Orbital and Naso-Orbital Fractures

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Orbital fractures may occur independently or may be associated with other facial fractures. One should obtain specific details as to where and how the accident occurred, and if there was a period of unconsciousness, loss of vision, or diplopia. A history of prior medical or surgical treatment is also important.

Prior to the primary treatment of the fracture, an ophthalmologic examination should be done routinely to verify visual acuity and check the possibility of intraocular or corneal injury. Obviously, if the patient is unconscious, the examination is limited. In the conscious patient, a delay of the operative procedure for a few days will allow the edema to subside and permit a more thorough examination. Facial bone fractures are not surgical emergencies unless the airway is obstructed and a tracheotomy is indicated. Treatment of an orbital fracture should not be delayed more than necessary, however, as fibrosis of the entrapped extraocular musculature, loss of orbital fat, and organization of the fracture occur rapidly and complicate the treatment.

When feasible, extraocular muscle movements should be checked at far and near distance and in all cardinal fields, as ocular muscle imbalance is often associated with fractures. A fundus examination often uncovers congenital anomalies or macula or optic nerve injuries that may limit the postoperative result because of diminution or loss of vision and may reduce the probability of satisfactory extraocular muscle fusion.

In trauma involving the medial canthus, when there is an associated naso-orbital fracture, an evaluation of the lacrimal system should be obtained, if feasible, depending on the magnitude of the injury. Irrigation of the secretory system showing drainage of the fluid into the nose demonstrates that the continuity of the lacrimal apparatus has not been interrupted.

Orbital fractures occur in association with zygomaticomaxillary, naso-orbital, and high maxillary (Le Fort III) fractures, as well as in pyramidal (Le Fort II) fractures, where the line of fracture traverses the orbital floor (see Chapter 24). The backward displacement of the fractured thick inferior orbital rim comminutes the thinner portion of the orbital floor. The downward displacement of the zygoma results in a separation at the frontozygomatic junction and a lowering of the orbital floor.

Orbital fractures complicated by diplopia are frequently associated with midfacial fractures. McCoy, Chandler, Magnan, Moore, and Siemsen (1962) found an incidence of 15 per cent of ocular complications is a series of 855 patients with facial fractures. Morgan, Madan, and Bergerot (1972), in a review of 300 cases of midfacial fractures, found persistent diplopia in 11 per cent of the patients.
The Orbit: Anatomical Considerations

The orbits are paired bony structures separated in the midline by the interorbital space. The interorbital space, the portion of the nasal cavity situated between the orbits, is delimited above by the floor of the anterior cranial fossa, formed in this portion by the roof of each ethmoid sinus laterally and by the cribriform plate medially. The orbits are situated immediately below the floor of the anterior cranial fossa, a portion of the fossa being formed by the roofs of the orbits (see Chapter 56).

The orbital contents are protected by strong bony abutments: the nasal bones, the nasal spine of the frontal bone, and the frontal processes of the maxilla medially; the supraorbital arch of the frontal bone above; the frontal process of the zygoma and zygomatic process of the frontal bone laterally; and the thick infraorbital rim formed by the zygoma and maxilla inferiorly.

The Skeletal Anatomy of the Orbit. The skeletal components of the orbital cavity are the frontal bone, the lesser and greater wings of the sphenoid, the zygoma, the maxilla, the lacrimal bone, and the ethmoid.

The bony orbit has been described as cone-shaped or pyramidal in shape. Both of these analogies are somewhat inaccurate. The widest orbital diameter is not located at the orbital rim but approximately 1.5 cm within the orbital cavity. The medial wall has a quadrangular rather than a triangular configuration. The optic foramen lies on a medial and slightly superior plane in the apex of the orbit. In children, the orbital floor is situated at a lower level in relation to the orbital rim because the maxillary sinus has not reached full development.

The floor of the orbit, a frequent site of fracture, has no sharp line of demarcation with the medial wall because the orbital floor tilts upward in its medial aspect, while the lower portion of the medial wall has a progressively lateral inclination. The floor is separated from the lateral wall by the inferior orbital (sphenomaxillary) fissure. The floor of the orbit (the roof of the maxillary sinus) is composed mainly of the orbital plate of the maxilla, a paper-thin structure medial to the infraorbital groove, and partly by the zygomatic bone anterior to the inferior orbital fissure. The infraorbital groove (or canal) traverses the floor of the orbit beginning at about the middle of the inferior orbital fissure. Anteriorly it penetrates the thick inferior orbital rim as the infraorbital canal, which opens on the anterior surface of the maxilla as the infraorbital foramen.

The orbital floor has a general upward inclination; the anterior portion is concave and the posterior portion is convex. In the blowout fracture mechanism, the force transmitted to the orbital contents tends to fracture the floor at its weakest point and may be nature's way of protecting the ocular globe from rupture by decompressing the orbit. In the posterior portion of this inclined plane there is an area of thin bone. This "weak area" represents the...
thinnest bone of the orbit; its medial extension is the lamina papyracea of the ethmoid, a portion of the medial orbital wall which, as its name implies, is a plate of bone of paperlike thinness. The medial half of the orbital floor is also weakened by the canal (or groove) for the passage of the infraorbital nerve.

The inferior oblique muscle arises from the medial aspect of the orbital floor, lateral to the lacrimal groove, near the anterior margin of the orbit.

The *medial wall*, reinforced anteriorly by the frontal process of the maxilla, is relatively fragile and is formed from the frontal bone, the lacrimal bone, the lamina papyracea of the ethmoid, and part of the lesser wing of the sphenoid around the optic foramen. The lamina papyracea is the largest component and accounts for the structural weakness of the medial wall. The lesser wing of the sphenoid and the optic foramen are posterior to the lamina papyracea. Thus the optic foramen is located close to the posterior portion of the ethmoid sinus, not at the apex of the orbit. Consequently, in severe fractures involving the medial wall in its posterior portion, the optic nerve can be injured.

The groove for the lacrimal sac is a broad vertical fossa lying partly on the anterior aspect of the lacrimal bone and partly in the frontal process of the maxilla; the anterior and posterior margins of the lacrimal groove form the respective lacrimal crests. The groove is continuous with the nasolacrimal duct at the junction of the floor and medial wall of the orbit, passing down into the inferior meatus of the nose.

Between the roof and medial wall of the orbit are the anterior and posterior ethmoidal foramina which lead into canals communicating with the medial part of the anterior cranial fossa.

The *lateral wall* is relatively stout in its anterior portion. It is formed by the greater wing of the sphenoid, the frontal process of the zygomatic bone, and the lesser wing of the zygomatic bone, and the lesser wing of the sphenoid lateral to the optic foramen. The superior orbital fissure is a cleft which runs forward and upward from the apex between the roof and lateral wall. The fissure, which separates the greater and lesser wings of the sphenoid, gives passage to the three motor nerves to the extraocular muscles of the orbit and leads back into the middle cranial fossa. The lateral wall of the orbit is situated in an anterolateral and posterior medial plane. It is related to the temporal fossa; posteriorly a small part of the wall lies between the orbit and the middle cranial fossa and temporal lobe of the brain. Between the floor and lateral wall of the orbit is the inferior orbital fissure which communicates with the infratemporal fossa.

The *roof of the orbit* is composed mainly of the orbital plate of the frontal bone, but posteriorly it receives a minor contribution from the lesser wing of the sphenoid. The fossa lodging the lacrimal gland is a depression situated along the anterior and lateral aspect under shelter of the zygomatic process of the frontal bone. The anterior portion of the roof can be invaded by the supraorbital extension of the frontal sinus or by an extension of the ethmoid sinus, a frontoethmoidal cell (see also Chapter 56). The roof separates the orbit from the anterior cranial fossa and from the middle cranial fossa on the posterolateral aspect.
Often consisting of brittle bone, the orbital roof varies in thickness and may be quite thin in its medial portion.

The supratrochlear and supraorbital nerves and, more medially, the trochlea of the superior oblique muscle are located along the superior rim of the orbit. The tendon of the superior oblique muscle functions in a cartilaginous pulley or trochlea, which is fixed by ligamentous fibers immediately behind the superomedial angle of the orbital margin. Fractures involving the superior rim of the orbit may result in compression of the supraorbital nerve, with consequent anesthesia of its area of distribution. Diplopia may also result from injury to the pulley of the superior oblique muscle, thus affecting the balance of the extraocular musculature.

The Orbital Fat and the Ocular Globe. The ocular globe is surrounded by a cushion of orbital fat within the orbital cavity. The ocular globe occupies only the anterior half of the orbital cavity; the posterior half of the orbital cavity is filled with orbital fat, muscles, vessels, and nerves supplying the ocular globe. The two halves of the orbital cavity, anterior and posterior, are separated by Tenon's capsule, which subdivides the orbital cavity into an anterior or precapsular segment and a posterior or retrocapsular segment.

The Septum Orbitale. The orbital contents are maintained in position by the septum orbitale (orbital septum), a fascia inserted on the inner aspect of the rim of the orbit. The septum orbitale attaches to and blends with the levator aponeurosis in the upper eyelid for a distance of a few millimeters above the upper border of the tarsus; in the lower eyelid, the septum orbitale is attached to the lower border of the tarsus.

The Periorbita. The periosteum lining the periphery of the orbit is known as the periorbita. The periorbita is continuous with the dura at those sites where the orbit communicates with the cranial cavity, eg, the optic foramen, the superior orbital fissure, and the anterior and posterior ethmoidal canals.

The Optic Foramen and the Optic Canal. The optic foramen is situated at the junction of the lateral and medial walls of the orbit. The foramen is not located on a horizontal plane with the orbital floor but above it.

The optic canal, 4 to 10 mm in length, is the passage through which the optic nerve and ophthalmic artery pass from an intracranial to an intraorbital position. The canal is framed medially by the body of the sphenoid and laterally by the lesser wing and is thus in close approximation to the sphenoid sinus and the posterior ethmoidal cells.

Blowout Fractures of the Floor of the Orbit

A blowout fracture is caused by a sudden increase in the intraorbital pressure, resulting from the application of a traumatic force to the soft tissues of the orbit (Converse and Smith, 1957, 1960). The fracture is often complicated by diplopia, which is caused by a vertical muscle imbalance secondary to entrapment of the orbital contents which may include the inferior rectus and inferior oblique muscles and the surrounding fascial expansions into the dehiscence in the orbital floor. The escape of orbital fat through the blowout dehiscences is a major cause of enophthalmos.
One of the first descriptions of a blowout fracture was given by King and Samuel in 1944: "We would like to add one other type of fracture of great importance, which is not infrequent. In this there is a downward displacement of part of the orbital floor, unassociated with any damage to the margin of the orbit surrounding the facial bones. The cause of such a fracture is difficult to visualize. The most ready explanation is trauma transmitted through the eye to the orbital floor".

After the application of a traumatizing force over the orbital contents by a nonpenetrating object, such as a tennis ball or the human fist, the orbital contents are forced backward into the narrower portion of the orbit (Table 25-1). The increased intraorbital pressure thus exerted causes a blowout at the weakest area of the orbital floor without fracturing the orbital rim. This type of fracture may be referred to as "pure" blowout fracture (Table 25-2) (Converse and Smith, 1957). The strong rim of the orbit protects against objects with a radius of curvature greater than 5 cm; an object having a curvature of less than 5 cm may penetrate this protective barrier and damage the globe. Such objects are golf balls, hockey pucks, and the tip of a football. Damage to the globe leading to blindness may occur.

Table 25-1. Etiologic Factors in 100 Blowout Fractures

<table>
<thead>
<tr>
<th>Factor</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Automobile</td>
<td>49</td>
</tr>
<tr>
<td>Human fist</td>
<td>18</td>
</tr>
<tr>
<td>Human elbow</td>
<td>4</td>
</tr>
<tr>
<td>Wooden plank</td>
<td>1</td>
</tr>
<tr>
<td>Ball</td>
<td>5</td>
</tr>
<tr>
<td>Snowball</td>
<td>2</td>
</tr>
<tr>
<td>Ski pole</td>
<td>2</td>
</tr>
<tr>
<td>Edge of table</td>
<td>1</td>
</tr>
<tr>
<td>Hit by blunt object</td>
<td>1</td>
</tr>
<tr>
<td>Shoe kick</td>
<td>2</td>
</tr>
<tr>
<td>Steel bar</td>
<td>1</td>
</tr>
<tr>
<td>Hit by machinery</td>
<td>2</td>
</tr>
<tr>
<td>Boxing glove</td>
<td>1</td>
</tr>
<tr>
<td>Mop handle</td>
<td>1</td>
</tr>
<tr>
<td>Human buttock</td>
<td>1</td>
</tr>
<tr>
<td>Airplane accident</td>
<td>1</td>
</tr>
<tr>
<td>Water ski accident</td>
<td>1</td>
</tr>
<tr>
<td>Ice bank</td>
<td>1</td>
</tr>
<tr>
<td>Fall on face</td>
<td>4</td>
</tr>
<tr>
<td>Iatrogenic (surgical)</td>
<td>1</td>
</tr>
<tr>
<td>Military casualty (shell fragment)</td>
<td>1</td>
</tr>
</tbody>
</table>

A champagne bottle cork may damage the globe, because its radius is less than 5 cm. However, its lesser propulsive force also makes it a frequent cause of blowout fracture. Such an accident has marred many festive occasions.

Larger objects cannot enter the orbital opening and cause direct injury to the eye unless fragile spectacle lenses, which can shatter, are interposed between the traumatizing object and the eye.

Blowout fractures are more frequent on the right side than on the left because most individuals are right-handed and many blowout fractures are caused by the human fist. One should beware of the patient with a "black eye" who complains of visual disturbances: he may have suffered a blowout fracture of the orbital floor.

Mechanism of Production of a Blowout Fracture

Following the clinical findings of fracture of the orbital floor, entrapment of the structures, and diplopia without fracture of the rim (Converse and Smith, 1957), the
mechanism of production of the orbital blowout fracture was demonstrated experimentally. It was verified in a cadaver by duplicating a force similar to that which had produced a blowout fracture in one of our patients, who had been hit by a ball used in the Irish game of hurling (Smith and Regan, 1957). The dried out condition of the cadaver globe was corrected by the intraocular injection of normal saline solution. A hurling ball was placed over the closed lid of the cadaver orbit, and the ball was struck sharply with a mallet. A cracking sound was heard and was interpreted as having been caused by fracturing bone. An exploratory incision through the skin of the infraorbital margin and elevation of the orbital contents from the floor exposed a depressed comminuted fracture of the floor of the orbit. Exenteration of the orbital contents exposed the fracture in its entirety. There was also a comminuted fracture without displacement involving the lamina papyracea of the ethmoid bone. No fracture of the orbital rim or zygomatic arch was observed. This experiment duplicated almost exactly the injury sustained by several of our patients.

Table 25-2. Classification of Orbital Fractures

1. Orbital blowout fractures
   A. Pure blowout fractures: Fractures through the thin areas of the orbital floor, medial and lateral wall. The orbital rim is intact.
   B. Impure blowout fractures: Fractures associated with fracture of the adjacent facial bones. The thick orbital rim is fractured, and its backward displacement causes a comminution of the orbital floor; the posterior displacement of the orbital rim permits the traumatizing force to be applied against the orbital contents, which produces a superimposed blowout fracture.

2. Orbital fractures without blowout fracture
   A. Linear fractures, in upper maxillary and zygomatic fractures. These fractures are often uncomplicated from the standpoint of the orbit.
   B. Comminuted fractures of the orbital floor with prolapse of the orbital contents into the maxillary sinus is often associated with fracture of the midfacial bones.
   C. Fracture of the zygoma with frontozygomatic separation and downward displacement of the zygomatic portion of the orbital floor and of the lateral attachment of the suspensory ligament of Lockwood.

In a second experiment, the opposite orbit of the cadaver was exenterated. The soft tissue covering the orbital rim was excised to allow direct contact of the bony orbital rim with the surface of the hurling ball. Repeated blows of similar force with the hammer failed to fracture either the floor or the rim of the orbit. However, when the striking force was sufficiently increased, the orbital rim and orbital floor were comminuted simultaneously.

The mechanism of blowout fracture (an increased hydraulic pressure) has been questioned by a number of authors (Rény and Stricker, 1969; Fujino, 1974a, b). Rény and Stricker suggested the following hypothesis: the traumatic force striking the inferior orbital rim, which is sufficiently resilient to transmit the force to the orbital floor, fractures the latter while the rim rebounds without fracturing. Fujino (1974a, b), in a series of experiments in collaboration with engineers, demonstrated on a dried human skull, without orbital contents, that a brass striker weighing 120 g with a flat silicone plate, when dropped on the infraorbital
margin from a height of 15 cm, produced a linear fracture of the orbital floor. When the weight was dropped from a height of 20 cm, a punched-out fracture in the convex portion of the orbital floor was produced. Both of these fractures occurred without fracture of the orbital rim.

While Rény and Stricker's statement is obviously conjectural, Fujino's experiments, performed with mathematical precision, fail to demonstrate the most important clinical consequence of the blowout fracture: the entrapment. How does he explain the entrapment of the orbital contents without the increased intraorbital pressure forcing the tissues into the site of fracture? And how is it possible for the increased infraorbital hydraulic pressure to occur without contact of the orbital contents with the traumatizing object?

Whatever the theory of the mechanism of the blowout fracture, the fact remains that, in the presence of diplopia due to entrapment and inability to rotate the globe by means of the forced duction test, release of the entrapment is the only means of relieving the patient of the extraocular muscle imbalance and diplopia.

"Impure" Blowout Fracture

According to Garrett (1963), ocular-orbital damage occurs in approximately 10 per cent of all head injuries sustained in automobile accidents in the United States. In the typical automotive injury in which the passenger's face is projected against the dashboard, the thick orbital rim is fractured and backwardly displaced, resulting in an eggshell comminution of the orbital floor. The continuing momentum and the pressure against the orbital contents produce a superimposed blowout fracture. It is to this type of orbital fracture that the term "impure" blowout fracture, as suggested by Cramer, Tooze, and Lerman (1965), may be applied (Table 25-2). The human fist was the principal factor in the causation of pure blowout fractures in the series studied by Emery and his associated (1971). In the series studied by Converse and his associates (1967), automobile accidents caused the largest proportion of fractures; most of these were impure and complicated fractures (see Table 25-1). Impure blowout fractures often occur in association with midfacial fractures.

Pyramidal maxillary fractures (Le Fort II) and craniofacial disjunction (Le Fort III) are characterized by fracture lines extending through the orbital floor, findings which further contribute to the comminution of the bone produced by the fractured orbital rim. Massive prolapse of the floor into the maxillary sinus may occur.

Not All Orbital Floor Fractures Are Blowout Fractures! The term "blowout" fracture (Converse and Smith, 1957) has been used to refer to all orbital floor fractures. Not every fracture of the floor of the orbit is a blowout fracture (see Table 25-2). The term "blowout" fracture defines a particular type of fracture mechanism. Orbital floor fractures, as stated earlier in the text, occur in fractures of the zygoma and in upper maxillary fractures, and comminuted fractures of the floor may result in a downward sagging of the orbital contents into the maxillary sinus.

Blowout Fracture in Children. The maxillary sinus is undeveloped in children. As a result, the floor of the orbit is situated in a low position, dipping downward from the orbital
rim. Despite the resiliency and elasticity of young bones, blowout fractures are not infrequently seen in children. The mechanism of entrapment is similar to that seen in adults.

**Surgical Pathology**

**Diplopia.** Extraocular muscle imbalance and subjective diplopia are the result of deviation of the visual axes. The deviation has several causes: the major one is entrapment of the soft tissue structures in the blowout fracture area, a finding which explains the constancy of vertical muscle imbalance (Table 25-3). These soft tissue structures may include the inferior rectus muscle, inferior oblique muscle, suspensory ligament of Lockwood, periorbita, and fascial expansions.

A downward displacement of one ocular globe does not always result in diplopia. Massive comminution of the orbital floor will cause a downward displacement of the ocular globe without entrapment: there is no diplopia.

The most common site of the blowout fracture is the portion of the floor that is weakened by the infraorbital canal or groove. The inferior oblique muscle arises from the maxillary portion of the orbital floor lateral to the lacrimal groove, and the inferior rectus muscle is situated immediately above the infraorbital canal on the undersurface of the orbital contents. It is not surprising, therefore, that these two muscles are frequently involved in the blowout fracture. Absence of elasticity in the impounded inferior rectus muscle restricts rotation in the field of action of its antagonist, the superior rectus. Because the inferior rectus and the inferior oblique muscles are intimately connected at the point where the inferior oblique crosses beneath the inferior rectus, disturbance of function of the inferior oblique muscle is usually observed in blowout fractures. When the fracture is located lateral to the infraorbital groove or canal, the inferior rectus and inferior oblique muscles may not be involved. These variations in the site of the blowout fracture explain variations in the symptoms and clinical signs in these fractures.

**Nerve Injury.** Injury to the motor nerves of the inferior oblique and inferior rectus muscles must also be considered. The inferior oblique and the inferior rectus muscles are innervated by the inferior division of the third cranial nerve. The branch to the inferior rectus muscle passes along its upper surface to pierce it at the junction of the posterior and middle thirds of the muscle. The branch to the inferior oblique muscle runs along the lateral edge of the inferior rectus muscle, enters the ocular surface of the inferior oblique muscle, and is exposed to injury in blowout fractures. The relatively short course of the nerve to the inferior rectus muscle renders it less vulnerable to injury. Electromyographic examination will assist in determining whether nerve conduction has been interrupted by the injury.

**Other Causes of Diplopia.** Other causes of diplopia are injury to the third, fourth, or sixth cranial nerves, direct injury to the extraocular muscles, laceration of the muscle by bone fragments, disruption of the muscle attachments, hemorrhage into the muscle, or muscle imbalance caused by a change in orbital shape. Secondary muscle imbalance occurs when ptosis of the globe is associated with enophthalmos. Secondary muscle imbalance occurs when ptosis of the globe is associated with enophthalmos. Secondary deviations are commonly due to overaction of the yoke or conjugate muscles of the opposite eye. A factor to be remembered is that no extraocular muscle acts singularly to produce ocular movements.
Ocular rotation is the sum of the action, counteraction, and relaxation of 12 extraocular muscles (6 per ocular globe). It is not within the scope of this chapter to discuss the complex subject of the physiology of oculorotary muscles; only the aspects which pertain to the problem under discussion are explained.

Not only do paralytic deviations occur, but also trauma may often uncover tropias (constant imbalance) or phorias (latent imbalance occurring only with disruption of fusion) after temporary immobilization of the injured eye. These are usually horizontal in nature.

The typical blowout fracture is usually not seen in fracture-dislocation of the zygoma if the bone is displaced as a single fragment. In such a fracture, the site of impact is lateral to the orbital cavity and the orbital contents are usually not directly involved. In zygomatic fractures which are severely comminuted following an exceptionally strong impact, the lateral orbital wall and the rim and floor of the orbit may also be severely comminuted; the ocular globe is injured to the extent that enucleation may be required. A blowout with entrapment may have occurred, and release is necessary, even if the ocular globe is enucleated, to permit movement of the ocular prosthesis for purely esthetic reasons.

Enophthalmos. Enophthalmos, the second major complication of the blowout fracture, is the result of a number of causative factors. The first, the escape of fat from the orbital cavity, occurs when the periorbita is ruptured and the orbital fat escapes into the maxillary sinus. A second cause of enophthalmos is the retention of the ocular globe in a backward position when the structures are entrapped in the fracture site. A third causative factor is the enlargement of the orbital cavity resulting from the fracture and the downward displacement of the orbital floor; the orbital fat is distributed in a large cavity and is no longer sufficient in quantity to prevent a sinking in of the globe. A fourth factor is orbital fat necrosis resulting from pressure caused by orbital hematoma and a low grade inflammatory process.

The mechanism opposite to a blowout fracture is the "blown-in" fracture, in which penetration of bone fragments into the orbit diminishes the size of the orbital cavity, resulting in exophthalmos.

Enophthalmos, when it is conspicuous and particularly when orbital contents are downwardly displaced, results in a pseudoptosis of the upper eyelid, a deepening of the supratarsal fold, and a shortening of the horizontal dimension of the palpebral fissure. The deformity becomes more complex in orbital fractures associated with fractures of adjacent bones, especially a concomitant naso-orbital fracture.

Lower animals possess an orbital muscle which spans the floor of the orbit, covering the inferior orbital fissure; this muscle protrudes the eyeball for purposes of focusing vision. In man a vestige of this muscle has been designated as the orbital muscle. Some anatomists discount its importance; others claim that paralysis of the muscle is a factor in the production of enophthalmos in conditions such as Horner's syndrome, caused by paralysis of the third cranial nerve. Others feel that atrophic changes in the orbital fat due to injury of the sympathetic innervation may be responsible for the production of enophthalmos. Other factors which have been held responsible for the development of traumatic enophthalmos are dislocation of the trochlea of the superior oblique muscle, cicatricial contraction of retrobulbar tissue, and rupture of the orbital ligaments or fascial bands.
Enlargement of the orbital cavity as a factor in the causation of enophthalmos was suggested by Lang (1889): "I suggest that the injury may have produced a fracture and a depression of a portion of the orbital wall; the orbital fat would then be no longer sufficient in quantity to fill this enlarged postocular area without a sinking-in of the globe from atmospheric pressure and a resulting limitation in ocular movements."

The enlargement of the orbital cavity from the depression of the floor is a frequent factor in the production of enophthalmos. However, there may be no entrapment of the orbital structures and no diplopia (see Table 25-3).

**Enophthalmos in Orbital Fractures Without Blowout Fracture.** Fractures of the bones of the midfacial area often involve the orbital floor: Le Fort III fractures lines traverse the floor of the orbit; Le Fort II fractures also involve the orbital floor in its medial portion (see Table 25-2).

**Variations in Diplopia and Enophthalmos in Orbital Fractures.** Table 25-3 classifies the variations which occur in orbital fractures according to the anatomical damage suffered by the orbit and its contents.

**Examination and Diagnosis**

**Clinical Examination.** In the typical blowout fracture, the patient complains of diplopia in the primary position which increases in the upward gaze. The patient may not recognize diplopia early if the eye is temporarily closed by edema of the lids or dressings or if there is an intraocular injury. When examined during the first hours after the fracture, the ocular globe appears displaced backward and downward, and the supratarsal sulcus is deepened. Edema and hematoma may obscure such clinical findings when the patient is not examined during the first hours after injury. Ocular globe injury, eyelid damage, and lacerations and hematoma in the levator muscle or aponeurosis are not infrequently observed.

Diplopia is the most frequent complaint of the patient but is not necessarily an indication for operation; diplopia may be caused by hematoma and edema and may resolve spontaneously. Subjective diplopia is not an indication for surgical exploration.

When an object is held approximately two feet from the patient's eye and the patient is asked to look at the object, the affected eye is not able to rotate upward in the normal range as does the unaffected eye; restriction in rotation in other directions is also observed. The function of the inferior rectus and inferior oblique muscles is restricted by their entrapment in the floor of the orbit, and the superior rectus cannot rotate the globe because of the resistance offered by the short rein of the entrapped structures; when released, the globe is again able to rotate upward. In a child, the authors have observed nearly complete fixity of the globe.

When the infraorbital rim is fractured (impure blowout fracture), one may observe that the lower lid is shortened vertically and everted. The infraorbital rim is displaced backward and in its forced retreat carries with it the insertion of the septum orbitale. This finding accounts for the vertical shortening of the lower eyelid.
Indications for Surgical Intervention. Although diplopia is the most frequent complaint of the patient, it is not an indication for operation, as diplopia may be caused by hematoma, edema, and neurogenic factors.

Indications for operation are: (1) limitation of forced rotation of the eyeball, (2) radiologic evidence of fracture, and (3) enophthalmos.

1. Limitation of forced rotation of the eyeball. This test, known as the "traction test" or the "forced duction test", provides a means of differentiating entrapment of the inferior rectus muscle from weakness or paralysis of the superior rectus, and it is the pathognomonic sign of a blowout fracture of the floor of the orbit. A few drops of local anesthetic solution instilled into the conjunctival sac provide sufficient anesthesia to permit grasping the eyeball with forceps at the insertion of the inferior rectus muscle at a point of approximately 7 mm from the limbus.

2. Radiologic evidence of fracture. Radiologic diagnosis is essential, and tomography is of additional assistance in locating the area of the blowout fracture. Careful roentgen examination will show a variety of findings and define the location, size, and type of the fracture site.

3. Enophthalmos. Clinically obvious enophthalmos is another indication for surgical exploration, as it suggests a gross derangement of the orbit - enlargement of the volume of the orbit resulting from the fracture of the floor or escape of orbital fat.

Sensory Nerve Conduction Loss. In a suspected orbital floor blowout fracture, anesthesia or hypoesthesia in the area of distribution of the infraorbital nerve is suggestive evidence of a blowout fracture involving the infraorbital groove or canal. This finding assists in locating the site of the blowout fracture. Absence of infraorbital anesthesia implies that the fractured area is either lateral, medial, or posterior to the infraorbital groove or canal and is not a sign that the orbital floor is not fractured. In the patient shown a linear fracture occurred posterior to the infraorbital canal, entrapping the soft structures.

Radiologic Examination. Because of the superimposition of both thick and thin bones, the roentgen picture is apt to be difficult to interpret. The diagnosis of orbital fracture by roentgenography is made by means of a variety of positions: the Caldwell position, the Waters position, the fronto-occipital position, the anteroposterior projection, the reverse Waters position, and the oblique orbital-optical foramen view.

Diagnosis of blowout fracture of the orbit is frequently missed if the radiologic examination is not comprehensive. Fracture lines may be mistaken for superimposed bony septa or suture lines, or they may be hidden by disease processes in the underlying maxillary sinus. The thin orbital floor, partially transparent on radiographs, may be obscured against the background of other bones of the skull. Tomography will often disclose the presence of a blowout fracture and its location (Zizmor and coworkers, 1962).

Polytomography, as well as hypocycloidal movement, is advocated in all skull radiography. It brings into focus a 1-mm thin layer of tissue with reasonable clarity and sharpness. It is far superior to the curvilinear tomography formerly used.
With adequate technique, blowout fractures of the orbit can be diagnosed in over 90 per cent of the cases with conventional radiography. Polytomography has a similar degree of diagnostic accuracy and, in addition, can delineate the location, depth, and extent of the fracture with a degree of clarity and accuracy not possible with conventional radiography.

The type of blowout fractures varies: a lowering of the orbital floor; the "hanging drop", seen in the blowout fracture through which the orbital fat has extruded into the maxillary sinus; the trapdoor fracture, one or two bone fragments hanging into the sinus on a periosteal hinge; the massive extrusion of orbital contents into the maxillary sinus; associated fracture of the medial wall. Such positive radiologic signs, combined with positive clinical signs, are indications for surgical intervention. Crikair, Rein, Potter, and Cosman (1972) have drawn attention to the danger of "overoperating" when only presumptive radiologic signs (such as opacity of the maxillary sinus) are present. Certainly in the absence of positive clinical signs, surgical intervention should not be undertaken on the basis of presumptive radiologic signs alone.

**Treatment**

The three main purposes of surgical treatment are to (1) disengage entrapped structures and restore oculorotary function; (2) replace orbital fat into the orbital cavity if it has prolapsed into the maxillary sinus; and (3) restore orbital cavity size and form to minimize extraocular muscle imbalance and enophthalmos.

Once these primary objectives of treatment have been achieved and the bony architecture of the orbit is restored, additional surgery may be required to restore oculorotary function and to correct residual deformities or malfunction of the ocular adnexa.

**Timing of Surgery.** It is not necessary to operate immediately, particularly if posttraumatic edema is present. It is usually advisable to wait a few days, as subsidence of edema can be expected in this period of time. Delay beyond seven days is dangerous, particularly in children, as bone regeneration is rapid and the freeing of incarcerated orbital contents becomes more difficult. If treatment is postponed for two or three weeks, complications consisting of late motility problems as well as enophthalmos may be encountered. Undue delay, therefore, is not advocated.

In a large series of facial fractures studied by Hakelius and Pontén (1973), 21.8 per cent of the patients with midfacial fractures had double vision. By comparing a series of cases treated within two weeks after the accident and another series in which treatment was delayed, Hakelius and Pontén, in a follow-up study, found that 16 per cent of the patients in the first group reported the presence of diplopia only when they were tired (93 per cent were completely free of diplopia); in the second group, 24 per cent still had unchanged diplopia. As a result of the study an early, active surgical approach was recommended. It is significant that in a series of 50 patients with blowout fracture and other complications referred following unsuccessful, delayed treatment (mean time between trauma and surgery 3.5 weeks), 43 patients showed extraocular muscle imbalance (Converse and coworkers, 1967). Emery, von Noorden and Schlemmitzauer (1971) also reported the clinical findings in 159 patients with orbital floor fractures. They reported late diplopia in 60 per cent of patients with untreated blow out fractures when the diplopia was still present 15 days after injury.
Surgical Treatment: Yes or No? The authors have had a few patients in whom the entrapment was relieved by the forced duction test; these patients often do not require any further treatment. It is difficult to agree with Putterman, Stevens, and Urist (1974), who advocate the nonsurgical management of blowout fractures of the orbital floor. Putterman and his associates reported 25 per cent residual diplopia in a retrospective study and 27 per cent residual diplopia in a prospective study. Enophthalmos occurred in 65 per cent of the patients in the retrospective study and in 36 per cent of the patients in the prospective study.

Each case should be considered individually, and the decision for or against exploratory surgery is made on the basis of the criteria set forth above. One must bear in mind that diplopia is not the only major consequence of a blowout fracture; enophthalmos can be a major complication if the surgeon is content to watch the ocular globe sink progressively into the orbital cavity.

Operative Technique. A number of questions are usually asked concerning the method of treating blowout fractures. First and foremost is whether the method of approach to the orbital floor is through the eyelid or through the canine fossa and the maxillary sinus.

Although the eyelid or conjunctival approach to the orbital floor is preferred because it facilitates disengagement of the entrapped orbital tissues, the authors recognize that the approach to the orbital floor through the canine fossa and the maxillary sinus is indicated occasionally for the removal of bone fragments in the maxillary sinus and has merit in comminuted fractures of the maxilla and other bones of the midfacial area. Indeed, the placing of gauze packing or an inflatable balloon may be the only method of maintaining the contour of the orbital floor when these bones are fragmented into small pieces. A trapdoor type of fracture can be supported by gauze packing once the entrapment has been relieved.

In the absence of a blowout fracture without entrapment, intramaxillary sinus packing may effective support the comminuted orbital floor at a suitable level. However, maxillary sinus packing may be dangerous when it is excessive. In the patient shown, the globe was pushed upward under considerable pressure. Simultaneous observation of the floor of the orbit through the eyelid approach at the time of maxillary sinus packing would have prevented this complication. Suppuration has also been observed after gauze packing of the maxillary sinus, and blindness has been reported following this procedure. McCoy and associates (1962) reported a case in which packing of the maxillary sinus caused fragments of bone to damage the optic nerve with ensuing blindness. They condemn the method as dangerous, archaic, and ineffective in giving support to the fragments.

The maxillary sinus approach alone is not satisfactory for the release of the entrapped orbital soft tissues or for placing the orbital floor graft or implant. In a follow-up study of a series of 50 complicated cases, eight patients whose fractures had been repaired through the maxillary sinus alone required the trans-eyelid approach to release the incarcerated orbital contents from the surrounding impacted healed bony fragments (Table 25-4).
Table 25-4. Analysis of Complications in Orbital Fractures Persistent after Floor Repair* (50 cases)

<table>
<thead>
<tr>
<th>Complications</th>
<th>Preoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>3 months</td>
</tr>
<tr>
<td>Extraocular muscle imbalance</td>
<td>43</td>
<td>30</td>
</tr>
<tr>
<td>Enophthalmos</td>
<td>27</td>
<td>15</td>
</tr>
<tr>
<td>Ptoasis</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Medical canthal deformity</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Lacrimal obstruction</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Vertical shortening of the lower lid</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Visual impairment</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Trichiasis-symblepharon</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

* These complications were observed in patients referred to us after unsuccessful treatment.

The use of gauze packing or an inflated balloon is indicated when the orbital floor is comminuted and prolapsed into the maxillary sinus. Support of the comminuted fragments restores the floor of the orbit to the level of the floor on the contralateral side. The orbital floor should be explored, however, through an eyelid incision in order to check the level of the supported floor, to release an entrapment, if present, and to introduce an implant or transplant.

Exposure of the Orbital Floor

**The incision.** Various types of incisions through the lower eyelid may be employed. The one stroke incision to the orbital rim has the disadvantage of causing a unified line of cicatricial tissue which may result in a retracted scar and vertical shortening of the lower lid.

Although the subciliary incision near the margin of the lid about 3 mm below the lashline leaves an inconspicuous scar, an incision through one of the skin folds of the lower lid is also inconspicuous after healing and requires less dissection. The subciliary incision is made through the skin and orbicularis muscle until the tarsus is reached. At this point, the muscle (pretarsal portion of the orbicularis) is raised from the tarsus, the septum orbitale comes into view blow the tarsus, and the dissection over the septum orbitale is continued to the rim of the orbit. An incision below the tarsus obviates the need for as much dissection and is less liable to cause vertical shortening of the lid.

The conjunctival approach is another incision which has been advocated by Tessier (1973) in craniofacial anomalies and by Converse, Firmin, Wood-Smith and Friedland (1973) in post-traumatic deformities. If careful dissection is done according to the technique shown, it permits the surgeon to avoid perforation the septum orbitale with consequent extrusion of the orbital fat.
A simplified technique employed by Tenzel and Miller (1971) consists of a direct incision in the fornix which reaches the orbital rim and a retroseptal approach which exposes the orbital fat. A Desmarres retractor is used to retract the lower eyelid away from the globe. A malleable retractor placed posterior to the orbital rim gives adequate exposure. The incision is made through the conjunctiva to the orbital rim and includes the periosteum. This incision of necessity penetrates through the septum orbitale and exposes orbital fat. Tenzel and Miller have employed this type of incision in patients with small blowout fractures without restriction of ocular rotary movements of the globe. They did not employ the incision in patients with massive fractures with herniation of the orbital contents into the maxillary sinus.

The conjunctival incision avoids an external scar, albeit inconspicuous, and claims have been made that it prevents postoperative lower lid lagophthalmos in the upward gaze.

Because of the need to preserve the orbital fat and recuperate fat which has extruded into the maxillary sinus, it is preferable to avoid extrusion of fat through the septum orbitale, whenever possible.

When the orbital rim has been reached by following the septum orbitale downward, an incision through the periosteum is made immediately below the orbital rim. Subperiosteal elevation is extended backward until the area of the blowout fracture is identified. The infraorbital nerve should be respected; with the aid of visual magnification with binocular loupes, the nerve may be carefully dissected from the herniated and entrapped soft tissues.

The orbital structures should be raised using a caterpillar technique with two retractors. Dural pliable retractors with rounded edges minimize pressure on the ocular globe. Retraction of the intraorbital contents should be relaxed periodically. When necessary, a wide exposure can be obtained through the eyelid incision.

If the infraorbital rim is also fractured (impure blowout fracture), the fragments are realigned and fixation is maintained by interosseous wiring. If the orbital rim has been displaced backward, it is essential that the fragments be realigned in their former position. This measure will prevent postoperative vertical shortening of the lower eyelid.

The inferior rectus muscle and orbital structures are liberated from the areas of the blowout. The floor must be explored sufficiently far back into the orbit until the posterior edge of the defect can be identified. Verification that the ocular globe is freed from the fracture site is obtained by the forced duction test; it is essential to demonstrate the full range of all oculorotary movements. The most common cause of failure to release the entrapped structures is inadequate exposure of the floor in depth. The fracture may be far back.

**Restoration of the Continuity of the Orbital Floor.** Restoration of the continuity of the orbital floor is required in all orbital floor fractures, except in small fractures in which the entrapped structures can be freed readily and the forced duction test shows that free rotation of the eyeball has been reestablished.

**Bone grafts.** An iliac bone graft (a split rib graft in children) taken from the smooth inner aspect of the ilium is preferable in fractures in which there is a wide area of communication between the orbit and the maxillary sinus. The bone graft, as it becomes
vascularized, is better able to resist bacterial invasion than an inorganic implant. The authors have given preference to the bone graft in all major fractures with disruption of the orbital floor. We have also used the anterior wall of the maxillary sinus in the area of the canine fossa, the perpendicular plate of the ethmoid, and the septal cartilage for the restoration of small defects of the floor. Costal cartilage has also been employed and constitutes an excellent, seldom used transplant. Irradiated cartilage allografts have been employed by Dingman and Grabb (1961).

The mucous membrane lining of the subjacent maxillary sinus, often ruptured, will repair itself and line the undersurface of the transplant.

**Inorganic implants.** Inorganic materials employed in the orbital region have included solids, sponges, gels, and liquids. Tantalum, stainless steel, Vitallium, Paladon, methylmethacrylate, polyvinyl sponge, polyurethane, polyethylene, Teflon, Silastic, and Supramid have been commonly used.

Ballen (1964) employed Cranioplast, a rapidly polymerizing methylmethacrylate, which is prepared by mixing powdered acrylic with a liquid catalyst. The material is molded in situ and hardens by a process of polymerization, which gives off considerable heat. Ballen has used this procedure in 31 patients but does not mention complications. Miller and Tenzel (1969) have employed prefabricated Cranioplast implants, which are prepared in various sizes and thicknesses, in over 300 patients (Tenzel, 1974). The prefabrication has the advantage of eliminating the time interval required for the polymerization of the methylmethacrylate.

Freeman (1962) implanted sheets of Teflon in 36 patients with orbital floor fracture, despite communication with the maxillary sinus in several; Browning and Walker (1965) have reported the successful use of Teflon in 45 patients with orbital blowout fractures. Our own experience confirms these findings. Teflon is available in sheets 1-mm thick, and Silastic may also be carved to fit the specific defect. Supramid sheets 0.3-mm thick are also available.

The inorganic implant offers the advantage of obviating the need for an additional concomitant operation for the removal of a bone graft, and it has been satisfactory in most simple fractures. The authors have also had successful results in large defects with a wide area of communication with the maxillary sinus. In the course of secondary operations, regeneration of the maxillary sinus lining under the implant and, in moderate-sized defects, bone regeneration have been observed.

The purpose of the orbital floor insert, whether bone graft or inorganic implant, is to reestablish the continuity of the floor, seal off the orbit from the maxillary sinus, and restore the volume of the orbital cavity. The orbital floor insert should bridge the defect and rest on the stable adjacent portions of the floor. Smooth materials such as Teflon tend to slide forward and protrude under the skin of the eyelid. A tongue is prepared by making two cuts in the implant; the tongue is introduced under the anterior edge of the bony defect in the orbital floor, thus maintaining it in position and avoiding forward displacement and extrusion. Care should also be taken to avoid dead space between the inorganic implant and the bone of the orbital floor, as the accumulated fluid in the dead space constitutes a favorable medium for the growth of bacteria.
Blowout Fractures: Variations

A concomitant naso-orbital fracture suggests the possibility of an associated blowout fracture through the lamina papyracea of the medial orbital wall.

A major portion of the floor may be collapsed into the maxillary sinus. More limited blowout fractures are located medially, centrally, or laterally. The central blowout is typical of the pure blowout fracture. The medial blowout often occurs in the impure type associated with a naso-orbital fracture, a fracture of the medial orbital wall, and a blowout through the lamina papyracea. The lateral blowout occurs in the impure type associated with fracture of the zygoma.

There is no standard pattern in blowout fractures; fixity of the eyeball may be observed in massive blowout fractures as well as in small blowout fractures. In one case, the inferior orbital contents were pierced and pinned to the floor by a sharp bone fragment. The entire orbital floor may be fragmented and hanging hammocklike into the maxillary sinus, and the patient suffers no diplopia because oculorotary action is only slightly impaired. The prolapse of the orbital contents into the maxillary sinus may be extreme; in some of these cases, the ocular globe is difficult to find. The authors recall a patient in whom a long nasal speculum was necessary to retract the edematous eyelids in order to locate the ocular globe. The most dramatic case of this sort involved a fireman who inadvertently turned on the full power of his fire hose as he was inspecting the inside of the nozzle. The resultant blowout fracture was of such extent that the ocular globe disappeared from the orbital cavity and was presumed to have been enucleated by the force of the projected blast of water. The eyeball, which underwent several choroidal tears, was subsequently located in the maxillary sinus and replaced in position. After repair of the orbital floor and a normal postoperative course, the patient's vision was 20/20 for reading and 20/60 for distant vision.

Fractures of the Medial Orbital Wall. Medial orbital wall fractures usually occur in conjunction with an orbital floor fracture or a naso-orbital fracture. A special etiologic factor is the ski pole, the tip of which has struck the medial canthal area.

Clinical Findings. Rougier (1965) reported tethering of the medial rectus muscle following a blowout fracture, strongly suggesting an associated fracture of the medial orbital wall into the ethmoid sinus with entrapment of the medial rectus by a blowout mechanism similar to that which occurs in the orbital floor. Fractures of the medial wall were also noted by Miller and Glaser (1966), Edwards and Ridley (1968), Trochel and Potter (1969), Dodick, Galin, Littleton and Sod (1971), and Rumelt and Ernest (1972). The clinical signs were progressively increasing enophthalmos, narrowing of the palpebral fissure, horizontal diplopia with restriction of abduction, and increasing enophthalmos on abduction.

It has been suggested that medial orbital wall fractures are associated with orbital floor blowout fracture in an incidence varying from 5 per cent to 50 per cent (Gould and Titus, 1966; Jones and Evans, 1967; Dodick and coworkers, 1971). The high percentage can be explained by the structural relationships between the orbital floor and the medial wall described in an earlier section of the chapter. Our own experience, as well as that of Prasad (1973), is that entrapment of the medial rectus muscle is rare and that many of these fractures are found on radiographic examination to be in association with a naso-orbital fracture. The
cellular structure of the ethmoid bone offers resistance which the hollow maxillary sinus beneath the orbital floor does not. The possibility of a concomitant blowout fracture of the medial orbital wall should be suspected, however, if enophthalmos develops following adequate treatment of an orbital floor fracture.

Radiologic Findings. The diagnosis is often made by radiography; the presence of air within the orbit, clouding of the ethmoid sinus, and medial displacement of the medial orbital wall or displaced fragments of bone often seen on tomography demonstrate a medial wall fracture. The radiologic examination is invaluable in verifying the integrity of the medial wall preoperatively so that adequate measures can be taken at the time of surgery.

Treatment. Depending on the severity of the fracture, exposing the medial orbital wall, freeing the medial rectus muscle if it is entrapped, and placing an inorganic implant over the area of fracture will usually suffice to restore the architecture of the orbit. Primary bone grafting may be required in massive comminuted fractures. The treatment varies, therefore, according to the clinical and radiological findings; abstention from surgical treatment is indicated when only a linear fracture is present.

Progressively developing enophthalmos may be the price to pay for the failure to diagnose a medial wall fracture.

Fractures of the Lateral Orbital Wall. The lateral orbital wall consists of a strong, resistant anterior frontozygomatic rim which is exposed to facial trauma and a thinner posterior portion formed by the orbital process of the greater wing of the sphenoid.

The most severe fractures of the lateral wall of the orbit occur in conjunction with massive trauma to the zygomatic area with frontozygomatic disjunction and downward displacement of the lateral portion of the orbital floor. The lateral canthus is dislocated downward, with ectropion of the lower eyelid. This type of fracture requires a direct approach similar to that employed in multiple fractures of the midfacial skeleton (see Chapter 24). Direct interosseous wiring of the fragments and primary bone grafting to restore the orbital floor, lateral wall, and zygomatic osseous framework are indicated. In such severe fractures, the ocular globe suffers injury of varying degree, and loss of vision is not infrequent.

Lateral wall fractures are probably more frequent that is generally assumed. The authors have noted a number of such fractures during craniofacial operations performed for the correction of grossly malunited fractures. In some cases, orbital fat was found in the temporal fossa, suggesting a blowout fracture of the posterior portion of the lateral orbital wall. Behind the thick, lateral orbital rim is an area of thin bone; fracture of the rim may comminute this thin portion of the lateral orbital wall, facilitating a blowout of the area. Such a fracture may also be an unsuspected cause of persistent postoperative enophthalmos.

Fractures of the Orbital Roof. LaGrange (1918), in his classic monograph, showed that the thin medial portion of the orbital roof is fractured and displaced in its posterior part in the region of the superior orbital fissure and optic foramen. A fracture of this type can lead to serious complications, such as optic nerve atrophy and injury to the nerves to the extraocular muscles which enter the superior orbital fissure. Dodick, Galin, Littleton and Sod (1971), in a series of 22 cases of suspected blowout fractures of the orbit, obtained radiologic
evidence of fracture of the orbital floor in 15 cases; in two cases there was a concomitant fracture of the orbital roof.

Fracture of the orbital roof may also occur in conjunction with naso-orbital fractures, as the medial portion of the orbital roof is thinner and more susceptible to fracture.

If the superior rim of the orbit is fractured and the trochlea of the superior oblique muscle is displaced, consequent impairment of the function of the superior oblique muscle may result in diplopia, which is usually temporary.

Fractures of the orbital roof usually occur in conjunction with fractures of the supraorbital rim and frontal bone. A combined craniofacial approach is required in these fractures. The dura, which may be torn or penetrated by comminuted fragments, is raised and retracted. In such cases, after exposure of the anterior cranial fossa and neurosurgical repair, the orbital roof is repaired by a suitable thin bone graft.

**Orbital Floor Fractures Without Blowout Fracture**

**Examination and Diagnosis**

**Clinical Examination.** The symptoms and signs of fractures of the floor of the orbit without blowout fracture are similar to those of a blowout fracture, with the fundamental difference that the patient is able to effect oculorotary movements in an essentially normal fashion. The forced duction test is negative. There may be transitory diplopia.

**Roentgenographic Examination.** Tomograms will fail to reveal the characteristic excrescence of the orbital contents into the maxillary sinus, although in crushing fractures of the orbital rim and the floor of the orbit may have collapsed into the sinus. Maxillary and zygomatic fracture lines involving the orbital floor and irregularity of the contour of the infraorbital rim are noted on roentgenograms in these types of fractures.

**Treatment**

Fractures of the orbital floor occurring in zygomatic and maxillary fractures often do not require orbital intervention other than realignment and wiring of the fragments of the fractured orbital rim. The treatment is that required for the maxillary or zygomatic fracture. Verification should always be made, however, that the patient does not have a blowout fracture. Careful checking of the oculorotary movements and radiologic examination will eliminate this possibility.

In fractures with bony displacement, the risk of enlargement of the orbital cavity and of consequent enophthalmos is an important consideration. Treatment of an orbital fracture is done in conjunction with the reduction and fixation of fractures of other bones of the midfacial area. Measures are taken to restore the bony continuity of the displaced or fragmented bones. This is best done by direct exposure of the fractured area, reduction, and interosseous wire fixation.
When there is doubt as to the integrity of the orbital floor, exploration is indicated in order to eliminate an occult comminuted and depressed fracture.

**Comminuted Fractures of the Orbital Floor.** In cases of exceptionally severe "crush" and "crash" injuries seen following accidents in automobiles, helicopters, or airplanes and usually associated with other fractures of the midfacial skeleton, the orbital rim and floor may be completely demolished. The fragments of bone, most of them suspended hammocklike from the periosteum, and the orbital contents sink into the maxillary sinus. The bone is pulverized or reduced to small particles. If bone fragments can be salvaged, they are used to reconstruct the orbital rim. The lateral wall must be stabilized by interosseous fixation prior to reconstruction of the orbital floor. Ocular globe injury often requires enucleation. Opening the maxillary sinus through the canine fossa provides an approach to the fragmented orbital floor, which is elevated by packing the maxillary sinus with gauze impregnated with antibiotic ointment. Small portions of the orbital floor may remain laterally and medially and serve to support the bone graft used to restore the orbital floor and rim. Wire fixation is often required to stabilize the graft. In one of our patients whose eye was enucleated, a shelf of bone was found only in the posterior reaches of the orbit.

**Naso-Orbital Fractures**

Severe injuries of the midfacial area associated with fractures of the maxilla, nasal bones, zygomas, or orbits may also be complicated by fracture of the bones of the frontoethmoidal area of the facial skeleton. The bones of the middle third of the face are also in close anatomical relationship to the floor of the anterior cranial fossa and the frontal lobes of the brain through the frontal and ethmoid sinuses and the cribiform plate. The possibility of a concomitant blowout fracture of the orbital floor has also been discussed.

Because of the possibility of brain damage, patients suffering these fractures should be observed for neurologic complications, such as progressive loss of consciousness, and signs of epidural hematoma, aerocele, and chronic subdural hematoma. Fracture of the odontoid process, which requires early reduction, has been reported. Pulmonary edema is another complication of head injuries.

**Structural Aspects**

The thin areas of the medial orbital wall transilluminate readily and thus contrast with the heavier abutments formed by the nasal process of the frontal bone, the frontal processes of the maxilla, and the thick upper portion of the nasal bones. Posterior to the frontal process of the maxilla, the thinner lacrimal bone and the delicate lamina papyracea are vulnerable to trauma. The anterior and posterior ethmoidal foramina are situated along the upper border of the lamina papyracea in the frontoethmoidal suture, where the orbital plate of the frontal bone and the lamina papyracea of the ethmoid are joined. The anterior ethmoidal foramen transmits the nasociliary nerve and the anterior ethmoidal vessels; the posterior ethmoidal foramen gives passage to the posterior ethmoidal nerve and vessels. The rupture of these vessels in naso-orbital fractures with backward penetrating fragments is one of the causes of orbital hematoma, a complication which may require immediate incision and drainage.
The most posterior portion of the medial orbital wall is formed by the body of the sphenoid immediately in front of the optic foramen. In severe skeletal disruption of this area, the fracture lines involving the optic foramen and the optic nerve may result in blindness.

**The Interorbital Space.** The term "interorbital space" designates an area between the orbits, beneath the floor of the anterior cranial fossa. The interorbital space contains the two ethmoidal labyrinths, one on each side.

The interorbital space is roughly cuboidal, being wider anteriorly than posteriorly. It is limited above by the cribriform plate in the midline and by the roof of each ethmoidal mass on the sides and is divided into two approximately equal halves by the nasal septum. The interorbital space is limited below at the level of a horizontal line through the lower border of the ethmoidal labyrinth. The lateral walls of the interorbital space are the medial walls of the orbit. Anteriorly the interorbital space is limited by the frontal processes of the maxilla and by the nasal process and spine of the frontal bone.

The interorbital space contains cellular bony structures, the ethmoidal cells; spongy bony structures, the superior and middle turbinates; and a median thin plate of bone, the perpendicular plate of the ethmoid bone which forms the posterosuperior portion of the nasal septal framework.

**The Frontal Sinus.** The size of the frontal sinus varies greatly; it may be that of an ethmoidal cell, or it may be a very large sinus, pneumatizing the frontal bone. Occasionally it is absent.

The sinus has the shape of a pyramid with inferior, anterior, and posterior walls. The inferior wall or floor of the frontal sinus corresponds to the roof of the orbit and is the thinnest portion of the frontal sinus. The anterior wall is thickest and is composed of cancellous bone. The posterior wall is thinner than the anterior wall and is entirely composed of compact bone which separates the sinus from the frontal lobe.

**The Ethmoidal Sinus.** The ethmoid bone occupies the lateral portion of the interorbital space. Below the interorbital space, the lower half of the nasal cavity is flanked by the maxillary sinuses. Each lateral mass of the ethmoid is connected medially to the cribiform plate; the roof of each ethmoidal mass is inclined upward from the cribiform plate and projects, in its lateral portion, about 0.25 cm above the cribiform plate.

The ethmoid is pyramidal or cuboidal, measuring 3.5 to 5 cm long and 1.5 to 2.5 cm wide. It is cellular in structure and contains eight to ten cells with thin lamellar walls; these cells drain into the middle meatus of the nose. The frontal sinus drains through the ethmoid, either through a distinct duct or by emptying into an anterior ethmoidal cell and into the middle meatus. Thus there is an intimate anatomical relationship with the frontal sinus through the frontonasal duct. It will be recalled that, in embryological development, the frontal sinus is formed by an outcropping ethmoidal cell. A large ethmoidal cell, the frontoethmoidal cell, may be seen in the frontal bone between the frontal sinus and the roof of the orbit.
Surgical Pathology

Situated in the upper and central part of the middle third of the face, anterior to the anatomical crossroads between the cranial, orbital, and nasal cavities, the bones forming the skeletal framework of the nose may be projected backward between the orbits when they are subjected to a strong traumatic force. The term "naso-orbital" is employed to designate this type of fracture (Converse and Smith, 1963, 1964, 1966). A typical cause of naso-orbital fracture is an impact force applied over the upper portion of the bridge of the nose caused by the projection of the face against the dashboard or steering column of an automobile when it comes to a crash stop. A crushing injury with comminuted fractures is thus produced. Bursting of the soft tissues due to the severity of the impact and penetrating lacerations of the soft tissue resulting from projection through the windshield may transform the closed fracture into a compound fracture.

If the impact force suffered by the strong anterior abutments is sufficient to cause backward displacement of these structures, no further resistance is offered by the matchbox-like structures of the interorbital space; indeed, these structures collapse and splinter as would a pile of matchboxes struck by a hammer. The roof of the interorbital space is frequently involved in these fractures, and the anterior cranial fossa is penetrated, the fracture occurring either medially through the cribiform plate or laterally through the roof of the ethmoid sinus.

Some of the neurologic complications resulting from naso-orbital fractures are laceration of the dura covering the frontal lobes, laceration of the tubular sheaths enveloping the olfactory nerves as they perforate the cribiform plate, penetration of the brain by a sharp-edged ethmoidal cell wall, and necrosis of brain tissue.

An additional point of interest in the skeletal structure of this area is the continuity of the thin lamina papyracea of the medial orbital wall with the thin portion of the floor of the orbit. The splintering of the lamina papyracea facilitates a blowout fracture in this area and may occur in patients who suffer a blowout fracture of the floor of the orbit concurrently with a naso-orbital fracture.

Lacerations of the soft tissues may sever the levator palpebrae superioris or penetrate through the medial canthal area, severing the medial canthal tendon and the lacrimal canaliculi or sac.

Fractures of the other facial bones, particularly of the midfacial skeleton, are frequently seen. In some of our patients, the frontal bone was also involved.

The Nasal Area: The Weakest Portion of the Facial Skeleton. Studies confirm that the nasal area is the weakest portion of the facial skeleton; fractures occur in this area with an impact load of 35 to 80 g. In Swearington's study (1965), 45 impacts were made on cadaver heads to determine the fracture points of the various portions of the facial skeleton. The comparative forces that can be tolerated over the various facial areas without fracture are illustrated. With the exception of the neck of the condyle, the zygomatic area is the next weakest area, being unable to sustain impact forces greater than 50 g. The upper portion of the middle third of the face, which includes both the nasal and orbital areas, is structurally susceptible to fracture. In contrast, the lower portion of the maxilla sustains impact forces of
up to 100 g, and the major portion of the body of the frontal bone, with the exception of the central portion which is weakened by the frontal sinus cavities, sustains impact forces of up to 200 g.

Although padding of the rigid dashboard decreases the severity of the injuries sustained by the right front passenger, the padded dashboard lip in many automobiles has a contour suitable for the production of the "pushback" of the nasal structures between the orbits. Such fractures occur even though the passenger is wearing a lap seat belt; they occur less frequently when he is protected by the shoulder harness type of belt. The passenger without a seat belt is often projected through the windshield and suffers various types of soft tissue lacerations, including penetrating lacerations of the naso-orbital tissues.

**Traumatic Telecanthus.** In many naso-orbital fractures, the patient has a characteristic appearance; the bony bridge of the nose is depressed and widened, and the eyes appear far apart as in orbital hypertelorism. The appearance of the patient is the result of traumatic telecanthus, an increase in the distance between the medial canthi (intercanthal distance) as a result of displacement by fracture of the bones forming the skeletal framework of the nose and medial orbital walls. Naso-orbital fractures may be unilateral but are most often bilateral following severe trauma. Traumatic orbital hypertelorism is a deformity characterized by an increase in the distance between the orbits and ocular globes and occurs in massive disruption of the midfacial skeleton and frontal bone (Converse, Smith and Wood-Smith, 1975).

Traumatic telecanthus is produced by two varieties of backward displacement of the bony structures. In the first, the frontal processes of the maxilla and the nasal bones penetrate the interorbital space, comminuting the ethmoid cells and out-fracturing the medial wall of the orbit. The medial canthal tendon attachments are displaced with the bones, and the medial canthus is displaced laterally and deformed, assuming a rounded shape.

In the second type of fracture, the nasal bones and frontal processes of the maxilla are splayed outward and projected backward into the medial portion of the orbital cavity along the lateral surface of the medial orbital wall, severing the medial tendon, transecting the lacrimal sac, or severing the canaliculi. Thus traumatic telecanthus is also caused by increase in the thickness of the medial orbital wall from the overlapping of bone fragments.

Loss of bone in the area may result from injudicious debridement and removal of bone fragments or the expulsion of bone fragments into the nasal cavity at the time of fracture.

Occasionally the medial portion of the inferior rim of the orbit is also fractured and displaced backward.

**Examination and Diagnosis**

**Clinical Examination.** The appearance of the patient who has suffered a naso-orbital fracture is typical: the nose is flattened, appearing to have been pushed between the eyes; the medial canthal areas are swollen and distorted, the caruncles and plicae semilunares being covered by the edematous and displaced structures; ecchymosis and subjunctival hemorrhage are usual findings.
Intranasal examination shows the findings observed in fractures of the nasal bones and septum. Fracture of the septum is suggested by septal hematoma. Naso-orbital fractures are often accompanied by signs of orbital blowout fracture or fracture of the maxilla or zygoma, which are frequently associated fractures. Edema and hematoma often mask the extent of the skeletal distortion of the area, particularly if the patient is not seen during the first hours after the accident.

The patient may be unconscious or have had loss of consciousness of long or short duration. Loss of consciousness is suggestive of brain injury. The patient may be irritable, restless, or even thrashing about after a severe injury. As in other fractures around the orbit, extensive edema of the lids and orbital structures may cause mechanical limitation of eyeball movements.

There may be little evidence of skeletal deformity because of hematoma or swelling. In some cases the deformity is evident when the frontal bone has been crushed inward or the nasal structures have been projected into the interorbital space. The bones may be loose, and crepitation may be felt when they are mobilized. The entire upper jaw may be movable, and motion may be felt in the bones of the interorbital space. A portion of the forehead skin may be avulsed in compound fractures, exposing the bone and revealing the site of fracture.

Clear fluid escaping from the nose is strongly suggestive of cerebrospinal fluid rhinorrhea show an initial escape of blood from the fracture site; the fluid then becomes brownish in color and finally clear. The fluid may be seen to pulsate within the nose.

**Roentgenographic Examination.** Roentgenograms and tomograms are required to estimate the amount of damage. As stated earlier in the text, a fracture of the medial orbital wall may be associated with a blowout fracture of the orbital floor. Careful tomographic study will show suggestive signs of medial wall fracture. Fractures of the cribriform plate may be impossible to detect by ordinary radiographic examination. The presence of air in the subdural, subarachnoid space or in the ventricle is a sign of communication with the nasal cavity or sinuses, establishing a direct pathway to infection, and is an indication for neurosurgical intervention. Air may not be detected during the first 24 hours. Roentgenograms should therefore be repeated if the patient shows increasing signs of frontal lobe dysfunction in the nature of mental changes.

Fragmentation and a "buckled" appearance of the cribriform plate are suggestive of penetration of fragments of ethmoidal cells into the brain; this is an additional indication for neurosurgical operation. Tomograms may be of help in evaluating the location of the damage and the degree of displacement of the fragments.

**Treatment**

Brain injury should be suspected when the patient has been unconscious. Neurosurgical intervention is required in patients who have suffered destructive lesions of the brain or penetration of bone fragments into the brain. Careful neurologic and radiologic examination is required. Cerebrospinal fluid rhinorrhoea should not be a contraindication for treatment of the fractures. A delay of a number of days may be required to allow for subsidence of
swelling and hematoma and clarification of the neurosurgical status of the patient; such a delay does not jeopardize an ultimate satisfactory result.

The technique of the treatment of naso-orbital fracture consists of the elevation of the comminuted fragments by means of an instrument placed inside the nasal cavity. External digital pressure and, if necessary, realignment of the fragments restore the position of the medial orbital walls. In naso-orbital fractures, the wired plate technique and the figure-of-eight wire suspension have their selective applications. In severe cases of naso-orbital fractures, the open-sky method is the treatment of choice.

The Open-Sky Technique (Converse and Hogan, 1970). In compound naso-orbital fractures, the external wound permits direct inspection of the area, and the comminuted fragments can be realigned under direct vision. In severely comminuted naso-orbital fractures that are not compound, an open reduction is indicated. Bilateral vertical incisions through the skin over the lateral wall of the nose provide exposure; if additional exposure is required, the vertical incisions are joined by a transverse incision placed over the root of the nose. The bilateral vertical incisions, if they are of adequate length, give adequate exposure in most cases, and the resulting scars are not conspicuous, usually being hardly perceptible. After the fragments are dissected one by one, each lacrimal sac and the nasolacrimal duct exposed and preserved, and the medial canthal tendon identified, subperiosteal exposure of the medial walls of the orbit is extended posteriorly behind the area of fracture. It is then possible, under direct vision, to replace the fragments in acceptable alignment, aided by an instrument placed intranasally. Repair of the lacrimal apparatus in any of its sections can also be done under direct vision. Fragments of the nasal bones and the frontal processes of the maxilla may be joined to each other by interosseous wiring as well as to the frontal bone. Direct exposure of the fracture area is indispensable in fractures involving the cribriform plate with cerebrospinal rhinorrhea, as it permits inspection of the cribriform plate through the area of fracture.

The principle of the open treatment of naso-orbital fractures is the preservation of all fragments of bone, even where they are detached from soft tissues.

A frequent occurrence is the lateral displacement of the portion of the medial orbital wall containing the anterior lacrimal crest and the attachment of the medial canthal tendon. The orbicularis oculi muscle exerts a lateral traction, deforming the medial canthal area; this is another mechanism in the production of traumatic telecanthus.

Primary Bone Grafting. Primary bone grafting is indicated to restore bone continuity when the medial orbital wall is reduced to a pulp. The bone graft establishes an area of purchase for the attachment of the medial canthal tendon and also restores the medial orbital wall and the size and shape of the orbit (see Chapter 28).

In the preceding chapter (see Chapter 24), certain indications for primary bone grafting were given - namely, when bone is destroyed, and when assistance is required in maintaining the projection of the maxilla by bone grafting in the pterygomaxillary area. Primary bone grafting has also been advocated for the repair of defects of the floor of the orbit, of the median and lateral walls, and of the orbital roof. Moreover, primary bone grafting in severely comminuted naso-orbital fractures is indicated when the clinical conditions are favorable.
Supraorbital And Glabellar Fractures And Fractures Involving The Frontal Sinus

Either independently or in conjunction with naso-orbital fractures, fractures of the lower portion of the frontal bone in the supraorbital and glabellar regions are relatively infrequent. Schultz (1970) estimated the incidence of such fractures at approximately 5 per cent of those patients suffering fractures of the facial bones. Furthermore, he noted that patients suffering supraorbital and glabellar fractures require a longer average hospital stay than other facially injured patients irrespective of the cause.

Fractures of the supraorbital ridge are clinically evident by the observation of an area of local depression, especially in the early stages. In later stages, edema and periorbital ecchymosis may mask the deformity. The degree of depression of the supraorbital arch may depend on the size of the frontal sinus, which is extremely variable from one individual to another. Individuals with large frontal sinuses are more susceptible to fracture. When the depressed area involves the trochlea and the pulley of the superior oblique muscle, the patient may complain of diplopia, which is usually transitory. Some of our patients with supraorbital fractures had penetrating injuries which severed the levator palpebrae superioris. The resulting ptosis of the upper eyelid was masked in the early stages by edema and ecchymosis. Restriction of global movements secondary to the ecchymosis might lead one to the suspicion of a concomitant blowout fracture. If the lid, however, is carefully raised with a small elevator, unrestricted upward rotation of the globe can be observed and can be confirmed by the forced duction test. These findings eliminate the presumptive diagnosis of blowout fracture of the orbital floor.

The authors have observed concomitant fracture of the supraorbital arch, depressed fracture of the glabellar region, and naso-orbital fracture in the same patient. More frequently, however, the glabellar fracture is associated with a severe naso-orbital fracture, and the supraorbital arch fracture occurs independently.

When the causative impact is extremely violent and when an associated brain injury is present, the fragmented bones may be so badly comminuted that their realignment is not possible; some of them may even be ejected by the explosive force. In such cases, the type of deformity illustrated may be seen, with loss of nasal, glabellar, and supraorbital arch skeletal framework.

Roentgenographic diagnosis may be difficult, particularly in attempting to demonstrate a fracture of the posterior wall of the frontal sinus by lateral, Waters, or posteroanterior views. Tomograms, however, may be helpful in evaluating the extent of the damage and fracture.

Treatment nearly always involves open reduction through the wound or through a surgical incision. Exposure for supraorbital arch fractures can be achieved through the eyebrow if the incision is correctly placed. Objections have been made to such incisions because of subsequent hairless scars separating the upper and lower portions of the eyebrow. The hair follicles of the eyebrow are implanted in an oblique fashion; the incision should be slanted downward in order to parallel the direction of the hair follicles. In this manner, an inconspicuous scar will result in a well-furnished eyebrow, particularly in male patients. In
female patients with plucked eyebrows, it may be preferable to make the incision immediately below the orbital margin.

The approach to glabellar fractures, often associated with the telescoping type of naso-orbital fracture, can be achieved through the incisions made for the open-sky approach to naso-orbital fractures. If wider exposure is required, a transverse incision at the root of the nose is extended laterally on each side immediately below the orbital margin.

The best technique of exposure in major fractures involving the frontal bone is the coronal flap advocated by Tessier. In severe fractures involving the anterior cranial fossa, an intracranial approach and neurosurgical collaboration are indicated.

Reduction and realignment of fragments depend essentially upon the type of fracture. When large fragments are present, they may be levered upward and will often remain in position without interosseous wire fixation. When smaller fragments are present, direct interosseous wiring of the multiple fragments, as in the treatment of naso-orbital fractures, may be necessary.

**Treatment of Fractures Involving the Frontal Sinus.** Excluding the possibility of a concomitant head injury which may require craniotomy, fracture with backward crushing of the anterior wall of the frontal sinus may not cause any functional disturbance, but it leaves the patient with a depression deformity if no treatment is applied.

Open reduction of the depressed anterior wall of the frontal sinus, in a manner similar to that employed in glabellar fractures, is the method of choice. The depressed bone is elevated and maintained by wire fixation. When the anterior wall of the frontal sinus is comminuted, the following approach can be practiced.

In fractures of the anterior wall of the frontal sinus, an incision is made along the upper portion of the lateral wall of the nose from the point where the root of the nose joins with the supraorbital arch to a few centimeters below. The incision is through the skin and periosteum, equidistant between the dorsum of the nose and the medial canthus of the eye. Subperiosteal elevation is extended upward to below the supraorbital arch. The lacrimal sac is temporarily elevated and retracted from the lacrimal groove. Posterior to the area of the lacrimal groove, the lamina papyracea of the ethmoid is cut through, and the ethmoidal sinus is penetrated. By working upward from the ethmoidal sinus with a small Kerrison punch, it is possible to follow the ethmoidal cells until the medial aspect of the frontal sinus is reached. Part of the floor of the frontal sinus is removed, and the frontal sinus is entered. The fractured anterior wall of the frontal sinus is pried forward with a blunt probe or a hard rubber catheter. Often, if the depressed fracture consists of one large fragment, the fragment will maintain its position after it has been repositioned.

If the anterior wall of the frontal sinus is severely comminuted and if the general condition of the patient is satisfactory, a thin iliac bone graft is resected and placed over the frontal sinus to restore contour, if a satisfactory soft tissue covering can be assured.

If clinical and radiologic examination shows intracranial injury and a posterior wall fracture of the frontal sinus, neurosurgical exploration and treatment should be undertaken.
Complications of Orbital and Naso-Orbital Fractures

The type of injury, the force of the impact, associated tissue damage, and inadequate or delayed treatment are the major causes of complications in orbital and naso-orbital fractures.

Early diagnosis and adequate repair of the orbital floor result in few late complications. Depending on the quality of the initial treatment and despite adequate treatment, some cases are subjected to an inexorable evolution toward late complications (see Table 25-4).

The sequelae and their treatment have been discussed in some detail (Converse and coworkers, 1967; Kazanjian and Converse, 1974), and enumeration of these sequelae follows: structural deformities; complications with the implant or transplant; muscular imbalance and enophthalmos; ocular complications; lacrimal system disturbances; hematoma and blindness; blepharoptosis; medial and lateral canthal deformities; vertical shortening of the lower eyelid; and infraorbital nerve anesthesia. These complications and their treatment will be discussed in Chapter 28. A discussion of a few of the early complications follows.

**Structural Deformities.** Deformities and functional impairment are late complications which can be reduced by early diagnosis and treatment, but often the diagnosis is obscured by more severe cranial and facial injuries which demand primary treatment. The unconscious patient cannot experience diplopia, and orbital edema, hemorrhage, and ptosis can mask the enophthalmos. After several weeks, fibrous cicatrization is established, and reconstruction of the orbital cavity and restoration of symmetrical ocular function in a malunited fracture must be undertaken in the presence of scarred, atrophic orbital fat and muscles.

**Complications with the Implant or Transplant.** Dead space between the inorganic implant and the bone of the orbital floor should be avoided, as the accumulated fluid in the dead space constitutes a favorable medium for the growth of bacteria. Orbital infection and suppuration are indications for incisions, drainage, and removal of the implant, which is replaced after the infection has subsided. Antibiotic therapy should be routinely employed in all cases to avoid this complication.

In the average blowout fracture caused by a fist punch, a 1-mm Teflon implant is adequate to restore the continuity of the floor. Excessive thickness of the implant may cause the eyeball to be elevated. If the latter complications occurs, the implant should be removed and replaced by a thinner one. Furthermore, excessive overcorrection may result in elevation of the ocular globe and excessive compression on the orbital contents and the optic nerve.

Occasionally in severe trauma, hematoma may elevate the ocular globe. Gradual resorption of the hematoma will reduce the ocular globe to a level commensurate with the contralateral ocular globe.

An implant of excessive anteroposterior dimensions may compress the optic nerve and cause blindness. Extrusion of the implant by progressive forward migration can occur and can be avoided by the technique shown.
There is no perfect substitute for the orbital floor at the present time. The material used, however, should fit certain criteria. It should be well tolerated by the patient, sufficiently strong to support the orbital contents, relatively nonreactive to prevent adhesions to the orbital capsule, easily obtainable at the time of surgery, and workable into the desired shape. In early fracture repairs, the thin inorganic prosthesis is best, as exophthalmos can result from overcorrection. While autogenous bone is well tolerated by the patient and is the most physiologic substitute, it is not immune to absorption. Every effort should be made to ensure close contact of the bone graft with the bone of the orbital floor by carefully shaping the bone graft and eliminating dead space with small slivers of cancellous bone. Teflon, Silastic, Supramid, and Cranioplast implants are the preferred inorganic implants presently available. They can be shaped or carved and fitted to the floor of the orbit without preliminary preparation or delay.

**Muscular Imbalance and Enophthalmos.** Despite adequate early treatment of the orbital fracture, progressive ocular muscle imbalance and enophthalmos may ensue. Many adequately treated orbital floor fracture patients do not recover complete extraocular muscle function. Slight limitation in the upward gaze results in diplopia in this position, a relatively slight handicap. Extraocular muscle surgery on the affected eye or on the contralateral unaffected eye is a frequent requirement to restore eye muscle balance.

The complicated blowout fracture is often accompanied by multiple fractures of the facial bones and injuries of the soft tissues. Many of the fractures with complications treated by us have fallen into this category. These patients show impairment of ocular rotation action and diplopia, enophthalmos, depression of the zygomatic prominence, ptosis of the upper eyelid, downward displacement of the orbital contents, medial canthal deformities, shortening of the horizontal dimension of the palpebral fissure, shortening of the vertical dimension of the lower eyelid, saddle deformity and/or widening of the nasal bony bridge, and occasionally deformities of the supraorbital arch (see Table 25-4). Most complicated orbital fractures require extraocular muscle surgery during the months following the injury.

**Ocular Complications.** Ocular injury following orbital fractures has been reported as varying between 14 per cent (Milauskas and Fueger, 1966), 17 per cent (Miller and Tenzel, 1967), and 29 per cent (Jabaley and coworkers, 1975) in different series. Ocular globe injury also varies in severity from a corneal abrasion to loss of vision from a ruptured globe or a fracture involving the optic canal. Blindness or loss of the eye is remarkably infrequent, in view of the severity of some of the injuries sustained.

The importance of the ophthalmologic examination in all fractures of the orbit has already been discussed. Vitreous hemorrhage, dislocated lens, rupture of the sclera, traumatic cataract, choroidal rupture and hemorrhage, ruptured iris sphincter, glaucoma, retinal detachment, and diminution or loss of vision are some of the complications which may be avoidable if treatment is instituted early.

Verification of vision is essential in the course of an ophthalmologic examination. An excellent prognostic sign is the Marcus Gunn pupillary sign. A light is moved rapidly from one eye to the other alternately. If conduction of the optic nerve is lessened, the pupil on the involved side will appear to dilate as the light is brought from the sound eye to the involved eye. Monocular vision should also be considered.
The need for preliminary ophthalmologic examination is dramatically illustrated in a case reported by Miller (1968). Miller reported a patient with a midfacial fracture in whom vision in the left eye was 20/70 a few hours after the injury but dropped to no light perception by the fifth day. No surgery had been done. If the surgery had been performed before the fifth day, the resulting blindness could have been attributed to the operation.

Blindness has never resulted from repair of the orbital floor in any of the patients treated by the authors. We have seen patients whose vision was lost following repair by others. Nicholson and Guzak (1971) reported six cases in which vision was lost in a series of 72 patients who underwent orbital floor repair by means of silicone implants inserted by various surgeons in the same hospital. This high rate of visual loss, occurring in a reputable hospital, may be explained by the fact that the patients were operated upon by a number of different surgeons. It is preferable to place such patients under the care of specialized experienced surgeons.

**Lacrimal System Complications.** Interruption of the continuity of the lacrimal apparatus, chronic inflammatory condition of the lacrimal sac, or cystic dilation (known as a mucocele) with ensuing epiphora requires dacryocystorhinostomy or other procedures (see Chapter 28).

**Hematoma and Blindness.** Hematoma is unusual. It occurs if continuous bleeding from the anterior and posterior ethmoidal arteries is not spontaneously arrested in fractures involving the medial orbital wall. Blindness may be the consequence of a hematoma occurring under a firm pressure dressing. The use of continuous suction drainage is recommended if bleeding is excessive at the end of the operation. The treatment of hematoma of the orbit is usually conservative (see Chapter 37).

**Blepharoptosis.** True ptosis of the upper lid is to be differentiated from pseudoptosis resulting from the downward displacement of the eyeball and enophthalmos. True ptosis results from loss of function of the levator palpebrae superioris. This may occur either as a result of transection of the levator aponeurosis or of intramuscular hematoma and subsequent fibrosis, or from an injury to the third cranial nerve. In most cases the levator aponeurosis is transected, usually not through its entire width. The levator aponeurosis can usually be successfully repaired (see Chapter 28).

**Vertical Shortening of the Lower Eyelid.** Vertical shortening of the lower eyelid with baring of the sclera below the limbus of the globe in the primary position (scleral show) may result from downward and backward displacement of the fractured inferior orbital rim. Release of the septum orbitale attachment from the orbital rim and restoration of the position of the orbital rim after osteotomy may be required. If such operative procedures fail, a tarsocconjunctival graft from the same or opposite upper eyelid will elevate the lower lid margin up to 4 mm. Often pseudoptosis and depression of the supratarsal fold accompany vertical shortening of the lower lid; these three problems can also be at least partly resolved by means of a tarsocconjunctival graft.

The authors have received a number of personal communications concerning vertical shortening of the lower lid following the approach to the floor through a subciliary incision.
We are not using a slightly lower incision, below the inferior tarsus, to eliminate such a complication.

**Infraorbital Nerve Anesthesia.** Infraorbital nerve anesthesia may be very disconcerting to some patients. The area of sensory loss usually extends from the lower lid over the cheek and lateral ala to the upper lip. Release of the infraorbital nerve from the pressure of bone fragments within the canal may be indicated (see Chapter 28). Sensation may return spontaneously as late as one year after fracture.

**Cerebrospinal Fluid Rhinorrhea.** This complication occurs in naso-orbital fractures. The present trend is toward a conservative approach, influenced by a diminished fear of meningitis which results from the protection provided by antibiotic therapy. However, there have been reports of cases of traumatic cerebrospinal fluid rhinorrhea with recurring bouts of meningitis 15 years after injury.

When the radiologic examination fails to show evidence of damage other than a fracture line, the patient is treated conservatively while being observed for signs of impending complications, such as meningitis or extradural or intracerebral abscess. No packing is placed in the nasal fossae, smoking is forbidden, and the head of the bed is elevated to an angle of 60 degrees. The patient should be warned against blowing his nose, because the leakage might recur, or he might force tissue or air into the cranial cavity. If the cerebrospinal fluid rhinorrhea is prolonged, an operation to close the fistula should be considered; this is a decision to be made by the neurosurgeon. Collins (1973) stated that spinal fluid drainage is confirmed by the presence of glucose in amounts of more than 30 mg of glucose per 100 mL of fluid. The use of glucose oxidative paper is not reliable as a test for glucose. As high as 75 per cent positive reactions have been obtained when using the oxidative paper test in patients with normal secretions. The fistula may be located by isotope dyes or by dyes placed in the lumbar or ventricular cerebrospinal spaces.

As stated in Chapter 24, Collins (1973) advocated early reduction of facial fractures in the presence of cerebrospinal fluid rhinorrhea (Dingman, 1974). He stated that the objective is to obtain reduction and fixation of the fractured bones, thus providing support of the area of injury. In 19 patients treated by him in whom the facial bone fractures have been reduced in less than 48 hours, only two patients required operative repair of the dural fistulae, and in both the fistulae were not in proximity to the facial bone fractures.