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Double Vision Revisited: Evolving Concepts Of Diplopia In Maxillofacial Trauma

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Abstract:
Diplopia is a common and functionally significant complication of orbital trauma, affecting binocular vision and quality of life. This review synthesizes evidence from 13 peer-reviewed studies addressing the etiology, underlying pathophysiology, patterns of diplopia, diagnostic evaluation, and management strategies of diplopia post orbital fractures. Post-traumatic diplopia in maxillofacial injuries has traditionally been regarded as a mechanical problem related to muscle entrapment. Clinically, post-traumatic diplopia presents in vertical, horizontal, or torsional patterns reflecting restrictive or paretic mechanisms. Recent insights have expanded this understanding to include neurogenic, myogenic, and orbital volume–related mechanisms, as well as central sensory factors. A structured diagnostic evaluation and improved imaging techniques and a better understanding of orbital biomechanics have influenced contemporary management strategies, emphasizing individualized treatment planning and judicious surgical intervention to optimize postoperative ocular function. Diagnosis requires clinical orthoptic assessment, forced-duction testing and high-resolution imaging. Management ranges from conservative observation to surgical reconstruction and secondary strabismus procedures, depending on fracture type and severity of motility limitation. This review provides a practical framework to aid oral and maxillofacial surgeons in diagnosis, surgical decision-making, and outcome optimization in orbital trauma.

Keywords: *orbital trauma; post traumatic diplopia; extraocular muscle dysfunction; orbital floor fracture; medial wall fracture; orbital reconstruction; orthoptic assessment*

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I. Introduction

Diplopia is a common and functionally debilitating sequela of maxillofacial trauma, particularly in fractures involving the orbital floor and medial wall. Once considered primarily a manifestation of extraocular muscle entrapment, current evidence demonstrates that post-traumatic diplopia represents a complex clinical entity arising from a combination of mechanical restriction, intrinsic extraocular muscle injury, neural dysfunction, globe malposition, and disturbances of binocular sensory fusion.[1,2] Advances in orthoptic evaluation have shown that simple bedside tests frequently underestimate ocular motility disorders, while comprehensive functional assessments reveal a significantly higher prevalence of clinically relevant diplopia, thereby influencing both diagnosis and treatment planning.[1] Concurrent improvements in high-resolution imaging and biomechanical analysis have refined understanding of orbital volume alterations and globe displacement as independent contributors to binocular misalignment, even in the absence of overt muscle entrapment.[3] Contemporary management strategies increasingly favor algorithm-based, etiology-driven decision-making, distinguishing patients who benefit from orbital reconstruction from those requiring strabismus surgery or conservative orthoptic rehabilitation.[4] Furthermore, recognition of uncommon but clinically important mechanisms such as extraocular muscle rupture and post-traumatic muscle palsy has shifted emphasis toward individualized, multidisciplinary care aimed at restoring functional binocular single vision rather than merely achieving anatomical correction.[5,6] Together, these evolving concepts necessitate a reappraisal of diplopia in maxillofacial trauma, integrating anatomical, functional, and sensory perspectives to improve diagnostic accuracy and optimize patient outcomes.

Pathophysiology of Post-Traumatic Diplopia

Post-traumatic diplopia is a multifactorial consequence of maxillofacial and orbital injury, resulting from disruption of the finely coordinated anatomical and neuromuscular mechanisms responsible for binocular single vision. Trauma to the orbit alters ocular alignment through mechanical, neuromuscular, and sensory mechanisms, either in isolation or in combination, and the resulting diplopia may be transient or persistent depending on the severity and nature of injury[7,8]

One of the most common mechanisms is mechanical restriction of extraocular muscle movement, typically associated with orbital wall fractures, particularly of the orbital floor and medial wall. In these injuries, herniation of orbital fat and extraocular muscles into adjacent sinuses leads to tethering or incarceration, most

frequently involving the inferior rectus or medial rectus muscles. This produces incomitant diplopia that worsens in specific gaze positions, classically vertical diplopia on upgaze in orbital floor fractures. Edema, hemorrhage, and ischemia further exacerbate muscle dysfunction in the acute phase [1,2,4]

In addition to frank entrapment, restrictive diplopia may result from post-traumatic scarring and fibrosis of periorbital tissues. Even after successful fracture reduction, fibrotic healing around extraocular muscles can limit their excursion, resulting in persistent diplopia. This mechanism explains delayed or recurrent diplopia after initial improvement and underscores why clinical findings may underestimate injury severity in the early post-traumatic period[8,9]

Intrinsic extraocular muscle injury represents a less common but clinically significant pathophysiological mechanism. Blunt orbital trauma can cause partial tears, longitudinal flap tears, or complete rupture of extraocular muscles—most notably the inferior rectus—either at the muscle belly or near the tendon insertion. Unlike entrapment, muscle rupture may present with severe motility deficit but minimal resistance on forced-duction testing. Such injuries may be missed on routine CT imaging unless thin-slice reconstructions or MRI are performed, leading to persistent diplopia despite anatomically successful orbital reconstruction [5,6]

Another important contributor is neurogenic diplopia, resulting from traumatic injury to the oculomotor (III), trochlear (IV), or abducens (VI) nerves. Neuropraxia, axonotmesis, or nerve compression can lead to isolated or combined extraocular muscle palsies. Fourth nerve palsy is a well-recognized cause of vertical diplopia following blunt trauma, while inferior rectus palsy may mimic mechanical restriction. Neurogenic diplopia often improves gradually but may become permanent when axonal damage is severe [5,10]

Globe malposition is a major anatomical factor contributing to post-traumatic diplopia. Enlargement of orbital volume due to bony defects results in enophthalmos, hypoglobus, or vertical dystopia, altering the relative positions of the visual axes and leading to binocular misalignment. Even in the absence of muscle entrapment or palsy, subtle globe displacement can disrupt binocular fusion and produce symptomatic diplopia. The severity of diplopia correlates with the extent of orbital volume change rather than fracture size alone[3,7,9]

Post-traumatic diplopia also has a functional and sensory component. Disruption of binocular sensory fusion may occur following prolonged diplopia, orbital edema, or central nervous system adaptation. Reduced fusional reserves, suppression, or abnormal retinal correspondence may persist even after anatomical correction, explaining why some patients continue to experience diplopia despite restoration of ocular alignment [7,9]

Importantly, multiple mechanisms often coexist. For example, a patient may have orbital floor fracture–related globe malposition combined with inferior rectus contusion and transient nerve injury. This multifactorial nature explains the variability in clinical presentation, recovery patterns, and treatment outcomes observed in post-traumatic diplopia and highlights the need for comprehensive diagnostic evaluation and etiology-based management[7–9]

Patterns of Diplopia

As for etiology, no significant associations were observed between etiological categories and presence of diplopia. While the presence of diplopia alone does not reliably predict fracture site, specific patterns of diplopia show strong anatomical correlations. Diplopia on upward gaze (elevation) is significantly associated with orbital floor fractures, reflecting involvement of the inferior rectus muscle, surrounding edema, or tethering of orbital fat. Horizontal diplopia correlates strongly with medial orbital wall fractures, often due to displacement or dysfunction of the medial rectus muscle.[11,12] Combined floor and medial wall fractures frequently present with complex or multidirectional diplopia, reflecting greater orbital disruption.[13]. **Thus, careful ocular motility assessment can act as a clinical guide to fracture localisation, complementing CT imaging.**[11,12]

Diplopia may be transient, resolving as edema subsides, or persistent when associated with muscle entrapment, fibrosis, or significant orbital wall displacement.[12]

Diplopia involving primary gaze or central 30^{o} gaze is considered more clinically significant due to its interference with daily activities than diplopia occurring only at extremes of gaze.[7,12] A significant proportion of patients demonstrate diplopia only in secondary or extreme gaze positions, especially upgaze. Such diplopia may not be subjectively reported unless actively tested, highlighting the importance of objective motility assessment.[7]

No consistent associations were found between diplopia and patient age, sex, or mechanism of injury, emphasizing the role of fracture anatomy rather than demographic factors.[12]

Mild residual diplopia, especially in extreme upgaze, may persist postoperatively despite optimal reduction and implant positioning, but is often symptomatic. This is commonly related to prior muscle contusion or fibrosis rather than residual entrapment[7]

Diplopia patterns that correlate with CT-demonstrated orbital abnormalities (floor defects, scarring, malposition) are more likely to improve with orbital surgery, whereas non-correlative patterns suggest intrinsic muscle injury requiring strabismus management[9]

Diplopia resulting from enophthalmos or vertical dystopia reflects altered globe position rather than intrinsic muscle injury. These patients often present with vertical diplopia and show high rates of improvement following revision orbital reconstruction[9]

Diagnostic evaluation

Diplopia is a crucial clinical sign of an underlying ocular motility disorder following orbital blow-out fractures and plays a pivotal role in determining the need for conservative versus surgical management.[3] All midfacial fractures involving the orbit warrant a thorough ophthalmological assessment, as the presence of diplopia may signal a potentially vision-threatening orbital injury.[5]

A detailed clinical history should include the timing and mechanism of trauma, any pre-existing visual or ocular abnormalities, and the onset, nature, and progression of diplopia.[5] Initial examination must document the presence of diplopia in primary gaze, its exacerbation on up-gaze or eccentric gaze positions, and the adoption of compensatory head postures—such as chin elevation or head tilt—used to maintain single binocular vision.[9]

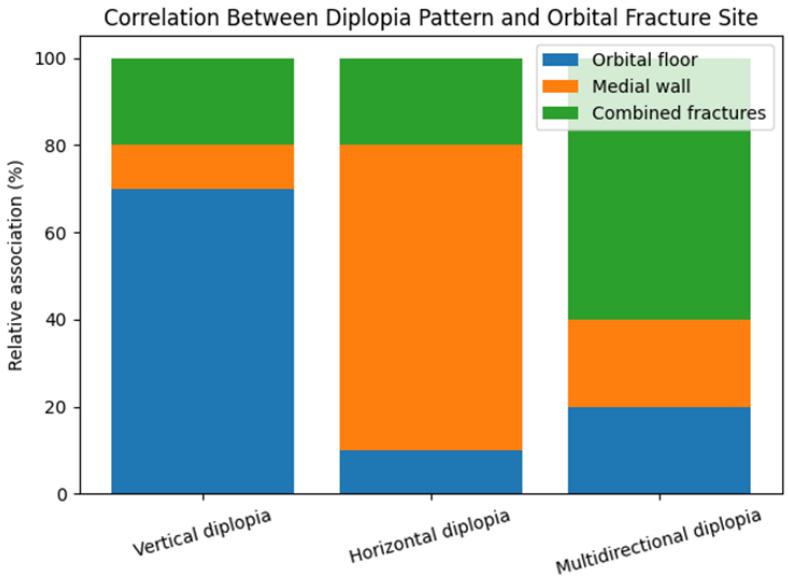
Systematic palpation of the orbital rims should be performed to identify step deformities or localized tenderness suggestive of orbital wall disruption.[5] Differentiation between monocular and binocular diplopia is essential, as monocular diplopia typically reflects lens or globe pathology, whereas binocular diplopia is more commonly trauma-related and associated with orbital wall injury or extraocular muscle involvement.[5]

Bedside ocular motility assessment using the finger-following test across eight cardinal positions of gaze remains the most frequently employed clinical method; however, it significantly underestimates ocular motility disorders, detecting abnormalities in only 23–35% of cases.[3] Comprehensive assessment of extraocular movements in all gaze directions is therefore essential to identify limitations of elevation, depression, or horizontal movements, as well as incomitant strabismus—findings that strongly suggest orbital wall fracture with muscle involvement.[4]

Early post-traumatic diplopia may be obscured by periorbital soft-tissue edema or eyelid ptosis, leading to missed diagnoses when reliance is placed solely on simple clinical tests.[3] Orthoptic evaluation enables objective quantification of ocular motility disorders, differentiation between incomitant and concomitant deviations, identification of pre-existing strabismus, and monitoring of recovery following conservative or surgical intervention.[3]

Comprehensive orthoptic assessment is regarded as the diagnostic gold standard, detecting ocular motility disorders in approximately 65% of patients with CT-confirmed orbital floor fractures. The orthoptic test battery includes the finger test, assessment of fixation, accommodation and convergence, colour filter testing for binocular disparity, Worth four-dot test, Bagolini striated glasses test, prism cover test, synoptophore evaluation, and the Lancaster red-green screen test.[3]

No single orthoptic test is adequate in isolation; a combined battery approach is necessary to minimize false-negative and false-positive findings, particularly in the presence of edema or pre-existing strabismus. Among individual modalities, the Lancaster screen test demonstrates the highest sensitivity, detecting up to 97.7% of ocular motility disorders when combined parameters are analysed. The colour filter test exhibits high sensitivity (95%) and specificity (100%) for identifying binocular diplopia and is particularly valuable for detecting subtle binocular disparity.[3]



The forced duction test is useful in distinguishing neurogenic palsy from mechanical restriction, with a positive test indicating incarceration of extraocular muscle or periorbital tissue within the fracture site.[4] The cover–uncover test assists in detecting ocular misalignment and latent strabismus that may contribute to diplopia.[5] The red-glass test provides a simple clinical method for diplopia detection, wherein placement of a red glass before one eye and movement of a light source through nine gaze positions allows confirmation of diplopia and identification of gaze dependency.[5]

Diplopia charting using red-green or red-glass testing facilitates localization of the affected extraocular muscle, objective documentation of the severity and direction of diplopia, and monitoring of post-treatment recovery.[4] Assessment of globe position is integral, as hypoglobus and enophthalmos serve as indirect indicators of orbital floor or medial wall defects, with globe malposition contributing to altered visual axes and diplopia.[4] Objective measurement of enophthalmos using a Hertel exophthalmometer is particularly valuable once edema subsides, improving diagnostic accuracy.[5]

Evaluation of periorbital sensory deficits, such as infraorbital nerve hypoesthesia, is recommended, as these findings frequently coexist with orbital floor fractures associated with diplopia.[4] Persistence or recurrence of diplopia after an initial period of improvement represents an important diagnostic indicator, suggesting progressive fibrosis, residual mechanical restriction, or altered vertical fusional reserves following orbital trauma.[9]

Radiological assessment is mandatory in all suspected cases. While the Waters view may be used for initial screening, computed tomography with axial and coronal sections remains the imaging gold standard for identifying orbital wall fractures, extraocular muscle entrapment, and orbital volume expansion. [5]

Treatment Approaches for Post-Traumatic Diplopia

Management of post-traumatic diplopia requires an etiology-based and staged treatment approach, as diplopia may result from orbital wall defects, extraocular muscle entrapment, intrinsic muscle injury, restrictive fibrosis, cranial nerve palsy, or globe malposition. In the acute phase, conservative observation is appropriate in many patients, since diplopia may resolve with reduction of edema, hematoma resorption, and recovery from neuropraxia. Diplopia confined to extreme gaze positions or outside the central binocular field usually does not require immediate intervention and may be managed with reassurance, prism correction, or orthoptic therapy, provided imaging excludes muscle entrapment or significant orbital volume change[7,8]

Early surgical intervention is indicated when clinical and radiological findings demonstrate extraocular muscle entrapment, large orbital wall defects, significant motility restriction, oculocardiac reflex, or progressive enophthalmos. The primary goal of orbital fracture repair is restoration of orbital anatomy through repositioning of herniated tissues, normalization of orbital volume, and re-establishment of unrestricted extraocular muscle movement. Accurate and timely orbital reconstruction significantly reduces the incidence of persistent diplopia, with most postoperative diplopia—when present—being limited to extreme gaze and not functionally disabling[7,9]

When diplopia persists despite technically adequate orbital repair, revision orbital surgery should be selectively considered. Patients with globe malposition, such as enophthalmos or vertical dystopia, and those with restrictive strabismus due to scarring or fibrosis are most likely to benefit from revision procedures. High-resolution CT imaging and objective motility assessment are essential for patient selection. The use of patient-specific orbital implants and computer-assisted planning, including biomechanical and finite-element modeling, improves predictability in restoring globe position and minimizing residual diplopia while avoiding over- or under-correction[3,9]

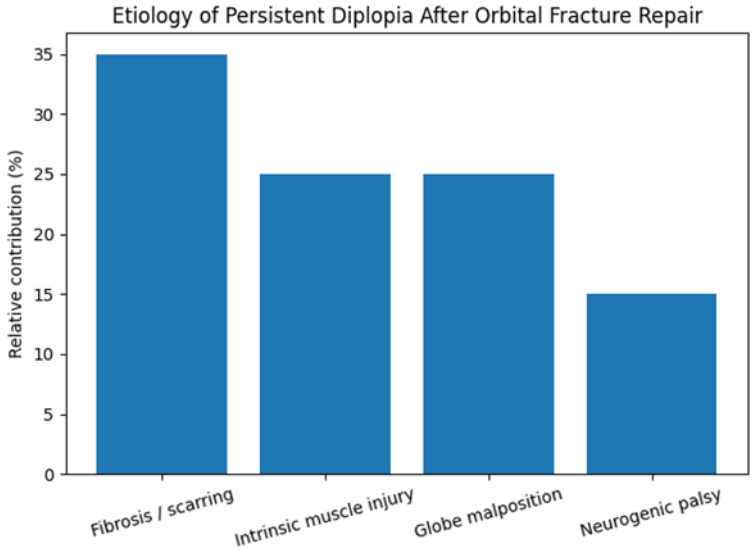
A distinct subgroup of patients develops diplopia due to intrinsic extraocular muscle injury, including partial tears, complete ruptures, or isolated muscle palsy—most commonly involving the inferior rectus muscle. In these cases, diplopia may persist despite anatomically successful orbital reconstruction and absence of mechanical restriction on forced-duction testing. Early recognition is critical, as primary reattachment of a ruptured muscle offers the best prognosis when feasible. However, not all muscle ruptures require reoperation; selected patients may achieve satisfactory functional outcomes with orthoptic therapy and prismatic correction, avoiding unnecessary secondary surgery[5,6]

In patients with permanent muscle palsy or severe incomitant deviations, staged strabismus surgery may be required once orbital anatomy has stabilized. Procedures such as muscle transposition, antagonist recession, or contralateral eye surgery aim not at perfect ocular alignment but at restoration of a central, functional field of binocular single vision, which represents a realistic and meaningful treatment endpoint in complex post-traumatic diplopia[5]

Overall, optimal management of post-traumatic diplopia depends on multidisciplinary collaboration between maxillofacial surgeons, ophthalmologists, and orthoptists. A stepwise strategy—beginning with observation, followed by targeted orbital reconstruction when indicated, and reserving strabismus surgery or orthoptic management for residual or non-mechanical causes—maximizes functional outcomes while minimizing unnecessary surgical intervention[7–9]

II. Discussion

Post-traumatic diplopia following orbital fractures is no longer best understood as a purely mechanical consequence of extraocular muscle entrapment but rather as a multifactorial disorder involving restrictive, neurogenic, myogenic, anatomical, and sensory components. The evolving literature consistently demonstrates that diplopia patterns correlate more strongly with fracture anatomy and orbital volume alteration than with fracture size alone, highlighting the importance of functional assessment alongside radiological evaluation. Advances in orthoptic testing have revealed that traditional bedside motility examinations significantly underestimate the prevalence and complexity of diplopia, whereas comprehensive orthoptic assessment provides superior diagnostic sensitivity and enables accurate differentiation between restrictive and paretic mechanisms. This distinction is critical, as diplopia that correlates with CT-demonstrated orbital abnormalities shows higher rates of improvement following orbital reconstruction, while non-correlative or persistent diplopia is more frequently attributable to intrinsic muscle injury, fibrosis, or neurogenic palsy. Importantly, persistent postoperative diplopia is often unrelated to technical failure of fracture repair, emphasizing that revision orbital surgery should be selectively reserved for patients with demonstrable globe malposition or residual mechanical restriction. An algorithm-based, etiology-driven approach—integrating clinical screening, functional differentiation, and anatomical correlation—allows targeted intervention, minimizes unnecessary surgery, and shifts the therapeutic goal from anatomical perfection toward restoration of a functional field of binocular single vision. Such a multidisciplinary, individualized strategy is essential for optimizing long-term visual outcomes in patients with maxillofacial orbital trauma.



III. Conclusion

Post-traumatic diplopia remains a common and clinically significant complication of orbital fractures, reflecting a complex interplay of mechanical restriction, extraocular muscle injury, neurogenic dysfunction, globe malposition, and sensory fusion disturbance. Contemporary evidence challenges the traditional view of diplopia as a solely mechanical problem and underscores the need for comprehensive functional assessment in all patients with orbital trauma. Pattern-based evaluation of diplopia, combined with detailed orthoptic testing and high-resolution imaging, allows accurate etiological differentiation and guides appropriate management. Early orbital reconstruction yields favourable outcomes in patients with anatomically correlated diplopia, whereas persistent or non-correlative diplopia often requires orthoptic rehabilitation or staged strabismus surgery rather than revision fracture repair. An algorithm-driven, multidisciplinary approach focused on restoration of functional binocular single vision—rather than anatomical correction alone—optimizes patient outcomes and minimizes unnecessary surgical intervention in the management of post-traumatic diplopia.

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