FACIAL TRAUMA

VOLUME II

Orbit, Frontal Sinus, and Anterior Skull Base

Vision, orbital mechanics, reconstructive decision-making, sinus function, and intracranial separation

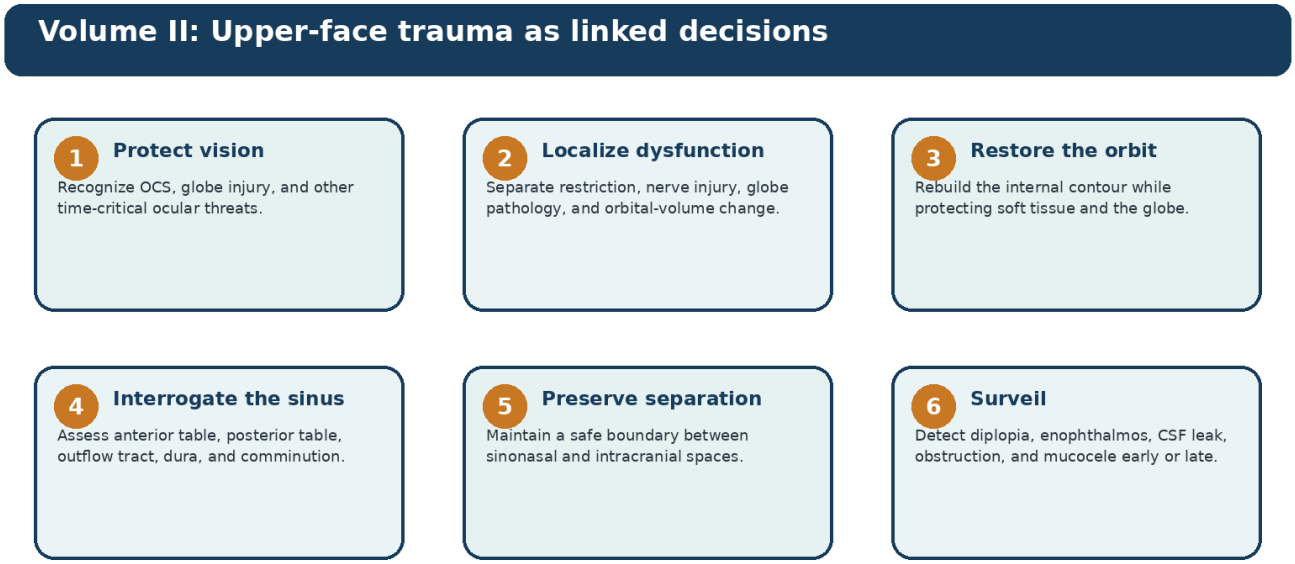

Figure 0.1. Volume II follows the upper-face trauma pathway from time-critical ocular threats to reconstruction, sinus decision-making, and long-term surveillance.

CENTRAL QUESTION

How do you protect vision, restore a functional orbit, and preserve a durable boundary between the sinonasal tract and the intracranial space?

Resident-level study guide • Prepared July 2026

How to Use This Volume

Volume II begins where the initial facial-trauma consultation becomes regionally specific. The orbit and frontal sinus are linked by more than geography: both are thin-walled compartments at the interface of vision, sinonasal spaces, and the cranial cavity. A resident must therefore reason simultaneously about function, time, three-dimensional anatomy, and the consequences of violating a barrier.

The guide is organized as a progression. Chapters 1 and 2 establish the orbital mental model. Chapters 3 and 4 teach the threatened eye and the localization of diplopia. Chapter 5 develops operative planning and reconstruction. Chapters 6 through 8 move superiorly into the frontal sinus, anterior skull base, CSF leak, and long-term surveillance. Chapter 9 integrates the entire workflow in oral-board cases.

SCOPE

This volume assumes the initial trauma survey and complete facial examination from Volume I. It focuses on the upper face. Detailed NOE reconstruction belongs in Volume III, although its relationship to the frontal recess and medial orbit is repeatedly highlighted.

Learning objectives

- Explain orbital anatomy as a tapered container and predict how wall defects alter globe position and motility.
- Distinguish orbital compartment syndrome, open globe, traumatic optic neuropathy, entrapment, edema-related restriction, and cranial neuropathy.
- Interpret orbital CT by fracture geometry, soft-tissue relationship, stable boundaries, and anticipated volume change.
- Choose observation, urgent repair, or interval reconstruction based on symptoms, age, examination reliability, and structural consequence.
- Plan orbital exposure, implant contour, posterior support, and intraoperative verification while anticipating eyelid and visual complications.
- Analyze frontal sinus trauma using the anterior table, posterior table, outflow tract, dura/CSF status, and comminution.
- Distinguish sinus preservation, contour repair, obliteration, and cranialization as goal-driven pathways rather than rigid labels.
- Diagnose and manage traumatic CSF rhinorrhea and design surveillance for late obstruction, mucocele, and infection.
- Present common upper-face trauma scenarios in a written-boards and oral-boards format.

EVIDENCE CALIBRATION

Orbital and frontal sinus management contains strong consensus around emergencies but less certainty around many elective thresholds. Numerical cutoffs can be helpful shorthand, yet symptoms, serial examination, defect geometry, and reconstructive feasibility often matter more. The guide labels established standards, evidence-supported trends, and institution-dependent practice.

Abbreviations

Abbreviation	Meaning	Why it matters
CT	Computed tomography	Primary modality for orbital and frontal sinus fracture geometry.
EOM	Extraocular muscle	Motility can be limited by entrapment, swelling, nerve injury, or poor fixation.
FSOT	Frontal sinus outflow tract	Determines whether a preserved sinus can ventilate and drain.
IOP	Intraocular pressure	Useful in suspected OCS only after open globe is reasonably excluded.
OCS	Orbital compartment syndrome	Clinical emergency requiring prompt decompression.
RAPD	Relative afferent pupillary defect	Evidence of asymmetric retinal or optic-nerve dysfunction.
TON	Traumatic optic neuropathy	Optic-nerve injury distinct from OCS and open globe.
CSF	Cerebrospinal fluid	A leak indicates failure of the skull-base barrier.

Contents

CHAPTER 1

The Upper Face as a Shared Boundary

CENTRAL QUESTION

Why should orbital and frontal sinus injuries be understood as failures of linked compartments?

OPENING CASE

A patient arrives after a high-speed collision with a displaced orbital roof fracture, pneumocephalus, and periorbital swelling. The CT contains three injuries in one image: a damaged orbit, a disrupted anterior cranial floor, and an open communication with a sinus. Treating each fracture line as a separate diagnosis obscures the central problem - the barriers between compartments have failed.

1.1 Three compartments, two barriers, one clinical problem

The upper face is organized around three adjacent compartments: the orbit, the paranasal sinuses, and the anterior cranial cavity. The orbital walls protect the globe and organize the extraocular muscles, nerves, vessels, and fat. The frontal and ethmoid sinuses form aerated spaces beneath and between the orbits. The anterior cranial base supports the frontal lobes and separates sterile intracranial contents from a colonized sinonasal tract. Trauma can violate one compartment or connect several of them.

This arrangement explains why the same mechanism may produce diplopia, pneumocephalus, CSF rhinorrhea, frontal contour deformity, and delayed sinus obstruction. It also explains why the correct consultation often crosses specialties. Ophthalmology addresses the globe and visual pathways; neurosurgery addresses the dura, brain, and unstable cranial boundary; otolaryngology or facial trauma teams address the orbit, sinus, drainage pathways, and skeletal framework. The resident's task is not to assign ownership first. It is to identify the threatened function and the failed boundary.

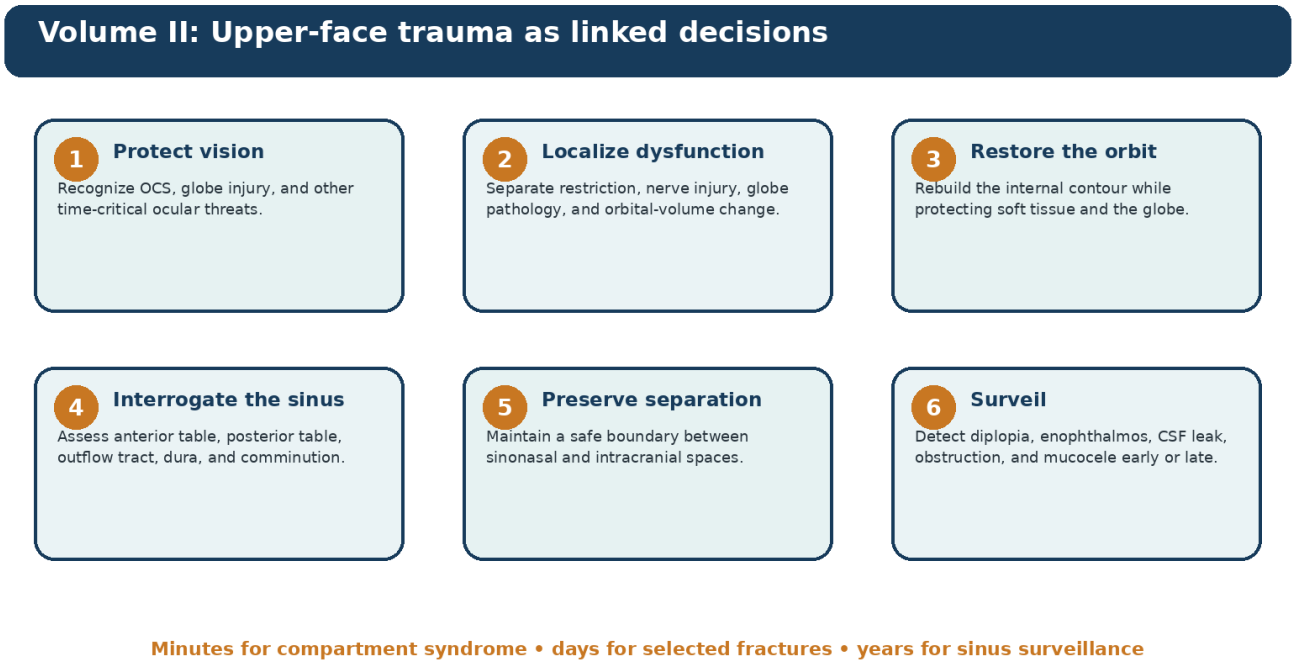

Figure 1.1. The upper-face trauma sequence. The early clock is dominated by vision and intracranial safety; the later clock is dominated by reconstruction and surveillance.

1.2 Four functions that organize the volume

Table 1.1. The upper face as a set of functions rather than fracture names.

Function	Normal structure	Traumatic failure	Clinical consequence
Protect vision	Rigid rim, compliant internal walls, intact globe and optic pathway	Hemorrhage, open globe, entrapment, apex injury	Potential irreversible visual loss or persistent diplopia
Maintain globe position	Three-dimensional internal orbital contour	Expanded cavity, lost inferomedial support, malpositioned implant	Enophthalmos, hypoglobus, asymmetry
Ventilate the frontal sinus	Patent frontal recess and mucociliary clearance	Obstruction, scarring, displaced fragments	Frontal sinusitis, mucocele, late infection
Separate brain from nose	Posterior table, skull base, dura	Fracture and dural tear	CSF leak, pneumocephalus, meningitis risk

A useful injury description therefore contains more than anatomy. “Orbital floor fracture” should become “orbital floor and inferomedial defect with tissue herniation, improving edema-related diplopia, no enophthalmos, and no evidence of entrapment.” “Frontal sinus fracture” should become “comminuted anterior-table and nondisplaced posterior-table injury with a patent-appearing outflow tract, no CSF leak, and acceptable contour.” The added words are the management variables.

1.3 The clocks of upper-face trauma

Upper-face injuries operate on at least three clocks. The emergency clock measures minutes to hours: orbital compartment syndrome, open globe, active visual decline, severe pediatric entrapment with systemic signs, and unstable intracranial injury. The reconstructive clock measures days to a short interval: persistent functionally important diplopia, displaced orbital fractures, unacceptable frontal contour, and injuries in which delayed fibrosis or loss of landmarks will complicate repair. The surveillance clock measures months to decades: progressive enophthalmos, implant problems, frontal outflow obstruction, mucocele, recurrent sinusitis, and delayed CSF leak.

CLINICAL BRIDGE

A patient can leave the emergency clock without leaving the disease process. “No operation tonight” must be followed by a plan for repeat examination, definitive decision-making, and long-term surveillance.

1.4 The resident’s recurring questions

- Is vision objectively normal, stable, and measurable?
- Is restricted motility mechanical, neurogenic, globe-related, or nonspecific?
- Has the orbital container enlarged, collapsed, or lost a reliable boundary?
- Which frontal sinus variables are injured: anterior table, posterior table, outflow tract, dura, or comminution?
- Is the sinonasal-intracranial barrier intact?
- What must happen now, what should happen after swelling settles, and what requires surveillance years later?

RESIDENT SYNTHESIS

The orbit, frontal sinus, and anterior skull base should be understood as neighboring functional compartments. The treatment plan follows the threatened function, the failed barrier, and the time to harm - not simply the number of fracture lines.

CHAPTER 2

Orbital Anatomy and Fracture Mechanics

CENTRAL QUESTION

How does the orbit maintain vision, motility, and globe position - and how does trauma disrupt those relationships?

2.1 The orbit is a tapered container

The bony orbit resembles a four-sided pyramid with a broad anterior aperture and a narrow posterior apex. The orbital rim is relatively strong and protects the globe from direct impact. Posterior to the rim, the roof, floor, medial wall, and lateral wall converge toward the optic canal and superior orbital fissure. The internal walls are not equivalent: the medial wall and floor are thin and adjacent to aerated sinuses; the lateral wall is thick and provides a stable reference; the roof separates the orbit from the anterior cranial fossa.

This geometry matters clinically because the orbit is not an empty box. It contains the globe, extraocular muscles, optic nerve, motor nerves, vessels, lacrimal structures, and fat. When a wall fractures outward into a sinus, the bony cavity can become larger. Early edema may temporarily mask the resulting loss of support. As swelling resolves, the globe may drift posteriorly or inferiorly. Conversely, displaced fragments, hemorrhage, emphysema, or an implant can reduce effective orbital space and raise pressure.

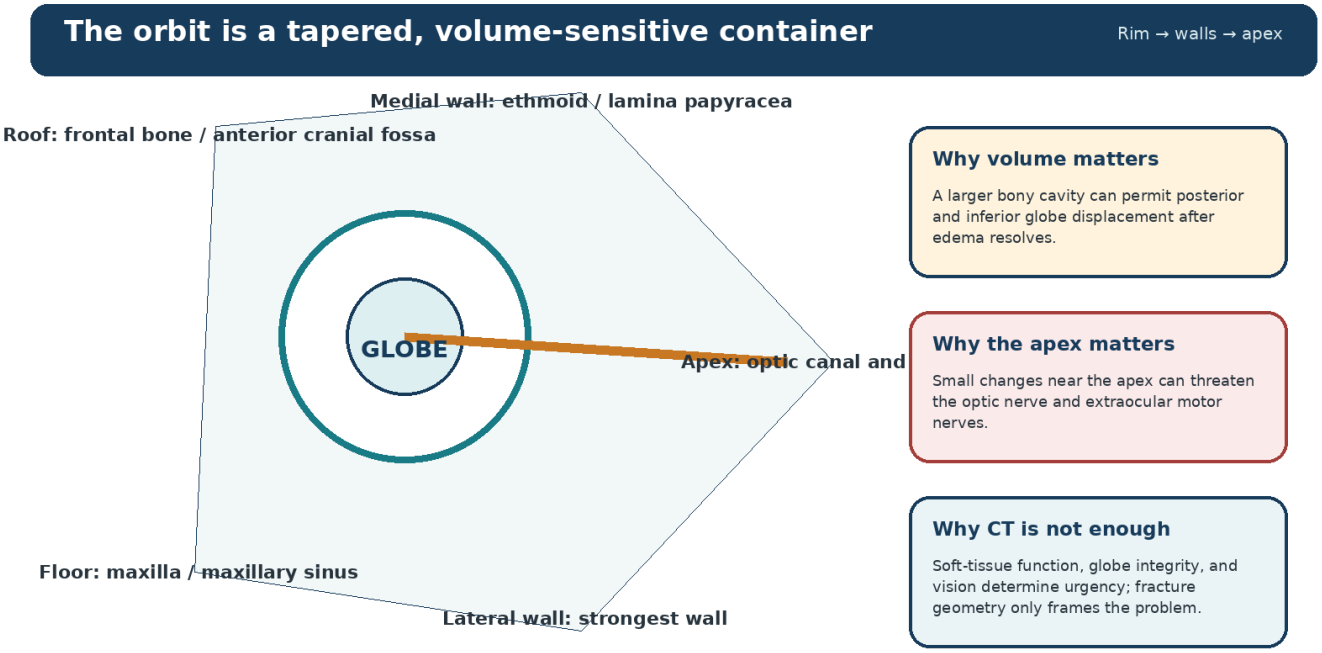

Figure 2.1. The orbital mental model. The rim protects the globe, the internal walls determine volume and contour, and the apex concentrates the optic nerve and motor pathways.

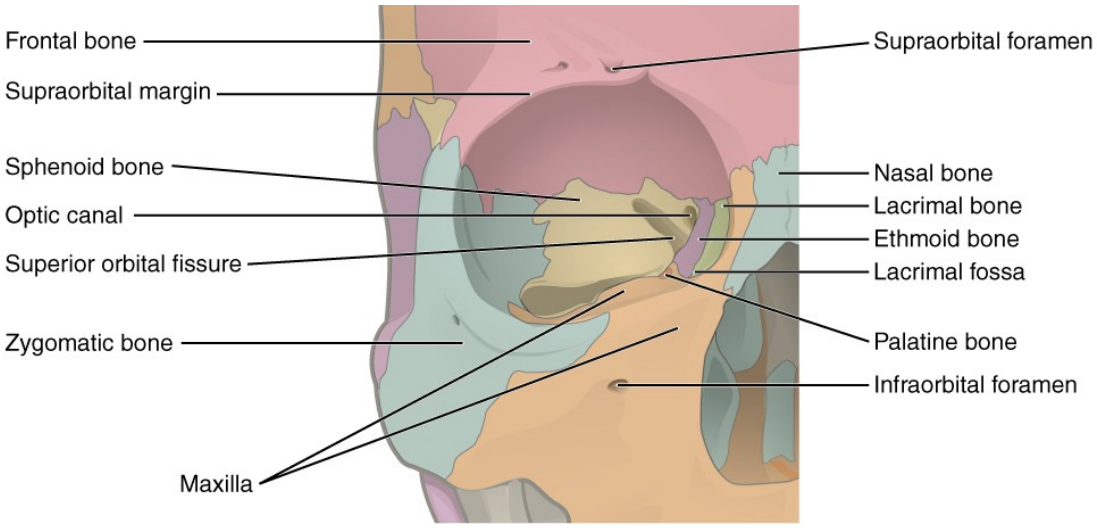

Figure 2.2. Bones forming the orbit. Learn the walls as boundaries to adjacent compartments rather than as an isolated anatomy list. Source: OpenStax, Wikimedia Commons, CC BY 3.0.

2.2 The orbital walls and their neighbors

Table 2.1. Orbital boundaries and why each matters clinically.

Region	Key anatomy	Neighbor	Trauma implication
Roof	Frontal bone; lesser wing of sphenoid posteriorly	Anterior cranial fossa, frontal sinus laterally	Roof fractures may coexist with intracranial injury, CSF leak, or pulsatile proptosis.
Floor	Maxilla, zygoma, palatine contribution	Maxillary sinus	Defects can increase volume and involve the infraorbital nerve or inferior rectus complex.
Medial wall	Lamina papyracea, lacrimal bone, sphenoid posteriorly	Ethmoid cells and nasal cavity	Thin wall permits fracture and emphysema; large inferomedial defects are easy to under-reconstruct.
Lateral wall	Zygoma and greater wing of sphenoid	Temporal fossa and middle cranial fossa posteriorly	A reliable reduction landmark in ZMC injury; displacement changes orbital width and projection.
Apex	Optic canal and superior orbital fissure region	Middle cranial fossa and central skull base	Small hematomas or fragments can affect vision or multiple cranial nerves.

2.2A Operative landmarks that change reconstruction

The resident should be able to identify the optic canal, superior and inferior orbital fissures, anterior and posterior ethmoidal foramina, infraorbital groove and canal, inferomedial strut, and posterior ledge. These landmarks determine where dissection becomes dangerous, which boundaries can support an implant, and whether a defect can be reconstructed through a limited corridor.[26]

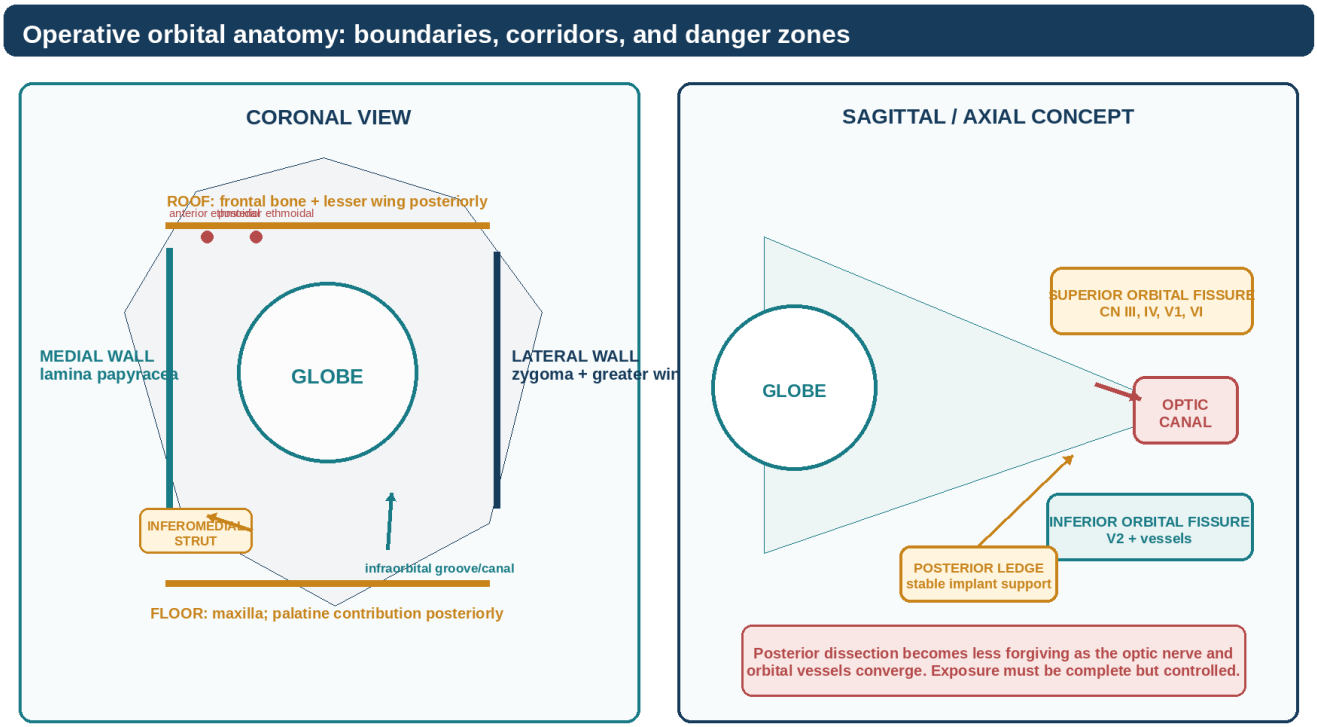

Figure 2.2A. Operative orbital landmarks. The posterior roof is the lesser wing of the sphenoid; the greater wing contributes to the lateral wall. The inferomedial strut and posterior ledge are high-value reconstructive references.

2.3 Floor and medial wall mechanics

Two models explain how a relatively intact rim can coexist with an internal orbital fracture. In the hydraulic model, force transmitted to the globe and orbital contents creates a transient pressure wave that ruptures a thin wall. In the buckling model, force applied to the rim travels posteriorly through bone until the floor or medial wall fails. These models are complementary rather than mutually exclusive. Their educational value is that they shift attention from the visible rim to the hidden internal orbit.

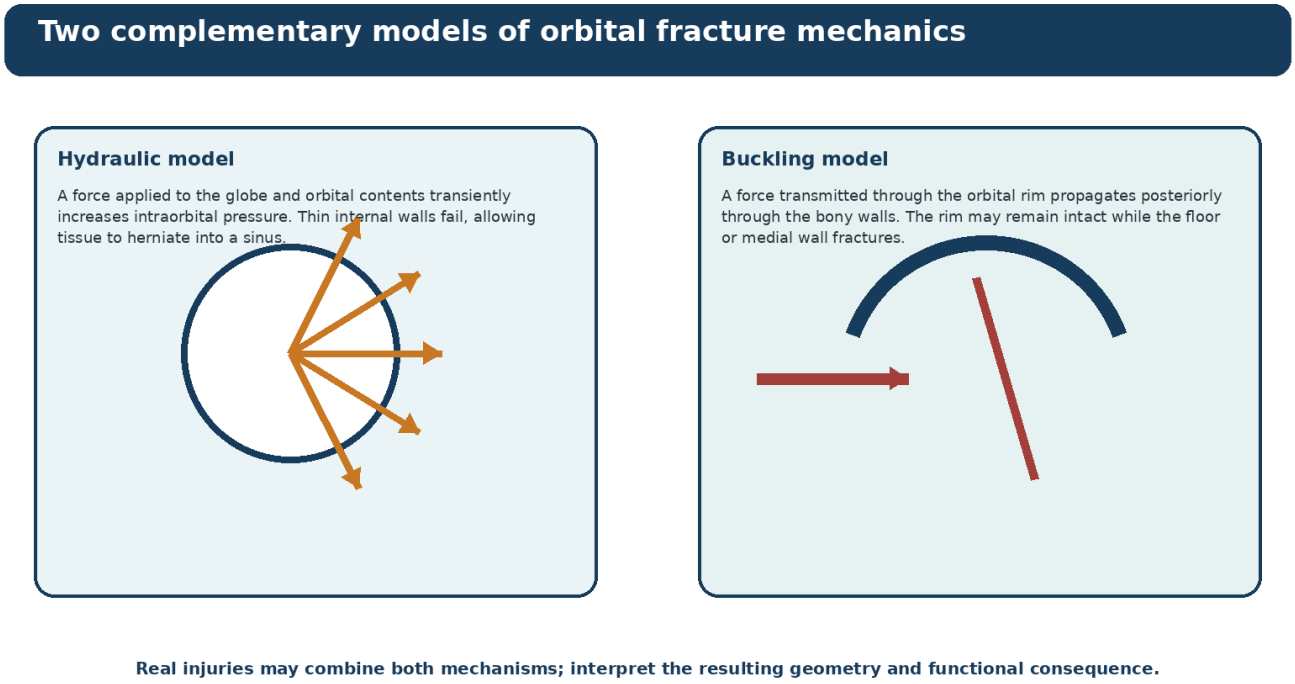

Figure 2.3. Hydraulic and buckling mechanisms. Real injuries often combine both, and either can produce a clinically important internal defect despite an intact rim.

The inferomedial transition deserves special attention. A combined floor and medial wall defect can remove a substantial portion of the internal orbital cone while leaving no obvious external deformity. If reconstruction restores the flat floor but fails to recreate the inferomedial contour, residual volume expansion and late enophthalmos can persist. The posterior limit is equally important: an implant that stops short of stable posterior support may sag into the maxillary sinus or allow soft tissue to remain displaced.

2.4 Extraocular muscles are not simple strings

The rectus muscles arise near the orbital apex and travel forward within a system of connective-tissue pulleys. Periorbital tissue, intermuscular septa, and fascial attachments coordinate globe rotation and maintain muscle position. A fracture can therefore cause dysfunction without trapping the belly of a muscle between two sharp fragments. Tethered fascia, displaced orbital fat, edema, hematoma, pain, or a nerve palsy may all limit movement.

This is why the phrase “muscle entrapment on CT” should be used cautiously. CT can show muscle shape, location, herniation, and relationship to a defect; it cannot directly prove that the observed motility deficit is caused by mechanical incarceration. Conversely, pediatric trapdoor fractures can produce severe restriction even when the bony defect appears small or nearly closed.

2.5 The optic pathway and orbital apex

The optic nerve passes through the intraconal space to the optic canal, accompanied by the ophthalmic artery. The oculomotor, trochlear, and abducens nerves enter through the superior orbital fissure region. Trauma near the apex can therefore produce visual loss, ophthalmoplegia, sensory change in V1, or combinations described as superior orbital fissure or orbital apex syndromes. The pattern is more important than the eponym: optic-nerve dysfunction plus multiple ocular motor deficits localizes posteriorly and should prompt urgent multidisciplinary assessment.

> **DIAGNOSTIC TRAP**
> A normal-appearing anterior globe does not exclude posterior visual pathway injury. Conversely, reduced acuity in a swollen eye is not automatically traumatic optic neuropathy; first exclude open globe, corneal injury, hyphema, retinal

2.6 Orbital volume is a useful model, not a single threshold

Orbital volume helps explain enophthalmos, but management cannot be reduced to one defect-size threshold. Interpret location, shape, inferomedial-strut integrity, soft-tissue herniation, preinjury asymmetry, and reconstructive accuracy together. Posterior and combined floor-medial defects may be more consequential than anterior or single-wall defects of similar area.

2.7 Localize posterior orbital deficits

Multiple ocular motor deficits are a localization problem, not a synonym for edema. Examine visual acuity, color, pupils/RAPD, fields, CN III/IV/VI function, V1/V2 sensation, corneal reflex, globe position, and venous-congestion signs.

Table 2.2. Posterior orbital localization.

Pattern	High-yield findings	Immediate implication
Superior orbital fissure syndrome	CN III/IV/VI ophthalmoplegia or ptosis with V1/corneal sensory loss; optic-nerve function is preserved.	Review thin-section posterior orbital/skull-base imaging and obtain urgent multidisciplinary assessment.
Orbital apex syndrome	Superior-fissure pattern plus optic neuropathy: reduced acuity, color, or field and/or an RAPD.	First exclude open globe and OCS; obtain urgent ophthalmologic assessment and targeted imaging.
Cavernous sinus / traumatic CCF	Multiple motor palsies with V1 +/- V2 loss; chemosis, proptosis, arterialized vessels, high IOP, pulsatile tinnitus, or bruit.	If vascular injury is suspected, obtain urgent CTA and involve the neurovascular team; angiography may be required.

BOARD PEARL

CN II dysfunction separates an orbital apex pattern from isolated superior orbital fissure syndrome. Venous congestion, pulsatility, or bruit should redirect the workup toward traumatic carotid-cavernous fistula.

Resident synthesis

POSTERIOR-ORBIT RULE

The orbit is a tapered container. Localize the deficit before choosing the scan or operation: vision threat, motor pattern, trigeminal pattern, and venous-congestion signs determine which posterior compartment is at risk.

CHAPTER 3

The Threatened Eye

CENTRAL QUESTION

Which ocular problems demand immediate action, and how should the resident distinguish them?

OPENING CASE

After orbital fracture repair, a patient reports rapidly increasing pressure and reduced vision. The eyelids are tense, the globe is proptotic, and a new RAPD is present. The postoperative CT has not yet been obtained. The key decision is not which scan to order; it is whether the clinical syndrome is sufficient to decompress now.

3.1 Vision is a measured variable

Every upper-face trauma evaluation begins with objective visual assessment. Record acuity in each eye separately using the best feasible method: a chart, near card, printed text, counting fingers, hand motion, or light perception. Test pupils and look for an RAPD. Assess color saturation when practical, confrontational fields, motility, diplopia, globe position, and the anterior eye. Repeat the examination whenever symptoms change, after reduction or repair, and after the patient becomes more cooperative.

The words “vision intact” should never mean only that the patient can see something. A patient with unilateral acuity loss, red desaturation, a new RAPD, or a constricted field has a visual pathway problem until proven otherwise. Document the limitations of the examination explicitly when intubation, sedation, developmental age, pain, or language barriers prevent reliable testing.

Table 3.1. A syndrome-based approach to the threatened eye.

Finding	Likely localization	Immediate concern
Peaked pupil, visible wound, shallow chamber, severe anterior-eye injury	Globe	Open globe; shield and avoid pressure
Proptosis, tense orbit, RAPD, rising IOP, visual decline	Orbital compartment	OCS; decompression is time critical
Reduced acuity or color with RAPD, relatively quiet globe	Optic nerve / retina	TON or retinal injury; urgent ophthalmic evaluation
Diplopia with nausea/bradycardia in a child	Mechanical entrapment / oculocardiac reflex	Urgent release may be required
Multiple ocular motor palsies plus optic dysfunction	Orbital apex / superior fissure region	Posterior orbital or skull-base injury

3.2 Suspected open globe

An open globe changes the examination. Clues include a full-thickness corneal or scleral wound, uveal prolapse, an irregular or peaked pupil, markedly reduced vision, a shallow or asymmetric anterior chamber, dense circumferential subconjunctival hemorrhage, or abnormal globe contour on CT. A normal CT does not exclude a small rupture. Avoid tonometry, forced eyelid opening, ocular ultrasound, or any maneuver that places pressure on a suspected open globe.

Place a rigid eye shield rather than a pressure patch, control nausea and pain, keep the patient NPO, update tetanus prophylaxis, and involve ophthalmology urgently. Prompt empiric systemic broad-spectrum antibiotics are commonly initiated because post-traumatic endophthalmitis is vision-threatening; regimen, route, and duration should follow the treating ophthalmology service, allergy profile, contamination pattern, and local open-globe protocol.[28] Facial fracture repair is coordinated around globe protection; it does not take priority over closure of the eye.

HIGH-RISK OVERLAP

Open globe and OCS are not mutually exclusive. If rupture is suspected, shield the eye and avoid tonometry or globe pressure, but do not let progressive orbital ischemia become an observation problem. Involve ophthalmology immediately; when the OCS syndrome is compelling, use the fastest safe decompression pathway and do not delay solely for CT. Coordinate later fracture exposure, forced duction, and implant timing around globe protection.

3.3 Orbital compartment syndrome

OCS is a pressure syndrome in a confined space. Hemorrhage, edema, emphysema, or a postoperative collection raises intraorbital pressure, reducing perfusion of the retina and optic nerve. The diagnosis is clinical. The most concerning pattern is acute visual decline with a tense proptotic orbit, RAPD, painful or restricted motility, and elevated IOP when measurement is safe. Not every retrobulbar hematoma produces OCS, and not every OCS patient displays every classic sign.

When the clinical picture is compelling, decompression should not be delayed solely for imaging. Lateral canthotomy with inferior cantholysis releases the lower eyelid from the lateral canthal tendon and allows the globe to move forward. The resident must know the local protocol, equipment, and escalation pathway. Persistent pressure or visual compromise after an adequate bedside release may require superior cantholysis, operative exploration, removal of an implant, or evacuation of a localized hematoma. AO guidance similarly emphasizes emergency decompression for a tense proptotic globe with acute visual disturbance.[1,2]

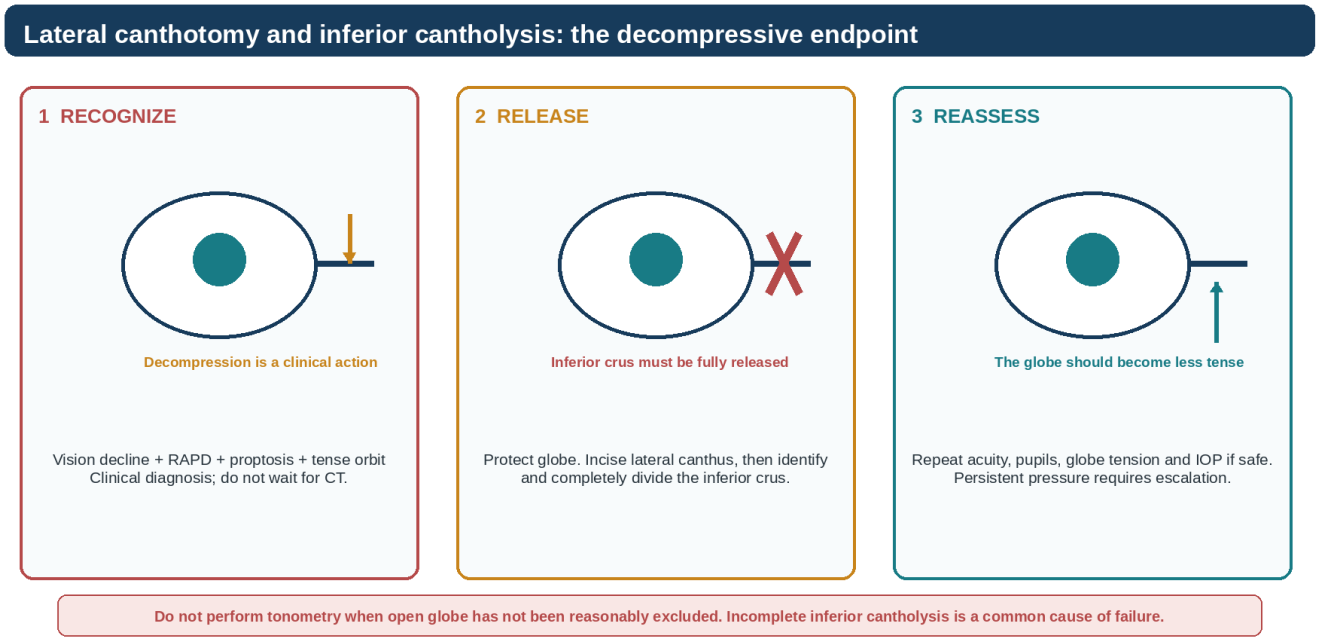

Figure 3.1. Lateral canthotomy with complete inferior cantholysis is a decompressive procedure. The endpoint is a lax lower eyelid and a less tense orbit, followed by immediate reassessment of acuity, pupils, globe tension, motility, and IOP when safe.

PROCEDURE SEQUENCE

Protect the globe, infiltrate local anesthetic when time permits, crush the lateral canthus briefly, incise laterally, identify and divide the inferior crus of the lateral canthal tendon, then reassess eyelid laxity, globe tension, acuity, pupils, and pressure. Incomplete release is a common cause of failure.

3.4 Traumatic optic neuropathy

TON describes optic-nerve dysfunction caused by trauma. Direct TON results from laceration, avulsion, penetrating injury, or clear compression by a fragment. Indirect TON is thought to arise from transmitted forces and secondary injury without obvious disruption of the nerve. The presentation is reduced acuity, color vision, or field with an RAPD when the injury is asymmetric; the anterior globe may appear relatively normal.

TON is not OCS. OCS has a treatable pressure mechanism and requires urgent decompression. For indirect TON without compartment syndrome, contemporary evidence does not support routine megadose corticosteroids or optic-canal decompression. A 2024 systematic review found no evidence of benefit for medical or surgical treatment and emphasized evidence of harm from megadose intravenous methylprednisolone in traumatic brain injury populations.[3] Management therefore centers on accurate diagnosis, exclusion of reversible ocular and orbital causes, neuro-ophthalmic input, and treatment of associated injuries rather than reflexive steroid administration.

EVIDENCE NOTE

Established: decompress OCS. Evidence-supported caution: do not conflate indirect TON with a steroid-responsive compression syndrome. Direct compression by a displaced fragment, foreign body, or hematoma is a different anatomic problem and requires individualized multidisciplinary reasoning.

3.5 Pediatric trapdoor entrapment and the oculocardiac reflex

Children have more elastic bone. A wall may hinge open, allow tissue to prolapse, and recoil toward its original position, incarcerating muscle or fascia. External ecchymosis and CT displacement may be modest. The child may present with painful gaze restriction, diplopia, nausea, vomiting, bradycardia, or syncope. These vagal symptoms can represent an oculocardiac reflex triggered by traction on orbital tissues.

3.5 Pediatric trapdoor injury: urgency can hide behind a quiet eye

A "white-eye" presentation should not reassure the examiner. Severe motility restriction with nausea, vomiting, bradycardia, syncope, or other oculocardiac symptoms should be treated as ischemia-producing trapdoor entrapment until proven otherwise. Notify ophthalmology and the facial-trauma service immediately, keep the child NPO, control nausea, and coordinate urgent release based on the mechanical and oculocardiac syndrome.

A subtle or initially negative CT report does not exclude incarceration or justify delay; review thin-section images with radiology and ophthalmology when needed. If bradycardia, syncope, or rhythm disturbance is present, obtain ECG assessment and continuous monitoring while release is coordinated. Forced-duction testing under anesthesia can confirm the mechanical component and should be repeated after release.[27]

1 Recognize
Restriction plus vagal symptoms is an ischemic mechanical syndrome.

2 Stabilize
NPO, antiemetic control, ECG/monitoring when symptomatic, urgent consultation.

3 Release
Do not let a subtle CT report delay release; verify free motion after repair.

3.6 Orbital emphysema

Air commonly accompanies fractures that communicate with a sinus. Most cases are self-limited, but one-way-valve physiology after nose blowing or positive pressure can produce rapidly progressive proptosis and OCS. Avoid nose blowing and forceful Valsalva; reassess immediately for sudden visual symptoms after sneezing or nose blowing.

3.7 Bedside sequence for acute visual change

- Recheck acuity, pupils/RAPD, color, fields, globe position, motility, and pain against the last examination.
- Look for open-globe signs before IOP measurement or eyelid manipulation.
- If OCS is clinically likely, decompress immediately and summon ophthalmology; do not wait for CT.
- If the globe is intact and the orbit is not tense, localize optic, retinal, motor-nerve, and mechanical causes.
- After intervention, document the same objective variables again.

RESIDENT SYNTHESIS
Open globe requires protection and closure; OCS requires decompression; pediatric entrapment can require urgent release despite a quiet external examination and subtle imaging.

CHAPTER 4

Orbital Fracture Evaluation

CENTRAL QUESTION

How do examination and CT findings become a defensible decision to observe, reassess, or repair?

OPENING CASE

A patient with an isolated floor fracture has diplopia in upgaze and downgaze on the day of injury. CT shows a moderate defect with herniated fat and an elongated inferior rectus. The radiology report calls this “entrapment.” The patient has no nausea, no bradycardia, and can move through the full range with discomfort. The management question is whether the deficit represents a time-sensitive mechanical problem or an edema-dominated examination that should be repeated.

4.1 Begin with the symptom pattern

Diplopia is not one diagnosis. Ask whether it is binocular or monocular, constant or gaze-dependent, present in primary gaze or only at extremes, and improving or worsening. Binocular diplopia resolves when either eye is closed and usually reflects misalignment. Monocular diplopia persists with the fellow eye closed and points toward optical or globe pathology. Functional disability is greatest when diplopia affects primary gaze, reading gaze, or the patient’s occupational field of view.

Pain with movement is common early and does not prove entrapment. Nausea, vomiting, bradycardia, or marked gaze restriction in a child increases concern for mechanical incarceration. New enophthalmos or hypoglobus suggests loss of structural support. Facial numbness localizes injury near the infraorbital canal but is not, by itself, a routine indication for orbital reconstruction.

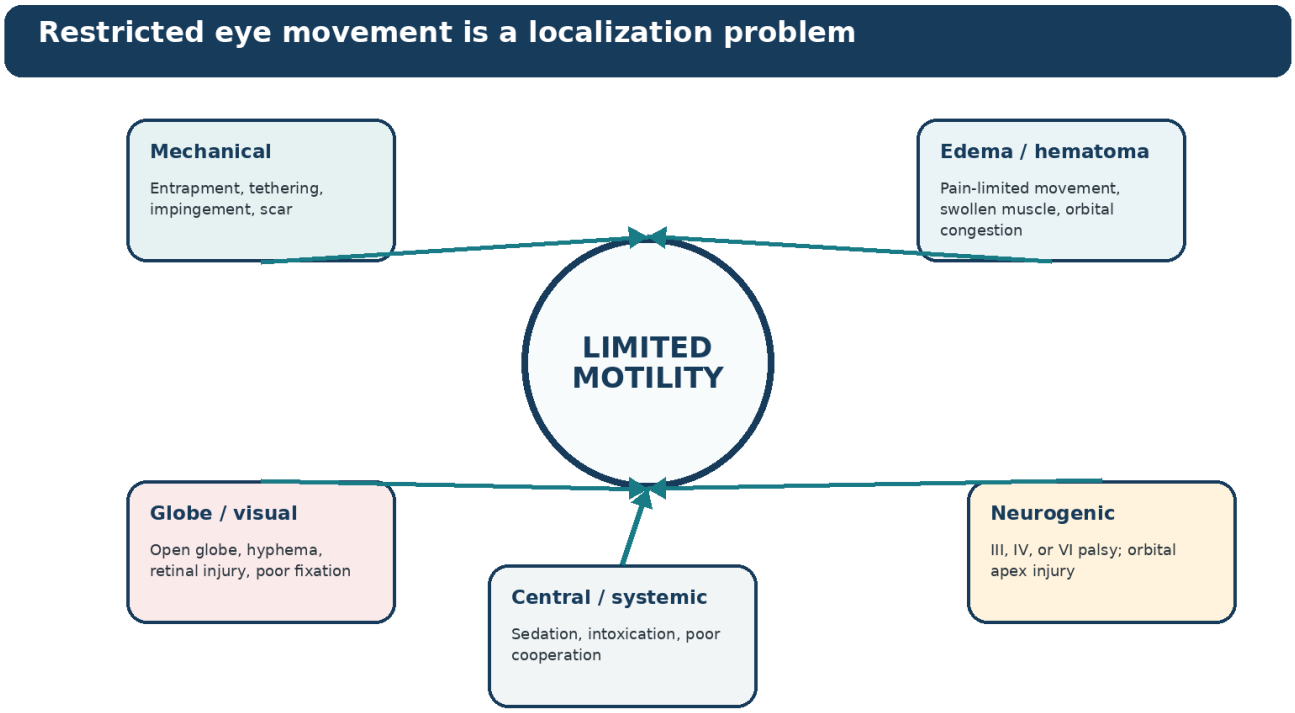

Figure 4.1. Restricted motility should trigger a differential diagnosis. “Entrapment” is a mechanism that must be demonstrated clinically, not a synonym for any abnormal CT appearance.

Table 4.1. Motility patterns and localization.

Pattern	Mechanism to consider	Clues	Response
Severe restriction with vagal symptoms	Trapdoor entrapment	Child or young patient, small defect, nausea or bradycardia	Urgent specialty evaluation and likely early release
Painful limitation in several directions	Edema or hematoma	Early after injury, diffuse swelling, improving serial exam	Observe closely and repeat examination
Isolated abduction deficit	CN VI dysfunction	No clear tether at defect, posterior injury possible	Evaluate nerve and orbital apex / skull base

Pattern	Mechanism to consider	Clues	Response
Poor movement with severe visual loss	Globe, retina, optic nerve, or inability to fixate	Abnormal acuity, RAPD, anterior-eye findings	Treat visual pathology; do not label as entrapment
Persistent vertical diplopia with positive forced duction	Mechanical tethering	Reproducible restriction, compatible defect	Repair if functionally important and clinically appropriate

4.2 Forced ductions: what the test can and cannot tell you

Forced-duction testing asks whether the globe can be passively rotated through the restricted direction. It should not be performed until open globe has been reasonably excluded, is not a routine maneuver for every swollen traumatic eye, and should be undertaken gently by a clinician familiar with ocular trauma. Resistance supports a mechanical component; free passive movement suggests paresis, pain, poor cooperation, or central causes. Awake testing may be limited by guarding even with topical anesthesia. The most reliable test is often performed under anesthesia before and after release and reconstruction.

A positive forced duction does not name the trapped structure. Muscle belly, fascia, fat, scar, or an implant can all tether movement. A negative test performed poorly does not exclude entrapment. The result belongs within the larger syndrome rather than standing alone.

4.2A Quantify globe position and diplopia when possible

Clinical impressions become more useful when they are reproducible. Hertel exophthalmometry or an equivalent projection measurement should record the base and both eyes; vertical dystopia can be measured from the pupils or limbi; and binocular single-vision fields, prism measurements, or Hess/Lancaster testing can characterize functionally important diplopia. Baseline photographs and preinjury images are particularly helpful because edema can temporarily mask enophthalmos and because facial asymmetry is common.

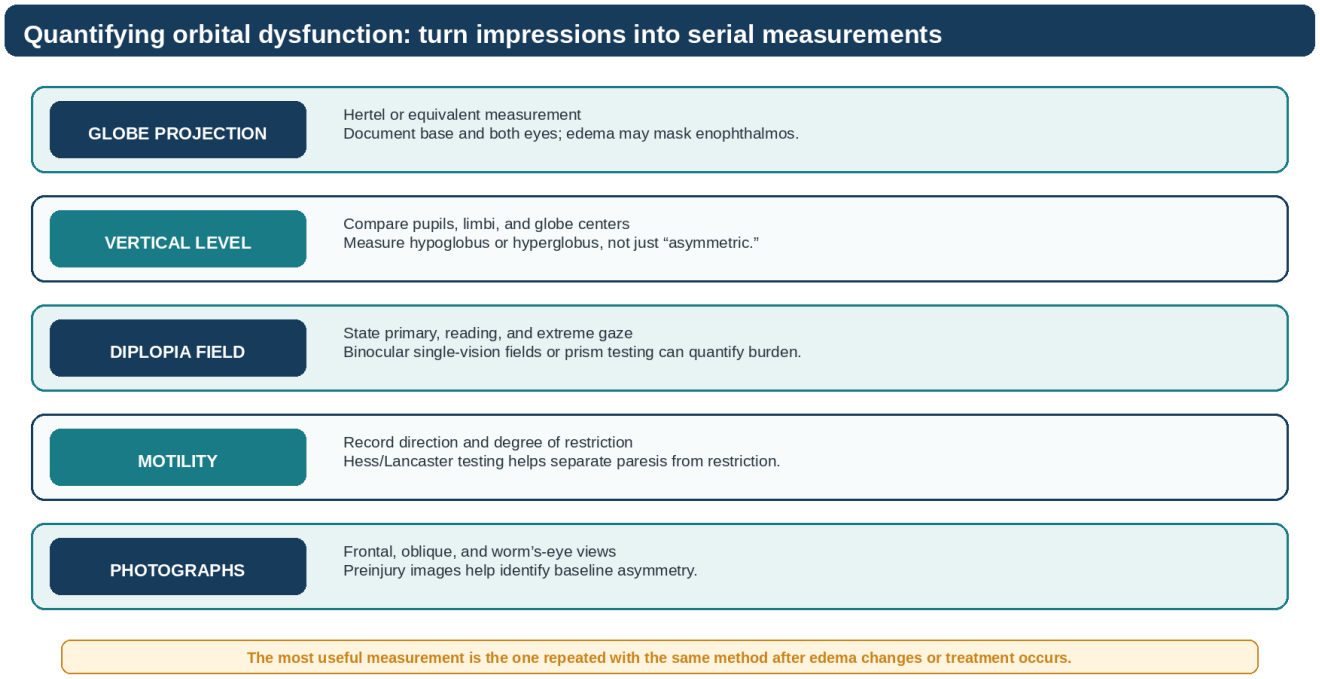

Figure 4.1A. Quantitative orbital follow-up. Serial measurements are most useful when the same method is repeated after edema evolves or after treatment.

4.3 Read orbital CT by geometry and soft tissue

Thin-cut CT in axial and coronal planes is the foundation of orbital fracture assessment; sagittal reformats clarify the floor and posterior ledge. Three-dimensional reconstructions help communicate rim and ZMC displacement but are inferior for the thin internal walls. Read both bone and soft-tissue windows. Begin with the globe, retrobulbar space, optic canal, and orbital apex before measuring the wall defect.

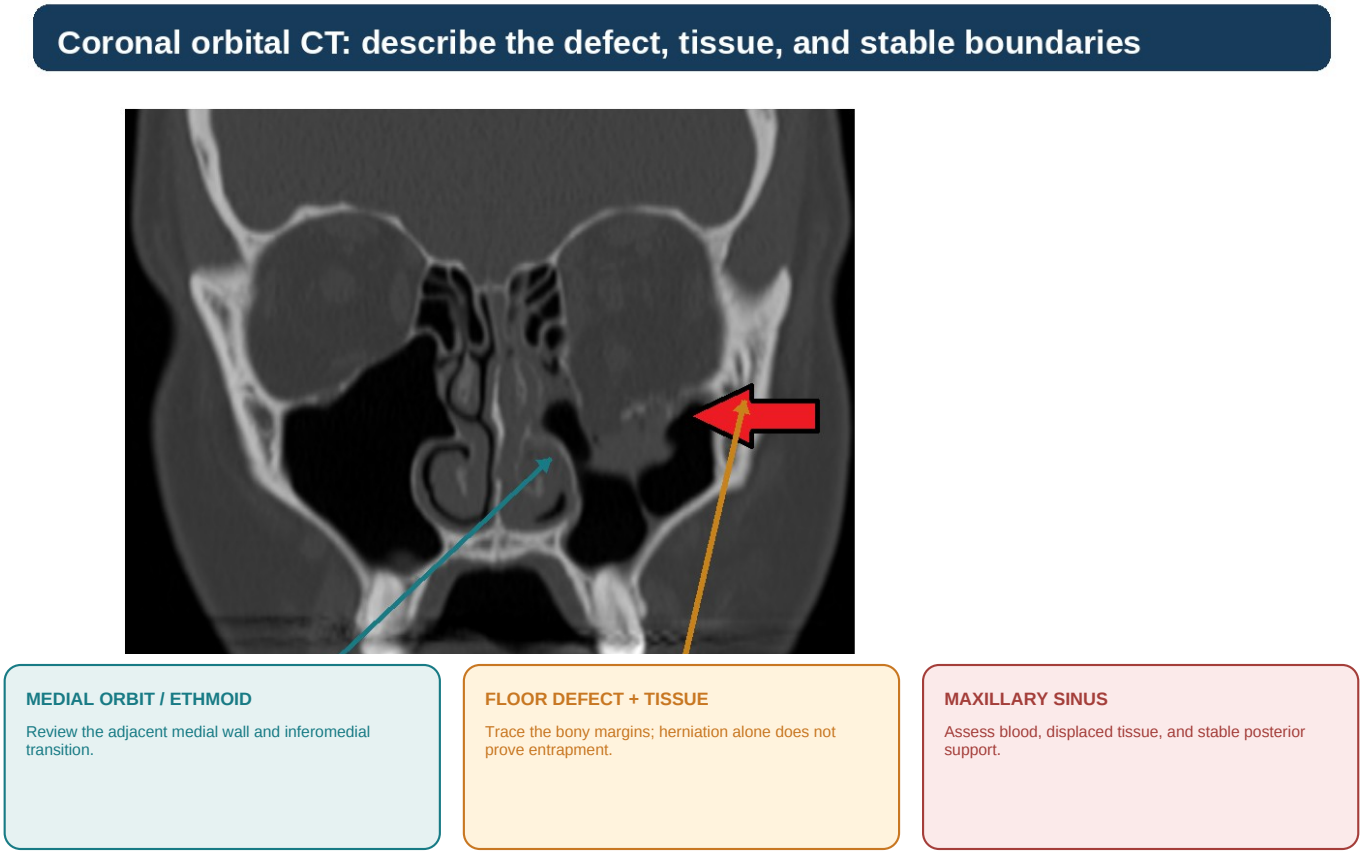

Figure 4.2. Coronal CT of a left orbital floor fracture. Describe the defect, tissue crossing it, globe level, maxillary sinus, inferomedial transition, and stable posterior boundary separately; CT proximity does not prove clinical entrapment. Source: James Heilman, MD, Wikimedia Commons, CC BY-SA 3.0; annotations adapted.

Table 4.2. A management-centered orbital CT review.

CT question	Why it matters	Common trap
Is the globe intact and normally shaped?	Open globe or lens injury changes the entire plan.	Focusing on the fracture before inspecting the eye.
Is there a retrobulbar collection or apical crowding?	May explain pressure, vision loss, or nerve dysfunction.	Calling every retrobulbar hematoma OCS without clinical correlation.
Which walls and junctions are disrupted?	Combined floor-medial defects lose more contour than an isolated small defect.	Describing only the largest single fracture line.
Is the inferomedial strut preserved?	Provides support and a landmark for reconstruction.	Underestimating junctional defects.
Is a posterior ledge visible?	Determines whether an implant can rest on stable bone.	Placing an implant without understanding posterior support.
What tissue crosses the defect?	Muscle shape, fat herniation, and tethering guide localization.	Equating proximity or herniation with proven entrapment.
Has the orbital cavity expanded?	Predicts risk of late globe malposition.	Using a single area threshold without considering location or shape.

4.4 Fracture patterns

An isolated floor fracture may preserve the orbital rim and lateral wall. A medial-wall fracture can cause emphysema and volume expansion with less obvious external deformity. Combined floor-medial fractures disrupt the inferomedial contour and can be technically difficult to reconstruct. ZMC injuries alter the lateral orbital wall and rim as part of a larger malar displacement. Roof fractures should trigger evaluation for frontal sinus, skull-base, and intracranial injury. Apex fractures raise concern for optic and cranial nerve dysfunction.

The term blowout fracture is useful descriptively but incomplete. A pure blowout fracture spares the rim; an impure fracture includes rim or broader facial framework disruption. What matters to management is whether the fracture creates a vision threat, mechanical restriction, durable volume change, or loss of stable skeletal references.

4.5 Observation is an active strategy

Most uncomplicated orbital wall fractures do not require emergency surgery. Observation allows edema, pain, and transient neuropraxia to improve and reveals whether diplopia or globe-position change will persist. An active observation plan includes baseline photographs, objective vision and motility documentation, sinus precautions, symptom control, and a defined reassessment interval. The follow-up examination should specifically compare diplopia fields, globe position, visual function, and sensory symptoms.

Patients must know which changes bypass routine follow-up: reduced vision, increasing proptosis or pressure, worsening pain, new nausea or bradycardia with gaze, sudden symptoms after nose blowing, or inability to close the eyelids over the globe. Observation without access to reassessment is not equivalent to conservative management.

4.6 Indications for repair are functional and structural

Common reasons to repair include urgent mechanical entrapment, persistent functionally important diplopia with evidence of restriction, clinically meaningful enophthalmos or hypoglobus, and a defect expected to produce unacceptable late globe malposition. Large defect size, substantial soft-tissue herniation, loss of the inferomedial support, and posterior extension increase concern, but no isolated CT measurement should automatically determine surgery.

The decision is more difficult when symptoms and imaging disagree. A large defect with normal motility and symmetric globe position may be observed with careful follow-up. A small pediatric trapdoor defect with severe restriction may require urgent release. A patient with preexisting strabismus, monocular vision, or thyroid eye disease requires individualized goals and ophthalmologic input.

> **CONTROVERSY**
> Traditional teaching often cites enophthalmos greater than 2 mm, involvement of more than half the floor, or persistent diplopia as thresholds. These remain useful orientation points, but contemporary reviews emphasize clinical trajectory, defect geometry, combined-wall injury, and patient-specific functional burden rather than rigid application of one cutoff.[4–6]

4.7 Timing of nonemergent repair

Once an indication is established, timing balances two competing goals. Waiting permits edema to recede and the examination to become more reliable. Excessive delay can permit fibrosis, persistent tissue displacement, and loss of straightforward planes. Many adult repairs are performed during the first one to two weeks, but this is an orientation interval rather than a universal deadline. Observational work suggests that most isolated floor-fracture patients who ultimately develop an operative indication declare it during early follow-up, while selected less-severe fractures may still be repaired later without automatically producing a poor result.[6,21] The correct interval depends on the syndrome, soft tissues, associated injuries, reconstructive complexity, and surgeon judgment.

Timing is not synonymous with urgency. A patient with stable vision and improving diplopia can usually be reassessed. A child with oculocardiac symptoms, a patient with a nonprotectable globe, or a patient with a clear mechanical syndrome occupies a different clock. The plan should name the reason for the timing rather than recite a date.

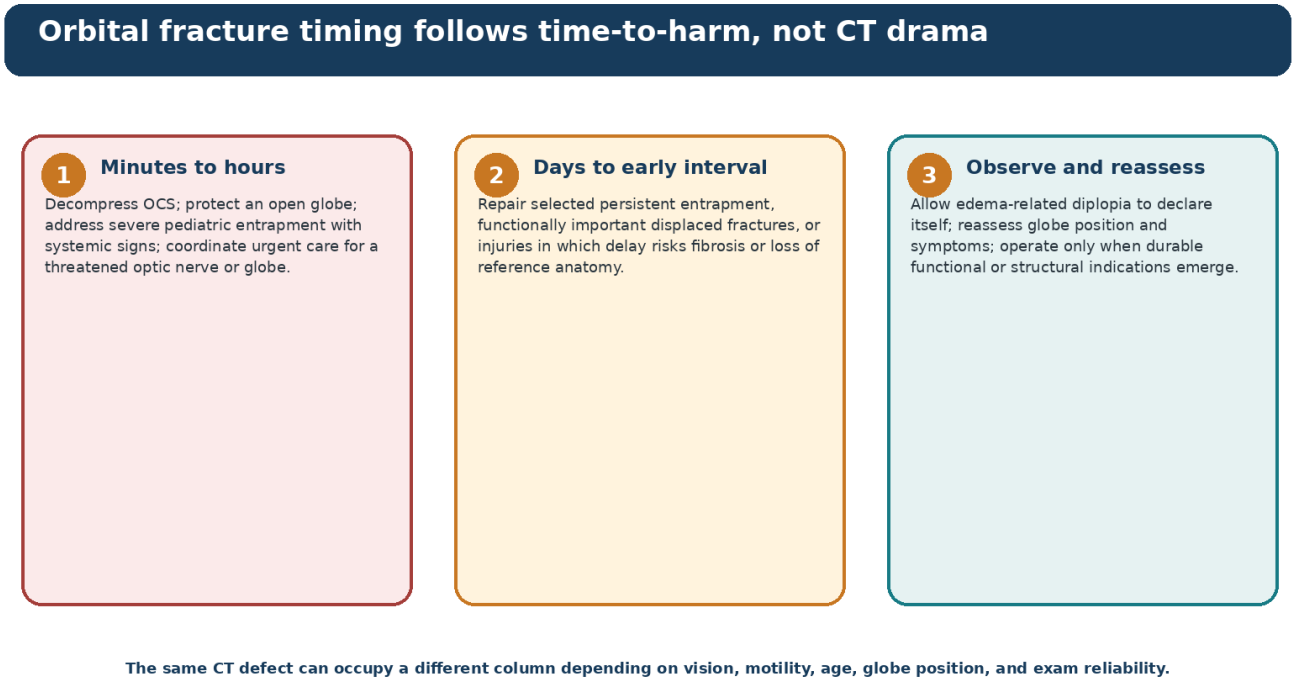

Figure 4.3. Timing is assigned by time to harm. The fracture image alone does not determine the column.

4.8 The decision statement

A useful assessment sounds like this: “The patient has a combined left floor-medial wall fracture with preserved vision, no RAPD, improving edema-related vertical diplopia, no oculocardiac symptoms, and no current enophthalmos. CT shows loss of the inferomedial contour but a visible posterior ledge. We will observe with sinus precautions and repeat motility and globe-position examination in one week; persistent primary-gaze diplopia or evolving enophthalmos would support reconstruction.”

RESIDENT SYNTHESIS

Orbital fracture management begins with a syndrome, not an area measurement. Localize diplopia, read the defect in three dimensions, identify stable boundaries, and assign a timeline based on vision, mechanical dysfunction, globe position, and anticipated durable deformity.

CHAPTER 5

Orbital Reconstruction

CENTRAL QUESTION

How do you restore the internal orbital contour while protecting vision, motility, and the eyelids?

OPENING CASE

During repair of a combined floor-medial defect, the anterior portion is easy to see, but the posterior ledge is indistinct. A broad implant appears stable anteriorly. Forced ductions are improved but not free. The temptation is to accept the reconstruction. The safer response is to ask whether tissue remains trapped, whether the implant truly reaches stable posterior bone, and whether the inferomedial contour has been recreated.

5.1 Define the reconstructive target before choosing the implant

Orbital reconstruction is not simply patching a hole. The target is restoration of the preinjury internal contour, release of displaced soft tissues, preservation of the globe and neurovascular structures, and stable support that will not migrate or sag. The desired endpoint is symmetric globe position and unrestricted movement, not radiographic coverage alone.

Preoperative planning should identify the anterior rim, intact medial and lateral boundaries, the posterior ledge, the inferomedial strut, the optic canal, and the full extent of the defect. Contralateral anatomy can serve as a template but is not perfectly symmetric. In combined facial injuries, the orbital rim and lateral wall may first require accurate reduction of the ZMC or NOE framework before internal reconstruction can be trusted.

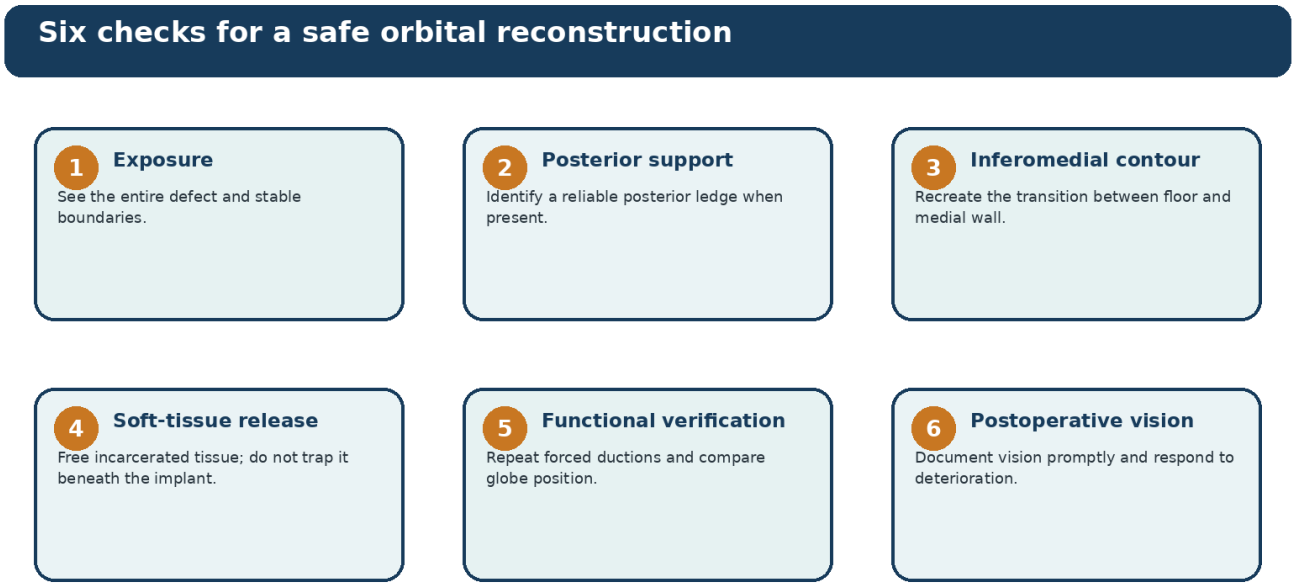

Figure 5.1. Six checks for orbital reconstruction. Most technical failures can be understood as inadequate exposure, incomplete release, inaccurate contour, or insufficient verification.

5.2 Surgical approaches are chosen by exposure and morbidity

Table 5.1. Orbital approaches. The best approach is the least morbid exposure that permits complete visualization and safe reconstruction.

Approach	Access	Advantages	Limitations / risks
Transconjunctival	Inferior rim and floor; extend retroseptally or preseptally	No external skin scar; low ectropion risk in experienced hands	Requires familiarity; possible entropion, conjunctival scar, or lower-lid retraction

Approach	Access	Advantages	Limitations / risks
Transcaruncular extension	Medial wall and inferomedial orbit	Direct medial access without external scar	Lacrimal, medial canthal, and posterior orbital structures require careful protection
Subtarsal	Inferior rim and floor through a natural crease	Direct exposure with a relatively inconspicuous scar	Visible scar and lower-lid malposition remain possible
Subciliary	Wide inferior orbital exposure	Familiar and direct	Higher concern for ectropion, scleral show, and lid retraction in comparative literature
Upper-eyelid / brow	Superior rim and selected roof or lateral wall injuries	Uses upper-lid crease or brow region	Limited for deep central roof; scar and supraorbital nerve considerations
Coronal	Broad frontal, superior, and lateral orbital exposure	Useful in panfacial, frontal sinus, and complex roof injuries	Larger operation; scalp numbness, alopecia, temporal branch risk, longer recovery

Incision choice and dissection plane are inseparable. The surgeon must preserve lower-lid support, identify the orbital septum and retractors, and obtain enough exposure to see the posterior defect rather than merely slide an implant into a tunnel. Comparative evidence generally favors transconjunctival access over subciliary incisions for ectropion and visible-scar risk, but technique and injury pattern remain decisive.[7,8]

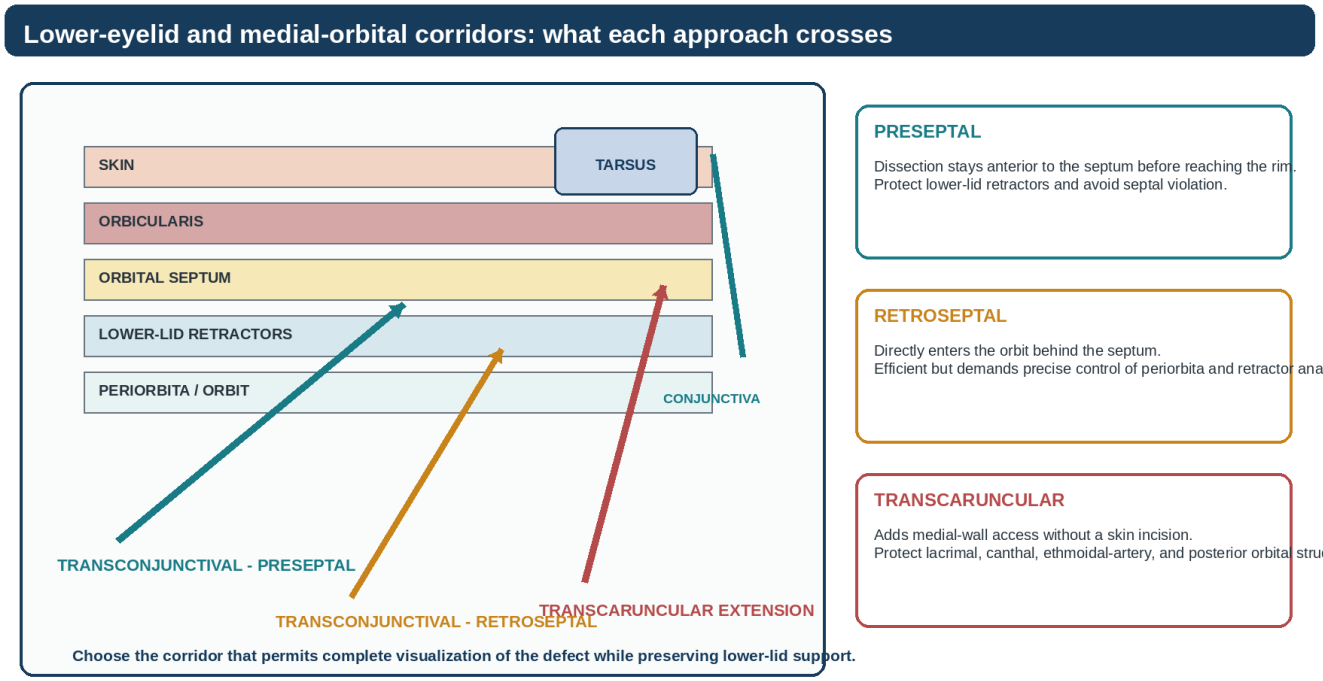

Figure 5.1A. Lower-eyelid and medial-orbital corridors. Preseptal and retroseptal transconjunctival dissections reach the rim through different relationships to the orbital septum; a transcaruncular extension adds medial-wall access.

Comparative studies generally associate transconjunctival approaches with less visible scarring and lower ectropion risk than subciliary incisions, although surgeon technique, injury pattern, canthotomy, and combined approaches influence outcomes.[7,8] The approach should not be selected by habit alone. A small incision that prevents visualization of the posterior defect is not minimally invasive if it produces an inaccurate reconstruction.

5.3 Exposure and release

After the rim or facial framework is reduced, elevate the periorbita carefully from stable bone toward the defect. Identify the entire fracture boundary. Prolapsed fat and muscle-fascial tissue are gently mobilized back into the orbit. Sharp fragments should be controlled rather than blindly swept. Posterior dissection becomes progressively less forgiving as the optic nerve and orbital vessels converge at the apex.

A common technical error is to create a tunnel just large enough to slide an implant across the visible anterior defect. This can leave posterior tissue incarcerated, misjudge the defect's true size, or place the implant on unstable fragments. Complete exposure is not an aesthetic preference; it is the foundation for safe support.

5.4 Implant principles

Available materials include titanium mesh, porous polyethylene, hybrid titanium-polyethylene constructs, resorbable materials, patient-specific implants, and selected bone grafts. No single material is ideal for every defect. The choice depends on defect size and location, need for contourability, visibility on imaging, infection environment, surgeon experience, cost, and whether removal would be difficult.

Table 5.2. Implant selection is a geometry and support decision, not a brand decision.

Material concept	Strength	Limitation	Best use logic
Titanium mesh	Rigid, contourable, radiopaque, stable for larger defects	Can adhere to tissue; sharp edges and difficult removal if malpositioned	Large or complex defects requiring durable, contourable support when precise placement can be verified
Porous polyethylene	Smooth handling; tissue incorporation; available in shaped forms	Less visible on standard CT; infection or removal can be challenging	Selected defects with stable support when surgeon experience and removal considerations favor it
Hybrid implant	Combines structural mesh with a smoother surface	Bulk and cost; still requires accurate contour	Complex defects where rigidity and soft-tissue interface both matter
Resorbable sheet	Avoids permanent implant	Limited long-term strength; inflammatory or contour concerns	Selected small defects with reliable bony boundaries; institution- and material-dependent
Patient-specific implant	Mirrors planned anatomy; useful in large or delayed defects	Planning time, cost, dependence on segmentation and reduction accuracy	Complex combined-wall, secondary, or bilateral reconstruction

5.4A Implant choice is subordinate to geometry

No implant material has a universal “best” indication. Material selection is influenced by defect size and shape, stable boundaries, contamination, revision risk, cost, local inventory, and surgeon experience. The essential requirements are accurate contour, stable support, complete soft-tissue release, safe distance from the apex, and verification that movement has not worsened.

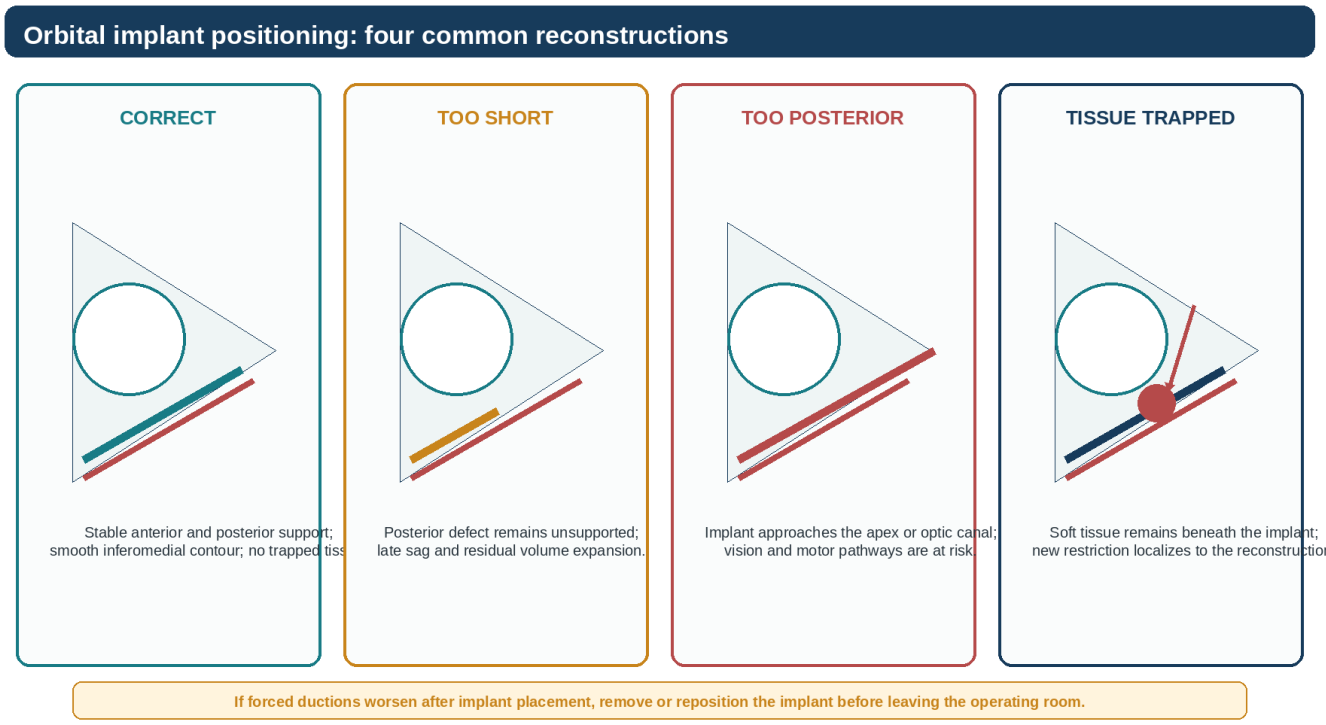

Figure 5.2A. Orbital implant errors are geometric. A reconstruction can fail because it is too short, extends too close to the apex, sags without posterior support, or traps tissue despite covering the visible defect.

Regardless of material, the implant should bridge stable bone, reproduce the internal contour, avoid the optic canal, and remain stable without trapping tissue. Fixation may be unnecessary for a well-seated implant with broad support; other constructs

require rim fixation. The implant must not project into the orbital apex or nasal cavity, impinge the lacrimal system, or create a sharp edge beneath mobile tissues.

5.5 The posterior ledge and inferomedial transition

The posterior ledge is the stable posterior floor on which an implant can rest. It may be narrow, comminuted, or absent. Inadequate posterior support allows the reconstruction to fall into the sinus, leaving the posterior orbit expanded. The posterior dissection should remain subperiosteal and controlled; unnecessary pursuit toward the apex can threaten the optic nerve.

In combined floor-medial defects, the implant must recreate a smooth transition rather than two disconnected flat surfaces. The inferomedial strut, when preserved, can guide and support the reconstruction. When absent, preformed or patient-specific implants and navigation may improve reproducibility, but technology does not replace direct confirmation that the implant sits on stable boundaries.

5.6 Intraoperative verification

- Inspect the full implant perimeter when possible and ensure no orbital tissue lies beneath it.
- Repeat forced-duction testing after release and again after implant placement.
- Compare globe position, projection, and vertical level with the contralateral side while accounting for edema.
- Palpate or visualize posterior support and the inferomedial contour.
- Use intraoperative CT, cone-beam CT, navigation, or endoscopic visualization selectively in complex defects.
- If motility worsens after implant placement, remove or reposition the implant before leaving the operating room.

OPERATIVE PEARL

A free forced duction before implant placement followed by restriction afterward localizes the problem to the reconstruction until proven otherwise.

5.7 Medial wall and endoscopic adjuncts

Medial-wall defects may be approached through the transcaruncular corridor, an extended transconjunctival approach, or selected endonasal endoscopic routes. Endoscopy can help visualize the medial orbital boundary, reduce herniated tissue from the ethmoid side, or confirm implant position. The surgeon must protect the medial rectus, anterior and posterior ethmoidal arteries, skull base, lacrimal structures, and optic nerve.

Endoscopic assistance is most valuable when it improves visualization of a boundary that would otherwise be uncertain. It should not encourage blind pressure from the sinus side or substitute for control of the orbital contents.

5.8 Orbital roof fractures

Roof fractures range from minimally displaced injuries to blow-in fragments that reduce orbital volume or blow-out injuries that communicate with the cranial cavity. Associated frontal sinus, dural, brain, and supraorbital rim injuries are common. Indications for intervention include vision or globe compromise, significant displacement affecting orbital volume, CSF leak requiring repair, pulsatile proptosis, growing skull fracture in children, or the need for cranial access for associated injuries.

Repair may require collaboration with neurosurgery through a coronal or cranial approach. The reconstructive goal is not merely a smooth roof; it is safe separation of orbit and intracranial contents without compressing the globe or optic pathway. Postoperative vision must be checked as soon as feasible because retrobulbar hematoma is a recognized high-consequence complication after orbital surgery.[1,9]

5.9 Postoperative care

Table 5.3. Postoperative orbital care prioritizes vision before appearance.

Domain	Immediate check	Ongoing care
Vision	Acuity, pupils/RAPD, color when feasible, pain, proptosis	Repeat for any symptom change; low threshold for urgent decompression
Motility	EOMs and diplopia pattern when awake	Expect edema; investigate new or worsening restriction
Eyelid	Closure, position, corneal protection	Lubrication, edema control, monitor ectropion/entropion
Wound / sinus	Bleeding, drainage, emphysema	Sinus precautions, nasal care, institution-specific antibiotics
Imaging	Selected postoperative CT for complex or uncertain reconstructions	Use to confirm contour and implant position, not as a substitute for exam

Supportive care includes head elevation, cold compresses, analgesia, antiemetics, and sinus precautions. Avoid prolonged prophylaxis for closed, nonoperative fractures; AAST guidance recommends none.[10] For operative injuries, tailor perioperative prophylaxis to contamination and local protocol; courses beyond 24 hours are generally unsupported in uncomplicated cases.

5.9 Postoperative orbital care

The first postoperative question is whether vision is stable. Recheck acuity, pupils/RAPD, color when feasible, pain, proptosis, motility, and eyelid position. New visual decline, a tense orbit, or worsening restriction demands immediate assessment for pressure, hemorrhage, tethering, or implant malposition.

For operative fractures, perioperative prophylaxis varies with contamination and local protocol, but courses beyond 24 hours generally lack supporting evidence in uncomplicated cases.[10]

5.10 Complications and how they arise

Table 5.4. Orbital complications are traceable to pressure, tethering, contour, or soft-tissue injury.

Complication	Mechanism	Prevention / response
Vision loss / OCS	Bleeding, edema, tight closure, implant compression	Objective checks; immediate decompression for clinical OCS.
Persistent diplopia	Residual tethering, muscle/nerve injury, impingement, scarring	Complete release, forced ductions, accurate implant; reassess trajectory.
Enophthalmos / hypoglobus	Under-reconstructed volume, missed medial defect, implant sag	Restore posterior and inferomedial contour; revise when functionally important.
Exophthalmos	Overcorrection, hematoma, implant displacement	Urgent pressure assessment; revise malposition.
Eyelid malposition	Approach, scarring, canthal disruption, retraction	Gentle technique, appropriate corridor, early care or later revision.
Infection / extrusion	Contamination, sinus disease, exposure, dead space	Treat infection, drain when needed, and remove implant selectively.

Mechanism-first review

1 Pressure
Vision falls; orbit becomes tense. Decompress the syndrome.

2 Tethering
Restriction changes after release or implant placement.

3 Contour
Globe position reflects posterior and inferomedial support.

4 Soft tissue
Scarring, muscle injury, and eyelid support shape function.

RESIDENT SYNTHESIS
Successful reconstruction requires exposure, complete soft-tissue release, restoration of posterior and inferomedial contour, and objective verification. After surgery, vision stability outranks appearance.

CHAPTER 6

Frontal Sinus and Anterior Skull Base Anatomy

CENTRAL QUESTION

Which structures determine whether a frontal sinus can be preserved safely?

OPENING CASE

CT shows an anterior-table depression, a subtle posterior-table fracture, and opacification in the frontal recess. The patient has no visible CSF leak. A single label - “frontal sinus fracture” - cannot answer the management question. The resident must determine whether the contour is acceptable, whether the intracranial boundary is stable, and whether the sinus will remain able to drain.

6.1 The frontal sinus is an air cell system with a narrow exit

The paired frontal sinuses develop variably and are frequently asymmetric. They sit between an anterior table that shapes the forehead and a posterior table adjacent to the frontal lobes and dura. Inferomedially, each sinus narrows toward the frontal recess and drains into the middle meatus through a pathway shaped by the frontal beak, agger nasi region, ethmoid cells, and surrounding mucosa. The pathway is not a simple rigid duct, which is why the term frontal sinus outflow tract is more useful than a single “nasofrontal duct.”

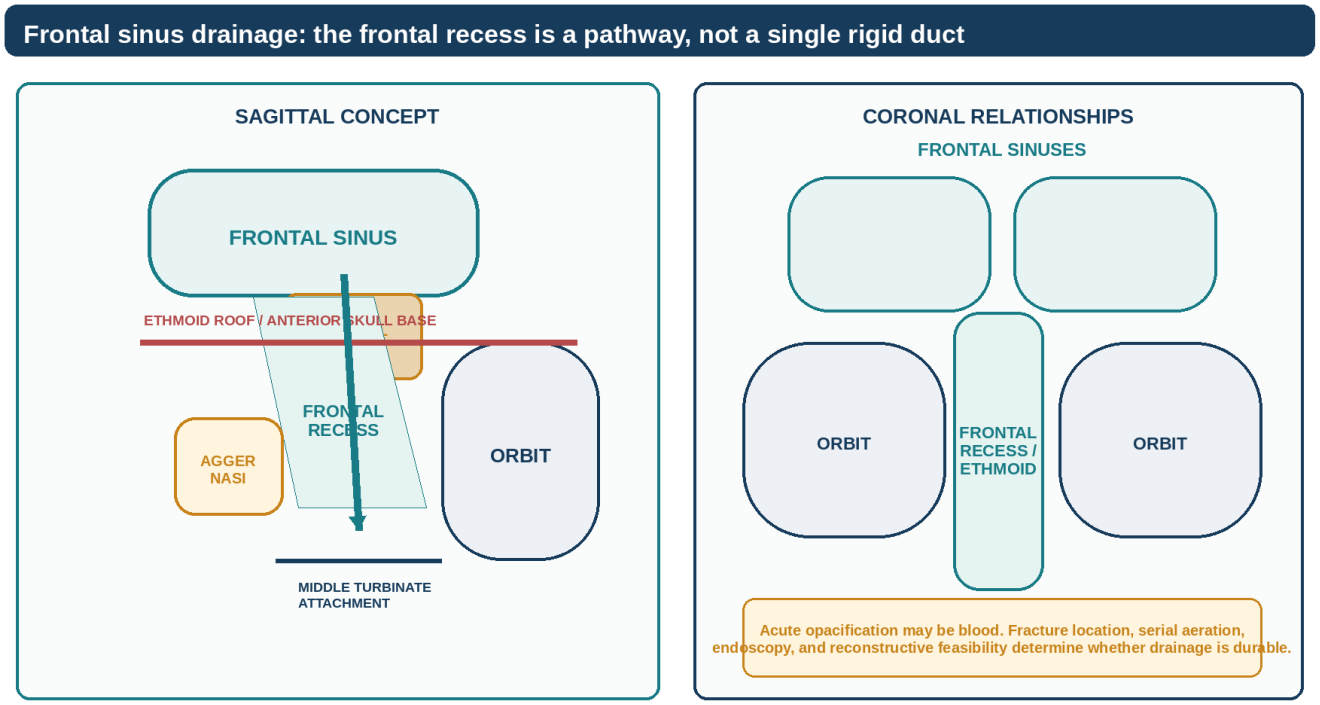

Figure 6.1. Frontal sinus drainage funnels inferomedially through the frontal recess, whose shape depends on the frontal beak, agger nasi region, ethmoid cells, skull base, orbit, and middle-turbinate attachment. It is a pathway rather than a single rigid duct.

6.2 Anterior table: contour and access

The anterior table is thicker than the posterior table and provides the visible forehead contour. Displacement can produce a palpable or visible depression, but acute edema may hide the deformity. The anterior table also serves as a surgical access route to the sinus. Its management is therefore driven primarily by contour, comminution, soft-tissue injury, and whether broader sinus or skull-base intervention is required.

A minimally displaced anterior-table fracture with acceptable contour and no other sinus variable may be observed. A displaced defect may be reduced and fixated to restore contour. The need for contour repair does not automatically require sacrificing sinus function. Conversely, a cosmetically acceptable anterior table does not prove that the posterior table or outflow tract is safe.

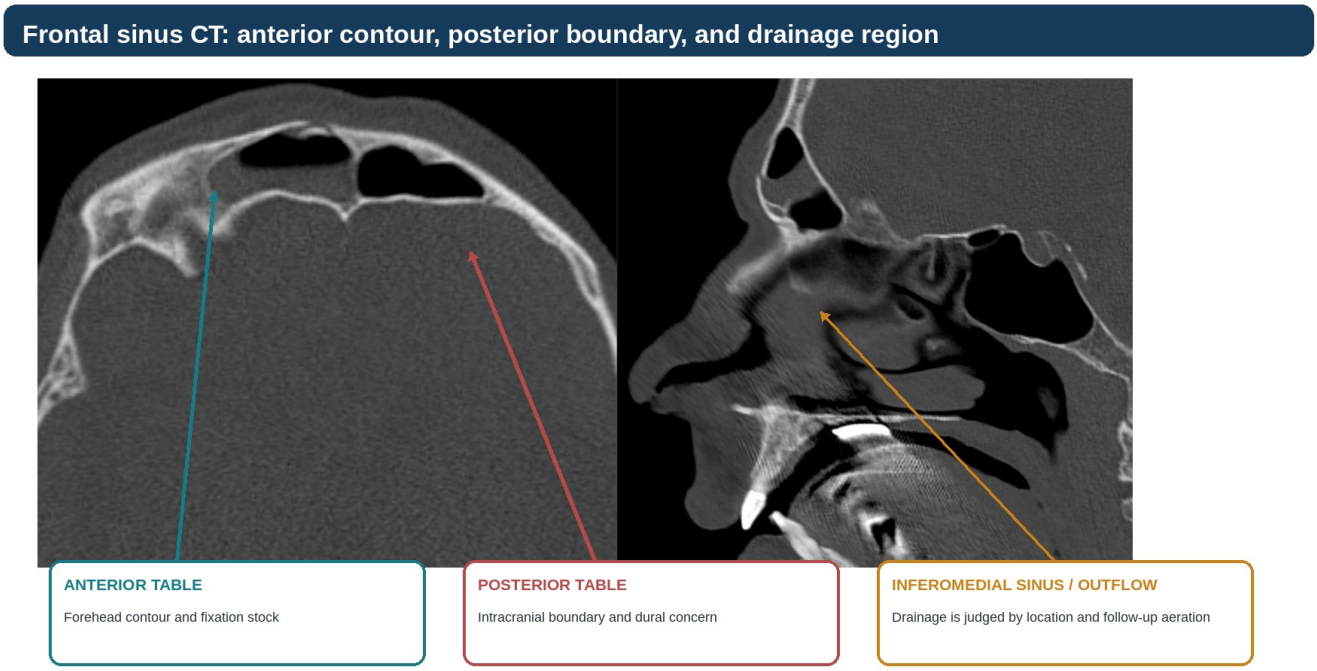

Figure 6.2. Annotated axial and sagittal CT views of an anterior-table frontal sinus fracture. The report should separately address contour, posterior-table integrity, inferomedial drainage anatomy, dura/CSF status, and comminution. Source: Hellerhoff, Wikimedia Commons, CC BY-SA 4.0; adapted with annotations.

6.3 Posterior table: intracranial boundary

The posterior table forms part of the anterior cranial floor. Displacement, comminution, and associated pneumocephalus raise concern for dural violation. Yet posterior-table involvement exists on a spectrum. A nondisplaced fracture without CSF leak or intracranial complication may be observed in selected patients. Severe comminution, major displacement, persistent CSF leak, or a posterior boundary that cannot be reconstructed reliably pushes management toward cranialization or combined repair.

The historical practice of treating any posterior-table fracture aggressively has softened as imaging, endoscopic techniques, and surveillance have improved. The contemporary question is whether the sinus can remain safely separated from the cranial cavity - not simply whether a fracture line touches the posterior wall.

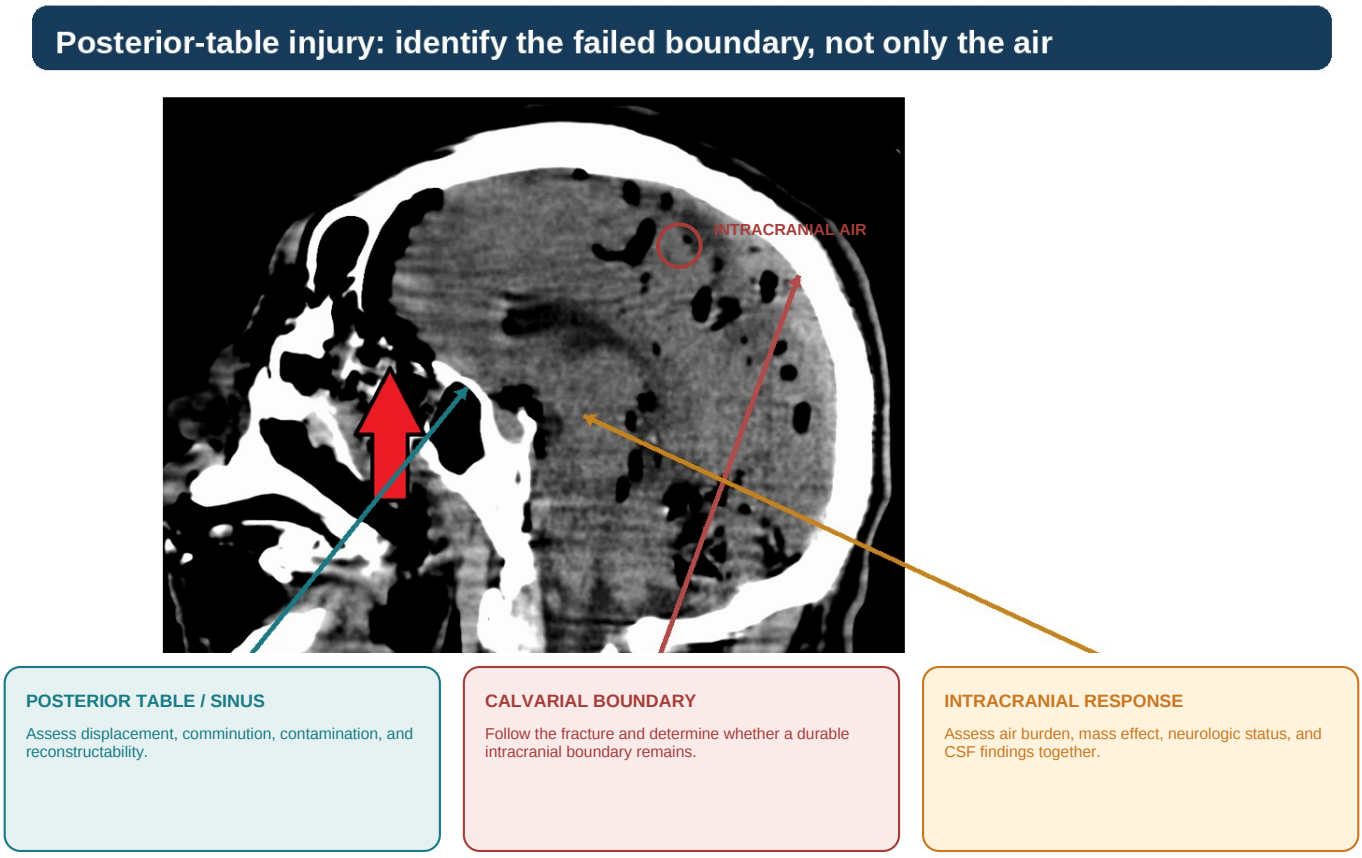

Figure 6.3. Sagittal CT with extensive pneumocephalus and comminuted frontal sinus/skull-base injury. Air confirms boundary failure, but urgency and operative planning depend on neurologic status, mass effect, posterior-table stability, dura/CSF status, outflow anatomy, contamination, and associated brain injury. Source: James Heilman, MD, Wikimedia Commons, CC BY-SA 4.0; annotations adapted.

6.4 Frontal outflow tract: the determinant of late sinus health

A preserved sinus remains safe only if mucosa can clear secretions through a patent outflow pathway. Fractures through the inferomedial sinus, frontal beak, or adjacent NOE and ethmoid complex can obstruct drainage directly. Even when the bony channel appears open, edema, displaced mucosa, scarring, and neo-osteogenesis can produce delayed stenosis. Conversely, CT opacification in the acute setting may represent blood rather than permanent obstruction.

Evaluation therefore combines fracture location, sagittal and coronal CT anatomy, endoscopic findings when available, and follow-up evidence of re-aeration. Contemporary series support observation of selected FSOT injuries with serial CT, reflecting a broader shift toward sinus preservation when drainage can recover.[11–13] This strategy depends on reliable surveillance and a willingness to intervene later if the sinus remains obstructed.

6.5 Dura and CSF

A CSF leak proves that the barrier has failed, but absence of visible rhinorrhea does not exclude a dural tear. Blood, edema, recumbency, and intermittent flow can conceal leakage. Pneumocephalus, intracranial air adjacent to a fracture, or a posterior-table fragment displaced intracranially increases suspicion. When clear drainage is present, laboratory confirmation and targeted localization are discussed in Chapter 8.

6.6 Mucosa is the source of both function and late disease

Frontal sinus mucosa supports mucociliary clearance when the sinus is preserved. The same mucosa can generate a mucocele if trapped behind an obstructed outlet or retained within an incompletely obliterated or cranialized sinus. This creates a central operative principle: either preserve viable mucosa with dependable drainage, or remove mucosa meticulously when excluding the sinus. A halfway operation - damaged drainage with retained secretory mucosa - creates the conditions for delayed disease.

6.7 The five-variable framework

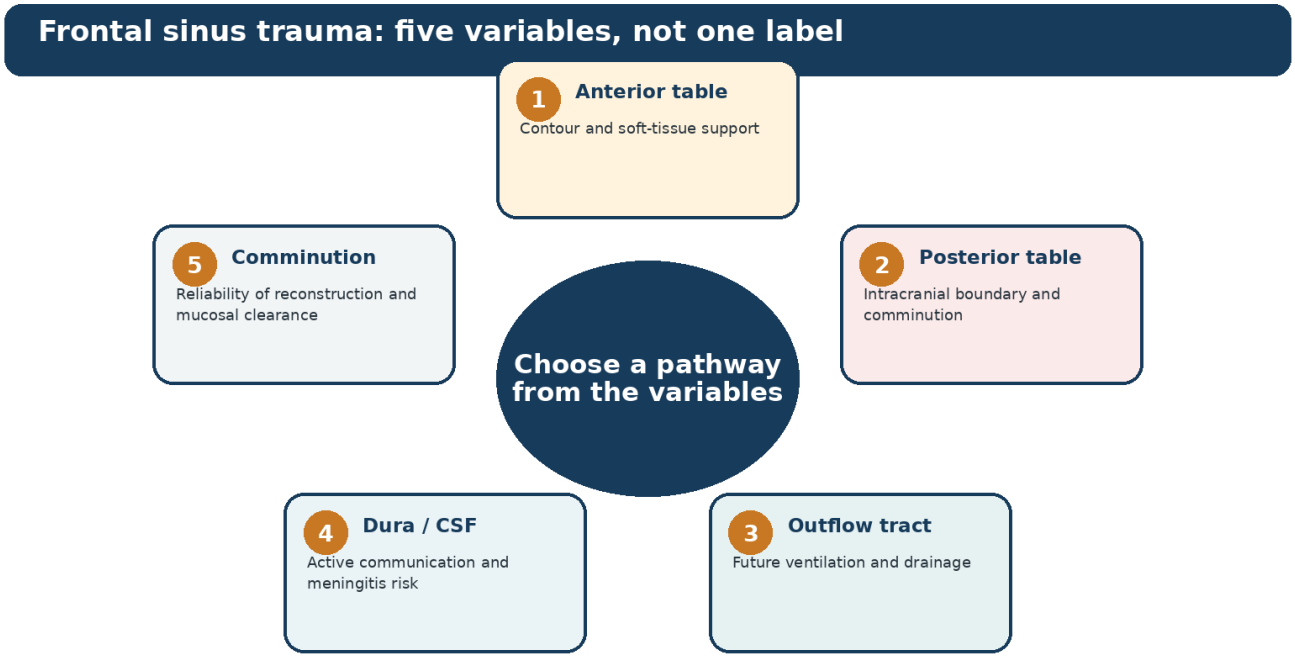

Figure 6.4. Five variables organize frontal sinus management: anterior table, posterior table, outflow tract, dura/CSF status, and comminution. AO Surgery Reference similarly frames decisions around these anatomic parameters.[14]

Table 6.1. The five-variable frontal sinus assessment.

Variable	Question	If favorable	If unfavorable
Anterior table	Is the forehead contour acceptable?	Observe or preserve during other repair	Reduce/fix or reconstruct contour
Posterior table	Is the intracranial boundary stable and reconstructable?	Observe or repair selectively	Cranialization or combined cranial repair may be required
Outflow tract	Will the sinus ventilate and drain?	Preserve with surveillance	Endoscopic restoration, obliteration, or other exclusion strategy
Dura / CSF	Is there an active communication?	Observe selected early leaks with monitoring	Repair persistent or complicated leak
Comminution	Can mucosa and bony boundaries be managed reliably?	Anatomic repair more feasible	Higher likelihood of ablation or cranialization

6.8 CT review of the frontal sinus and skull base

- Review axial bone windows for anterior/posterior displacement, comminution, and intracranial air.
- Use sagittal images to follow the frontal beak and outflow pathway inferiorly.
- Use coronal images to assess the frontal recess, ethmoid roof, cribriform region, and associated NOE injury.
- Inspect soft-tissue windows for frontal lobe contusion, epidural collection, orbital injury, and sinus contents.
- Distinguish acute blood and edema from a persistently non-aerated sinus on follow-up imaging.
- Look beyond the frontal sinus for multifocal anterior skull-base defects.

DIAGNOSTIC TRAP

An opacified frontal sinus immediately after trauma is not equivalent to an obstructed outflow tract. The question is whether the sinus re-aerates and drains after acute blood and edema resolve.

RESIDENT SYNTHESIS

The frontal sinus is safe when contour is acceptable, the intracranial boundary is reliable, drainage is functional, and mucosa is either preserved appropriately or removed completely. The five-variable framework prevents one fracture label from hiding several independent decisions.

CHAPTER 7

Frontal Sinus Fracture Decision-Making

CENTRAL QUESTION

How do the five variables lead to observation, contour repair, sinus preservation, obliteration, or cranialization?

OPENING CASE

A patient has a displaced anterior-table fracture and CT concern for frontal outflow tract injury, but the posterior table is intact and there is no CSF leak. One surgeon proposes immediate obliteration. Another proposes anterior-table repair and surveillance for sinus re-aeration. The disagreement is not simply technical; it reflects a broader shift from prophylactic sinus ablation toward selective preservation.

7.1 Begin with goals, not procedures

Frontal sinus procedures are best understood by the problem each solves. Observation accepts the existing anatomy and monitors healing. Anterior-table repair restores contour. Sinus-preserving management maintains or re-establishes ventilation and drainage. Obliteration excludes a sinus that cannot be trusted to drain while preserving the posterior table. Cranialization removes the posterior table and incorporates the former sinus into the intracranial space while sealing the sinonasal tract.

Figure 7.1. Frontal sinus pathways are goal-driven. Contemporary management increasingly preserves the sinus when drainage and intracranial separation are reliable.

7.2 Observation

Observation is appropriate when the anterior contour is acceptable, the posterior table is stable, no persistent CSF leak exists, and the outflow pathway is intact or reasonably likely to recover. Observation is not discharge from responsibility. The plan should define clinical follow-up, symptoms of obstruction or infection, and the timing of repeat CT to document re-aeration when the outflow tract is in question.

Selected posterior-table and FSOT injuries can also be observed when displacement is limited, intracranial complications are absent, and surveillance is dependable. The threshold varies by institution and by the ability to obtain long-term follow-up. A strategy that is safe in a mature skull-base program with endoscopic rescue capacity may not translate unchanged to every setting.

7.3 Anterior-table contour repair

The indication for isolated anterior-table repair is usually an unacceptable contour deformity rather than sinus disease. Acute edema can conceal depression, so preinjury photographs and delayed examination help. Approaches include repair through an existing laceration, limited brow or upper-eyelid access, endoscopic assistance, and coronal exposure for broad comminution or combined injuries. The goal is restoration of the forehead contour without unnecessary violation of the sinus or supraorbital neurovascular structures.

Comminuted fragments can be reduced and stabilized with low-profile fixation or reconstructed with mesh, bone graft, or patient-specific implants when native contour cannot be restored. If the sinus remains functional, the mucosa and outflow tract should be protected. If the fracture is part of a larger operation requiring sinus entry, the surgeon must still decide deliberately whether the sinus will be preserved or excluded.

7.4 Sinus-preserving management and delayed endoscopic rescue

Sinus preservation has become more common because many acute FSOT injuries re-aerate and because endoscopic frontal sinus techniques can address selected delayed obstruction. A preservation strategy may consist of fracture observation or repair, avoidance of unnecessary mucosal stripping, and interval CT. If the sinus fails to re-aerate or the patient develops frontal symptoms, endoscopic frontal sinusotomy, trephination-assisted evaluation, or other targeted treatment may restore drainage without ablating the sinus.

This strategy is most defensible when the posterior boundary is safe, the patient can return for imaging, and the anatomy remains amenable to rescue. It is less attractive when the outflow tract is destroyed, follow-up is unreliable, severe comminution prevents confident mucosal management, or the sinus must already be opened widely for posterior-table or dural repair.

> **EVIDENCE NOTE**
>
> Recent retrospective studies document successful conservative management and re-aeration in selected FSOT injuries, but high-quality comparative trials remain limited. The trend toward preservation is evidence-informed rather than absolute.[11–13]

7.5 Obliteration

Obliteration removes sinus mucosa, seals the outflow tract, and fills or excludes the sinus cavity while leaving the posterior table in place. Historically, it was used broadly for FSOT injury. Contemporary use is more selective because retained mucosa and incomplete outflow-tract closure can produce mucocele, and because some drainage injuries recover or can be managed endoscopically.

When obliteration is chosen, meticulous technique matters more than the filler material. All visible mucosa must be removed, including within recesses and fracture lines. The outflow tract must be sealed. The cavity may be filled with autologous fat, bone, muscle, or other materials, or managed according to institutional technique. The operation should leave no functioning mucosal pocket without drainage.

7.6 Cranialization

Cranialization is used when the posterior table and sinus-brain boundary cannot be preserved safely. The posterior table is removed, dura repaired, sinus mucosa eliminated, the outflow tract sealed, and vascularized tissue separates the intracranial and sinonasal spaces.

Indications generally include severe posterior-table comminution or displacement that prevents a reliable boundary, a large unreconstructable defect, persistent CSF leak requiring open cranial access, or an associated neurosurgical indication. A 2026 systematic review and meta-analysis found no significant overall complication-rate difference among cranialization, noncranialization, endoscopic repair, and observation. Because the evidence is heterogeneous and treatment is selected by anatomy, this is not proof that pathways are interchangeable: displacement alone should not replace assessment of dura/CSF status, FSOT function, contamination, reconstructive feasibility, and the independent need for craniotomy.[14-16,31]

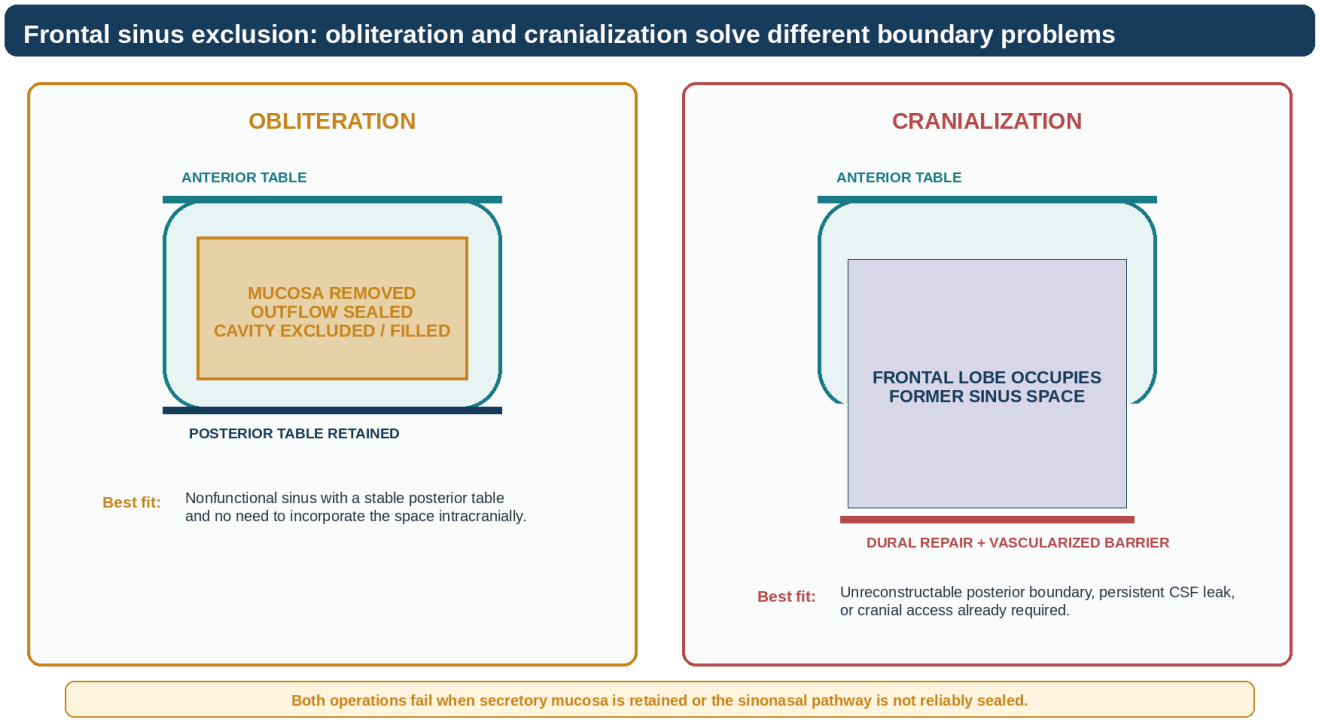

Figure 7.1A. Obliteration preserves the posterior table while excluding a nonfunctional sinus; cranialization removes the posterior table and reconstructs the dura and sinonasal barrier so the frontal lobes can occupy the former sinus space.

Table 7.1. Frontal sinus procedures and the failure each is designed to prevent.

Pathway	Primary goal	Essential technical requirement	Principal failure mode
Observation	Allow safe healing and recovery of drainage	Defined clinical and radiographic surveillance	Missed persistent obstruction or delayed complication
Contour repair	Restore forehead shape	Stable anatomic reduction while preserving sinus strategy	Contour irregularity, hardware visibility, sensory symptoms
Sinus preservation	Maintain mucociliary function	Patent or recoverable FSOT and intact intracranial barrier	Delayed stenosis, sinusitis, mucocele
Obliteration	Exclude a nonfunctional sinus	Complete mucosal removal and reliable FSOT closure	Retained mucosa or incomplete isolation
Cranialization	Create durable intracranial-sinonasal separation	Posterior-table removal, dural repair, FSOT seal, vascularized barrier	CSF leak, infection, intracranial complication

7.7 Pericranial flap and vascularized separation

A vascularized pericranial flap is a workhorse for open anterior skull-base separation. It is elevated through a coronal approach and based anteriorly on the supraorbital and supratrochlear vascular supply. The flap can cover repaired dura, the cranialized sinus, and the sealed frontal recess. Prior surgery, laceration, radiation, or avulsion can compromise its reliability, making alternative tissue necessary.

The operative principle is layered closure: repair the dural defect when possible, eliminate mucosal contamination, seal the communication, and interpose vascularized tissue between intracranial contents and the sinonasal tract. Hardware and bone grafts should not substitute for a soft-tissue barrier when the dura and sinus have communicated.

7.8 Antibiotics and meningitis prevention

Open contaminated wounds and operative craniofacial procedures may justify short-course perioperative prophylaxis according to trauma and institutional protocols. Prolonged antibiotics do not compensate for an unclosed CSF fistula or devitalized infected tissue, and routine prophylaxis has not consistently prevented meningitis in uncomplicated traumatic CSF leaks. Once a cranial CSF leak is confirmed, review pneumococcal vaccination history and follow current CDC risk-based recommendations; the indicated product and interval depend on age, prior vaccines, and other risk conditions.[10,29]

7.9 Follow-up after frontal sinus trauma

Follow-up must match the chosen pathway. For observed frontal sinus outflow tract injury, interval CT documents re-aeration after acute blood and edema resolve; a practical framework is an early checkpoint around 6 weeks and a later checkpoint around 12 months, adjusted to anatomy, symptoms, and local protocol. Contemporary reviews report that many selected mild-to-moderate outflow injuries re-aerate, but surveillance is part of treatment, not an optional add-on.[11,22,32]

A practical surveillance timeline

1 Immediate
Document injury, treatment, symptoms, olfaction limits, and a return plan.

2 ~6 weeks
Clinical review and CT for observed FSOT injury when used by local protocol.

3 ~12 months
Confirm durable aeration or identify persistent obstruction in selected patients.

4 Lifelong
New swelling, pain, infection, diplopia, proptosis, or neurologic symptoms require imaging.

Pathway-specific follow-up

Pathway	Early priorities	Late failure to teach
Observed FSOT injury	Symptom review and interval CT to document re-aeration.	Persistent obstruction, recurrent sinusitis, mucocele.
Anterior-table repair / preserved sinus	Contour, wound, hardware, drainage, and sinus aeration.	Contour or hardware symptoms; delayed obstruction.
Obliteration / cranialization	Wound, neurologic, infection, CSF, and contour assessment.	Retained-mucosa mucocele or delayed infection can still occur.

PATIENT COUNSELING
A mucocele may present years or decades later. New forehead swelling, frontal pain, recurrent infection, proptosis, diplopia, CSF symptoms, or neurologic change should prompt imaging even when the original trauma is remote.

RESIDENT SYNTHESIS
Frontal sinus management is a choice among goals: accept safe anatomy, restore contour, preserve drainage, exclude a nonfunctional sinus, or rebuild the intracranial boundary. Follow-up verifies that the chosen goal remains durable.

CHAPTER 8

CSF Leak, Complications, and Surveillance

CENTRAL QUESTION

How do you confirm a skull-base leak, decide when it can be observed, and prevent early and late failure?

OPENING CASE

A patient with an anterior skull-base fracture has intermittent clear drainage when leaning forward. The first sample is mixed with blood and cannot be tested. CT shows ethmoid-roof and posterior frontal sinus fractures, but no single obvious defect. The patient is neurologically stable. The next steps are to confirm that the fluid is CSF, localize the communication, and decide whether an early traumatic leak is likely to close or requires repair.

8.1 Recognizing CSF rhinorrhea

Traumatic CSF rhinorrhea may appear immediately or after edema and clot resolve. The drainage is typically clear and watery, may increase with leaning forward or straining, and can be associated with a salty or metallic taste. Unilateral drainage is easier to recognize than posterior drainage into the pharynx. The classic halo sign on gauze and bedside glucose testing are not sufficiently specific to establish the diagnosis.

The history should include meningitis, prior sinus or skull-base surgery, previous trauma, and symptoms of elevated or low intracranial pressure. Examination includes nasal endoscopy when safe, inspection for pulsatile drainage or skull-base defects, cranial nerve assessment, and review of all fracture sites. Avoid provocative maneuvers that deliberately increase intracranial pressure simply to produce a leak.

8.2 Laboratory confirmation

Beta-2 transferrin is highly specific for CSF and perilymph and is widely used to confirm rhinorrhea. Beta-trace protein is another useful marker where available, often with faster processing. A clean sample improves diagnostic yield; blood contamination, insufficient volume, and intermittent leakage can delay confirmation. A negative test in a poor sample does not override a compelling syndrome.

8.3 Localizing the defect

High-resolution thin-cut CT of the skull base is the first-line localization study because it demonstrates bone defects, pneumocephalus, sinus opacification, and associated fractures. MRI or MR cisternography can identify meningoencephaloceles and soft-tissue pathways, particularly when CT shows several possible defects. CT cisternography or intrathecal contrast studies are reserved for selected cases because they are invasive and perform best when the leak is active.

Intrathecal fluorescein can assist intraoperative localization in experienced centers but is off-label in many jurisdictions and carries dosing-related risks. It should be used under a defined institutional protocol rather than as a casual diagnostic test.

OLFACTION DOCUMENTATION

When the patient is awake and nasal testing is safe and interpretable, ask about preinjury function and, when feasible, test each side with a familiar nonirritating odor. If bleeding, obstruction, cognition, or skull-base precautions prevent testing, record that limitation and reassess later. Counsel patients that traumatic hyposmia or anosmia may persist.

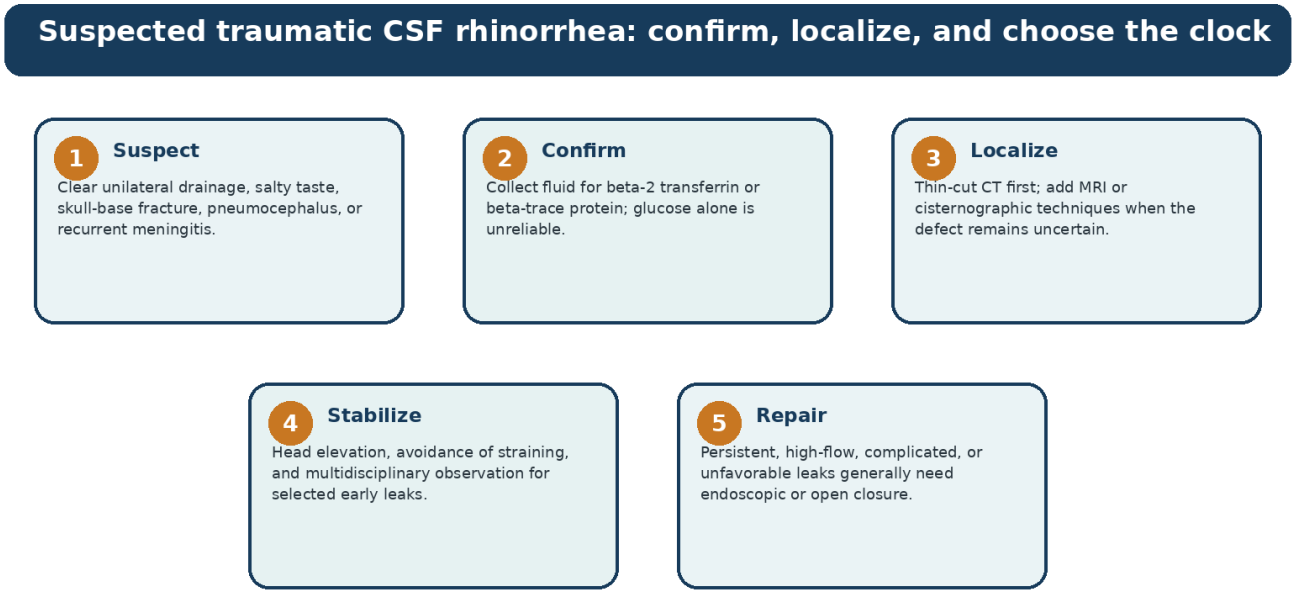

Figure 8.1. The CSF leak workflow: suspect, confirm, localize, stabilize, and repair according to persistence, flow, anatomy, and associated injury.

8.4 Initial conservative management

Many early low-flow traumatic leaks close as edema resolves and dural edges seal. In a neurologically stable patient without an independent surgical indication, a short observation interval may include head elevation, avoidance of straining and nose blowing, stool softening, antiemetics, and close monitoring for meningitis or increasing pneumocephalus. Lumbar drainage is selective; overdrainage can worsen headache, pneumocephalus, or herniation and must not postpone repair of a high-flow leak, large defect, encephalocele, or injury already requiring cranial surgery.

8.5 Indications to repair

- Persistent leak despite an appropriate observation interval.
- High-flow leak, large bony/dural defect, or meningoencephalocele unlikely to close reliably.
- Recurrent meningitis, delayed leak, progressive pneumocephalus, or tension physiology.
- Penetrating or contaminated injury, associated intracranial operation, or inability to create durable separation.

> **DO NOT MISS - TENSION PNEUMOCEPHALUS**
> Progressive intracranial air with neurologic deterioration or mass effect is a neurosurgical emergency. Stabilize the patient and obtain immediate neurosurgical assessment for decompression and definitive closure. Avoid nonessential CPAP/BiPAP, unnecessary high airway pressures, and nitrous oxide while an active communication is suspected. Airway and oxygenation still take priority; when positive-pressure ventilation is necessary, use the lowest effective pressures.

Contemporary endoscopic endonasal repair is central for many accessible anterior skull-base leaks. A trauma-specific systematic review reported lower complication and fistula-recurrence rates after endoscopic than open repair, although case selection and defect location affect that comparison. Open or combined approaches remain necessary for lateral frontal sinus defects, extensive comminution, injuries requiring craniotomy, or anatomy inaccessible from below.[17,30]

8.6 Principles of repair

Localize the defect, prepare a healthy circumferential bed, and build a multilayer closure matched to defect size, flow, intracranial pressure, access, and available vascularized tissue. The endpoint is durable separation of intracranial and sinonasal spaces.

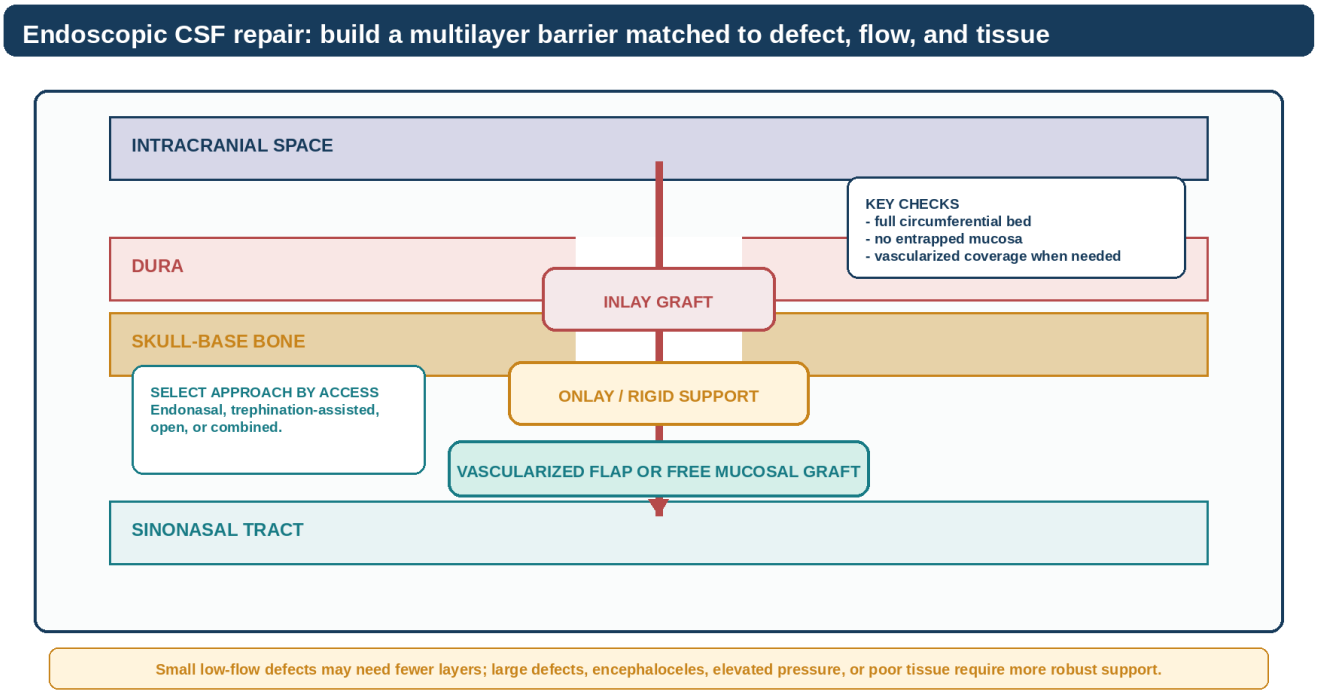

Figure 8.1A. Multilayer endoscopic skull-base repair. Layer number and material are matched to defect size, location, flow, intracranial pressure, and available vascularized tissue; the durable endpoint is complete separation of intracranial and sinonasal spaces.

A leak through the posterior frontal sinus or lateral recess can be less accessible endonasally and may require frontal trephination, Draf procedures, an osteoplastic approach, or cranialization. The correct approach is the one that permits definitive visualization and closure, not the one that is least invasive on paper.

8.7 Meningitis and infection

A persistent communication exposes the subarachnoid space to sinonasal organisms. Fever, meningismus, altered mental status, worsening headache, or systemic illness requires urgent evaluation and empiric treatment according to meningitis protocols. Prophylactic antibiotics have not reliably prevented meningitis in closed basilar skull fractures or uncomplicated CSF leaks; durable closure and early recognition of infection are more important than prolonged suppressive therapy.

8.8 Early orbital and frontal complications

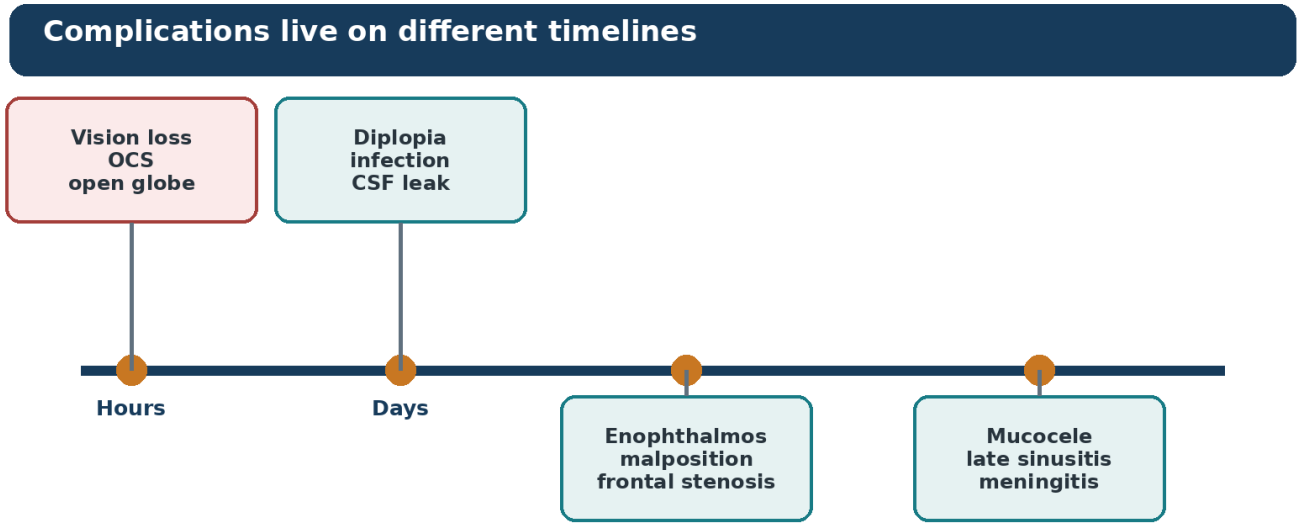

Figure 8.2. Upper-face complications occur from hours to decades. The follow-up plan must span more than the immediate postoperative period.

Table 8.1. Complications by clock.

Time	Complication	Mechanism	Response
Hours	OCS or visual decline	Hemorrhage, edema, implant compression	Immediate exam and decompression when indicated
Days	CSF leak or pneumocephalus	Unsealed dural/skull-base defect	Observe selected low-flow leak; repair persistent or complicated defects
Days-weeks	Infection	Contamination, devitalized tissue, retained collection	Drain/source control and culture-directed antibiotics
Weeks-months	Persistent diplopia or enophthalmos	Tethering, nerve injury, under-reconstructed orbit	Serial measurement; prism, strabismus input, or revision as appropriate
Months-years	Frontal stenosis, sinusitis, mucocele	Obstructed drainage or retained mucosa	CT/endoscopy; endoscopic or open drainage/reconstruction
Years-decades	Late mucocele with orbital/intracranial extension	Slow expansion of trapped secretory mucosa	Definitive drainage and removal of obstructing pathology

8.9 Mucocele: the signature late complication

A mucocele is an epithelium-lined, mucus-filled expansile lesion caused by obstructed sinus drainage or trapped mucosa. It can erode bone and extend into the orbit or cranial cavity. Presentation may include frontal pressure, forehead swelling, proptosis, diplopia, visual change, recurrent infection, or neurologic symptoms. Because expansion is slow, the original trauma or surgery may be decades in the past.

CT shows an expansile opacified sinus with bony remodeling or erosion; MRI helps distinguish secretions, inflammation, and intracranial or orbital extension. Treatment restores drainage, usually endoscopically when accessible, while addressing retained foreign material, obliteration substance, or inaccessible lateral disease when present. The possibility of a mucocele is the reason every frontal sinus operation requires a lifelong historical footprint in the medical record.

8.10 Surveillance plan

Table 8.2. Surveillance is tailored to the failure mode of the chosen strategy.

Injury / procedure	Early follow-up	Long-term surveillance
Observed orbital fracture	Repeat vision, motility, diplopia, and globe position after edema recedes.	Return for delayed enophthalmos, diplopia, sensory, or eyelid symptoms.
Orbital reconstruction	Early visual/motility checks; image complex repairs selectively.	Assess persistent diplopia, globe position, implant, and eyelid symptoms.
Observed FSOT injury	Interval CT to document re-aeration; practical checkpoints may include ~6 weeks and ~12 months.	Evaluate frontal pain, swelling, infection, obstruction, or orbital symptoms.
Preserved sinus after repair	Contour, wound, drainage, hardware, and aeration assessment.	Counsel regarding delayed obstruction and hardware symptoms.
Obliteration / cranialization	Wound, neurologic, infection, CSF, and contour assessment.	Maintain lifelong awareness of retained-mucosa mucocele or infection.

EMERGENCY EXCEPTION - TENSION PNEUMOCEPHALUS
Neurologic deterioration or CT mass effect from progressive intracranial air is not routine surveillance. Stabilize, obtain immediate neurosurgical assessment for decompression and closure, and avoid nonessential positive pressure or nitrous oxide while the communication remains active.

When new symptoms appear

1 Re-examine
Vision, pupils, orbit, neurologic status, wound, and drainage.

2 Re-image
CT first for bone/air; MRI for soft tissue or intracranial/orbital extension.

3 Re-localize
Pressure, obstruction, CSF communication, infection, or mucocele.

4 Re-escalate
Ophthalmology, neurosurgery, neurovascular, or skull-base team as indicated.

RESIDENT SYNTHESIS
A CSF leak is confirmed chemically, localized anatomically, and managed according to flow, persistence, neurologic risk, and reconstructive feasibility. Upper-face trauma follow-up continues long enough to detect delayed orbital deformity, obstruction, infection, or mucocele.

CHAPTER 9

Integrated Cases and Final Workflow

CENTRAL QUESTION

Can the resident integrate vision, orbital function, sinus physiology, and skull-base safety under oral-board pressure?

Case 1 - The tense postoperative orbit

CASE

Two hours after repair of a large floor-medial wall fracture, the patient develops increasing orbital pain, proptosis, reduced acuity, and a new RAPD. The eyelids are tense. CT is not immediately available.

Questions to answer

- What is the diagnosis?
- What should happen before imaging?
- What findings must be documented after intervention?
- What operative causes must be considered if bedside release is insufficient?

Reasoning

The syndrome is orbital compartment syndrome until proven otherwise. The combination of acute visual decline, RAPD, proptosis, pain, and a tense orbit is more important than confirmation of a retrobulbar hematoma on CT.

Perform immediate lateral canthotomy and inferior cantholysis while summoning ophthalmology and preparing for operative exploration. Reassess acuity, pupils, pressure when safe, globe tension, and motility immediately. If the orbit remains tense or vision does not improve, confirm that the inferior crus was fully released and escalate.

Postoperative causes include diffuse hemorrhage, a localized collection, tight closure, implant malposition, tissue incarceration, or an implant extending too posteriorly. Removal or repositioning of reconstructive material and hematoma evacuation may be necessary.

BOARD PEARL

A new RAPD after orbital surgery is a time-critical physiologic finding, not a reason to wait for routine postoperative imaging.

Case 2 - The child with a white eye

CASE

A 9-year-old is struck by a baseball. There is little ecchymosis, but the child cannot elevate the eye and vomits when attempting upgaze. Heart rate falls during the examination. CT shows a small linear floor fracture with minimal displacement.

Questions to answer

- Why is the benign external appearance misleading?
- What mechanism explains the systemic symptoms?
- How urgent is management?
- What should be checked intraoperatively?

Reasoning

Elastic pediatric bone can create a trapdoor fracture that incarcerates tissue after recoiling. The defect can appear small even when the mechanical syndrome is severe.

Nausea, vomiting, and bradycardia suggest an oculocardiac reflex from traction on extraocular muscle or associated fascia. This is not routine edema-related diplopia.

Notify ophthalmology and the facial-trauma service immediately and coordinate urgent release. Prolonged incarceration risks ischemia and fibrosis. Under anesthesia, document forced ductions before release, directly free tethered tissue, reconstruct only as necessary, and confirm free ductions afterward.

Case 3 - Diplopia on day one

CASE

A 31-year-old has a moderate isolated floor fracture after assault. Visual acuity and pupils are normal. Vertical diplopia occurs at extremes of gaze, but primary gaze is single. Motility is full with discomfort, and there are no vagal symptoms. CT shows fat herniation and an inferior rectus that lies near the defect.

Questions to answer

- Does CT prove entrapment?
- Would you operate today?
- What is the observation plan?
- Which findings would change the recommendation?

Reasoning

CT demonstrates relationship, not causation. The full painful range and lack of vagal symptoms favor edema and soft-tissue distortion over severe incarceration.

Immediate repair is not required. Provide sinus precautions and symptom control, document the diplopia field and globe position, and arrange early reassessment after edema begins to improve.

Persistent primary- or reading-gaze diplopia with objective mechanical restriction, evolving enophthalmos or hypoglobus, or a defect judged likely to create durable deformity would support reconstruction.

BOARD PEARL

Observation is a serial diagnostic test, not therapeutic neglect.

Case 4 - Visual loss with a quiet globe

CASE

After a forehead impact, an adult has acuity of counting fingers on the injured side, red desaturation, and an RAPD. The globe contour is normal, IOP is not elevated, and there is no proptosis. CT shows an optic-canal fracture without a clearly compressive fragment.

Questions to answer

- What is the likely syndrome?
- What diagnoses still must be excluded?
- Should megadose steroids be started?
- How should the plan be communicated?

Reasoning

The pattern is compatible with indirect traumatic optic neuropathy, but retinal injury and subtle globe pathology require ophthalmologic assessment. OCS is unlikely because the orbit is not tense and pressure is not elevated.

Current evidence does not support routine megadose IV methylprednisolone or optic-canal decompression for indirect TON without a specific reversible compressive lesion. Explain the uncertainty, obtain neuro-ophthalmic input, and treat associated brain and orbital injuries.

If imaging demonstrates direct compression by a displaced fragment, hematoma, or foreign body, the problem becomes a specific anatomic lesion and requires individualized multidisciplinary discussion.

BOARD PEARL

Do not treat the words “optic canal fracture” as an automatic steroid or decompression indication.

Case 5 - The depressed forehead with a functioning sinus

CASE

A patient has a displaced comminuted anterior-table fracture causing visible contour depression. The posterior table is intact, there is no CSF leak, and the frontal recess appears open. The patient is otherwise stable.

Questions to answer

- Which of the five variables are unfavorable?
- What is the operative goal?
- Does the sinus need obliteration?
- How should follow-up be framed?

Reasoning

The unfavorable variable is anterior contour. The intracranial boundary, dura, and apparent drainage pathway are favorable.

The goal is anatomic contour restoration through the least morbid exposure that permits stable reduction. Sinus mucosa and the frontal recess should be preserved if viable.

Obliteration is not required solely because the anterior table is repaired. Follow contour and wound healing, and reassess sinus aeration if the outflow pathway was near the injury or surgical field.

BOARD PEARL

Repair the problem the patient has. An anterior-table deformity does not automatically imply a nonfunctional sinus.

Case 6 - The uncertain frontal outflow tract

CASE

A patient has a fracture through the inferomedial frontal sinus and adjacent NOE complex. The posterior table is intact and there is no CSF leak. The sinus is opacified on the acute CT, and the patient can return reliably for follow-up.

Questions to answer

- Does acute opacification prove permanent obstruction?
- What are the preservation and ablation options?
- What surveillance is necessary?
- What finding would trigger delayed intervention?

Reasoning

Acute blood and edema commonly opacify the sinus. Fracture location raises concern for FSOT injury, but obstruction should be evaluated over time rather than inferred from density alone.

A sinus-preserving strategy with repair of the facial framework and interval imaging is reasonable in an appropriate program. Immediate obliteration remains an option when the tract is destroyed or follow-up and rescue are unreliable.

Obtain interval CT to document re-aeration. Persistent non-aeration, frontal symptoms, recurrent infection, or evolving mucocele would prompt endoscopic evaluation and drainage-restoring intervention when feasible.

BOARD PEARL

The safety of observation depends as much on a reliable surveillance system as on the first CT.

Case 7 - Posterior table comminution and CSF leak

CASE

A high-energy injury produces severe posterior-table comminution, pneumocephalus, a persistent high-flow CSF leak, and a frontal lobe contusion requiring craniotomy. The outflow tract is disrupted.

Questions to answer

- Which five variables are unfavorable?
- What operation best matches the goals?

Case 7 - Operative reasoning and priorities

Reasoning

The posterior table, outflow tract, dura/CSF status, and comminution are unfavorable; the anterior table may also require reconstruction.

Cranialization fits the need for cranial access and a posterior boundary that cannot be preserved. The operation removes the posterior table, repairs the dura, eliminates sinus mucosa, seals the outflow tract, reconstructs contour as needed, and interposes vascularized tissue such as pericranium.

Postoperatively, monitor neurologic status, CSF leakage, wound infection, frontal contour, and orbital function. The patient still requires long-term awareness of late mucocele or infection from retained mucosa.

BOARD PEARL

Cranialization is a barrier-reconstruction operation, not merely removal of the posterior table.

Case 8 - Delayed proptosis decades later

CASE

A patient presents with progressive unilateral proptosis and frontal pressure 22 years after a frontal sinus fracture treated through a coronal approach. CT shows an expansile opacified frontal sinus with erosion into the orbit.

Questions to answer

- What is the likely diagnosis?
- Why can it present so late?
- What imaging and treatment are needed?
- What lesson should be carried into current trauma counseling?

Reasoning

The likely diagnosis is a frontal sinus mucocele with orbital extension. Secretory mucosa trapped behind an obstructed pathway expands slowly and remodels bone.

CT defines bony expansion; MRI helps characterize contents and intracranial or orbital extension. Treatment requires definitive drainage, usually endoscopic when accessible, with open or combined approaches for lateral, complex, or previously obliterated disease.

The original trauma history remains clinically relevant for life. Every frontal sinus patient should receive written counseling about delayed symptoms and the need to disclose prior injury or surgery.

BOARD PEARL

The surveillance clock for frontal sinus trauma is measured in decades.

9.1 The final eight-step resident workflow

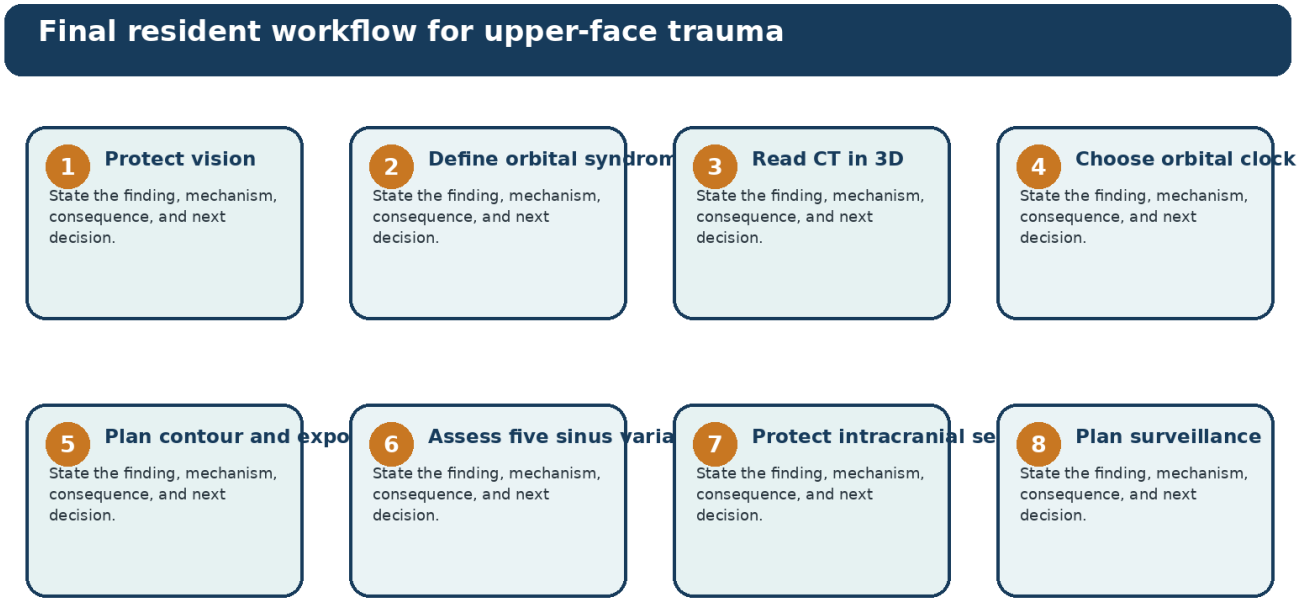

Figure 9.1. Final workflow for orbital, frontal sinus, and anterior skull-base trauma.

Table 9.1. The oral-board sequence for upper-face trauma.

Step	Question	Resident output
1. Protect vision	Is there open globe, OCS, active visual decline, or pediatric entrapment?	Name the emergency and act before reconstructive planning.
2. Define the orbital syndrome	Is dysfunction mechanical, pressure-related, neurogenic, or globe-related?	State acuity, pupils, motility, diplopia, and globe position.
3. Read CT in three dimensions	Which walls, stable boundaries, apex structures, and adjacent compartments are involved?	Describe geometry, tissue relationship, and anticipated volume change.
4. Choose the orbital clock	Emergent decompression, urgent release, interval repair, or active observation?	Give the reason and reassessment plan.
5. Plan reconstruction	Can the full defect be exposed, released, supported, and verified?	Choose approach and implant by geometry, not habit.
6. Assess five sinus variables	Anterior table, posterior table, FSOT, dura/CSF, comminution?	Convert “frontal sinus fracture” into five decisions.
7. Protect intracranial separation	Can the sinus be preserved safely, or must it be excluded/cranialized?	State the barrier and drainage goals.
8. Design surveillance	Which complication can occur later, and how will it be detected?	Specify examination, imaging, symptoms, and duration of follow-up.

FINAL RESIDENT TAKEAWAY

Protect vision before interpreting fracture thresholds. Reconstruct the orbit as a three-dimensional contour, not a flat defect. Treat frontal sinus trauma as five variables, preserve function when it is safe, and never forget that late sinus complications can outlive the original medical record.

Practical Appendices

Appendix A - Orbital trauma checklist

Domain	Document
Vision	Acuity in each eye, pupils/RAPD, color or red saturation, fields when feasible
Globe	Shape, anterior chamber, pupil, conjunctiva/sclera, concern for rupture
Pressure syndrome	Proptosis, tense orbit, pain, IOP only if globe is intact
Motility	Range, painful directions, diplopia fields, nausea/bradycardia, forced duction if appropriate
Position	Enophthalmos/exophthalmos, hypoglobus, eyelid closure, corneal protection
Sensation	V1 and V2; infraorbital distribution
CT	Globe, retrobulbar space, apex, involved walls, inferomedial strut, posterior ledge, herniated tissue
Clock	Emergency, urgent, interval repair, or active observation
Limitations	Sedation, swelling, age, language, intoxication, intubation; state repeat plan

Appendix B - Orbital CT reading template

- Globe: contour, lens, anterior chamber, intraocular foreign body.
- Retrobulbar compartment: hemorrhage, emphysema, optic-nerve stretch or apical crowding.
- Rims and facial framework: ZMC, NOE, frontal bar, and reduction references.
- Internal walls: roof, floor, medial wall, lateral wall; combined defects and junctions.
- Support: inferomedial strut, posterior ledge, intact lateral boundary.
- Soft tissue: muscle shape and course, fat herniation, suspected tethering.
- Volume and position: globe level, projection, anticipated expansion or compression.
- Adjacent spaces: maxillary and ethmoid sinuses, frontal sinus, skull base, intracranial air.

MODEL IMAGING SUMMARY

“Combined left floor and medial-wall fracture with loss of the inferomedial contour, herniation of fat into the maxillary sinus and ethmoid cells, preserved posterior ledge, no retrobulbar hematoma, and no apical fracture. Globe contour is intact.”

Appendix C - Frontal sinus five-variable worksheet

Variable	Favorable description	Unfavorable description	Plan consequence
Anterior table	Nondisplaced or acceptable contour	Visible depression, comminution, soft-tissue compromise	Observe versus contour repair
Posterior table	Nondisplaced, stable boundary	Marked displacement, comminution, intracranial fragment	Observe/repair versus cranialization
FSOT	Patent or likely to recover/re-aerate	Destroyed, obstructed, inaccessible, unreliable follow-up	Preserve/surveil versus restore or exclude
Dura/CSF	No leak or early low-flow leak resolving	Persistent/high-flow leak, encephalocele, pneumocephalus progression	Observation versus definitive repair
Comminution	Mucosa and stable bone manageable	Retained mucosal pockets likely, no reliable boundary	Raises need for ablation/cranialization

Appendix D - Postoperative orbital orders and checks

Time	Minimum assessment	Escalation trigger
PACU / awakening	Acuity or best feasible vision, pupils/RAPD, pain, proptosis, eyelid tension	Any new visual decline, RAPD, tense orbit, or disproportionate pain
Early floor/ward	Repeat vision and motility; inspect eyelid closure and wound	Progressive swelling, vomiting with gaze, new restriction, bleeding
Before discharge	Stable vision, tolerable pain/nausea, instructions understood	Unreliable exam, worsening symptoms, inability to access urgent care
Clinic	Diplopia field, globe position, sensation, eyelid position, CT when indicated	Persistent functional diplopia, enophthalmos, infection, implant concern

- Head elevation and cold compresses as appropriate.
- Analgesia and antiemetics to limit Valsalva.
- No nose blowing; sneeze with mouth open during the acute healing interval.
- Lubrication or eye protection when closure is incomplete.
- Antibiotics according to contamination and perioperative protocol, not reflexive prolonged courses.
- Immediate bedside review for any visual complaint.

Appendix E - Oral-board presentation template

“This is a [stable/unstable] patient after [mechanism]. The airway and neurologic status are [state]. Visual acuity is [state] with [no/a] RAPD; the globe is [intact/at risk], and there is [no/evidence of] orbital compartment syndrome. Motility shows [pattern], which I localize to [mechanical/edema/neurogenic/globe] dysfunction. CT demonstrates [walls and geometry], with [stable boundaries/volume implication/apex involvement]. I would place the orbital injury on the [emergent/urgent/interval/observation] clock because [reason]. The frontal sinus assessment shows [anterior table], [posterior table], [FSOT], [dura/CSF], and [comminution]. My management goal is [preserve drainage/restore contour/repair barrier/obliterate/cranialize], with [consultations] and [surveillance plan].”

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REFERENCE APPROACH

These sources prioritize contemporary reviews, consensus guidance, and practical operative references. Management must still be reconciled with current institutional protocols, ophthalmology/neurosurgery collaboration, injury pattern, and patient-specific anatomy.

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