

The TFOS International Workshop on Contact Lens Discomfort: Executive Summary

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See the tables in the Introduction for the members of the TFOS International Workshop on Contact Lens Discomfort.

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Contact lens discomfort (CLD) is a frequently experienced problem, with most estimates suggesting that up to half of contact lens wearers experience this problem with some frequency or magnitude. This condition impacts millions of contact lens wearers worldwide. Yet, there is a paucity of consensus and standardization in the scientific and clinical communities on the characterization of the condition, including the definition, classification, epidemiology, pathophysiology, diagnosis, management, influence of contact lens materials, designs and care, and the proper design of clinical trials.

The Tear Film & Ocular Surface Society (TFOS), which is a nonprofit organization, has conducted two prior international, consensus building workshops, including the Dry Eye WorkShop (DEWS; available in the public domain at <http://www.tearfilm.org/tearfilm-reports-dews-report.php>) and the Meibomian Gland Dysfunction Workshop (MGD; available in the public domain at <http://www.tearfilm.org/tearfilm-reports-mgdreport.php>). To that end, TFOS initiated the process of conducting a similar workshop in January 2012—a process that took approximately 18 months to complete and included 79 experts in the field. These experts participated in one or more topical subcommit-

tees, and were assigned with taking an evidence-based approach at evaluating CLD. Eight topical subcommittees were formed, with each generating a related report, all of which were circulated for presentation, review, and input of the entire workshop membership.

The entire workshop originally is being published in this issue of IOVS, in English, with subsequent translations into numerous other languages. All of this information is intended to be available and accessible online, free of charge. This article is intended to serve as an Executive Summary of the eight subcommittee reports, and all information contained here was abstracted from the full reports.

DEFINITION AND CLASSIFICATION OF CLD

While clinicians practicing in the area of contact lenses all are familiar with CLD, a variety of terms and verbiage have been used to describe this problem. Typically these patients present with symptoms of ocular discomfort of some sort (e.g., dryness, irritation, discomfort, fatigue, and so forth), and it is common

that these symptoms usually increase over the day while the patient is wearing the contact lenses. However, beyond this, no standard definition has been agreed upon globally with consensus as to what this problem is. As such, the definition of “CLD” is the following:

Contact lens discomfort is a condition characterized by episodic or persistent adverse ocular sensations related to lens wear, either with or without visual disturbance, resulting from reduced compatibility between the contact lens and the ocular environment, which can lead to decreased wearing time and discontinuation of contact lens wear.

The CLD Workshop membership characterized each of the terms in the definition, considering many other concepts in the development of the final definition. The rationale for the specific terminology included in the definition, and related terminology, can be found detailed in this subcommittee report. However, it is important to note that the CLD Workshop recognizes that CLD occurs while a contact lens is worn, and that the removal of contact lenses mitigates the condition (in particular the adverse ocular sensations). However, CLD is a condition that occurs after the initial “adaptation” period a neophyte goes through when first adjusting to contact lens wear. Physical signs may, or may not, be present in accompanying the adverse ocular sensations. Moving forward, the condition should be recognized as noted above, and the terms “contact lens dry eye” or “contact lens-related dry eye” should not be used when talking about contact lens discomfort. These terms should be reserved for an individual who has a preexisting dry eye condition, which may or may not be exaggerated when contact lenses are worn. Contact lens dropout refers to discontinuation of contact lens wear for a sustained period of time.

Classification of CLD was challenging, as classifying a disease relates to the ability to categorize it based on knowledge of the etiology. In addition, to our knowledge there has not been a previous classification scheme, and an understanding of etiologic factors has been identified in the other subcommittee reports as significantly lacking for CLD. The CLD Workshop felt that the two major categories of CLD were the contact lens and the environment (Fig. 1). The contact lens category was divided further into four subcategories: material, design, fit and wear, and lens care. The environment category also was broken down further into four subcategories: inherent patient factors, modifiable patient factors, ocular environment, and external environment. Details of each of these subcategories can be found within the Definition and Classification Report.

Lastly, very little is agreed upon regarding the temporal progression of CLD, as this relates to contact lens dropout (or permanent cessation of contact lens wear). As such, the modes of progression also are presented in Figure 1, showing the temporal progression of CLD as patients begin to struggle, which is followed by the adoption of management strategies (e.g., reducing wearing time), and ultimately by contact lens dropout.

EPIDEMIOLOGY OF CLD

The epidemiologic assessment of CLD faces many challenges, not least of which is the accurate assessment of the frequency of the condition. Since the first publication in 1960 linking hygienic contact lens care and comfortable lens wear, the issue of CLD remains a major reason for discontinuation of contact lens wear. It is estimated that there currently are more than 140 million contact lens wearers worldwide. It is much more

difficult to estimate the number of individuals who previously have worn contact lenses and then abandoned lens wear as a result of CLD. Studies report that between 12% and 51% of lens wearers “drop out” of contact lens wear, with CLD the primary reason for discontinuation.

While there have been tremendous developments in lens polymers, designs, replacement modalities, and care regimens over the last 50 years, the challenge of preventing or managing CLD still is a problem in clinical practice. A major deficiency in the literature is the lack of information derived from contact lenses that differ in only one parameter.

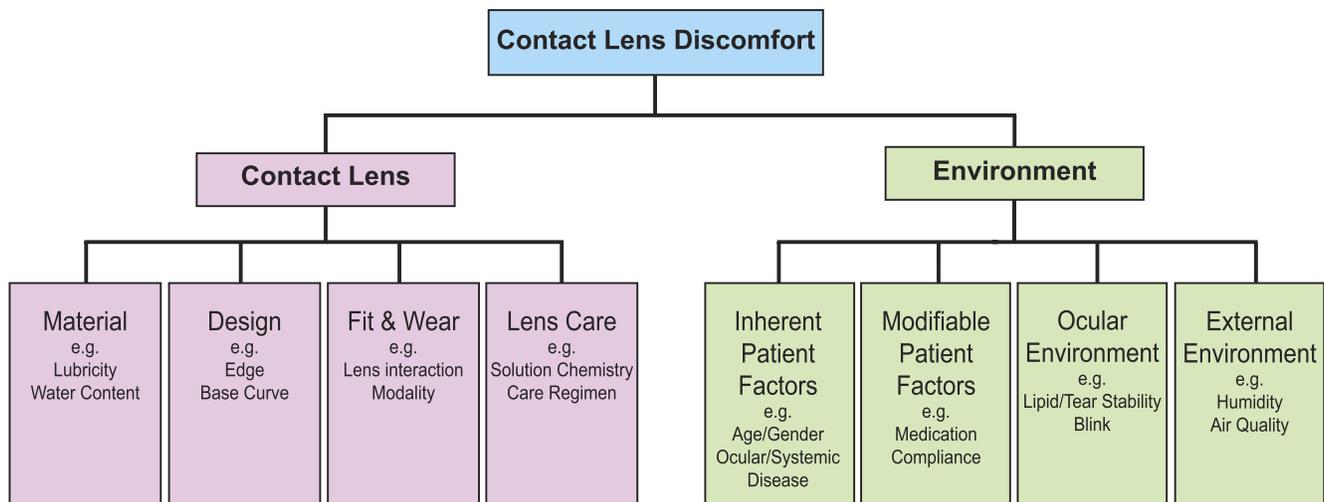
Our limited understanding of the etiology and correlation between signs and symptoms makes it all the more difficult to diagnose and manage CLD. The tools used to diagnose CLD and the expectations of contact lens wearers continually change, making it difficult to draw conclusions over time and to compare results from multiple studies. There are few validated instruments for assessing comfort in contact lens populations, and these tend to produce data that are highly variable, as most rely on a patient’s recall. In addition, the lack of postmarket surveillance studies, which would address many of the issues related to CLD in a longitudinal fashion, prevent drawing meaningful conclusions regarding the impact of technological advances on CLD. Future epidemiologic work designed to clarify the natural occurrence and evolution of CLD in rural and urban population settings, and in various countries and races are very much needed to enrich our understanding of CLD and associated risk factors.

As CLD is reported primarily by symptomatology as opposed to the observation of signs, and while the precise etiology of CLD is yet to be determined, the use of symptoms as an outcome measure is appropriate, because it relates directly to the patients’ experience with contact lenses, and the motivation to seek and use treatment, regardless of the presence of observable signs. The frequency and intensity with which these symptoms are reported can be assessed with the use of questionnaires. Further research and agreement of a universal adoption of a single measure of CLD is needed. The Contact Lens Dry Eye Questionnaire has been well received and, perhaps, is the most likely candidate for widespread CLD assessment.

CONTACT LENS MATERIALS, DESIGN, AND CARE

The influence of contact lens materials and designs, including rigid and soft contact lenses in these aforementioned areas, has been of significant controversy in terms of their association or etiologic influence in CLD. Further, there also has been great interest in the role of contact lens care solutions, regimen practices in caring for contact lenses, and wearing schedule differences in terms of their influence on CLD.

The vast majority of today’s market is made up of soft contact lenses (~90%), while rigid lenses make up the remainder of the market. Of soft lenses used, silicone hydrogel lenses now make up the majority of the market share within most major worldwide markets. Through the years, there has been a question about the role of materials and designs on the problem of CLD. This issue was first recognized in the peer reviewed literature in the early 1970s for rigid lens materials and in the 1980s for soft lens materials. Since that time, practitioners and scientists have questioned the influence of polymer chemistry, and various other material attributes that can be measured and quantified. The attributes considered have included the bulk (e.g., water content, dehydration, ionicity, oxygen transmissibility, modulus, and mechanical factors) and the surface (e.g., friction, wettability, surface modification) of contact lens materials. To date, almost none of



Progression of CLD



FIGURE 1. Classification of CLD.

these attributes, with the possible exception of friction based on early evidence, appears to be associated directly with CLD. Studies evaluating these factors, however, can be difficult to draw conclusions from in that they are confounded with differing designs, lack of rigor, lack of consistent definitions (e.g., of discomfort), and an inability to hold the design constant when testing the influence of a material or its attribute. Lastly, contact lens material chemistry also is known to influence tear film component deposition (proteins and lipid primarily), but the role of deposition in general is equivocal, perhaps again due to difficulties and inconsistencies in measurement and quantification of deposition.

Contact lenses vary in terms of their designs, and there has been some notion that the design of a lens influences the on-eye comfort during wearing. There is no question that the design of contact lenses influences their ability to fit the ocular surface properly, and this is influential in terms of overall performance. For instance, for soft contact lenses, moderate on-eye movement (with tear exchange) and corneal coverage are recognized as being important, but its overall association with CLD is not entirely clear. Likewise, in rigid lens fitting, the influence of the eyelid-edge interaction is recognized as being important in terms of patient comfort, but this relation again is not entirely clear in terms of its overall association with CLD. However, there is even less consensus when considering the influence of various design attributes on CLD. That said, the size, shape, and contour of lens edges appear to be some of the most influential determinants of contact lens comfort for soft and rigid contact lenses.

Lastly, contact lens care solutions, contact lens care practices, and contact lens wear schedules certainly are of

interest in terms of understanding their role(s) in CLD. To date, the peer-reviewed literature does not give a clear indication of specific formulations or components that may be associated either with increasing CLD or with improving contact lens comfort. However, most practitioners agree that regular contact lens care by contact lens wearers, including rub, rinse, and adequate soaking (disinfection and cleaning) are important in the success of lens wear. Further, most agree that increasing the frequency of replacement of soft contact lenses is ideal for ocular health and potentially improving comfort, although it is difficult to define the ideal replacement schedule. To our knowledge, large-scale, well-controlled studies using contemporary devices have not been conducted to provide insight into these issues.

NEUROBIOLOGY OF DISCOMFORT AND PAIN

Contact lenses interact with some of the most richly innervated areas of the body, such as the cornea, lid margin, and to a lesser extent the conjunctiva, and so it perhaps is not surprising that the eye can detect and sometimes react to the presence of the contact lens. The sensory (afferent) nerves (i.e., those that react to "pain" stimuli), which are derived from the ophthalmic and maxillary regions of the trigeminal ganglion, give rise to numerous intraepithelial terminals, some of which may extend to within a few micrometers of the ocular surface. The sensory nerves of the cornea consist of polymodal receptors (which can react to near-noxious or noxious mechanical energy, heat, cooling, chemical irritants, and by a large variety of inflammatory mediators), mechano-nociceptors

(which respond to mechanical forces of a magnitude close to that required to damage corneal epithelial cells), and cold-sensitive thermoreceptors (which react to temperature drops produced by evaporation of tears at the corneal surface, or application of cold and hyperosmolar solutions). Activation of these nociceptors is via specific ion channels; however, there appears to be no linear relationship between channel activation and contact lens discomfort.

Postreceptor propagation of the sensory nerve signal travels from the source through trigeminal ganglion to terminate in multiple spatially discrete zones along the rostrocaudal axis of the trigeminal brainstem sensory complex (TBSC) of the central nervous system. In this region, sensory nerves terminate mainly in the ventral aspect of the transition region between caudal interpolaris of the spinal trigeminal nucleus and caudalis of the same region (Vi/Vc) or at the spinomedullary junction (Vc/C1). Evidence suggests that ocular sensory neurons at Vi/Vc or Vc/C1 serve different functions in ocular homeostasis and sensation. Drying or detection of cold at the ocular surface stimulates the Vi/Vc region only. Transection of the spinal trigeminal tract at Vi/Vc eliminates pain sensation upon corneal stimulation, but a sense of corneal touch remains. Pharmacologic blockade of only Vi/Vc prevents reflex lacrimation evoked by chemical stimulation of the ocular surface. The ascending projections from second-order ocular neurons in the TBSC to higher brain centers are not well known and no systematic mapping study has been reported, even though the complex nature of many ocular perceptions, such as dryness, grittiness, itch, irritation, and fatigue, suggests interactions across multiple psychophysical channels that require integration at higher brain centers.

Contact lens wear may, or may not, alter nerve fiber density, tortuosity, branching, beading, thickness, or reflectivity. The large changes in morphology of the subbasal nerve plexus in the cornea during orthokeratology (OK) lens wear increase the threshold to sensation. Changes in corneal sensitivity with contact lens wear have been reported widely, but the underlying mechanism is not known, and the outcomes of studies may be very dependent on the type of instrument used to test sensitivity. The fact that tactile/pneumatic stimulus of the cornea after soft contact lens wear is reduced, but no associated change occurs in symptoms of discomfort during lens wear, suggests that the touch response in the cornea, and, hence, propagation of the stimulus through Vc/C1, is not associated with CLD. This then may implicate the cooling, osmolality differences detected through the Vi/Vc region. An alternate hypothesis, but not necessarily mutually exclusive, is the possibility of mechanical stimulation of the nociceptors in the lid wiper region of the eyelids. Stimulation of subacute inflammation of the ocular surface during lens wear may occur, and nerves can respond to the production of a variety of inflammatory mediators, including cytokines and arachidonic acid metabolites. The key neurotransmitters involved in the transmission of ocular sensations in human cornea and conjunctiva have been identified as substance P and calcitonin gene-related peptide (CGRP). No change in tear levels of substance P was found in a group of contact lens wearers compared to nonwearers, which may indicate no role for substance P in CLD. No reports on changes to CGRP were found. Conversely, the neurotrophin nerve growth factor (NGF) appears to be upregulated in CLD. As NGF is involved in survival and maintenance of sympathetic and sensory neurons, its upregulation suggests that nerves either are being damaged (and so need extra NGF for repair) or being altered in other ways during CLD.

Much more research needs to be performed to enable a comprehensive outline of the neurobiology of CLD. Better integration of the research from the peripheral and central

nervous system, with observations of nerve morphology/structural changes, and the biochemistry of the system could only be beneficial to our understanding of CLD. An important first step would be to design experiments to determine which tissue (e.g., corneal or lid margin) is the primary sensory location of CLD.

CONTACT LENS INTERACTIONS WITH THE OCULAR SURFACE AND ADNEXA

It would appear obvious that the interactions of a contact lens with the ocular surface and tear film are critical in the successful wear of the lens and the development of CLD. This subcommittee investigated the impact of contact lenses on the ocular surface and attempted to link these interactions to the development of CLD. A thorough review of the literature identified many dozens of ocular surface tissue alterations that may occur as a result of lens wear. While many of these result in frank pain (e.g., microbial keratitis), it was determined that such obvious pathologic complications were not the remit of this exercise and that the subcommittee would consider only potential tissue alterations that were associated with CLD (as defined above), and not pain that remained upon removal of the lens.

The cornea serves as the major surface on which the lens sits and could be a significant factor in CLD, particularly as it relates to its neurobiology. However, morphologic and apoptotic changes within the corneal epithelium have not been linked to CLD, nor have any changes in corneal epithelial barrier function. Despite many publications examining corneal staining associated with CL wear, overall, there appears to be, at best, a weak link between CLD and corneal staining, and it is not a major factor for most CL wearers. No stromal (keratocyte density, stromal opacities, stromal infiltrates, and stromal neovascularization), endothelial, or limbal (redness or stem cell deficiency) changes induced by lens wear were proven to be associated with CLD. While hypoxia can be a complication with many lens types or designs, no specific association with any hypoxic changes or marker of hypoxia could be linked directly to CLD.

The conjunctiva proved to be a tissue more closely linked to the development of CLD. Bulbar conjunctival staining, typically viewed using lissamine green, was found in some studies to be associated with CLD, particularly soft lens edge-related staining, and this may be related to lens edge design. While edge design and modulus may be linked to the development of conjunctival epithelial flaps, there appears to be no association between this tissue change and CLD. Bulbar hyperemia was not linked to CLD. Cytologic changes in the bulbar conjunctiva do occur in some wearers with CLD, but the many months it takes to reverse these changes obviously argues against a strong association with CLD, as CLD is relieved rapidly by removal of the lens from the eye.

The palpebral conjunctiva has an important role in controlling the interaction with the ocular surface and lens. Two specific issues potentially linked to CLD include alterations to the meibomian glands and to the leading edge of the palpebral conjunctiva as it moves across the lens surface (the so-called "lid-wiper" zone). Contact lens wear does appear to impact the function of the meibomian glands and reduced meibomian gland function has been associated with contact lens wear, but further studies are required for confirmation. Alterations to the lid-wiper area are more common in contact lens wearers who are symptomatic, and some studies have related these tissue changes to CLD. However, further work is necessary to investigate whether lid wiper epitheliopathy (LWE) is caused by specific properties of the lens material,

whether upper LWE is more or less relevant than lower LWE, whether making changes to contact lens properties, rewetting drops, or solutions can influence positively the degree of LWE, and to what extent modification of LWE will alleviate CLD. Finally, the lid margin is colonized more frequently with microbes than the conjunctiva, but the frequency of isolation varies between wearers. The role of lid microbiota has been studied only superficially during CLD and this also is an area worthy of future study, given that microbial toxins can impact ocular comfort.

In conclusion, some evidence is available to suggest a link between conjunctival and lid changes with CLD, with the strongest evidence being that related to meibomian gland and LWE changes. No convincing evidence of a link to CLD was unearthed with respect to any of the other forms of CL-associated tissue changes. Future studies would benefit from longitudinal designs that attempt to understand what pathophysiological changes occur in new wearers over time, and whether changes to lens materials, design, fit, or other factors impact these tissue changes. Studies also should examine whether the magnitude or timing of such changes can be related to the magnitude and timing of CLD.

CONTACT LENS INTERACTIONS WITH THE TEAR FILM

In evaluating contact lens interactions with the tear film and how those interactions might result in discomfort, the workshop considered the biophysical and the biochemical effects of contact lens wear on the tear film and their influence on discomfort.

The physical presence of a contact lens in situ divides the tear film into a pre- and postlens tear film, creating new interfaces with the ocular environment. Tear film changes occur upon lens application and during subsequent wear. In addition, biochemical differences are likely to exist between the pre- and postlens tear film layers. Partitioning of the tear film upon contact lens application and wear causes a series of compositional changes that result in a less stable tear film on the front surface of the lens and less well-defined changes to the postlens tear film layer. The resulting prelens tear film has reduced lipid layer thickness, reduced tear volume, and increased evaporation rate compared to the normal tear film. While the direct impact of these tear properties on discomfort has not been elucidated fully, the evidence to date specifically suggests that decreased tear film stability, increased tear evaporation, reduced tear film turnover, and tear ferning are associated with CLD. Further evidence is needed to support the associations between tear volume, surface tension, osmolarity, pH, and ocular surface temperature and CLD.

With respect to biochemical changes in tear film composition associated with contact lens wear, there appears to be no relationship between total protein, lactoferrin, and lysozyme with CLD. Current evidence suggests that levels of tear lipocalin-1, levels and activity of sPLA2, and levels of degraded lipids may be increased, and phospholipids decreased in CLD, which may be consistent with biochemical and functional changes in the tear lipid layer. Certain polar lipids, specifically the (O-acetyl)-omega-hydroxy fatty acids and their esters, have been associated with symptom reporting and may be important in CLD. Further evidence is needed to establish links between MUC5AC and other changes in the tear proteome with CLD. Given the potential evidence for frictional wear and lid wiper epitheliopathy in the pathophysiology of CLD, it may be expected that tissue and tear proteases, and inflammatory mediators would be increased in the tear film; however, such changes have not yet been demonstrated consistently.

There are significant gaps in our understanding of the extent to which tear film changes in contact lens wear are responsible for CLD. There is good evidence for associations between changes in tear lipids likely in the prelens tear film and CLD, although it is not clear if these changes are causal, or that they are present before contact lens wear. To understand these relationships better, it is important to use the definition of CLD as defined herein in future research and to study relevant subject groups using an appropriate study design. The lack of evidence for the postlens tear film in CLD likely relates to the current difficulties in evaluating this layer, in addition to the fact that this layer is relatively stagnant, as it largely is trapped and stagnant behind the contact lens.

Evidence also suggests that the parameters of the prelens tear film are interrelated and, therefore, it is difficult to identify a single component as being responsible for CLD. Tear film stability (via evaporation), however, is recognized as a key factor in CLD, and it appears to be a consequence of multiple tear film characteristics and their interactions. Given the relevance of prelens tear film stability in CLD, future research should focus on the development of novel materials or surface treatments to resist tear evaporation during wear, and on the development of wetting agents in care products to promote long-term contact lens wettability.

TRIAL DESIGN AND OUTCOMES

Design of clinical trials to determine the possible causes of CLD, for the most part, have not been optimal and numbers of participants in the trials generally small. Surprisingly, given the strong association of CLD with discontinuation of contact lens wear, the design of clinical trials has tended to focus on performance of certain contact lenses or lens care solutions, rather than the specific nature and etiology of contact lens discomfort. This may be due to the majority being industry-sponsored clinical trials.

Most clinical trials have evaluated the role of lens type (material differences), use of care systems, and effect of lens fitting, but they have been limited in their ability to isolate one factor from others. A significant limitation has been the lack of a consensus-based definition of CLD to date. Other limitations include lack of control of confounding variables or use of proper controls. An example of this is the problem often found when reports have been published on the results of changing wearers from their habitual lens of choice to a new (sometimes experimental) lens. Without appropriate masking and controls (for example, not only changing to the new lens type, but refitting a portion of subjects with or crossing over the subjects into their habitual lenses once masked), results tend to suffer from inherent bias.

This subcommittee report details many types of bias that should be considered in future work in this area. Further, prospective trial designs with randomization of subjects and double masking is optimal. Consideration of run-in and wash-out periods are important to avoid memory bias or changes that may occur to physiology during wear of lenses. Appropriate entry criteria and adequate sample size determinations a priori are critical.

Finally, it was determined that certain factors from clinical trials, at least potentially, had been associated with CLD. These included lid wiper epitheliopathy, tear film stability/volume, and lid parallel conjunctival folds. It was recommended that further appropriately designed clinical trials be performed to assess these factors (and others). Although no single outcome parameter of contact lenses was found to be validated fully, it was concluded that the Contact Lens Dry Eye Questionnaire currently was the most appropriate subjective outcome for

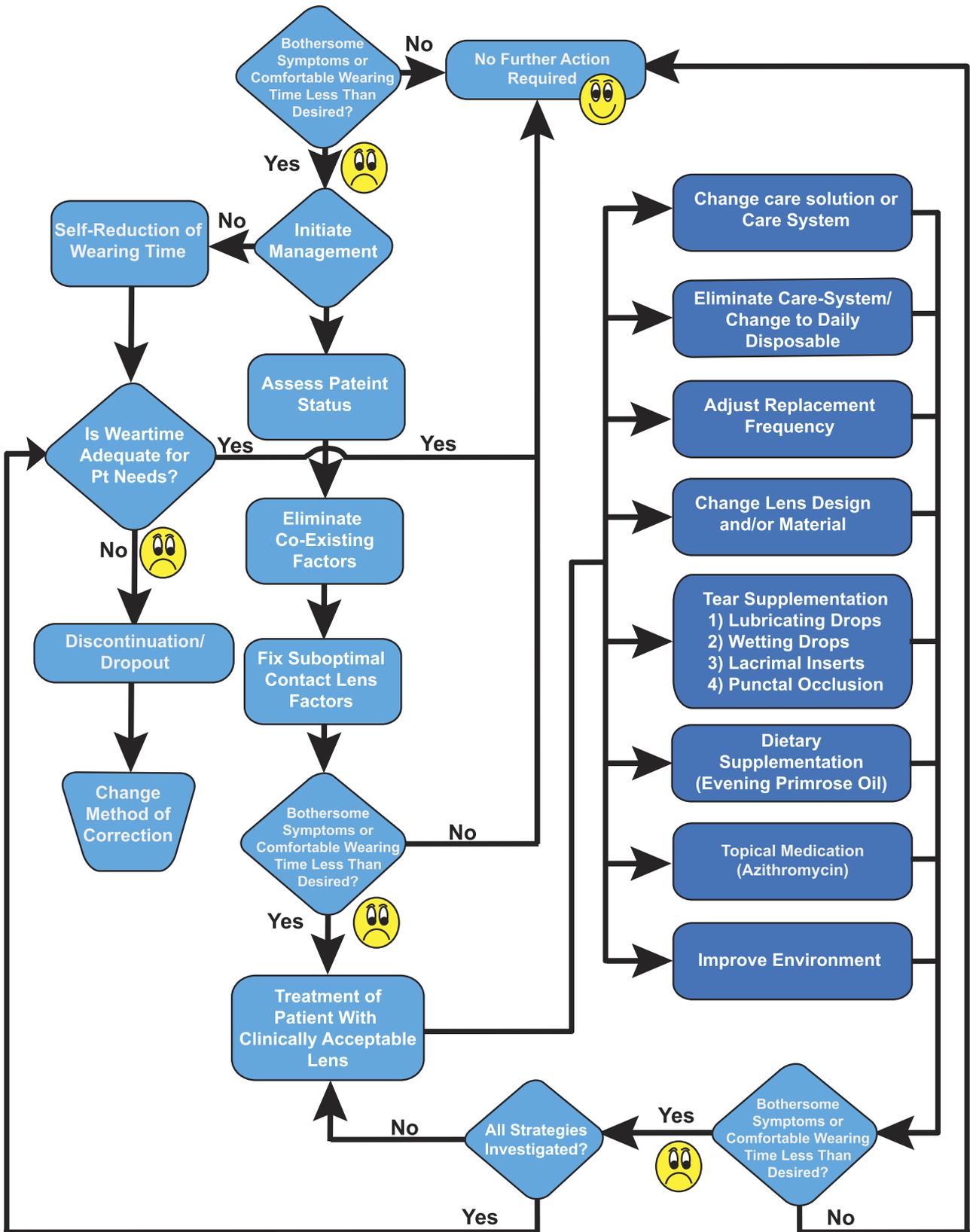


FIGURE 2. Summary of the management strategies for CLD.

CLD. An even more reliable and sensitive outcome parameter is needed for future work in this area.

MANAGEMENT AND THERAPY OF CLD

The condition of CLD is a considerable management and therapy challenge in clinical practice. While the causes of the short-term discomfort following difficulty with lens insertion generally are understood and appropriate remedies are straightforward, symptoms of discomfort and dryness that persist and increase toward the end of the day pose a more intractable problem. Managing wearers in these circumstances requires careful, individual assessment to eliminate concurrent conditions that may confuse the clinical picture, followed by a determination of the most likely cause or causes, and identification of corresponding treatment strategies (Fig. 2). The aim is to ensure that the contact lens is in a clinically acceptable ocular environment without obvious lens deficits of either a physical or behavioral nature.

A careful history of the presenting problem and the general status of the patient is a critical first step in the management process for CLD. Key elements in the evaluation include the age and sex of the wearer, timing and onset of symptoms, type of lens and lens material, care systems, lens replacement schedules, use of additional wetting agents, wear times and patterns, compliance and adherence to instructions, the occupational environment, coexisting disease, and current medications.

It is important to recognize that the symptom “discomfort” is relatively nonspecific, as discomfort can result from many sources other than the contact lens. Coexisting pathologies that may be responsible for the patient’s symptoms, such as ocular medicamentosa, systemic disease (autoimmune diseases and atopic disease), eyelid disease (blepharitis and anatomic abnormalities), tear film abnormalities, and conjunctival and corneal diseases, are important to identify and treat before focusing on the contact lens as the source of discomfort.

After noncontact lens causes of CLD have been identified and treated, the focus is on the contact lens and care system. Contact lens defects, such as edge chips and tears, deposits, and nonwetting surfaces, are typical causes of contact lens-related problems. Contact lens design properties (such as edge design), material properties, and on-eye fit, also are issues that must be considered. Care solutions and their components or improper care regimens also may at times contribute to CLD, and the benefits of daily disposable lenses may, in part, be due to elimination of these factors. However, the solution in the blister pack of disposable lenses also can be a source of CLD, particularly on application of contact lenses.

Frequent and appropriately-timed replacement of contact lenses may reduce or eliminate deposit formation. Switching to a different care system may have some effect on deposit formation. Although changing lens material may be helpful, it is difficult to separate material from design and surface effects as sources of CLD.

Fitting with steeper base curves, using larger diameter lenses, alternating the back lens surface shape, and using lenses with a thinner center thickness may improve CLD. However, it is difficult to manipulate lens parameters in isolation from each other, as altering one parameter may influence the other parameters.

The use of topical artificial tears and wetting agents, oral essential fatty acids (FA), punctal occlusion, and topical medications (e.g., azithromycin, cyclosporine A), along with avoiding adverse environments (e.g., aircraft cabins) and altering blinking behavior, all have been used in treatment of patients with dry eye and may be useful adjuncts in reducing CLD, although these require more substantial evidence in the future relative to their use (or lack thereof).

All these tactics may have limited effect on CLD and incremental improvements in CLD may be all that can be expected reasonably from any single intervention. The addition of treatments in a stepwise manner may be required to provide the maximum possible relief. Unfortunately, given the current state of knowledge of CLD, some patients will have residual levels of CLD that are sufficiently bothersome that it causes them to discontinue contact lens wear.

CONCLUSIONS

The TFOS International Workshop on CLD has addressed many areas of interest within the contact lens community as they relate to characterizing the ever-persistent problem of CLD. As noted, this international group of experts provided a framework that future studies and clinical activities can build upon when working in this area. It is critically important that the definition of CLD (as noted above) be applied in trials and studies that address CLD, including validated outcomes, such that there is consistency across research activities. Likewise, prospective natural history studies, which have not been performed to date, will help us better determine the incidence and risk factors for this condition, including factors that may relate to the patient or contact lenses in some way (e.g., material characteristics, designs, care system characteristics, care regimens). Etiologic considerations, including interactions with the ocular surface and tear film, need better models that will allow improved preclinical insight, and ultimately bench to the clinic translation in the development of novel products. Lastly, clinicians must be diligent in working with patients with CLD. It is important that the process of prevention and management of CLD starts early, perhaps even before the onset of symptoms, to improve the long-term prognosis of successful, safe, and comfortable contact lens wear.

Acknowledgments

Disclosure: Each workshop participant’s disclosure data can be found in the Appendix of the Introduction.