should be possible. While we differ from investigators[24,39] who advocate magnetic resonance imaging (MRI) as the primary diagnostic modality for orbital fractures, we recognize that its superior contrast sensitivity provides better soft-tissue detail. In ambiguous cases, therefore, MRI might differentiate herniated orbital fat from sinus mucoperiosteal hematoma. Multiplanar capabilities without changing patient position also allow coronal MRI when direct coronal CT is precluded by cervical injuries or dental artifacts.

Whether the postoperative outcome is irreversibly ordained by the initial soft-tissue trauma, or can be altered by a more individualized surgical approach, will be determined by prospective studies. Anticipating that early reversal of tissue crush or severe stretch might limit intrinsic fibrosis, we currently use the following guidelines. Patients with type B fractures (I, II and III) have surgery on an urgent basis, preferably within 1 to 3 days of the injury. We suspect that severe compression-ischemia might be more damaging than attenuation-ischemia, and we favor the earliest intervention for I-B and II-B fractures (eg, Figs 2, 3, and 5). Other inves-

tigators have also recommended early surgery for such fractures.[24,40,41] Clearly, our retrospective findings are not conclusive, but among patients with type B fractures and better than the median recovery of ocular motility, 60% had surgery within the first week of the injury. Among those with type B fractures and a poorer outcome than the median, only 9% had surgery within the first week.

Only 2 patients in our series had I-A fractures (trap-door injuries without orbital tissue visible in the sinus) (Fig 1). One patient, with surgery 63 days following trauma, had recovered only 48° of single binocular vertical excursion 1 month after surgery. The other, with surgery 19 days after injury, had recovered 86° in BVFs at 4 days and 2 years after surgery. On the basis of these results, we believe that the force exerted between trap-door fragments can be severely damaging, even if the soft tissues do not extend beyond the fracture site. We currently treat I-A fractures in the same manner as type B fractures, with intervention one to 3 days after trauma. Because entrapment may be equivocal in CT scans, added weight is given to the clinical findings, including patient discomfort and restriction of active and passive ductions.

Initial decisions for patients with II-A and III-A fractures are based on volume concerns. Hawes and Dortzbach[23] suggested that fractures involving more than one half of the floor or at least 15 "fracture volume units" are likely to produce enophthalmos. In these cases (eg, Figs 4, 7), we believe that surgery need not be delayed beyond the first 7 days. This earlier timing might preempt the *extrinsic* fibrosis between fibrofatty tissue and bone fragments/sinus mucosa that demands additional surgical manipulation and trauma. Patients with II-A or III-A fractures whose displacement does not portend late enophthalmos (eg, Fig 12) are observed for spontaneous resolution of their diplopia for up to 2 weeks. Substantial improvement during that interval permits continued observation, and surgery may be totally avoided depending on the final outcome. All patients who are observed for any period are advised to perform binocular muscle exercises.

If the motility pattern is inconsistent with the CT fracture subtype, coexistent neurogenic causes are considered, such as trochlear palsy.[42]

With regard to surgical technique, entrapped soft tissues are liberated as carefully as possible, to avoid compounding the traumatic injury with iatrogenic damage. This may require gentle fragmentation and extraction of bone fragments, particularly in I-B and II-B fractures. The value of depot corticosteroids in limiting intrinsic fibrosis within the inferior orbit has not been proved, but has theoretical merit. Koornneef,[31] in describing a secondary procedure for the dissection of a scarred fibrofatty-muscular complex, suggested using hyaluronic acid to minimize re-adhesion. This approach might be considered in primary fracture repair, as well.

In summary, we have analyzed the CT findings of patients with orbital blow-out fractures, classifying the relationship between fracture fragments and soft tissue. We believe that soft tissue damage — in the form of contusion, shearing, laceration, crush and attenuation — can lead to intrinsic fibrosis and tethered movement, despite successful surgical reduction of the entrapped fibrofatty-muscular complex. The extent of this soft-tissue damage can be estimated from coronal CT scans and should be considered in the treatment plan.

## REFERENCES


1. Mackenzie W. *A Practical Treatise on the Diseases of the Eye*. London: Longman, Rees, Orme, Brown & Green; 1830.
2. Lang W. Traumatic enophthalmos with retention of perfect acuity of vision. *Trans Ophthalmol Soc UK* 1889;9:41-45.
3. Lagrange F. Les Fractures de l'orbite par projectiles de guerre. Paris: Masson SA; 1917. Reprinted in *Adv Ophthalmic Plast Reconstr Surg* 1987;6:137-144.
4. Pfeiffer RL. Traumatic enophthalmos. *Arch Ophthalmol* 1943;30:718-726.
5. King EF, Samuel E. Fractures of the orbit. *Trans Ophthalmol Soc UK* 1944;64:134-153.
6. Schjelderup H. Some considerations concerning traumatic diplopia. *Acta Ophthalmol* 1950;28:377-391.
7. Smith B, Regan WF. Blow-out fracture of the orbit: mechanism and correction of internal orbital fracture. *Am J Ophthalmol* 1957;44(6):733-739.
8. Emery JM, von Noorden GK, Schlernitzauer DA. Orbital floor fractures: Long-term follow-up of cases with and without surgical repair. *Trans Am Acad Ophthalmol Otolaryngol* 1971;75:802-812.
9. Converse JM, Smith B, Obear MF, et al. Orbital blowout fractures: A ten-year survey. *Plast Reconstr Surg* 1967;39:20-36.
10. Greenwald HS, Keeney AH, Shannon GM. A review of 128 patients with orbital fractures. *Am J Ophthalmol* 1974;78(4):655-664.
11. Cole HG, Smith B. Eye muscle imbalance: a complication of orbital floor fractures. *Am J Ophthalmol* 1963;55:930-935.
12. Nicholson DH, Guzak SV. Visual loss complicating repair of orbital floor fractures. *Arch Ophthalmol* 1971;86:369-375.
13. Smith B, Converse JM. Early treatment of orbital floor fractures. *Trans Am Acad Ophthalmol Otolaryngol* 1957;61:602-608.
14. Lerman S, Cramer LM. Blow-out fractures of the orbit. *Am J Ophthalmol* 1964;57:264-267.
15. Putterman AM, Stevens T, Urist MJ. Nonsurgical management of blow-out fractures of the orbital floor. *Am J Ophthalmol* 1974;77:232-239.
16. Milauskas AT. *Diagnosis and Management of Blowout Fractures of the Orbit*. Springfield, Ill: Charles C Thomas; 1969.
17. Manson PN, Iliff N. Management of blow-out fractures of the orbital floor. II. Early repair of selected injuries. *Surv Ophthalmol* 1990;35:280-292.
18. Tadmor R, New PFJ. Computed tomography of the orbit with special emphasis on coronal sections: Part I. Normal anatomy. *J Comput Assist Tomogr* 1978;2:24-34.
19. Dulley B, Fells P. Orbital blow-out fractures: To operate or to not operate, that is the question. *Br Orthop J* 1974;31:47-55.
20. Wilkins RB, Havins WE. Current treatment of blow-out fractures. *Ophthalmology*

1982;89(5):464-466.
21. Dortzbach RK, Elner VM. Which orbital floor blowout fractures need surgery? (Editorial) *Adv OphthalPlast Reconstr Surg* 1987;6:287-289.
22. Nathanson A, Matthis SP, Tengvar M. Diagnosis and treatment of fractures of the orbital floor. A ten-year retrospective study. *Acta Otolaryngol* 1992;492(suppl):28-32.
23. Hawes MJ, Dortzbach RK. Surgery on orbital floor fractures: Influence of time of repair and fracture size. *Ophthalmology* 1983;90:1066-1070.
24. Tonami H, Yamamoto I, Matsuda M, et al. Orbital fractures: Surface coil MR imaging. *Radiology* 1991;179:789-794.
25. Biesman BS, Hornblass A, Lisman R, et al. Diplopia after surgical repair of orbital floor fractures. *Ophthalm Plast Reconstr Surg* 1996;12:9-16.
26. Koornneef L. Orbital septa: Anatomy and function. *Ophthalmology* 1979;86:876-878.
27. Koornneef L. Anatomy and function of orbital septa. In: *Plastic Reconstructive Surgery of the Head and Neck*. Vol 11. New York: Grune & Stratton; 1981:130-138.
28. Koornneef L. *Sectional Anatomy of the Orbit*. Amsterdam: Aeolus Press; 1981.
29. Hotte HA. *Orbital Fractures*. Assen, Netherlands: Royal Vangorcum; 1970.
30. Koornneef L. Current concepts on the management of orbital blow-out fractures. *Ann Plast Surg* 1982;9:185-200.
31. Koornneef L, Zonneveld FW. The role of direct multiplanar high resolution CT in the assessment and management of orbital trauma. *Radiol Clin North Am* 1987;25:753-766.
32. Feibel RM, Roper-Hall G. Evaluation of the field of binocular single vision in incomitant strabismus. *Am J Ophthalmol* 1974;78:800-805.
33. Gilbard SM, Mafee MF, Lagouros PA, et al. Orbital blow-out fractures: The prognostic significance of computed tomography. *Ophthalmology* 1985;92:1523-1528.
34. Millman AL, Della Rocca RC, Spector S, et al. Steroids and orbital blowout fractures—a new systematic concept in medical management and surgical decision-making. *Adv Ophthalm Plast Reconstr Surg* 1987;6:291-300.
35. Fujino T, Sato TB. Mechanisms, tolerance limit curve and theoretical analysis in blowout fractures of two and three-dimensional orbital wall models. *Proceedings of the Third International Symposium on Orbital Disorders*. Amsterdam 1977;240-247.
36. Metz HS, Scott WE, Madson E, et al. Saccadic velocity and active force studies in blow-out fractures of the orbit. *Am J Ophthalmol* 1974;78:665-670.
37. Saunders RA. Incomitant vertical strabismus. *Arch Ophthalmol* 1984;102:1174-1177.
38. Smith B, Lisman RD, Simonton J, et al. Volkmann's contracture of the extraocular muscles following blowout fracture. *Plast Reconstr Surg* 1984;74(2):200-209.
39. Ilankovan V, Hadley D, Moos K, et al. A comparison of imaging techniques with surgical experience in orbital injuries. A prospective study. *J Cranio-Maxillo-Facial Surg* 1991;19(8):348-352.
40. Jackson A, Whitehouse RW. Low-dose computed tomographic imaging in orbital trauma. *Br J Radiol* 1993;66:655-661.
41. Boxer Wachler BS, Holds JB. The missing muscle syndrome in blowout fractures: an indication for urgent surgery. *Ophthal Plast Reconstr Surg* 1998;14:17-18.
42. Ruttum MS, Harris GJ. Orbital blowout fracture with ipselateral fourth nerve palsy. *Am J Ophthalmol* 1985;100:343-344.

**DISCUSSION**

DR JOHN C. MERRIAM. It is a pleasure to open the discussion of this interesting paper by Dr Harris and colleagues. The management of orbital injuries has interested physicians for a very long time. Perhaps the most

celebrated patient was Henry II, King of France (1547-1559), who was struck by a lance above the right eye in a tilting match. Although he was attended by the eminent surgeon Ambrose Paré (1517?-1590) and the eminent anatomist Andreas Vesalius (1514-1564), the King died after 11 days, presumably from sepsis.[1] Happily, none of the patients from Milwaukee met a similar fate. We owe the first good clinical description of orbital floor fracture to William Lang (1852-1937) of London, who in 1889 described a 13-year-old boy named Hugh who "was struck on the right eyebrow, as he was running in the street, by the shaft of a cart driven at a trot."[2] Hugh bled from the nose and the lids swelled shut, but after recovery, "it was noticed that the right eye was sunken in the orbit." Hugh also had diplopia. Lang suggested that fracture of a portion of the orbital wall might account for the boy's appearance; but without roentgenography, the fracture could not be confirmed.

Lang made other contributions to ophthalmology, notably on the use of an orbital implant after enucleation to prevent enophthalmos and on the advantages of a clear corneal incision for needling of the lens in high myopia![3,4] He was 1 of the 113 founding members of the Ophthalmological Society of the United Kingdom in 1880 and was an enthusiastic but apparently indifferent golfer. It is easy to imagine that he would enjoy the informal competition of the American Ophthalmological Society. In 1937 his obituary appeared in both the *British Medical Journal* and the *British Journal of Ophthalmology*.[3,4] The tone of the obituaries suggests that Lang was both well liked and respected.

In 1943, Raymond Pfeiffer[5] from the Eye Institute at Columbia reported 120 patients with facial fractures, of whom 53 developed enophthalmos. In 24 cases, enophthalmos occurred without fracture of the orbital margin; in each of these, roentgenography confirmed fracture of the orbital floor, just as Lang had surmised. After experimenting with a cadaver and an Irish hurling ball, Smith and Regan suggested in 1957 that such fractures be called "blow-out fractures," and this colorful term has become part of the language of trauma.[6]

Dr Harris and his colleagues have studied coronal computed tomography (CT) images of 30 blow-out fractures to determine if the CT appearance helps to predict postoperative motility, and they conclude that greater soft-tissue displacement is associated with restriction of vertical gaze, whatever the size of the fracture. They now prefer to repair fractures with these characteristics within 1 to 3 days of injury.

The authors recognize the limitations of a retrospective study of patients who frequently do not return after the acute injury has resolved and whose injuries defy precise categorization. Although they had records for more than 100 patients who had undergone repair of orbital fractures, only 30 records met their study criteria. One may assume that the great majority of their

patients are young males, but the authors have given us no information about sex, age, or the nature of the injuries. Nor have they described surgical complications, enophthalmos, or the number of patients who needed prisms or strabismus surgery. The number in each of the 6 subgroups ranged from 2 to 9 — too small a sample for statistical comparison.

However, their data suggest that preoperative CT helps to predict postoperative motility, and this information may be useful when counseling patients before operation. When the 30 patients are split into just 2 groups — those with better motility and those with lesser motility — the type I and II fractures are nearly evenly divided, but more of the larger fractures had better motility after surgery. When the 9 best and worst outcomes are compared, it appears that the greatest risk of poor motility is associated with small to moderate fractures with obvious soft-tissue dispersion. Of the 9 patients with motility less than 65°, 1 was type I-A, 7 were type II-B, and 1 was type III-B. Of the 9 patients with motility better than or equal to 110°, 1 was type I-B, 3 were type II-A, 4 were type III-A, and 1 was type III-B.

Patients with blow-out fractures but no diplopia might have served as a control group. Based on the authors' data, one would expect these patients to have relatively less tissue dispersion. If true, the predictive value of their schema would be confirmed. I also wonder if the authors found a correlation between preoperative motility, or the lack of it, and postoperative result.

Dr Harris now prefers to operate on high-risk type B fractures within 1 to 3 days of injury. Time may prove them right, but their data do not appear to justify this urgency. The interval between injury and operation for the 9 patients with the worst motility outcomes ranged from 1 to 2,920 days, with a mean of 357 days! However, this group includes 2 obvious ringers with intervals of 180 and 2,920 days. Without them, the mean interval between injury and operation is only 16.4 days. For the 9 best outcomes, the time from injury to surgery ranged from 1 to 45 days, with a mean of 15.9 days.

A randomized, prospective trial may be necessary to determine if early surgery improves motility for high-risk blow-out fractures. Because at most institutions oral surgeons, otolaryngologists, plastic surgeons, and ophthalmologists compete for these cases, we are not likely to have a definitive answer soon. I congratulate the authors for their thoughtful analysis of a difficult clinical problem.

**REFERENCES**

1. MacKenzie W. Diseases of the orbit. Section 1: Injuries of the Orbit. In: *A Practical Treatise on the Diseases of the Eye*. 4th ed. Philadelphia: Blanchard & Lea; 1855:4-70.
2. Lang W. Traumatic enophthalmos with retention of perfect acuity of vision. *Trans Ophthalmol Soc UK* 1889;9:41-45.

3. William Lang, FRCS. (Obituary) *Br Med J* 1937(2);189-191.
4. William Lang. (Obituary) *Br J Ophthalmol* 1937; 21:568-572.
5. Pfeiffer RL. Traumatic enophthalmos. *Arch Ophthalmol* 1943;30:718-726.
6. Smith B, Regan WF Jr. Blow-out fracture of the orbit: mechanism and correction of internal orbital fracture. *Am J Ophthalmol* 1957; 44:733-739.

EDWARD L. RAAB, MD. I enjoyed this paper very much. One of the criteria was diplopia. Possibly a forced duction test would have been a more definitive way to evaluate these patients. Also, I'd like to sharpen the indication for imaging these patients. I wonder whether imaging is necessary if the patient has diplopia and the forced duction test shows restriction of movement. Obviously, imaging may be indicated in cases without diplopia because of the problem of an orbital cosmetic defect at a later date.

GEORGE BARTLEY, MD. I'd like to congratulate the authors of this fine paper. Dr Harris mentioned the effect of edema in these fractures. I wonder if he treated any of these patients with steroids. Also, did the placement of implants at the time of surgery have any effect on the ocular motility?

JOHN FLYNN, MD. I, too, would like to add my congratulations to the authors and compare treatment options between two cities. In Miami, the key factor to determine whether you have an operation or not is your first encounter in the emergency room. If you encounter a plastic surgeon, an ENT surgeon, a maxillofacial surgeon, an oral surgeon, you have surgery regardless of any other criteria. With these patients, the only time the ophthalmologist is called is when the physician suspects damage to the eye itself. We then get called after the surgery if patients have persistent diplopia or retinopathy with decreased vision. My question is, in Milwaukee are you successful in having all these various surgeons consult you and accept what appears to me to be very excellent criteria for operating on these patients? Thank you.

JOSEPH FLANAGAN, MD. In general, we have been very conservative in treating patients with blow-out fractures and operated only one-third of patients with radiologic evidence of a blow out fracture. However, over the past 5 years we have become more aggressive in our approach and I agree with Dr Harris that earlier surgery may give better results. We do most of these surgeries under local anesthesia with monitored anesthesia care, and we can also perform forced duction testing at the time of the surgery. We also used preoperative, perioperative, and postoperative systemic steroids. For Type I fractures he described, we would use an absorbable implant and for Type II and III fractures, non-absorbable implants. For these procedures we generally use an inferior cul-de-sac approach. I would ask Dr

Harris if he noticed any variability in results depending upon whether or not steroids were used, and whether the results depended upon the type of implant that was utilized. Thank you.

MARSHALL PARKS, MD. I too, did appreciate this paper. Many of the orbital fracture patients are unable to fuse in the primary position and must assume a torticollis to achieve fusion.

My question regards the amplitude of vertical version or duction described to judge success or failure of your initial orbital fracture surgery. Were the amplitudes measured according to the total vertical duction of the involved eye or according to maximal vertical version that permitted fusion?

May I submit that, in my opinion, the most meaningful postoperative measurement to evaluate success or failure is the supraversion and infraversion amplitudes that permit fusion, without torticollis, in the primary position. Therefore, supraduction improvement of the involved eye does not assess the criteria for success. If I correctly followed your presentation, I think this is what you did. Granted you did, I commend you for using the plotted binocular visual field as your criteria for success or failure rather than the amplitude of the supraduction or infraduction of the involved eye. However, the question remains, did some of the patients you classified as successful have to introduce a torticollis to fuse?

GERALD HARRIS, MD. I would like to thank all of the discussants for their careful analyses. I will try to address all of their questions, beginning in reverse order. Dr Parks, all of the vertical excursions in the study represent measurements of binocular fusion within binocular visual fields.

Drs Flanagan and Bartley, in our study we excluded any patients who were treated with corticosteroids in order to maintain a homogeneous group. We currently treat some patients with oral corticosteroids preoperatively, or during the period of initial observation. This treatment is intended to reduce the initial edema. However, we do not think it has the same effect as a small amount of depot corticosteroid in limiting intrinsic fibrosis as the pathological process evolves during the 4 to 6 weeks following injury. With regard to the surgical approach, we use a lower fornix incision almost exclusively. Our patients have general anesthesia. In virtually all cases, we use a very thin Nylamid implant, an extruded nylon-type material. Its' smooth surface may result in less adhesion compared with porous materials.

Dr Flynn correctly identified the ongoing battle between ophthalmic surgeons and other specialists who treat these injuries. I must confess that we are probably no more successful in Milwaukee than he is in Miami in converting the other specialties, but we continue to try.

Dr Raab asked how we approach malar complex fractures and how they differ from blow-out fractures. Other surgical specialties often lump malar fractures with blow-out fractures, but I think the mechanisms and appropriate management differ. Malar complex fractures generally involve a direct impact over the malar eminence. The inferior and lateral orbital rims fracture, and the zygoma is displaced. Because it is united to the bone by the periosteum, the fibrofatty-muscular complex is translocated, and ocular motility may be affected. However, this effect differs from the extrusion of soft tissue through a minimally displaced blow-out fracture that results in late intrinsic fibrosis. All patients had forced duction testing, either before or during surgery. As might be expected, patients with lesser degrees of soft tissue distortion on CT scanning had less clearly abnormal forced duction testing. Although I agree with Dr Raab that the physical findings can be very revealing, I find the CT scan to be very useful in the fracture repair, and am not quite ready to forego it.

Finally, I will address Dr Merriam's several points. We divided our cases not by fracture size, but by bone fragment configuration and soft tissue distortion. When the extent of fracture lines is considered, size and displacement are not equivalent. We did not provide epidemiological data, because this information has been repeatedly reported in the past, and was not relevant to our objective. Dr Merriam asked whether we considered categorizing patients without diplopia. We did review the scans of patients in this category, but the numbers were limited, because patients without diplopia or enophthalmos are rarely referred. Among the few patients without diplopia that we did review, there was definitely less soft tissue distortion compared with the cases that were included in the study. We feel that the bone fragment/soft tissue relationship represents a continuum. At one end are fractures without diplopia. Moving on, are fractures with diplopia that resolves spontaneously. Finally, we have fractures with diplopia that responds to a variable degree to surgical intervention, as included in our study. We excluded patients without diplopia from our study, because they did not require surgery. We attempted to compare only two variables: preoperative CT findings and postoperative motility. As much as possible, surgery was a "constant," which is why we limited cases to those performed by a single surgeon and required that all tissues be completely extricated from the surgical site. Dr Merriam wondered whether we found a correlation between preoperative and postoperative motility. We did not specifically look for such a correlation because we believe these reflect different factors. The preoperative factors include displacement of the fibrofatty-muscular complex, entrapment of any of the components, and to a lesser degree, edema and hematoma. Assuming complete release and reduction of entrapped and herniated orbital tissues, the postoperative motility reflects intrinsic fibrosis within the soft tissue

complex. Rarely, neurogenic paresis may be a common factor before and after surgery. That said, we did note anecdotally that patients with very poor outcomes had, on retrospective review of their preoperative findings, very limited movement preoperatively. Finally, Dr Merriam notes that the mean intervals between injury and operation were similar, at 16 days, for the best and worst outcomes, and concluded that an urgent approach is not justified. If only surgical timing is considered, fracture type and its influence on the outcome are ignored. Eight of 9 patients with the worst outcomes had greater soft tissue disruption (type B fractures). Seven of 9 with the best outcomes had lesser soft tissue disruption (type A fractures). That is precisely why we looked at timing within the *A* group and within the *B* group, and did note differences. Dr Merriam, perhaps inadvertently, makes our point. If all patients with blow-out fractures have surgery at 16 days, similar to the conventional approach of waiting up to 2 weeks, there will be marked variation in the clinical outcome. Sixteen days may be too long for type *B* fractures.

Thank you again for your attention.