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REVIEW

Ocular surface implications of upper and lower blepharoplasty: A DEWS III-guided review of mechanisms, risks, and management strategies

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Abstract

Upper and lower eyelid blepharoplasty is one of the most commonly performed oculoplastic surgeries worldwide, serving both aesthetic enhancement and functional purposes. While blepharoplasty enhances facial aesthetics and expands the visual field effectively, it carries significant ocular surface complication risks. These operations may impact ocular surface health by changing eyelid structure, influencing blinking behavior, and affecting tear film homeostasis, predisposing patients to or aggravating preexisting dry eye disease (DED). Therefore, a meticulous preoperative assessment for dry eye, cautious surgical approaches, and diligent postoperative care are necessary to prevent surgery-induced or aggravated DED and achieve optimal patient satisfaction and surgical success. This narrative review evaluates recent evidence and TFOS DEWS II and III consensus data to define causative mechanisms, recognize predisposing factors, and present management strategies to improve surgical safety and patient outcomes.

Keywords: Blepharoplasty; Dry eye disease; DEWS II; DEWS III; Blink Mechanics; Ocular Surface.

Blepharoplasty has become one of the most frequently performed procedures within oculofacial plastic practice, due to aging populations, increased sun and screen exposure, and changing aesthetic perceptions.^[1–3] Upper eyelid blepharoplasty is primarily performed to treat dermatochalasis, characterized by excess skin causing decreased vision and obstructing the superior and peripheral visual fields.^[2,4] In contrast, lower eyelid blepharoplasty is usually indicated to manage lid laxity, steatoblepharon, eyelid retraction, orbital fat prolapse, or tear trough deformities.^[2,5]

Despite high satisfaction rates and notable functional improvements, blepharoplasty may have risks. It may lead to ocular surface complications such as chemosis and dry eye disease (DED), along with blinking problems, which can negatively affect surgical results and patient quality of life.^[1,5–7]

The incidence of dry eye disease after cosmetic blepharoplasty is about 0–26.5%.^[5,8–11] Hamawy et al.^[8] proposed that dry eye after periorbital surgery is usually self-limited; however, despite protective surgical approaches, it may persist for longer than 2 weeks in over 10% of patients.



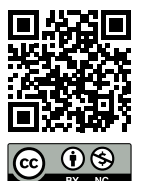
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Prischman et al.^[5] reported that simultaneous upper and lower blepharoplasty were more likely to cause dry eye disease compared to upper or lower blepharoplasty alone (31.3%, 12.9%, 21.4%, respectively). A study with 67 patients diagnosed with preoperative dry eye reported that the condition worsened in 5 (7.46%) patients, while 50 (74.63%) showed no change.^[9]

Aksu Ceylan and Yeniad detected a statistically significant decrease in Schirmer test results in blepharoplasty patients, suggesting a mechanism associated with the orbicularis muscle. The orbicularis muscle is believed to be weakened, resulting in a decrease in the blinking reflex and corneal sensation, as well as tear secretion due to damage to trigeminal nerve branches, leading to a decrease in basal and reflex tears.^[12]

According to the TFOS DEWS II definition, DED is a multifactorial disease involving tear film instability, hyperosmolarity, inflammation, and neurosensory deficits, alongside ocular symptoms.^[13] More recently, TFOS DEWS III classified iatrogenic dry eye as a specific DED subtype resulting from surgical or medical interventions that compromise eyelid integrity, blink function, or tear film dynamics.^[14]

Given that the eyelids, Meibomian and lacrimal glands, and blink mechanism form an integrated unit essential for ocular surface homeostasis^[13,15], any surgical modification to these structures can trigger ocular discomfort, visual changes, and chronic ocular surface impairment. Understanding the mechanisms behind blepharoplasty-induced ocular surface complications is essential for optimal preoperative evaluation, appropriate surgical planning, and postoperative care. This review synthesizes evidence regarding upper and lower blepharoplasty impacts and presents a TFOS DEWS III-based approach for risk assessment and management.

Methods

This review utilized a comprehensive narrative approach to compile current evidence on blepharoplasty-related ocular surface changes. A systematic search was performed in PubMed, Scopus, Web of Science, and Google Scholar for publications between January 2000 and June 2025. Boolean operators (AND, OR, NOT) were applied in combinations such as “blepharoplasty AND dry eye disease,” “oculoplastic surgery OR eyelid surgery AND ocular surface,” and “lagophthalmos NOT trauma.” Filters were applied to include only human studies, full-text availability, and articles in English. Randomized controlled trials (RCTs), prospective and retrospective clinical studies,

case series, and relevant narrative/systematic reviews were considered eligible. Case reports, editorials, non-peer-reviewed sources, and studies published in non-English languages were excluded.

Included studies evaluated ocular surface parameters before and after blepharoplasty, including tear breakup time (TBUT), Schirmer tests, ocular surface disease index (OSDI), corneal staining, Meibography, and in vivo confocal microscopy (IVCM) results.^[1,5,16–21] TFOS DEWS II & III consensus reports were also reviewed.^[6,13,14]

Findings were integrated within the TFOS DEWS III framework, particularly in relation to iatrogenic DED, ocular surface pathophysiology, and perioperative risk factors.^[6,14]

Results

Preoperative Risk Assessment

TFOS-Recommended Evaluation

In line with the TFOS DEWS II and III recommendations, a comprehensive preoperative evaluation is mandatory to reduce the risk of postoperative ocular surface complications. The assessment should include the Ocular Surface Disease Index (OSDI) questionnaire to identify subjective symptoms, as well as tear breakup time (TBUT), with values below five seconds indicating significant tear film instability.^[1,5] Schirmer test results of less than 10 mm over five minutes suggest aqueous tear deficiency.^[5] Meibography should also be performed to evaluate Meibomian gland dropout.^[14] Furthermore, blink rate and completeness analysis are essential^[13,16], along with eyelid laxity testing through snap-back and distraction assessments.^[2] The evaluation of Bell’s phenomenon is particularly important in candidates for lower eyelid blepharoplasty to anticipate postoperative lagophthalmos risk.

Identified Risk Factors

Patients with specific predisposing conditions are more likely to develop ocular surface complications after blepharoplasty. The presence of pre-existing dry eye symptoms or marginal tear production increases susceptibility to surgical trauma and delays postoperative recovery.^[1,5,18] A history of refractive surgery, especially LASIK, may compromise corneal innervation and tear film stability.^[2] Underlying autoimmune diseases such as Sjögren’s syndrome and rheumatoid arthritis contribute to ocular surface inflammation and dysfunction.^[5,18] Advanced age and female sex represent demographic risk factors associated with reduced tear secretion and meibomian gland dysfunction.^[1,18] Anatomical variations

such as proptosis or an insufficient Bell's phenomenon limit ocular protection during eyelid closure.^[2] Previous periocular botulinum toxin injections can interfere with blink mechanics or reduce lacrimal gland output.^[6] Long-term use of antiglaucoma medications is linked to ocular surface toxicity and tear instability due to preservative-related epithelial injury.^[22] Retrospective studies further highlight that preoperative scleral show and systemic hormone therapy are additional risk factors.^[5]

Upper Eyelid Blepharoplasty Outcomes

Upper eyelid blepharoplasty is primarily indicated for dermatochalasis, involving the removal of redundant skin and, in some cases, orbicularis muscle or periorbital fat. Although these procedures improve the visual field and cosmetic appearance, they may adversely affect ocular surface health.

Clinical studies report variable outcomes. Aygun et al. ^[1] documented increased OSDI scores and higher corneal fluorescein staining at both one and six months postoperatively, while Schirmer test values remained stable, suggesting a predominance of evaporative dry eye over aqueous deficiency. Sanad et al. ^[4] demonstrated a transient reduction in tear meniscus height one month postoperatively, which normalized by six months, indicating temporary disruption of tear volume. Mak et al. ^[16] found that blink rate and completeness remained unchanged, yet patients with pre-existing dry eye experienced worsened subjective symptoms, showing that subtle anatomical modifications can unmask subclinical disease even without major changes in blink mechanics. Conversely, Hollander et al. ^[17] found no significant changes in tear film parameters, underscoring the variability across studies and the influence of surgical technique and patient selection.

In a large retrospective cohort of 892 patients, 12.9% developed postoperative dry eye symptoms after upper blepharoplasty, 21.4% after lower blepharoplasty, and 31.3% after undergoing simultaneous upper and lower eyelid surgery, with procedures employing skin-muscle flap techniques.^[5]

Several mechanisms explain the ocular surface disturbances observed after upper blepharoplasty. Orbicularis muscle resection can weaken blink force and amplitude, reducing its role in tear distribution and meibomian gland compression.^[16] Even minor weakening may increase partial blink frequency, leaving the inferior ocular surface exposed, especially during sleep in patients with impaired Bell's phenomenon.

Excessive anterior lamellar removal may cause lagophthalmos, particularly in patients with a poor Bell's phenomenon.^[2] Temporary postoperative lagophthalmos has been associated with significantly higher dry eye prevalence (42.3% with lagophthalmos versus 22.9% without). Incomplete eyelid closure exposes the cornea to desiccating stress, leading to punctate epithelial erosions and exposure keratopathy.

Meibomian gland dysfunction is another critical mechanism. Reduced orbicularis contractility and higher partial blink rates impair lipid secretion, causing tear film thinning, increased evaporation, and hyperosmolarity, which drive ocular surface inflammation.^[16]

Surgical trauma further elevates inflammatory mediators such as interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor alpha (TNF- α), while oxidative stress markers, including 8-hydroxydeoxyguanosine (8-OHdG) and 4-hydroxy-2-nonenal (4-HNE), increase.^[1,19,20] Goblet cell dysfunction and epithelial barrier damage reduce mucin production, destabilizing the tear film.

Neurosensory alterations may result from manipulation of the supraorbital and supratrochlear nerves, producing temporary or permanent corneal hypoesthesia, reduced reflex tearing, and impaired blink responsiveness.^[2,14] TFOS DEWS III highlights neurosensory impairment as a key factor in iatrogenic dry eye.^[14]

Finally, microenvironmental factors such as incomplete blinking and lagophthalmos exacerbate tear evaporation, raising osmolarity and inducing epithelial apoptosis.^[23] Enlarged eyelid aperture during sleep predisposes patients to nocturnal exposure keratopathy.^[13]

Blink Dysfunction

Normal blink rates range between 10 and 20 per minute, and complete blinks are necessary for even tear distribution and lipid replenishment.^[16] After upper blepharoplasty, patients may experience reduced blink amplitude due to orbicularis weakening or scarring, partial blinking with incomplete eyelid closure, particularly affecting the inferior cornea, or transient blink infrequency caused by postoperative discomfort or edema. These changes disrupt the lipid layer, promote rapid tear breakup, and worsen evaporative dry eye. Patients with borderline tear function preoperatively are particularly susceptible to blink-related postoperative changes.^[16]

Lower Eyelid Blepharoplasty Outcomes

Lower eyelid blepharoplasty addresses steatoblepharon, lid laxity, and orbital fat herniation; however, it also

carries distinct ocular surface risks, similar to upper blepharoplasty. Reported complications include ectropion, characterized by outward turning of the eyelid margin with conjunctival and corneal exposure, leading to tearing, keratopathy, and irritation.^[2,4] Lid retraction, resulting from excessive skin or fat removal and anterior lamellar shortening, increases scleral show and corneal exposure.^[2] These conditions may lead to exposure keratopathy, punctate epithelial erosions, and keratitis, with persistent epithelial defects in severe cases.

Complication rates are influenced by surgical approach. Transcutaneous techniques are associated with higher rates of ectropion and scarring compared with transconjunctival methods.^[4,5] Ectropion rates range between 4% and 20% in patients undergoing aggressive skin resection without lateral canthal support^[24–26], whereas the transconjunctival approach shows markedly lower incidence, usually below 1%.^[26] Patients with pre-existing ocular surface abnormalities are more likely to develop postoperative dry eye, reinforcing the importance of detailed preoperative screening.

The transcutaneous approach involves incisions through skin and orbicularis muscle, increasing the risk of anterior lamellar shortening and cicatricial changes, whereas the transconjunctival approach preserves the anterior lamella and has a lower risk of ectropion.^[4]

The pretarsal orbicularis muscle is essential for blinking and tear drainage. Techniques such as the skin and skin–muscle flap approaches, when carefully executed, preserve the anatomical relationship with the tarsal plate. The skin flap over the pretarsal orbicularis continues as a skin–orbicularis flap over the preseptal orbicularis, reaching the orbital fat pads through dissection limited to the preseptal orbicularis oculi muscle.^[27]

Changes in eyelid position and muscle tone can impair blink mechanics. Even small alterations in lid-globe apposition may cause incomplete blinks or disrupt tear function along the inferior meniscus, thereby increasing evaporation. TFOS DEWS III emphasizes that lid malpositions directly compromise blink efficiency and aggravate evaporative dry eye.^[14] Loss of lid-globe contact also exposes the ocular surface to evaporation and destabilizes mucin–aqueous interactions, especially during visual tasks in downgaze, such as reading or computer use.^[23]

In contrast to upper eyelid blepharoplasty, which primarily affects blink force and completeness, lower eyelid surgery impacts blink efficiency through lid malposition.^[28,29] Ectropion prevents proper eyelid-globe apposition,

reducing the effectiveness of tear distribution during blinks.^[29] Lid retraction alters tear meniscus dynamics, decreasing tear film stability and promoting inferior corneal dryness. Excessive lid-tightening procedures can further impair blink function, highlighting the need for precise surgical planning to balance functional and cosmetic outcomes.^[29]

Discussion

Blepharoplasty remains one of the most frequently performed oculoplastic procedures worldwide, and while its aesthetic and functional benefits are well recognized, its impact on ocular surface health has become a subject of increasing academic interest. The last two decades have witnessed a growing body of literature describing postoperative ocular surface disease manifestations such as dry eye disease (DED), tear film instability, and corneal alterations, highlighting the importance of perioperative evaluation and management.^[4,12,13,28–30] The reported prevalence of postoperative DED varies considerably across studies, ranging from transient mild symptoms to persistent tear film dysfunction requiring long-term management. In a large retrospective cohort, Sanad and colleagues demonstrated that 26.5% of patients developed postoperative dry eye symptoms, with simultaneous upper and lower eyelid blepharoplasty and skin–muscle flap techniques being associated with significantly higher complication rates.^[4] Similarly, Aksu Ceylan and Yeniad reported significant reductions in tear breakup time (TBUT) and Schirmer test values after upper eyelid blepharoplasty, indicating compromised tear stability and aqueous production.^[12] Table 1 demonstrates post-blepharoplasty dry eye results in tabular form along with complication types and incidence rates.

These results provide mechanistic evidence that surgical disruption of eyelid and periocular anatomy may lead not only to tear film dysfunction but also to microstructural corneal changes that amplify ocular surface vulnerability.

The influence of surgical technique has been repeatedly emphasized in the literature. Procedures involving extensive skin–muscle flap dissection have been linked to a greater incidence of lagophthalmos and incomplete blinking, both of which exacerbate tear evaporation and lipid layer instability.^[1,4] Conversely, conservative resections that preserve orbicularis oculi integrity are associated with more favorable postoperative tear film outcomes, supporting the view that meticulous surgical planning is critical for minimizing ocular surface complications.^[1] Corneal biomechanics and refractive parameters also appear susceptible to periocular surgical

Table 1. Reported incidence of dry eye disease after blepharoplasty

Study (Year)	Population / Technique	Type of Surgery	Complications Reported	Incidence (%)
Prischmann et al. ^[5] (2013)	Retrospective, cosmetic blepharoplasty	Upper + Lower	Dry eye symptoms	31.3
		Upper only	Dry eye symptoms	12.9
		Lower only	Dry eye symptoms	21.4
Shao et al. ^[10] (2014)	Transcutaneous blepharoplasty	Lower	Dry eye symptoms	16.7
Floegel et al. ^[11] (2003)	Upper blepharoplasty (orbital fat was not removed)	Upper	Dry eye symptoms	21
Yan et al. ^[19] (2020)	Young Asian women, double-eyelid	Upper (double-eyelid)	DED (DEWS II definition: OSDI \geq 13 + NIBUT <10s)	Preop 12.5 1 wk 12.5 1 mo 32.5 3 mo 16.7
Saadat & Dresner ^[9] (2004)	Pre-existing dry eye patients	Upper / Lower	Worsening of dry eye	7.4 worsened 7.4 improved 74.6 unchanged
Mak et al. ^[16] (2020)	Blink dynamics study	Upper	OSDI worsening (12.5 vs 16.6)	85.7 worsened
Hamawy et al. ^[8] (2009)	Periorbital surgeries including blepharoplasty	upper and/or lower blepharoplasty±.additional facial cosmetic procedures	Dry eye persisting >2 weeks	10.9
			>2 mo	1.98
Zhang et al. ^[30] (2020)	Literature review	Upper + Lower	Overall range of postoperative dry eye	0–26.5
Sanad AA et al. ^[4] (2017)	Prospective, cosmetic upper blepharoplasty	Upper eyelid	Increased dry eye symptoms (OSDI \uparrow , TBUT \downarrow , Schirmer \downarrow) in early postoperative period; recovery by 3 months	30 symptomatic early, resolved by 3 months
Fan W et al. ^[28] (2021)	Prospective study, upper + lower cosmetic blepharoplasty	Upper & Lower eyelids	Observed increased DED symptoms postoperatively with in vivo confocal microscopy changes in corneal nerves and epithelial cells	Incidence up to 26.5
Aksu Ceylan & Yeniad ^[12] (2022)	Clinical study, upper blepharoplasty	Upper eyelid	Reported ocular surface alterations and corneal topography changes; Schirmer and TBUT reduced in early postop period	22 transient DED symptoms
Aygun et al. ^[1] (2021)	Prospective study, cosmetic upper blepharoplasty	Upper eyelid	Found significant changes in tear film stability and corneal microstructure after surgery	Dry eye symptoms in 22.9 early, improved later

manipulation. Several clinical studies have shown shifts in corneal astigmatism and keratometric values following blepharoplasty, alterations attributed to changes in eyelid tension and pressure distribution across the corneal surface.^[12,31] Although often subclinical, these modifications may destabilize the tear film and contribute to subjective visual complaints, particularly in patients with preexisting ocular surface disease or refractive errors.

Findings from Fan et al.^[28] and Zhang et al.^[30] confirm that blepharoplasty-related DED involves mechanical, neurosensory, inflammatory, and evaporative pathways. Persistent symptoms among patients with baseline DED highlight the necessity of thorough preoperative evaluations incorporating OSDI, TBUT, Schirmer tests, Meibography, and in vitro confocal microscopy (IVCM) where available.^[5,13,28,30] Zhang et al.^[30] suggest conservative resection strategies that maintain orbicularis function, staging of procedures where appropriate, and perioperative lubrication alongside anti-inflammatory therapy.^[28] Postoperative management should focus on lipid-based preservative-free tears, anti-inflammatory therapy, moisture chamber use, and early intervention for anatomical complications.^[13,14,28,30]

The perioperative risk profile is further influenced by baseline patient characteristics. Advanced age, female sex, prior history of ocular surface disease, and systemic hormone therapy have been identified as predisposing factors for worse postoperative outcomes, underscoring the multifactorial nature of blepharoplasty-related DED.^[4,31,32] Moreover, temporary postoperative lagophthalmos has been strongly associated with increased severity of dryness, with patients demonstrating nearly double the prevalence of symptoms compared to those without exposure-related complications.^[4]

The relevance of meibomian gland dysfunction (MGD) in the pathogenesis of iatrogenic dry eye has been highlighted by both experimental and observational studies. Gulal and Eroglu demonstrated that periocular interventions may exacerbate gland dropout and alter lipid secretion, thereby accelerating evaporative dry eye mechanisms.^[33] When combined with reduced blink efficacy following orbicularis manipulation, these glandular alterations contribute to persistent tear film instability and chronic ocular surface irritation.^[11] In addition to clinical parameters, quality-of-life implications should not be underestimated. Dryness, fluctuating vision, and foreign body sensation after blepharoplasty may significantly diminish patient satisfaction, even when aesthetic outcomes are objectively

successful. Prospective patient-reported outcome measures confirm that ocular surface symptoms exert measurable psychosocial and functional burdens in the postoperative period.^[28] These observations underscore the importance of integrating ocular surface health into the overall assessment of surgical success.

Taken together, current evidence demonstrates that blepharoplasty-related ocular surface complications arise from a combination of anatomical, functional, and inflammatory mechanisms. Disruption of orbicularis integrity, postoperative lagophthalmos, meibomian gland dysfunction, corneal biomechanical changes, and patient-specific vulnerabilities collectively shape the clinical course. Accordingly, comprehensive preoperative risk stratification, meticulous surgical technique, and vigilant postoperative care—guided by TFOS DEWS II and III recommendations—are indispensable to safeguarding ocular surface health in patients undergoing cosmetic or functional eyelid surgery.^[13,14]

Limitations and Future Directions

Although the body of literature on this subject has expanded, existing studies are still constrained by methodological limitations, including small patient cohorts, heterogeneity in surgical techniques, and inconsistency in diagnostic criteria. These factors restrict the generalizability of current findings and highlight the need for more robust evidence.

Future research priorities should include the incorporation of TFOS DEWS III subtyping to enable more precise stratification of surgical risk and to facilitate tailored perioperative management. Advanced diagnostic modalities, such as blink electromyography and tear proteomic profiling, hold promise in elucidating the functional and molecular mechanisms underlying iatrogenic ocular surface changes. Furthermore, randomized controlled trials directly comparing transcutaneous and transconjunctival techniques are required to clarify differences in outcomes and complication profiles. Preventive strategies also warrant systematic evaluation, particularly the potential benefits of lipid-based tear supplementation and perioperative anti-inflammatory regimens in reducing postoperative ocular surface morbidity. Equally important is the need for extended follow-up beyond twelve months, especially in older individuals with baseline meibomian gland dysfunction, as this group may be more susceptible to chronic sequelae. Long-term observational data will be critical to optimizing management strategies and ensuring sustained ocular surface health after oculoplastic interventions.

Conclusion

In conclusion, while blepharoplasty has considerable aesthetic and functional benefits, it carries inherent risks to ocular surface integrity through complex mechanical, anatomical, inflammatory, neurosensory, and microenvironmental mechanisms. Applying TFOS DEWS III-guided evaluations, refining surgical techniques, and implementing tailored postoperative care are essential for reducing iatrogenic DED and optimizing outcomes in oculoplastic practice.

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